Handbook of
Plant Disease Management
Series Editors:
Robert J. McGovern · Wade H. Elmer

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Robert J. McGovern
Wade H. Elmer *Editors*

Handbook of Florists' Crops Diseases

Springer

Handbook of Plant Disease Management

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Our objective for the Handbook of Plant Disease Management series is to provide up-to-date, field-tested information on integrated disease management that will be useful to a diverse technical audience interested in a wide variety of crops. The series will have an international focus and will address these questions: "What is the disease?" and "How can it be managed?"
The Handbook of Plant Disease Management series will include: Handbook of Florists' Crops Diseases, Handbook of Vegetable Diseases, Handbook of Tropical Fruit Diseases, Handbook of Temperate Fruit Diseases.

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Editors

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With 522 Figures and 49 Tables

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ISBN 978-3-319-39668-2 ISBN 978-3-319-39670-5 (eBook)
ISBN 978-3-319-39669-9 (print and electronic bundle)
https://doi.org/10.1007/978-3-319-39670-5

Library of Congress Control Number: 2017948983

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Printed on acid-free paper

This Springer imprint is published by Springer Nature
The registered company is Springer International Publishing AG
The registered company address is: Gewerbestrasse 11, 6330 Cham, Switzerland

Preface

Florists' crops production has evolved considerably through new technological advances in irrigation, media, and environmental control, along with the appearance of new centers of large-scale production of plant material. In addition, many florists' crops have changed their production cycles by shifting the initial production stage to offshore facilities. While this offers new benefits in product quality, quantity, and cost, it removes some opportunities to introduce management strategies to suppress future outbreaks. These changes have necessitated the development of innovative ways of suppressing pathogenic fungi, bacteria, viruses, and nematodes. The objective of the *Handbook of Florists' Crops Diseases* is to provide research-based information on the diagnosis and management of diseases of cut-flowers and potted flowering plants.

We believe that following such an integrated approach will help to minimize nontarget effects of flower production and increase its sustainability. The introductory chapters, "Components of Integrated Disease Management," present disease management strategies that are applicable to all florists' crops. The chapters on the individual crops that follow in the "Florists' Crops Diseases" section are grouped alphabetically within three subsections: "Cut-flowers," "Potted Flowers," and "Flowering Geophytes". The grouping of crops is somewhat arbitrary; some like orchid could fit into more than one subsection: cut-flowers and potted flowers. These chapters present information on the geographic occurrence and impact, symptoms/signs, biology and epidemiology, and integrated management of major diseases of these plants.

While it is not possible to cover the diagnosis and management of all diseases on every florists' crop, we believe that the extensive cross-referencing and indexing used in the handbook will provide assistance where specific information is lacking. We hope that the global perspective of the handbook and the detailed information provided will make it useful to florists' crops producers, researchers, extension personnel, and students around the world.

This volume is one of a series of *Handbooks of Plant Disease Management* being published by Springer.

Acknowledgments

We thank the many participating authors (over 60 from 11 countries) who freely gave of their time to share their considerable expertise on the management of florists' crops diseases. We appreciate not only the efforts of these individuals on their own chapters but also their generosity in providing diagnostic images to authors of other chapters. We also thank the many other individuals acknowledged throughout the handbook who provided images of and information on florists' crops diseases.

We are indebted to the Springer editorial team of Sylvia Blago, Simone Giesler, Zuzana Bernhart, and Priya Ponnusamy for their guidance in initiating and shaping the *Handbook of Florists' Crops Diseases*.

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About the Editors



Prof. Dr. Robert J. McGovern received his M.S. (1983) and Ph.D. (1986) in Plant Pathology from Cornell University. He joined Flower Time/Frank's Nursery & Crafts, Inc., the largest garden center chain in the USA, in 1985 and served first as its plant pathologist/academic liaison, and then as director of the Horticulture and Diagnostics Department. He joined the University of Florida, Institute of Food and Agricultural Sciences (UF-IFAS), Department of Plant Pathology in 1990. Dr. McGovern served as an assistant professor with extension, research, and teaching responsibilities for integrated disease management in vegetables and citrus (Southwest Florida Research and Education Center, 1990–1995) and as an associate professor focused on management of ornamental and vegetable diseases (Gulf Coast Research and Education Center, 1995–2002). He was promoted to full professor in 2002 and moved to the UF-IFAS main campus to become director of the Plant Medicine Program, which he led from 2002 to 2011. During this period, Dr. McGovern also helped to establish a number of other plant medicine/plant health programs in Asia and the USA, and served as co-director/director of the Southern Plant Diagnostic Network, a component of the USDA-coordinated National Plant Diagnostic Network. He retired from UF-IFAS in 2011 and is a professor emeritus of its Department of Plant Pathology. Currently, Dr. McGovern is a senior consultant with the NBD Research Co., Ltd., Lampang, Thailand, and an adjunct professor in the Chiang Mai University, Department of Entomology and Plant Pathology, Chiang Mai, Thailand. His research and teaching interests include integrated plant disease management, scientific writing and editing, and multidisciplinary training. Dr. McGovern is the author of over 150 scientific publications.



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Florists' Crops: Global Trends and Disease Impact

1

Robert J. McGovern and Wade H. Elmer

Abstract

The production of cut-flowers, potted flowering plants, and related propagative material is a dynamic, fast-growing, and highly lucrative global industry. In the past, flower production sites were primarily located in or near the major markets – developed countries in North America and Europe. However, because of lower production costs, including labor inputs, more favorable climatic conditions, and the development of efficient transportation methods, there has been a shift in production to exporting countries closer to the equator such as those in Latin America, sub-Saharan Africa, and Asia. The direct effects of pathogens on florists' crops are the extensive losses that can occur in their production and postharvest handling. Furthermore, the dissemination of pathogens and their vectors on cut-flowers and potted flowering plants and propagative material may also damage unrelated crops. Management of pathogens of florists' crops is very challenging and costly because these crops have an essentially zero pest damage threshold, and because of the increasing demand by the public and government agencies that all of agriculture, including floriculture, should adopt a sustainable approach which minimizes/eliminates its nontarget effects.

Keywords

Cut-flowers • Potted flowers • Production • International trade • Plant disease • Crop losses • Pathogen spread • Pest dissemination

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© Springer International Publishing AG 2018

R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_8

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1 Introduction

Flowers affect our emotions in a number of positive ways (Haviland-Jones et al. 2005). Flowers have apparently benefited humankind for millennia; the ceremonial use of flowers in burials has been documented as long as 60,000 years ago, during the Neanderthal era (Solecki 1975). Humans have actively sought positive psychological and environmental modification through flower cultivation for thousands of years (Ackerson 1967; Haviland-Jones et al. 2005). Today the production of cut-flowers, potted flowering plants, and related propagative material (seeds, cuttings, bulbs, etc.) is a dynamic, fast-growing, and highly lucrative global industry. Global trade volume of florists' crops has been estimated to be more than $100 billion annually (African Business Magazine 2012). In an industry where extremely high value criteria prevail, even a slight loss in quality can render the crop unmarketable. In Table 1, recent estimated losses caused by pathogens in representative florists' crops range up to 100%. If we assume a conservative estimate that pathogens reduce the value of florists' crops by 10%, it would suggest a staggering $10 billion in annual losses from floral diseases. This appears to be a very conservative estimate in that postharvest diseases alone have been reported to cause losses of 10–30% of the total florists' crops yield worldwide (Dole and Wilkins 2004).

Pathogens directly reduce the production and marketability of cut- and potted-flowers and propagative material. An indirect impact results from the dissemination of pathogens and their vectors as a result of the global movement of flowering ornamentals from offshore production facilities into new areas, which, in turn, may damage unrelated crops.

2 Global Production Trends

In the past, flower production sites were primarily located in or near the major markets, primarily the developed countries in Europe and North America. As of 2012, countries in the EU led by the Netherlands still accounted for a hefty 44% of world production of flowers and potted plants (European Commission Advisory Group 2012). However, because of lower production costs, including lower labor inputs, more favorable climatic conditions, and the development of efficient transportation methods, exporting countries located closer to the equator have steadily

Table 1 Estimated losses caused by pathogens in representative florists' crops

Crop	Disease/Pathogen	Country	Loss (%)[1]	Reference
Anthurium	Bacterial blight/ *Xanthomonas axonopodis* pv. *dieffenbachiae*	USA (Hawaii)	50–100	Alvarez et al. 2006
Caladium	Fusarium tuber rot/*Fusarium solani*	USA (Florida)	80–100	McGovern et al. 2003
Carnation	Sclerotinia stem rot/*Sclerotinia sclerotiorum*	India	38.5	Vinod Kumar et al. 2015
	Root knot/ *Meloidogyne incognita*	India (Bangalore area)	40–60	Parvatha Reddy 2014
Celosia	Yellows/' *Candidatus* Phytoplasma spp.'	Israel	up to 100	Tanne et al. 2000
China aster	Fusarium wilt/ *F. oxysporum* f. sp. *callistephi*	USA (Connecticut, Florida)	up to 70	Elmer and McGovern 2013
	Tobacco rattle virus	Japan	up to 63	Komuro et al. 1970
	Tomato spotted wilt virus	Greece	37.7	Chatzivassiliou et al. 2000
Chrysanthemum	White rust/ *Puccinia horiana*	Turkey (Izmir province)	80	Göre 2008
	Bacterial wilt/ *Dickeya chrysanthemi*	Hungary (Budapest area)	~100	Végh et al. 2014
	Tomato spotted wilt virus, Impatiens necrotic spot virus	Colombia	up to 80	Vasquez and Angarita 1999
Geranium	Bacterial blight/ *Xanthomonas hortorum* pv. *pelargonii*	USA	up to 100	Rosa and Moorman 2017
Gerbera	*Tomato spotted wilt virus*	Serbia, Venezuela	up to 30	Stankovic et al. 2011; Marys et al. 2014
Gladiolus	Fusarium corm rot/ *F. oxysporum* f. sp. *gladioli*	India	60–80	Chandel and Deepika 2010
Gypsophila	Powdery mildew/ *Erysiphe buhrii*	Korea (Iksan City)	~100	Choi et al. 2014
Lily	Fire/*Botrytis* spp.	Most lily-growing areas	up to 100	Chastagner et al. 2017

(*continued*)

Table 1 (continued)

Crop	Disease/Pathogen	Country	Loss (%)[1]	Reference
Lisianthus	Fusarium crown and stem rot/*F. avenaceum*	USA (Florida)	up to 70	McGovern et al. 1997
Orchid	Bacterial brown spot/*Burkholderia gladioli* Foliar nematodes/*Aphelenchoides besseyi* and *Aphelenchoides fragariae*	China USA (Hawaii)	10–25 up to 90–95	You et al. 2016 Kawate and Sewake 2014
Rose	Crown rot/*Phytophthora nicotianae*	Argentina	10–25	Wolcan et al. 2007
	Crown gall/*Agrobacterium tumefasciens*	Kenya	5–60	Real IPM/CSL 2007
	Gray mold/*Botrytis cinerea*	Ethiopia (East Shoa Zone)	up to 31 (postharvest)	Aman 2014
Snapdragon	Downy mildew/*Peronospora antirrhini*	USA	up to 100	Yarwood 1947
	Phytophthora stem rot and wilt/*Phytophthora* spp.	USA	50	Gill 1960
Multiple crops (Gerbera, Lisianthus, Rose)	Gray mold/*Botrytis cinerea*	The Netherlands Flower Exchange	15–30 (postharvest)	Vrind 2005

[1]Loss estimates refer to those observed in production unless stated otherwise.

increased their market share in cut-flowers, starting in the 1970s with Colombia and Ecuador, and more recently in sub-Saharan African countries including Kenya and Ethiopia, and Asian countries such as Malaysia (van Rijswick 2015). Although the export value of cut-flowers from the Netherlands rose by 2% to an industry-leading $7.2 billion in 2011, the number of Dutch exporters fell sharply to 724 compared to 857 in 2008 (ITC 2012).

In 2013, Colombia exported $1.34 billion worth of flowers (PMA 2015), and Ecuador exported $786 million (OEC 2013). Between 2000 and 2007, cut-flower exports from sub-Saharan Africa expanded by an average annual rate of 20% (WTO 2008). Africa's share of world exports doubled to 8% during this period. Kenya, Africa's leading exporter of cut-flowers, is the fourth-largest world supplier (behind the Netherlands, Colombia, and Ecuador) and the largest outside supplier to EU countries. The export value of Kenyan ornamentals (live trees, plants, bulbs, roots,

cut-flowers, etc.) was more than $720 million in 2013 (ITC 2013). From 2003 to 2007, exports of cut-flowers from Ethiopia increased by an average annual rate of 142%, while Zambia's exports increased at an annual rate of 44% between 2000 and 2006 (WTO 2008). With a compounded annual growth rate of 30%, the value of the Indian floriculture sector was projected to exceed $140 million by 2015 (Bahri 2012). Another Asian giant, China, is rapidly becoming one of the largest flower growing and consuming countries in the world with an annual production of 2.7 billion fresh flowers and sales worth about $260 million (Canada-China Agriculture and Food Development Exchange Centre 2015).

Interestingly, floriculture is also an important source of food security in developing countries because of the income it brings to thousands of workers – most of whom are women (FAO 2002). Although not a major exporter, the wholesale value of USA-produced floricultural crops remains very substantial, being estimated at $2.13 billion in 2012, up 1% from 2011 (PMA 2012).

3 Direct Disease Impact

Because of the inherent esthetics involved, florists' crops have an essentially zero pest damage threshold including even slight flower blemishes caused by pathogens. Noticeable residue from fungicides and other materials is also unacceptable. Disease management is additionally challenging because of the increasing demand by the public and government agencies that all of agriculture, including floriculture, should adopt a sustainable approach which minimizes/eliminates its nontarget effects. Therefore, along with other flower production practices, disease management must be equally precise, intensive, and sustainable.

The direct effects of pathogens on florists' crops are the losses that can occur in their production and postharvest handling. There have been many reports of extensive losses caused by pathogens in florists' crops. Table 1 presents recent disease loss estimate percentages for a number of these plants. However, estimates assigning a monetary value to such losses in these high value crops have been rare. The University of Georgia annually reports the financial impact of disease on the state's crops. In 2013, total losses due to diseases of floricultural crops in the state of Georgia, USA, were estimated to be about $42 million, or approximately 17% of the 2010 farm gate value for these crops (Martinez-Espinosa 2013).

4 Indirect Disease Impact

Increasing globalization of trade can rapidly introduce new ornamental pathogens and their biotypes. In addition, the dissemination of pathogens and their vectors on cut-flowers, potted flowering plants, and propagative material may also damage unrelated crops. *Ralstonia solanacearum* is a major bacterial pathogen of many horticultural crops. Race 3 biovar 2 (R3bv2) of the bacterium is considered to be

particularly damaging because of its ability to cause rot and wilt in eggplant, geranium, potato, and tomato, as well as persist in areas with temperate climates. The bacterium is considered a serious quarantine pest by national regulatory agencies like the USDA Animal and Plant Health Inspection Service (APHIS), and multinational regulatory organizations such as the North American Plant Protection Organization (NAPPO), and the European Plant Protection Organization (EPPO). In 2003 and 2004, R3bv2 was detected in geraniums that were imported as cuttings from Kenya and Guatemala and grown in greenhouses in Canada and the USA (USDA-CSREES 2004). The costly eradication measures mandated for the pathogen included quarantine of the affected greenhouses and total crop destruction.

In addition, movement of pesticide-resistant pathogens in global agricultural trade is an important issue. The pathogenic fungus *Botrytis cinerea* has acquired fungicide resistance due to the use of single-site mode of action fungicides to suppress Botrytis blight without rotation. The dissemination of fungicide resistant strains of *Botrytis* and other fungal pathogens in imported flowers and related materials to geographically distinct regions may compromise local production of vegetables, small fruit, and other crops.

The movement of viruses and other graft-transmissible agents in ornamentals has been a long-standing regulatory concern (Lawson and Hsu 2006). Preventing the dissemination of virus vectors is also of critical importance. The *Bemisia tabaci* whitefly complex is viewed as one of the most damaging agricultural pest groups because of the many viruses that it vectors and its large host range which includes many florists' crops (Lapidot and Polston 2007; Oliveira et al. 2001). It has been hypothesized that the B biotype of *B. tabaci* (aka silverleaf whitefly), which originated in the Middle East, was moved to the Western Hemisphere on ornamental plants (CABI 2015). Poinsettia infested with the B biotype may have further spread the pest within the USA (Parella et al. 1992). The Q biotype of *B. tabaci*, which is reported to be resistant to commonly used neonicotinoid insecticides, was recently spread from the Mediterranean region to China on ornamentals (Chu et al. 2006).

Regulatory organizations like the EPPO, NAPPO, USDA/APHIS, etc. maintain lists of quarantinable pathogens and other plant pests and their hosts. The USDA/APHIS also operates a remote program, the Offshore Pest Information System (OPIS), to gather information about new pest outbreaks of potential concern to US agriculture. Furthermore, the USDA/APHIS conducts preclearance of a number of crops in cooperation with export countries to help reduce the spread of quarantinable pathogens and other pests.

In addition, there are joint industry-government certification programs to insure that plant propagative material is free of pathogens and other pests. In the Netherlands such a program, the Naktuinbouw Elite certification system (http://www.naktuinbouw.eu/node/192), combines pathogen-free propagation methods with compulsory pathogen testing.

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Part I

Components of Integrated Disease Management

Fundamentals and Advances in Plant Problem Diagnostics

2

Tim Schubert, Ayyamperumal Jeyaprakash, and Carrie Harmon

Abstract

Plant disease/problem diagnostics and pathogen detection are fundamental components of successful agriculture. Any corrective action for poor plant performance should arise out of thorough assessment and diagnosis of the problem(s), to be followed by appropriate management suggestions. Among the factors to consider in the choice of diagnostic techniques are speed, cost, accuracy, availability of technology, training, value of the crop, and likelihood of generating a successful management strategy. Many exciting new technologies are entering the field of diagnostics, and plant disease/problem diagnostics and pathogen detection are not exceptions. Among the favorites are molecular-based tests that rely on the specificity of gene sequences, serological tests in various formats, direct and indirect biosensor technologies, and spectral imaging. We have entered an era in which the entire biome both inside and outside a plant can be identified, diseased plants can be analyzed non-destructively, and the overall health of an entire crop is quickly discernible. Now we must learn how to interpret all that data and put it to practical use. In spite of the enormous potential of all these new developments, successful disease diagnosis will still depend fundamentally on basic skills that have been honed and perfected in practice for more than a century. Without the foundational information from thorough and focused observation on-site and access to all horticultural information and inputs for the crop in question, no amount of advanced technology can reliably correct for deficiencies in the primary steps in diagnosis.

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R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_1

Keywords
Plant disease diagnosis • Plant problem diagnosis • Triage • Microscopy • ELISA • PCR

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1 Introduction

When a crop fails to perform up to expectations, it is logical to turn to a diagnostic process to determine what is preventing the attainment of full potential. This chapter will explain the full spectrum of options for determining what might be limiting the growth of a particular plant while considering various factors that influence which tools are most suitable for the particular diagnosis in question. In an era of rapidly advancing modern technologies promising extraordinary accuracy and precision

heretofore practically unimaginable, the wise deployment of the entire spectrum of available tools maximizes the accuracy and usefulness of the diagnosis for the client and the grower community with a minimum of expense in a reasonable amount of time.

There are three caveats at the beginning: Firstly, for this discussion, we use two different terms or phrases to describe the process of figuring out what is wrong with a plant – *plant problem diagnosis* and *plant disease diagnosis*. It is not necessary for the reader to accept any strict definitions here, as the two can have nearly the same meaning, and that is how we intend to use them. We draw attention to both terms to make certain that the reader understands that we are including some techniques for the diagnosis of plant problems that have no biotic (living) causal agent, i.e., they are due to some abiotic (nonliving) cause. Some consider plant disorders caused by abiotic influences something other than disease, with the term "disease" restricted to plant disorders caused by living causal agents (giving some latitude for the viruses and viroids and perhaps prions which are incapable of life functions outside a host cell). Others classify these disorders as noninfectious diseases or physiological diseases. In this abiotic category are factors such as excesses or deficiencies of nutrients, water, or light; poor-quality water, soil/media, or air in the growing environment; temperatures outside optimal ranges for plant growth; mechanical damage; etc. Though some may prefer to make a distinction, we will be using plant *problem* diagnosis and plant *disease* diagnosis as essentially the same thing.

Secondly, a further clarification of terms is helpful in our quest for plant health. Plant disease or plant problem diagnosis represents the final step or culmination of a process that is preceded first by plant disease surveillance, followed by plant disease or plant pathogen detection. The semantics here are important. A grower must engage in continual surveillance to monitor overall plant health. This answers the question, "Is the crop normal? Is it on schedule? Is there a problem?" Plant disease or plant pathogen detection is the process of following up on the findings of surveillance. It addresses the question, "Is there a problem?" by defining the nature of the problem found, and discovers what organisms or causal agents are possibly associated with the syndrome detected. Finally, plant disease or problem diagnosis is the term reserved for the process of assigning the blame for the problem detected in the surveillance. The techniques and technologies discussed in this chapter can help in one or all of these three steps (Stack and Fletcher 2007; Miller et al. 2009).

Thirdly, many clients of plant disease diagnostic services automatically assume that the diagnosis takes place entirely or mostly in the lab using esoteric methods, specialized knowledge, and complicated machinery. Actually, overlooking the field aspects of the diagnosis can derail the entire process and yield faulty conclusions. Though we will start our discussion on lab techniques, at several points through the chapter, we will remind the reader of what must be accomplished in the field with the goal of reinforcing the idea that good diagnostics requires more than modern equipment and skilled operators. The indispensable detective work in the field at the time the sample is taken sets the stage for an accurate and holistic diagnosis that leads to optimal management decisions. Furthermore, very often the sample-taker

does not participate in the lab work and vice versa, so good communication networks between all the parties (the client, the sample-taker, and the lab scientist) are vital.

2 General Laboratory Diagnostic Techniques

In general, the techniques normally restricted to the laboratory and used for plant disease or plant problem diagnosis fall into these four major categories:

- Microscopy – examining the afflicted plant's tissues at various magnifications to visualize symptoms and evidence of any pathogen(s). This would include a hand lens, dissecting (reflected light) and compound (transmitted light) microscopy, and scanning and transmission electron microscopies.
- Microbiology – using various culture methods to separate the pathogen from diseased host tissue to study it in isolation for identifying features. If a pathogen can be isolated from its host and grown in culture or, in the case of viruses and viroids, purified by various methods, proof of pathogenicity by reinoculation is possible using Koch's postulates. Failure to recover or witness any organisms associated with the syndrome would push the diagnosis in the direction of an abiotic causal agent.
- Serology – using antisera developed against specific antigens from suspect pathogens to identify the causal agent in whatever matrix it may be found.
- Molecular biology – using nucleic acid visualization and sequencing to find identifying sequences in DNA or RNA extracts from isolated suspect pathogens or taken directly from samples of diseased tissues suspected of containing the pathogen. This technology has the sensitivity to detect latent disease and inform management decisions proactively (Michailides et al. 2005).

These categories are what many clients in need of diagnostic services believe to be the major or even sole methods employed. Each is in a typical sequence of steps and in relative order of cost to perform, and each requires special equipment and training to be successful in their use and interpretation. As technology advances, some of the work formerly confined to the lab is being "field hardened" to permit quick deployment on-site (Boonham 2014). Examples are more sophisticated field microscopes, dipstick and lateral flow devices for serological detection of microbes, and field PCR units. These methods can be used separately or in combination to reach the desired level of specificity in the diagnosis. However, it is extremely important to remember that for the best diagnosis, much of the successful process takes place in the field, prior to sample processing in the lab. While still in the field at the time of sample taking, a diligent effort must be made to acquire background information about the problem. Further questions may arise during lab processing that will make it necessary to revisit the site, resample, discuss issues again with growers, etc. These frequently underappreciated, overlooked, or poorly performed primary aspects of the diagnostic process are covered in greater detail in Sect. 6.

3 Special Features Related to Ornamental Disease Diagnosis

At the outset, it is important to note that plant problems in ornamental crops as opposed to food, fuel, or fiber crops present some unique features that need special consideration in the diagnostic context. First, the value of ornamental plants is aesthetic – based upon appearance. Perfection (or near perfection) in appearance can make a huge difference in profitability of the enterprise, so much so that addressing any factor(s) that prevents attainment of that aesthetic standard could require the utmost in diagnostic expertise and technology. Plus, specifically ornamental floral crops are by their very nature more fragile than even other ornamental plants such as foliage or woody ornamentals. Conversely, cosmetic imperfections that blemish the non-harvested portions of food, fuel, or fiber crops (or even the harvested portions to a degree) can be tolerated with little or no impact on yield or product value. (In making note of this fact, we must at the same time admit much room for improvement in consumer tolerance of minor blemishes in food products in order to feed the growing masses and still provide a fair economic return to the grower.) Calculating the amount of pest damage to tolerate on any crop with no loss in value is fundamental to good stewardship and maximizing profits. So, in the final analysis, that allowance for imperfections while remaining profitable tends to be much smaller for the ornamental crop, especially floral products. Furthermore, commerce involving the propagative material of ornamental plants often comes with a much greater plant pest regulatory risk than for food, fiber, and fuel crops because the ornamental commodity is frequently destined *not* for some devitalizing use such as consumption as food, manufacture into an inanimate object, or burned as fuel. Instead, it is *planted* in a new location for a continued existence, complete with all the pests and pathogens that accompanied it on its original journey. The default "apparently free of pests and diseases" standard for most routine interstate and international phytosanitary certification leaves considerable latitude for cryptic organisms to accompany and disperse from propagative material. This added risk potential makes high-level diagnostics especially appropriate in the ornamental plant arena. And lastly, high-value ornamental crops as a rule receive much more attention in the way of horticultural inputs, each one designed to maximize quality production at minimum cost in the shortest amount of time. Crops "pushed hard" in this manner are more apt to suffer abiotic disorders from such things as excesses in fertilizer or growth regulators, phytotoxic pesticide applications and/or mechanical injury from repotting and handling for merchandizing.

4 An Argument for Continued Use of Fundamental Diagnostic Techniques

Before addressing what might be considered by many to be the main topic in this chapter (that of advancements in biotic disease diagnostic techniques), we consider it entirely fitting to present a strong case for continued employment of more fundamental standard diagnostic methods. For the most part, few diagnostic tests are

necessarily outmoded or automatically unsuitable with the passage of time. Especially neglected in this category are macroscopic and microscopic diagnostic approaches. Several reasons support this position, and these are addressed singly and in order.

4.1 Attention to Details

First, one's initial approach to a diagnostic puzzle should commence with fundamental and historically proven tactics which depend upon keen attention to all aspects of the disease syndrome. Unfortunately, in every aspect of modern life, forces work against a person's ability to simply give attention, to focus intently on a subject, and to notice pertinent details (Crawford 2015). It is at this initial stage where disciplined and polished observational skills and inquiries pay great dividends and start the diagnostic quest in the right direction. Furthermore, in most cases, the sample collection process is performed by someone other than the diagnostician, so good collaboration and communication with the sample collector is critical. It is no exaggeration to assert that the sample collector is an absolutely essential participant in the diagnostic process. Every available means of modern communication (both real time and recorded) should be made available and used to help the lab personnel grasp the disease presentation in the field and guide the sample collection process to full advantage. Done properly, much information will be gathered in these early stages. Much of it will prove unhelpful and tangential to the final diagnosis, but there is no substitute for a thorough job at this stage. The essential clues will be distilled from that voluminous assemblage of seemingly random information. A sloppy job here will frequently alter the course of the entire diagnosis, and advanced techniques have little or no inherent value to correct the situation.

4.2 Discerning Indications of Abiotic Disease

Second, it is important to remember that abiotic causes are responsible for about half the samples submitted to a typical plant disease diagnostic clinic. The fact that ornamental plants are subjected to a high degree of deliberate manipulation by caretakers translates into a quite reasonable expectation that abiotic diseases might be even more common here. Assignment of an abiotic cause is, of course, based on our current fragmentary understanding of what can cause various symptoms in plants, but it would be unwise to assume that our understanding does not remain fragmentary even with advanced diagnostic tools at our disposal. Abiotic diseases seldom leave signs of the causal agent in the classic sense (a recognizable part of the causal agent left behind at the scene), as is more often the case with a pathogen/biotic disease. Only symptoms remain, and symptoms are notoriously general with multiple paths of causation. Genomic tools are not as valuable under such abiotic disease circumstances, though one day, transcriptome (RNA molecules present) or

metabolomics (chemical fingerprints) characterization technologies might prove helpful in understanding how a syndrome developed, and that could yield clues as to what abiotic condition may be responsible (Adams 2013).

4.3 Many Advanced Techniques Yield Only Binary Answers

Third, many (not all) of the more advanced diagnostic techniques are limited to providing only yes/no answers inasmuch as they can only detect a single organism. These are more accurately deemed *detection* tools that contribute to a diagnosis and do not constitute a *diagnosis* in and of themselves. This is especially true of PCR techniques and also serological tests to a lesser degree. They are more suited and useful for diagnostic confirmation than for diagnostic exploration.

4.4 Interpretation Challenges with Genetic Sequencing Data

Fourth, more advanced nucleic acid sequencing techniques and metagenomics have enormous potential to reveal genetic clues about *all* the organisms present in a sample of diseased plant tissue. One must be careful in the interpretation of the data procured with sequencing methods however. Our understanding of apparently innocuous or beneficial endophytic and epiphytic organisms associated with plants is too rudimentary to jump to conclusions about causality for a plant disease syndrome. Furthermore, associating gene sequences of an organism and its ability to cause disease is still a loose and tenuous relationship for many pathogen (e.g., *Fusarium oxysporum*). Many surprises await us, and understanding the significance of the biome and microbiome associated with a diseased plant will probably be a long learning curve. The more traditional diagnostic skills of isolating a pathogen from diseased tissues offer a cleaner substrate to take to the lab for nucleic acid sequencing, but limitations in what can be successfully isolated from diseased tissues have been known at some level for a long time. The automatic bias against fastidious microbes as pathogens is something the diagnostician must consciously avoid. The more we learn about the plant biome/microbiome, the more we understand about the limitations of culturing or otherwise separating microbes from diseased plant tissue. Even though these limitations are greater than we may have imagined, it is still a good idea to attempt to isolate the suspected causal agent from the host tissues because the clarity of genomic analyses of the clean isolate is far greater than an extract taken directly from diseased plant tissues.

4.5 The Complications of Host Predisposition

Fifth, as diagnostic skills advance, it is becoming more apparent that, under certain (perhaps many) circumstances, predisposing abiotic events precede and may even be

essential for biological invasions leading to plant disease development. Since plant tissues can behave something like ecosystems in miniature, it is not unreasonable to assume that abiotic predisposing events and colonization events could very well take place in a somewhat orderly succession. Although it is helpful to know the biotic agent responsible for the eventual symptoms, the most effective plant disease management techniques would recommend the avoidance or minimization of the predisposing conditions to lessen or prevent the biotic phase(s) of the disease altogether. Starting with metagenomics in such a situation would render a very complicated and incomplete diagnosis and probably generate an ineffective management scheme. And if preliminary genomic findings have any early message for growers and diagnosticians, we should set aside notions of genetic stability of pathogens and, as a consequence, discount any parallel belief that a sequence-based diagnostic method will reach perfect performance and permanence. Clearly, pathogens are much more dynamic and adaptable than once thought. Fortunately for both client and diagnostician, a 100% accurate diagnosis is not always necessary to yield a helpful (if incomplete) determination(s) and provide recommendations that improve plant health.

4.6 Balancing Diagnostic Costs

Sixth, it remains imperative that any clinical methods be commensurate with the goal of the diagnosis, triage in one sense of the word. Every sample does not call for advanced techniques. With no intent to diminish the importance of any diagnosis, clinics need to maximize the resources at their disposal to provide as much service to society as possible without limiting their capacity to respond to extraordinary circumstances with every tool possible when the need arises. Most plant problem clinics operate with some level of public support, so there is an obligation to serve any tax-paying client to the fullest extent possible and warranted. Charging a fee for diagnostic services to supplement operating budgets and recoup costs may make larger staff and more costly procedures possible, but those same fees can also interrupt vital links to the very community that can serve as front-line detectors and “citizen scientists” for early disease detection and effective intervention. These fragile links need nurturing, and diagnostic fees as such tend to be counterproductive. The balance between affordability, applicability, accuracy, and sensitivity must be decided jointly by the clinician and client while keeping all these competing factors in view. One trend that helps in this regard is that of illustrated electronic communications to initiate a diagnosis. Assuming good photographic skills on the part of the client or disease surveyor/scout, a good diagnostician should be able to recognize the routine problems and dispatch them with minimum expense and time to save resources for the major events that will require “all hands on deck” and full spectrum diagnostics. In short, when it comes to advanced techniques, discretion in deployment may be as important as capabilities.

4.7 Diagnosticians Are the Major Ambassadors of Phytopathology

Seventh, related to the theme of how the costs of diagnostic services are met, we must further acknowledge that disease diagnostic services and the essential cooperative nature of this enterprise with the client are almost certainly the sole link between the phytopathology profession and most of the general public. That being the case, the diagnostic clinic is well advised to put its best foot forward for the reputation of the profession. Additionally, even though all life on planet Earth is based directly on plant health, the scarcity of providers of diagnostic plant pathology services contributes to the general obscurity of phytopathology in the public service arena. For comparison purposes, according to the US Bureau of Labor Statistics, the medical profession has in the neighborhood of 700,000 diagnostic physicians and surgeons and the veterinary profession about 70,000. Diagnostic phytopathologists, on the other hand, are very generously estimated to number only about 1000 nationwide based on the number of members in the American Phytopathological Society that participate in the Diagnostics Committee plus the estimated number of employees at state- and federally sponsored diagnostic labs and clinics around the country. Diagnosticians undoubtedly serve as the foremost ambassadors for the entire profession. Deciding on the method(s) to be used in a diagnostic project must take into account much more than cost and applicability but must also consider the representation of the profession in the public forum and the potential contributions to be made to the common good.

5 The Challenges of Abiotic Disease Diagnosis

In many respects, the diagnostic approach for any disease, biotic or abiotic, will follow the same pathways. Exceptions are granted when the diagnostician knows a crop extremely well and is fully aware of all the horticultural inputs and their effects. Familiarity with common biotic diseases for the crop also helps narrow the diagnostic focus. Usually, however, a thorough abiotic disease diagnosis is a meticulous process of eliminating possible biotic causal agents, then resorting to attempts to match abiotic causal agents to the symptoms observed.

6 A Universal Diagnostic Process

Stepwise, here is a rational process for any disease diagnosis. We emphasize that much of a successful and accurate diagnostic process occurs before moving into the lab. It gathers enough information to evaluate both categories, biotic and abiotic:

6.1 Step One: Assemble a Comprehensive Syndrome Description

Begin with a careful examination of the syndrome to record all symptoms plus any signs of a pathogen with the goal of fully describing the problem. Is the presentation actually abnormal? How should the crop appear at this stage in the production cycle? At this early juncture, it is essential that detailed record keeping be initiated and practiced throughout the diagnostic process. These documents are vital for legal disputes and for trace back – trace forward investigation.

6.2 Step Two: Check for Spatial Patterns

Look for any patterns in the symptom expression on both the individual plants and within the plant population as a whole. Do not confine the exam to just the single crop, but also evaluate neighboring flora, even weeds if they are present. Examine whole plants as much as possible, internally and externally both above and below ground, and be prepared to sacrifice representatives to supply samples for further lab work.

6.3 Step Three: Establish the Disease Chronology

Determine the chronology of symptom appearance and any changes in the syndrome over time. When did the syndrome start? Was the onset sudden? Do symptoms abate, stay the same, or get worse?

6.4 Step Four: Assemble a Comprehensive History of the Horticultural Treatment of the Crop

Find out as much as possible about how the crop has been grown. Is the crop new to the grower, a new variety, growing in a different structure, or has something changed in the horticultural regimen (physical and chemical qualities of the growing media, water quality and amount, fertility, light levels and quality, temperature, growth regulator use)? Pay particular attention to the mode of delivery of any horticultural treatments. Phytotoxic agents can reach a crop in any number of ways, including by direct and deliberate application, drift, contamination of pesticide or fertilizer product or application equipment, rate miscalculations, poor application techniques, contamination of soil or surroundings, mulch or soil amendments, in irrigation water or runoff, etc.

6.5 Step Five: Consult Literature

Check references on the plant in question to learn of any unusual sensitivities, preferred growing conditions, idiosyncrasies in growth cycles and plant appearance,

and reported diseases and disorders. APS compendia and reference manuals such as this one are good sources of information (Chase et al. 1995; Daughtrey et al. 1995; Gill et al. 2006; Gleason et al. 2009; Horst and Cloyd 2007; Horst and Nelson 1997; Horst 2013).

6.6 Step Six: Begin Laboratory Analysis

Based on the information gathered, determine what laboratory testing might be warranted to attempt confirmation. (It is here that the more technical laboratory work introduced earlier in Sect. 1 and addressed in more detail in Sect. 8 usually begins). Determine whether any biotic agents discovered using laboratory techniques and coupled with symptoms and disease chronologies are primary or secondary. Soil and tissue analysis are advised for confirmation of symptoms associated with known syndromes of nutrient deficiencies/excesses or soil pH problems. A bioassay using indicator plant species can confirm problems with suspected harmful soil residues. Testing of pesticide products for foreign components or testing of plant tissues for residues of pesticides is much more involved and expensive, usually requiring some idea of the nature of the residue sought.

6.7 Step Seven: Consolidate Information

Review all the gathered information to synthesize a likely diagnosis. The process for an abiotic disease diagnosis can eventually assemble large amounts of information because it is fundamentally a process of elimination, and much of the facts gathered may end up having little or no bearing on the final conclusions. Nevertheless, the quality of the diagnosis depends on thoroughness of the overall investigation. As stated earlier, using the most advanced diagnostic tools available cannot make up for deficiencies in steps 6.1 through 6.5.

7 A Special Diagnostic Category: Indexing Plant Propagative Material for Cryptic Pathogens

Indexing for plant pathogens in propagative material is a different sort of diagnostic challenge altogether. The most sensitive detection methods available deserve top consideration, especially if the propagative material is to be moved to a new location within a country or to another country and must meet the attendant phytosanitary requirements. The potential gains in overall plant health (and thus profitability) from indexing of asexually propagated perennial plants in particular can be substantial. The rewards for indexing annual plants are different but similarly valuable because the crop customarily starts from seed each year anyway. With annuals, indexed seed is the goal. Indexed seed and stock plants are extremely valuable, though they can be quite expensive to acquire and maintain. Furthermore, these costs come up front,

before any actualization of profits. Rewards may or may not prove to be worth the investment. Costs of the alternative (imperfect regulatory protection in place of starting with clean stock) are seldom fully considered in any cost-benefit ratio calculations; indeed such values are difficult to commodify without advanced economic skills which are outside the circle of most growers' acquaintances. Assigning costs to any of the parties involved, especially at the outset, is also controversial, but eventually the potential for a higher profit margin and premium price for the superior product could supply ample financial returns to cover the setup and day-to-day operating expenses. Acquiring the equipment for the most advanced indexing techniques is another matter. Prices for advanced sequencing equipment and subsequent data processing are becoming more affordable with the passage of time. Grants and sharing resources collaboratively with other professions needing similar services (e.g., medical/veterinary health and ecosystem services) may be a possible solution. Still, in a free market, there will always be a niche for the cheapest items of any commodity, so we should not fool ourselves into thinking that every grower will eagerly adopt propagation from high-quality indexed stock. Regulatory oversight and public funding to protect consumers and natural resources from the consequences of substandard plant material are still necessary and advisable.

8 Diagnosis of Biotic Diseases: Direct Methods

Historically, plant disease diagnosis has targeted direct detection of the organism (s) associated with a syndrome to arrive at a diagnosis. In the case of an abiotic disease, there may be no physical evidence of a causal agent to detect and obvious shortcoming. Still, direct methods can be used in a process of elimination toward an abiotic diagnosis, and some abiotic agents do indeed leave physical evidence amenable to direct methods. Lately, progress has been made in indirect methods of disease diagnosis. These are covered in Sect. 9.

8.1 Microscopic Methods for Diagnosis

The careful observation of diseased plants and tissues using various means of magnification is the oldest, potentially the quickest, lowest cost, and most accessible diagnostic method available. Unfortunately, in many respects, the more advanced microbiological, serological, and molecular methods have distracted diagnosticians from the inherent value of simple and thorough light microscopic techniques. Skill in microscopic methods definitely requires good equipment and the time and material for plenty of practice. An apprenticeship with an adept microscopist is a great advantage. The more one practices microscopy and the discriminating observation and attention skills required to spot signs of a pathogen, the more exciting discoveries appear. Some good advice when examining diseased plant tissues for clues about the pathogen: have patience and *expect* to discover useful information using the microscope, do not give up easily, and do not restrict your examination to just a

few pieces of symptomatic tissue. Carefully examine all stages of the symptoms insofar as you can reconstruct a time frame for the syndrome. If you see nothing noteworthy upon initial examination, try a moist chamber incubation for a few days to encourage fungal sporulation, and then reexamine. Inducing fungal sporulation to permit microscopic examination might require many weeks and manipulation of incubation conditions in some cases. In the emerging era of identification at the molecular level, there remains a significant place for morphometric identifications based on reproductive structures (Hyde et al. 2010) using fungal identification literature (Barnett and Hunter 1998; Leslie et al. 2006; Siefert et al. 2011). High-resolution digital photographic equipment for both macroscopic and microscopic images allows the sharing and cataloguing of images with and by experts over the Internet, thereby enhancing the technique even further. Good light microscopic equipment for both reflected and transmitted light microscopy equipped with digital cameras can cost from $3,000 to $4,000 at the low end to as much as $15,000 at the high end.

Electron microscopy provides even more imaging capacity which can prove especially useful for diagnosis of viral and viroid diseases. Virus and viroid particles can be fairly quickly detected in expressed sap dried down and negatively stained on a coated grid. Electron microscopes small enough for a lab bench or tabletop capable of both scanning and transmission electron microscopy can be obtained for around $175,000 to $200,000. However, the specialized training and consumables make this equipment a likely purchase only when in support of a larger program or one with an emphasis in virus diagnosis or morphologically supported taxonomy and systematics.

Microscopic detection methods suffer from the sensitivity inherent in the process. It is impossible to examine large volumes of host tissues or the environment using a light microscope to find suspected targets. The sampling problem is compounded when using the electron microscope. Extraordinary observation skills, training, and experience can lessen the inefficiency to a degree.

8.2 Microbiological Methods for Diagnosis

Several excellent treatises and lab manuals for the microbiological techniques traditionally used for plant disease diagnosis are available (Burns 2009; Dhingra and Sinclair 1995; Dugan 2006; Schaad et al. 2001; Shurtleff and Averre 1997; Streets 1979;Tuite 1969; Waller et al. 2002) plus there are many methods described online at http://wiki.bugwood.org/Diagnosticians_cookbook.

Bacterial differentiation to the genus level is possible with the use of a few semiselective culture media coupled with tests for oxidase, Gram reaction, anaerobic growth, and hypersensitive response. These tests are not particularly difficult, but selection of isolates to carry forward from streaked plant tissue is based on experience. Careful attention to the chemistry and recipes of the culture media will encourage success. One is reminded to use fresh solutions and young cultures for best results. A few useful additions to the diagnostician's bench include

semiselective media for fungi that can help to separate out oomycetes from true fungi, can encourage sporulation, and can be amended to identify fungicide resistance.

8.3 Serological Methods for Diagnosis

Serological methods rely on the specificity inherent between an antigen and antibody developed as they are naturally formed by the immune system after injecting the antigen into the bloodstream of an animal, usually a mammal. This specific recognition phenomenon can be exploited to great advantage to seek out pathogen-specific antigens in any matrix (expressed plant sap, ground tissue extracts, etc.) and coupled with stains for light and/or electron microscopy, spectrophotometry, etc. Antisera produced in this traditional fashion are polyclonal and a mixture of antibodies generated to recognize different parts of a surface protein molecule or antigen. Most antibodies are now produced using hybridoma technology in tissue cultured cells that can be kept indefinitely to produce more antisera as needed. Antisera produced via hybridoma technology are monoclonal and recognize as a specific part of the antigen.

Immunodiagnostics are quite reliable depending of the selectivity and sensitivity of the antiserum. Several enzyme-linked immunosorbent assays (ELISAs) have been developed to detect pathogens, especially bacteria and viruses. However, this does require that a species-specific antibody is already available to perform ELISA. The collective commercially available catalog of ELISA assays that have been developed so far to diagnose selected viral and bacterial pathogens is limited, but the availability of commercially available positive and negative controls encourages standardization of testing and verification of results. Interpretation of results must be done with care. Sutula et al. (1986) discuss the potential pitfalls and how to avoid erroneous interpretation. Once set up, an ELISA assay can be performed rapidly, but the costs of superior antigen identification and antibody development plus signal attachment can be a constraint. Once all reagents are developed and validated, a 96-well plate ELISA assay format can be utilized to reduce cost when the test volume is high. A microplate reader will be required to perform ELISA. Some serological assays have been incorporated into a very handy single sample dipstick or a lateral flow format that can be employed even in the field when and where the samples are collected. Both types of antisera, polyclonal and monoclonal, are often used in double-antibody sandwich ELISA (DAS-ELISA) and the aforementioned dipstick or lateral flow device. Monoclonal antibodies are more expensive to produce but since they are highly specific to the pathogen antigen, can detect single strains of a virus or a bacterial species or potentially subspecies. Results between tests are highly reproducible. Polyclonal antibodies are produced as a mixture, which has the advantage of being less expensive to produce as well as detecting multiple strains of a virus or multiple species of a bacterial genus. However, they are more likely to cross-react with other proteins. The lower specificity of polyclonal antisera may have an additional advantage in that they may provide clues to help identify unknown or as-yet uncharacterized viruses.

DAS-ELISA kits including all reagents and controls are available from many vendors globally, costing between $1–12 per test well in kit cost. This plate-based assay is scalable, making diagnosis of large numbers of samples relatively easy and decreasing cost per sample. Quick tests such as dipsticks and lateral flow devices are readily available but for a smaller number of pathogens than plate-based ELISA and cost $1–5 per test. The limits of detection using ELISA are 10^5 to 10^6 CFU/ml.

Serological specificity has been adapted a step further by attaching the antibodies to a fluorescent marker that emits a visible signal when viewed under a specially equipped microscope upon binding with its antigen while illuminated with a particular wavelength of light, usually in the UV range. This immunofluorescent microscopy depends on the availability and specificity of the antiserum. Flow cytometry (or FCM), used sparingly in plant pathology, can also take advantage of immunofluorescence technologies to detect pathogen antigens in large volumes of individualized plant cells. Flow cytometry offers enormous potential for indexing plant tissues for cryptic pathogens or a genetic sequence of interest, targeting any antigen or genetic sequence for which a specific antibody can be devised and a fluorescent probe attached (D'Hondt et al. 2011). It is even possible to sort the scanned cells based on the signal detected, thus permitting further work specifically with the selected tissues. Pathogen detection and viability in environmental samples is another application of flow cytometry (Chitarra and van den Bulk 2003). The need to develop antibodies and affix a fluorochrome is unnecessary to make use of flow cytometry if the target cells have an innate signal fluorescence when illuminated by a particular wavelength of light from the laser light source in the instrument. A basic FCM instrument without sorting capabilities costs from approximately $40,000 to $100,000. The sensitivity of FCM is in the range of 10^4 CFU/ml.

8.4 Metabolic Tests for Pathogen Identification

Microplate-based metabolic assays such as Biolog® have successfully guided bacterial diagnosis, and also limited fungal diagnosis of plant pathogens. This system records the ability of a given active culture to metabolize specific carbon sources; the resulting activity matrix is compared to a library of known responses for over 2000 bacteria and yeasts. A separate setup can identify a smaller number of filamentous fungi. Originally developed for assessment of bacterial mammalian pathogens, the current GenIII library includes many plant pathogens as well, and also tests for Gram reaction. However, the use of this technology to identify specific organisms may be limited to genus, with a suggestion as to the species. Additionally, this system is limited for obvious reasons to culturable organisms. But it requires little specialized expertise and can be piggy-backed onto a plate reader that can also be used for ELISA, creating some efficiency in equipment purchase and upkeep for both systems. Biolog® can cost about $20 per sample, assuming the use of the less-costly nonautomated equipment.

Identification of bacteria and fungi via fatty acid methyl ester (FAME) analysis is exemplified by the Sherlock™ Microbial ID system (MIDI). It is based on gas

chromatography and, in disease diagnosis, has been most heavily used for identification of culturable aerobic plant pathogens. This well-plate-based assay can be fully automated to resolve the identification of a microbe and compare its profile to a large library of known organisms. As with Biolog, this system generally resolves bacteria to genus reliably and suggests species and subspecies. The MIDI system develops a dendrogram of relatedness with references in the library, as chosen by the user, and generates a similarity index to the closest match. The MIDI tests generally cost $30–50 per sample, with savings possible for increasing numbers of samples run together.

Diagnosticians tend to couple data from Biolog® or FAME tests with other sequence-based, cultural, or plant-based tests to confirm the identified organism is capable of causing the disease symptoms of the affected plant. One thing these two tests can provide that molecular detection such as PCR and sequencing cannot is the verification that the detected organism is viable.

If any or all these traditional approaches fail to provide a complete and/or clear diagnosis, molecular diagnosis at DNA or RNA level (Sect. 8.5) is an appropriate next step.

8.5 Molecular Methods for Pathogen Detection and Identification

Plant pathogen identification has always been a challenging task requiring increasingly more sophisticated tools and techniques as our understanding of host-parasite relationships and pathogenicity advances. As stressed earlier, even with molecular methods, initial assessment still requires techniques of visual inspection to recognize the symptoms caused by the infection such as chlorosis, necrosis, stunting, distortion, dieback, etc. Properly targeted sampling and careful shipment of the selected plant parts or nucleic acid extracts to a diagnostic laboratory is required for a methodical analysis to identify the pathogens at genus, species, or strain level. Molecular analysis requires various pieces of equipment (water bath, refrigerator, freezer, microcentrifuge, tissue homogenizer, nucleic acid quantification equipment, PCR and real-time PCR machines, gel-documentation system, Sanger and next-generation sequencing platforms, sequence analysis software and computers), funding for the lab supplies (extraction kits, reagents, pipettes tips and tubes, etc.), and a skilled technician to do the work. Some labs charge a price for every sample submitted for analysis to cover the operational cost. Other labs cover costs using grants from grower associations and local and federal governments, thus providing diagnostic help without any additional charge to the sample submitter. Cost estimates are given under the individual subject headings.

8.5.1 DNA and RNA Extractions

The starting material for DNA and RNA extraction varies depending on the pathogen. Harvested bacterial/fungal cultures grown on plates or finely ground up infected

plant leaves, stem, or root tissues (100–200 mg) are extracted using spin-column kits or chemical purification such as CTAB methods kit to obtain genomic DNA. Elution using ultrapure, PCR-grade water (100–200 μl) is always preferred because the elution buffers provided in many extraction kits contain enzyme inhibitors which could interfere with downstream applications. The majority of the viral diseases are caused by RNA viruses, and their detection will require extracting total RNA from infected plant tissues (100–200 mg) using a kit such as a QIAGEN RNeasy Plant Mini Kit. A microcentrifuge is required to perform this task. The nucleic acids can be stored at −20°C until ready for analysis. The cost for the QIAGEN column is reasonable ($2–3). The quality of the DNA and RNA extracted must be quite good for successful downstream applications.

8.5.2 Molecular Identification

The precise strategy used for identification of plant pathogens can vary depending on the organism and the information available about the pathogen. The NIH GenBank® database (www.ncbi.nlm.nih.gov/genbank) has nucleic acid sequences for numerous well-known species, and the database is growing constantly as scientists from all over the world deposit sequences. Several million sequence deposits already exist in the GenBank, and that includes sequences from a large number of important plant pathogens. There is a good chance that the sequence information about a suspected plant pathogen could be already present in the GenBank. Sequence information for rare plant pathogens may not exist in the GenBank, so this would require using universal primers (ribosomal RNA or mitochondrial COX1) or degenerate primers (β-tubulin, actin, histone, etc.) to amplify a marker sequence by PCR and perform phylogenetic analysis to match to a closely related species for identification. Therefore, a strategy for molecular identification has to be selected carefully.

8.5.3 Fungal Species Identification

Species-Specific PCR

Positive identification of plant-associated fungi is tremendously aided by modern genetic analysis methods. One must remember, however, that simply identifying a plant-associated fungus based on a genetic sequence is not strictly the equivalent of a disease diagnosis, though it can be a step in the process. Pathogenicity testing will be required to confirm the virulence of any isolates from the diseased tissues. Proving pathogenicity can be a simple or very complicated process which may need to take into account groups of organisms which work in concert in a chronological set or physiological sequence. Furthermore, environmental predisposition of the host to achieve a susceptible state can be complicated to determine and recreate.

If a fungal plant pathogen is suspected to be a particular species based on morphology and culture characteristics, but a confirmation is required, then the sequences can be obtained from the GenBank for that species, and species-specific primers can be designed using CLUSTAL W sequence alignment generated by comparing to all closely related species and then selecting a unique sequence sites to design a species-specific primer. A straightforward PCR assay can be performed,

and the size of the DNA band amplified should match the sequence information already existing in the GenBank, thus providing confirmation. Further confirmation can be derived by digesting the PCR products with a restriction enzyme and developing a banding pattern to detect unique restriction fragment length polymorphisms (PCR-RFLP), which should match the expected sizes. This approach has an added advantage, as it can be used for plant pathogens that are difficult to culture because the DNA extracted from infected plant could be used for PCR. The primers and a PCR test per sample could cost around $20. A PCR machine and an electrophoresis gel system would be required. A basic gradient block PCR thermocycler can be purchased for as little as $5000; gel electrophoresis equipment can be $12,000 or more, depending on the camera and size of the setup.

Sequencing

Several nuclear or mitochondrial marker sequences can be used to identify plant pathogenic and other plant-associated fungi. This strategy requires using universal primers or degenerate primers to amplify a specific nuclear or a mitochondrial marker, sequencing, and then comparing the sequences to those already existing in the GenBank using a BLAST search tool. The markers generally used for fungal taxonomy are as follows.

Ribosomal RNA

The ribosomal RNA region (18S-ITS1-5.8S-ITS2-28S) has been well investigated from numerous fungal pathogens. 18S rRNA, 28S rRNA, and ITS1-ITS2 spacers are the markers that are extensively investigated from fungal pathogens. The ITS1 and ITS2 spacers are known to accumulate mutations, and it is often possible to find unique species-specific sequences in this region to generate phylogenetic trees for identification or to design species-specific primers. The ITS region is multicopy, but surprisingly all the copies that are present in a fungal species turned out to be identical. This was exploited by fungal taxonomists to build a rich collection of sequence information in the GenBank database. Universal primers are available that amplify well from both known and unknown fungal cultures grown on media (Table 1) (White et al. 1990). Culturing on a plate is necessary before performing this procedure to eliminate all the other background fungi present in the diseased tissue.

Other Markers

Mitochondrial cytochrome oxidase 1 (COX1), the nuclear elongation factor-1 α (EF1α), β-tubulin (Tub), histone (His), etc. could be used as well, but the sequence information library is not as rich for fungal species as it is for the ribosomal sequences. Multilocus sequence analysis (MLSA) involving several different markers greatly improves fungal species identification. The PCR products can be shipped to a commercial sequencing company or university core laboratory to obtain the sequence information. The cost for sequencing PCR products or cloned inserts is steadily decreasing, around $10 per sequencing run. Multiple sequence runs as well as sequencing both forward and reverse strands would greatly improve sequence

Table 1 Generic primers for amplifying fungal and bacterial pathogen DNA

Organism	Marker	Primer	Sequence 5′–3′
Fungi	18S	NS1	GTA GTC ATA TGC TTG TCT C
		NS8	TCC GCA GGT CAC CTA CGG A
	ITS	ITS1	TCC GTA GGT GAA CCT GCG G
		ITS4	TCC TCC GCT TAT TGA TAT GC
		ITS5	GGA AGT AAA AGT CGT AAC AAG G
	28S	LROR	ACC CGC TGA ACT TAA GC
		LR5	TCC TGA GGG AAA CTT CG
		LR7	TAC TAC CAC CAA GAT CT
Bacteria	16S	27f	GAG AGT TTG ATC CTG GCT CAG
		1495r	CTA CGG CTA CCT TGT TAC GA

accuracy and provide a clear identification. Occasionally, diagnostic labs do have their own Sanger sequencing machine (Applied Biosystems 3130XL Genetic Analyzer), with equipment costs starting around $150,000.

Real-Time PCR

The conventional standard PCR procedure can be quite labor-intensive, is prone to aerosol contamination, and is good for screening only a few samples. If the sample size is large, then a real-time PCR (also called quantitative PCR or qPCR) assay, such as one based on TaqMan technology, is ideal. This does require two primers (forward and reverse) and a species-specific fluorescent probe (6FAM or TET). This technology exploits the *Taq* DNA polymerase exonuclease activity, which shreds the probe bound to the species-specific site releasing the fluorescent marker and producing a positive result (Ct value 16–32). The probe fails to bind other non-target species DNA due to mismatches, producing a negative result (Ct value 32–40). The Ct values <32 produced from a test sample are a good indication that it is positive for this particular pathogen. The major advantage of the TaqMan assay is scalability and ease. A real-time TaqMan assay could cost $15 per sample for the master mix, primers, probe, and the reaction tube. Initial investment in a real-time PCR thermocycler machine is required ($10,000 or above). The detection sensitivity varies widely due to both technique and primer-probe sensitivity. Detection limits can be anywhere from a few cells to around 10^3 to 10^4 CFU/ml.

Fluorescence In Situ Hybridization (FISH)

Fluorescent *in situ* hybridization or FISH is also based on molecular detection of particular genetic sequences. The attachment of a fluorochrome to a DNA probe permits the detection of that specific sequence under the light microscope when illuminated by the proper wavelength of light. In this respect, it is similar to immunofluorescence microscopy; however, sensitivity is better – around 10^3 CFU/ml. – plus it has the advantage of being able to detect unculturable organisms. Autofluorescence of host tissue can interfere with this technique.

Flow Cytometry with Nucleic Acid Probes

Earlier the technique of using fluorochrome-labeled antibodies was mentioned as the basis of sorting cells for a particular trait while passing through a flow cytometer. Substituting a nucleic acid probe for the antibodies to perform the selectivity opens up new possibilities. As with any nucleic acid-based technology, an effective fluorescent probe must be designed that will operate under the ambient conditions of the instrument and have ready access to the dissociated nucleic acid of the target for the probe to attach and signal.

8.5.4 Bacterial Species Identification

The strategies used for bacterial identification are very similar to the fungal identification strategies, but the markers are different. The 16S rRNA gene has been used as a standard to identify bacterial pathogens successfully. GenBank has quite a large collection of 16S rRNA sequences. Universal primers (Weisburg et al. 1991) to amplify 16S markers are available for bacteria isolated from infected plants and grown on synthetic media. Plants can harbor several different species of unculturable/fastidious endophytes, and testing for a bacterial species infecting the plant from this bacterial DNA mix would require another approach. A species-specific PCR or real-time TaqMan assay would be sufficient to test for the bacteria that are not culturable or present as a mixed population. Keep in mind that the test detects an organism's unique target nucleic acid sequence but does not assess the viability of that organism.

8.5.5 Viral Identification

The genetic material for viruses could be DNA or RNA. Viruses containing RNA require a slightly different approach; a reverse transcription step is required prior to PCR. Kits are available to standardize this process, including the Qiagen one-step RT-PCR kit, containing two enzymes (reverse transcriptase and *Taq* DNA polymerase). PCR and RT-PCR work well for a plant infected with just one virus. Plants carrying multiple virus infections would require designing specific primer sets for each virus sequence separately. Then PCR or RT-PCR has to be performed on the mixed DNA or RNA templates using specific virus-primer sets separately to confirm different viruses present in the multiple virus infections. Development of primers for these methods works only when the virus sequences are known and available in the GenBank.

8.5.6 Metagenomics

If the molecular identification fails, then the fungal, bacterial, or viral pathogen could be a new, undescribed species. This requires a different strategy using sequencing equipment and considerable computational power for data analysis (Perkel 2011). The metagenomic approach is ideal for this purpose. The ITS or 16S PCR products or the genomic DNA are fragmented into 200–400 bp pieces and sequenced using a next-generation sequencing (NGS) platform like MiSeq Illumina or Ion Torrent PGM. No prior sequence information is required for this procedure. An adapter is attached to the DNA fragment, and an adapter-specific primer is used to sequence all

the random pieces. One million or more sequence reads of 200 to 400 bp sizes can be generated from random pieces and assembled. The strategy is quite robust, and BLAST searches of these random sequences will provide a good insight into all the species of fungi, bacteria, or viruses present. Since the amount of data generated is high, it is better to select the templates carefully. Viruses can be separated and isolated using a cesium chloride gradient with ultracentrifugation to reduce the contaminating plant RNAs. The 16S PCR products generated by universal primers for bacterial identification will help to eliminate the background DNA from plants and help focus on identifying all the bacterial species present in this infected plant specimen. The NGS strategy thus has the advantage of permitting PCR products from multiple markers to be sequenced simultaneously using different adapters. The current cost to perform a single NGS run is $200 including the enzymes, buffers, and other reagents. Of course, an NGS machine ($80,000) or access to one is required. Prior knowledge about the plant pathogen is not required to perform this procedure. In other words, this technology can identify unknowns rather than simply confirm or reject diagnostic premonitions as is the case with serological or PCR techniques.

These strategies as currently deployed in diagnostic laboratories represent the most advanced diagnostic methodology presently available, capable of making a definitive molecular identification of a plant pathogen at the genus, species, or strain level.

8.5.7 Interpretation of NGS Sequence Data

The discoveries to be made using deep sequencing technology on plant tissues are bound to uncover some surprises. Based on reports in other venues using this technology, one should expect to find gene sequences indicative of gene transfer and uptake from other organisms into the plant genome that will require some critical assessment to understand their significance. Interpretation and subsequent regulatory and management policies will be especially tricky in indexing plant tissues for freedom from plant pathogens. A host of endophytes, parasites, and pathogens may lurk in and on plant tissues, the impact and significance of which will require much study on interactions among these agents and the host plant. The consequences of altering the internal and external microflora of an apparently healthy host plant in the interest of "cleaning up" germplasm could have unexpected consequences, both favorable and unfavorable. Additionally, the commitment of resources to remove genetic material from elite germplasm should be evaluated in the light of the expected stability of the altered germplasm. If the excised sequence is likely to be quickly reacquired and reinserted under natural conditions by various means, the cost-benefit of such a procedure needs reconsideration.

8.5.8 DNA Microarray for Pathogen Detection and Identification

DNA microarray technology and other chip-based technologies are another excellent way to screen for well-characterized plant pathogens. This requires synthesizing DNA oligos (10–20 bp) and spotting them on to a microchip. Selected gene sequences from pathogens could be spotted on different regions of the chip. The DNA extracted from infected plant tissues could be hybridized to the oligos in the

microchip. If the hybridization is successful, a positive result for the pathogen is generated. There is an advantage with this technology because all known pathogens can be tested in one assay. The oligo synthesis and spotting to microchips are performed by commercial companies like Affymetrix for a price ($20 per microchip or lower for large volume orders). Hybridization equipment and a computer to read microchip results are required ($20,000).

8.5.9 The Advent of Biosensors

Biosensors represent the latest in pathogen/disease detection technology. The various approaches and technologies used to develop these detection devices defy any attempt to categorize and classify them meaningfully here. Suffice it to say that numerous agents of selectivity (serological, nucleic acid based, enzymes and enzyme products, metabolic substrates, phages) to produce any number or combination of signaling methods (electrical, chemical, optical, vibrational, magnetic, and audible) are under exploration (Fang and Ramasamy 2015). The FAME and Biolog technologies covered in Sect. 8.4 and the microarray tool discussed in 8.5.6 can be considered precursors and in the case of the microarray, an elaborate early form of a biosensor. Nanotechnology has proven to be extremely helpful in the development of biosensors to affix the selective agent to a matrix and to deliver the test medium through the sensor. Biosensors detect a single target, and sensitivity reflects the nature of the selective agent utilized.

9 Indirect Methods of Plant Pathogen or Disease Detection

All the previously discussed methods for solving a plant problem directly approach the causal agent. Indirect disease diagnostic and pathogen detection techniques are emerging that offer the possibility of diagnosis by searching for signals emitted by the pathogen itself or emitted by the diseased host plant as a consequence of the infection (Martinelli et al. 2015).

9.1 Detection of Volatile Organic Compounds

The fact that infected or stressed plant tissues release volatile organic compounds (VOCs) has been exploited by utilizing either keen animal olfactory senses (usually dogs) (Whattam et al. 2010) or electronic sensors to detect informative gaseous chemical signals indicating pathogen presence or a diseased host (Sankaran et al. 2010). These disease-specific volatile compounds can be presented in real time or on absorptive pads to dogs trained to be alert to specific compounds, or they can be analyzed by gas chromatography. In general, the VOCs known to date tend to be general and not disease specific in nature. Since the VOCs are sometimes released prior to visible symptom development, proactive management tactics could be undertaken before regular scouting detects any problems, even if the threat is not precisely known. The use of VOCs is hindered to some degree by the variability that

naturally occurs in emissions from a plant under normal circumstances and the dilution and blending of VOCs from various sources under natural outdoor conditions. Both of these hindrances should be less of a problem if applied to a uniform crop growing under greenhouse conditions.

9.2 Spectral Imaging of Host Plants

An analysis of the reflected and transmitted light from a crop of interest can yield valuable information about the health of that crop. Several basic approaches are finding some application in disease detection: thermography, biophotonics, hyperspectral imaging, and fluorescence imaging (Fang and Ramasamy 2015; Sankaran et al. 2010; Mahlein 2015; Martinelli et al 2015). Some of these techniques are sensitive enough to identify infection prior to symptoms expression, thus making better informed proactive management a possibility, though one must recognize that not all invisible and incipient infections invariably proceed to full-blown disease warranting intervention.

9.2.1 Thermography

Plants in the early stages of infection or stress can exhibit changes in individual leaf or whole canopy temperature. By using infrared wavelength detection instruments, the onset of a problem can be witnessed in real time and management strategies based thereon. The rise in temperature is usually a consequence of stomatal aperture closure and the resulting reduction in transpirational water loss, though some heat from additional metabolic activity during the infection process is likely too. The typical changes that occur due to ambient environmental conditions can confound the use of thermography for outdoor use. The technique is also nonspecific, mostly able to tell only healthy from diseased or stressed individuals or populations of plants.

9.2.2 Hyperspectral Imaging and Biophotonics

This technique, similar to thermography, makes use of more than just infrared emission from a leaf or a crop to provide clues about the overall health of the target plant; therefore, it enjoys a measure of added specificity to a degree (Sankaran et al. 2010). Coupled with accurate diagnosis by another method, one may theoretically compose a spectral image profile using multiple wavelengths between 350 nm (ultraviolet) and 2500 nm (far infrared). Biophotonics takes the use of light energy analysis further to measure photon emission, detection, absorption, reflection, modification, and creation from biological materials with the goal of identifying signals indicating a diseased state or pathogen presence.

9.2.3 Fluorescence Imaging

Photosynthesis in plant foliage can be monitored and evaluated by observing the fluorescence of chlorophyll (Sankaran et al. 2010). In some cases, the difference

between biotic and abiotic causal agents is discernible, but like other indirect methods mentioned previously, specificity is low, plus the technique is restricted to lab application on single plants/leaves for the most part.

10 Accurate and Timely Reporting of the Diagnosis

The method of communication of test results and final diagnosis often depends upon the consumer of the information. Diagnoses for specific pest/pathogen surveys made by regulatory agencies for growers, port inspection stations, or aimed at particular commodities are often communicated as "organism X detected/not detected." General regulatory survey and plant inspection diagnoses are more comprehensive but do not always include disease management or mitigation recommendations. The speed of the identification, specificity of the diagnosis, and the confidence in the diagnosis must all be to a high level, since decisions to release a shipment or quarantine a greenhouse are not taken lightly. Diagnostic reporting of plant diseases is subject to the same diverging ends as medical diagnostic reporting. A desire for confidentiality for growers with diseased crops must be balanced with the need for disclosure of particularly serious and damaging diseases, especially those adapted to dispersal on asymptomatic host material and contaminated personnel, equipment, or media. The diagnostic clinic must continually strive to serve clientele in such a way as to simultaneously protect and enhance overall industry integrity and success. Indiscrete reporting of disease incidence only complicates and interferes with the open channels of communication between growers and diagnosticians necessary to monitor and respond to commercial plant health issues on a local, national, and international scale. Reporting of diagnostic results should therefore protect the integrity and reputation of the grower while concurrently informing others that need to know and take protective precautionary measures against an emerging pathogen. Communications of these results are often via secure communications channels that may include email to a specific need-to-know group of recipients and may be automated as part of a communication chain.

Diagnoses made by university or private laboratories for commercial or extension clientele are often sent by email to the sample submitter and listed clientele only and usually include disease management recommendations. Specificity of the diagnosis may be to genus level only, but the cause of the plant problem is often described more fully and may include both regulated and nonregulated organisms. Reporting from university laboratories is often automated through their laboratory information management system (LIMS). High-impact or regulatory diagnoses made by partner laboratories in the National Plant Diagnostic Network are communicated via a codified chain of communication protocol to ensure the security of the detection data and the timeliness of the communication.

In the United States, important local diagnostic information is forwarded to the USDA National Agricultural Pest Information Service (NAPIS) and combined with data from the Cooperative Agricultural Pest Survey (CAPS) to comprise an

overview of the current pest and disease situation nationwide. The National Plant Board also serves to consolidate and communicate general pest and disease incidence data for the various state plant health regulatory agencies. Additionally, public mapping and reporting platforms exist online to support survey efforts, citizen-science-type projects, and regulatory reporting. One example, EDDmapS (https://www.eddmaps.org/), includes a verifier step to ensure accuracy in reporting and generates the findings as county-level maps. NAPPO releases detection information via its Phytosanitary Alert System (http://www.pestalert.org) to "provide up-to-date information on plant pest situations of significance to North America." The Florida Department of Agriculture and Consumer Services – Division of Plant Industry – reports on detection activities from plant nursery inspections and requests for identification of plants and pests from the public. These reports are publicly available on the division's website (http://www.freshfromflorida.com/Divisions-Offices/Plant-Industry/Plant-Industry-Publications/Tri-ology-FDACS-DPI). Many agencies use tools like these as well as blogs, alert LISTSERV, and social media outlets to communicate detections and rapidly exchange information to encourage early detection, accurate diagnosis, and effective mitigation or management of plant diseases.

Whenever a new pathogen is discovered, the diagnosis is shared among the regulatory, university, and USDA-APHIS-PPQ diagnostic laboratories, with DNA extracts and additional samples (if available) exchanged for confirmation. If and when confirmed, press releases and pest alerts to the agricultural community are distributed in a coordinated process. Grower meetings often follow in due time. Sequence information for deposit to the GenBank is kept confidential until a published manuscript is accepted in Plant Disease or other widely circulated plant pathology journal. If there is no urgent need to publish the work, the DNA sequences are deposited to GenBank immediately. Confidentiality of the ultimate source of the sample is maintained by providing only the collector's name and a city or county name where the sample was collected for the GenBank catalog.

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Nutritional Disorders of Florists' Crops

3

Rosa E. Raudales

Abstract

Nutrient disorders result from chemical imbalances in plants caused by prolonged insufficient or excessive concentrations of elements in plant tissue. Nutrient deficiencies develop when an essential element is below the critical concentration required for growth and development. Nutrient phytotoxicity occurs when an element, essential or nonessential, is present at concentrations higher than what the plant can metabolize. Both conditions can cause abnormal growth and development. Generalization of nutrient disorder symptoms in floriculture crops is complex because the response to nutrient concentration is specific to the plant genetics. Visual diagnosis of nutrient disorders in the field is conducted by matching nutrient function and mobility to distribution and characteristics of symptoms. Nutrient deficiencies in plants may result from insufficient nutrient levels at the root zone, interactions with other elements, or reduced absorption caused by other growth factors not directly related to nutrition. Diagnosticians can provide immediate on-site corrective recommendations based on visual diagnosis. However, recommendations should be adjusted when nutrient analyses become available. Nutrient management begins with planning a nutrient program based on water quality, growing media properties, and crops' requirements (genetics and growth stage). In general, corrective procedures for deficiencies include a single foliar or drench application of the element or adjustment of the pH or fertilization program. Corrective options for nutrient toxicity include discontinued application of the element, increased leaching with clear water, changed or blended water source (if water source is the problem), pH increase, or use of antagonistic elements to reduce uptake.

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R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_2

Keywords
Essential elements • Visual diagnosis • Critical concentration • Deficiency • Toxicity • Cations • Nutrient translocation • Immobile element • Mobile element

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1 Introduction

All physiological processes in plants depend on the incorporation of specific chemical elements into biochemical structures and reactions at a given concentration and time (McDonald 1994). Nutrient disorders result from chemical imbalances in plants caused by prolonged insufficient or excessive supply of chemical elements. Nutrient deficiency symptoms develop when an essential element for plant growth is below the optimum concentration required for growth and development. In general, in the deficiency stage, plants respond to external nutrient supply (Marschner 1995). Plants have limited nutrient uptake selectivity; therefore elements not required for growth can accumulate to concentrations that are harmful to plants (Marschner 1995). Phytotoxicity occurs when an element, essential or nonessential, is present at concentrations higher than what the plant can metabolize resulting in abnormal growth and development.

2 Systematic Approach for Visual Diagnosis of Nutrient Disorders

Generalization of nutrient disorder symptoms in floriculture crops is complex, because the response to nutrient concentration is specific to species and cultivar (Barnes 2010; Paparozzi 2003). However, visual diagnosis of nutrient disorders in the field can be effectively conducted by implementing a systematic approach (Lea-Cox 2015). Visual diagnosis is the interpretation of characteristic symptoms in leaves, flowers, fruits, or roots caused by mineral disorders (Bould 1984).

Visual diagnosis of nutrient disorders requires a systematic approach based on the following criteria:

1. Part of the plant with symptoms (i.e., new leaves, older leaves, flower, fruit, etc.)
2. Prevailing symptoms (i.e., general chlorosis, interveinal chlorosis, necrosis, distortion, etc.)

Visual diagnosis should be considered as only one of the several indications of nutrient deficiency. Some limitations to visual diagnosis of nutrient disorders are:

1. The same element causes different symptoms by crop species or cultivar.
2. Two nutrient elements cause similar symptoms (i.e., nitrogen and sulfur deficiencies cause general chlorosis).
3. Some nutrient disorders have indistinguishable visual symptoms in the presence of multiple problems (i.e., pH below 6.0 increases the solubility of micronutrient resulting in phytotoxicity caused by multiple micronutrients simultaneously).
4. Symptoms of the disorder are visible only when the deficiency is very advanced (i.e., K deficiency in *Phalaenopsis* visible at the flowering stage).
5. Other abiotic disorders may present symptoms similar to nutrient disorders (i.e., application of plant growth regulators (Fig. 1)).

The cause of nutrient disorders may or may not be revealed by nutrient levels in the root zone. Causes of the problems may be associated with other plant health issues, malfunction of equipment, changes in practices, or sourcing of materials, among others. The diagnostician should complement on-site visual diagnosis with in-house measurement of the pH and electrical conductivity (EC) of the growing media and water source and verify adequacy of fertilization practices, equipment functionality, irrigation practices, and visual diagnosis of plant diseases (for more information on the association of nutrients and diseases, see ► Chap. 10, "Mineral Nutrition and Florists' Crops Diseases"). Finally, tissue samples will confirm the diagnosis. As we will discuss in the section below, nutrient deficiency of some elements occurs because another element reduces the availability of the nutrient or directly competes for uptake. Hence nutrient analysis of growing media will help identify the specific cause of the problem and should be used to develop a management plan.



Fig. 1 Normal plant response caused by application of a plant growth regulator (dikegulac sodium) in chrysanthemum (*Chrysanthemum morifolium*) (C. Bográn © 2017. All Rights Reserved.)

3 Essential Elements for Plant Growth

Seventeen elements have been classified as essential elements for plant growth (Table 1). The characterization of essential elements has been postulated in terms of criteria for essentiality. Arnon and Stout (1939) established that an essential element should meet the following three criteria: (1) the absence of the element prevents the plant from completing its life cycle; (2) the function of the element is not replaceable by another element; and (3) the element must be involved directly in plant metabolism. However, some researchers argue that plants have the ability to complete their life cycle despite severe deficiencies of "essential nutrients" (Epstein 1999; Hoagland 1944). Therefore, Epstein and Bloom (2005) proposed that an essential element is one that is an intrinsic component of a structure or metabolism of a plant and that the absence causes abnormalities in plant growth, development, or reproduction. Essential elements for plant growth include seventeen elements (Table 1) (Epstein and Bloom 2005; Marschner 1995).

Essential elements are classified by the concentration in plant tissue as macro- and micronutrients (Table 1). Macronutrients include nitrogen (N), potassium (K), calcium (Ca), magnesium (Mg), phosphorus (K), and sulfur (S). Silicon (Si) is not an essential element for plants; however it is present in plants at concentrations comparable to macronutrients (>0.5%) and has been associated with improved plant

Table 1 Concentration of essential plant elements in foliar tissue[a]

Element	Chemical symbol	Concentration in dry matter (ppm or percentage)
Macronutrients		
Carbon	C	45%
Oxygen	O	45%
Hydrogen	H	6%
Nitrogen	N	0.5–6%
Potassium	K	0.8–8%
Calcium	Ca	0.1–6%
Sulfur	S	0.1–1.5%
Phosphorus	P	0.15–0.5%
Magnesium	Mg	0.05–1%
Micronutrients		
Nickel	Ni	0.05 ppm
Molybdenum	Mo	0.1 ppm
Copper	Cu	6 ppm
Zinc	Zn	20 ppm
Boron	B	20 ppm
Manganese	Mn	50 ppm
Iron	Fe	100 ppm
Chlorine	Cl	100 ppm

[a]Based on Epstein and Bloom 2005

health (Epstein 1994). Essential plant micronutrients include molybdenum (Mo), copper (Cu), zinc (Zn), boron (B), manganese (Mn), iron (Fe), nickel (Ni), and chlorine (Cl). Sodium (Na) is an essential micronutrient only for C4 and CAM plants (Brownell and Crossland 1972). Carbon (C), oxygen (O), and hydrogen (H) are also essential elements for plant growth. However, discussion of these elements is beyond the scope of this section. Further discussion will focus on the remainder of the essential elements for plant health, traditionally known as mineral elements.

3.1 Macro- and Micronutrients

In general, plants have very similar nutrient requirements. However, the small variances in nutrient needs are typically what result in nutrient disorders. Plants have specific needs on the quantity and type of nutrients required at a given time. Critical concentration is the nutrient concentration in plant tissue just below (typically 10%) the optimum level for plant growth, quality, and profit (Bates 1971). Deficiency symptoms develop when the concentration of an essential element is below the critical concentration. The optimum nutrient concentration is specific to plant genus, species, varieties, and cultivars. The typical concentration of nutrient elements is provided in Table 1. Nonetheless, it is recommended that specific

guidelines for each crop are used as a reference when matching tissue analysis results to optimum levels.

Micronutrient toxicity and deficiency are the most common nutrient disorders observed in floriculture. Micronutrients are required at such low concentrations that there is a fine line between toxic, optimum, and deficient levels. Fe, Mn, Cu, B, and Zn are more available when the pH is below 6.0 and less available at pH higher than 6.0 (Marschner 1995). Plants' sensitivity to micronutrient level is very specific and narrow. For example, petunias are inefficient at taking up micronutrients, especially iron, from the root zone and are prone to iron deficiencies when then pH is above 6.0 (Argo and Fisher 2002). Other crops, like marigolds, are very efficient at taking up iron and manganese from the root zone and are prone to Fe and Mn toxicity. When conducting visual diagnoses, it is important to know whether the plant genus or species is sensitive to minor changes of pH or nutrients in the root zone. A general initial practice when diagnosing micronutrient disorders is to measure the pH of growing media.

Nutrient deficiency is the prevalent nutrient disorder for macronutrients. Macronutrient deficiencies occur when the essential elements are below the critical concentration in the plant tissue caused by low nutrient levels in the root zone or incapacity of the plant to uptake nutrients. Toxicity caused by macronutrients is typically associated with high concentration cations (i.e., calcium, potassium). Extremely high concentrations of macronutrients result in imbalance nutrition and therefore suboptimal growth. In some instances high concentration of an element reduces uptake or availability of another element resulting in an indirect nutrient deficiency (specific examples are provided under each element in the section below).

3.2 Nutrient Function and Movement

Classification of the essential elements by function and movement provides a good understanding of plant physiology (Marschner 1995; Taiz and Zeiger 2006; Welch and Shuman 1995) and is also a strong tool to diagnose nutrient disorders (Table 2). By understanding the functions and translocation of nutrients in plants, we can discriminate between elements causing the problems by making inferences based on symptoms and the organs where the symptoms are observed. In practice, this is especially important in circumstances when there is no background information on a specific species or cultivar. For example, calcium is involved with cell structure and membrane integrity; therefore deficiencies lead to distortion of organs. Iron is involved in chlorophyll synthesis; therefore deficiencies lead to chlorosis. The functions of each element are listed in the section below and in Table 2.

Nutrient translocation is the movement of nutrients in plants from source to sink via the phloem; in other words it is the movement of nutrients from one part of the plant to another (Marschner 1995; Taiz and Zeiger 2006). Translocation of nutrient elements depends on whether the element moves exclusively via xylem or also via

Table 2 Function and translocation of essential plant elements

Element	Important functions	Translocation[a]	Common deficiency symptoms
Boron	Constituent of cell wall, involved in seed and flower development, nutrient absorption and cell division	Immobile	Distortion of tip and margins of new leaves, hard and dull growth, distorted flowers
Calcium	Constituent of cell wall, cell membrane structure and permeability, signaling and regulating enzyme activity	Immobile	Distorted and necrotic leaf tip, leaf scorching, strap-like growth, and blossom end rot in fruits and flowers
Chlorine	Activates enzymes, osmosis, and ionic balance	Mobile	Wilting of the leaf tips, followed by chlorosis and necrosis. Plants wilt during the day and recover at night or on cloudy days.
Copper	Enzyme component and activator	Immobile	Reduction of overall plant growth and distortion and necrosis of new leaves.
Iron	Involved in chlorophyll synthesis, nitrate reduction, and enzymatic reactions	Immobile	Interveinal chlorosis in younger leaves, advanced symptoms result in completely chlorotic leaves and necrotic margins.
Magnesium	Constituent of chlorophyll and enzyme activation	Moderately mobile	Interveinal chlorosis in older leaves
Manganese	Enzyme activator	Immobile	Necrotic leaf spots and mottling and leaf scorch
Molybdenum	Nitrogen fixation and nitrate reduction	Mobile	Interveinal chlorosis and necrosis in older leaves
Nickel	Component of urease and required in N-fixing plants	Mobile	Not known to be a problem in floriculture crops.
Nitrogen	Constituent of organic compounds, proteins, amino acids, enzymes, coenzymes, and nucleic acids	Mobile	General chlorosis and reduction of overall plant size and development
Phosphorus	Energy acquisition, storage, and utilization and component of genetic material	Mobile	Slow growth, stunted plants, and dark-green leaves turn reddish
Potassium	Enzyme activity, regulate cell turgor, sugar translocation, and stomatal opening	Mobile	Slow growth, marginal chlorosis, and leaf scorching
Sulfur	Components of amino acids, proteins, coenzymes, and vitamins and contribute to aroma of plants	Mostly immobile	General chlorosis and growth reduction

(*continued*)

Table 2 (continued)

Element	Important functions	Translocation[a]	Common deficiency symptoms
Zinc	Enzyme activator and involved in the formation of chlorophyll	Mobile	Short internodes, small and distorted leaves, rosette growth habit. Chlorosis of older leaves and flower abscission

Based on Epstein and Bloom (2005) Evans and Sorger (1966), Gibson et al. (2007), Marschner (1995), Mengel and Kirkby (2001), and Taiz and Zeiger (2006)
[a]Mobile elements translocate from older tissue to younger tissue; therefore, early deficiency symptoms appear on older leaves. Immobile elements do not translocate after absorption; therefore, deficiency symptoms first appear on younger leaves.

phloem. All inorganic elements move from the rhizosphere to the upper parts of the plant through the xylem via mass flow (unidirectional water movement driven by transpiration) (Taiz and Zeiger 2006). Not all elements move multidirectionally or translocate. Mobile elements are nutrients that translocate from older parts of the plant to younger tissue. If an essential element is mobile, deficiency symptoms are noticeable first on the older leaves. Immobile elements are elements that do not translocate in plants or do it slowly. If an essential element is immobile, deficiency symptoms tend to appear first on younger leaves. For example, Mg is a structural component of chlorophyll and Fe is involved in chlorophyll synthesis; consequently both Mg and Fe cause interveinal chlorosis in leaves. This pattern is developed because the chlorophyll in the vascular systems withstands deficiencies for prolonged periods (Taiz and Zeiger 2006). However, Mg is mobile and Fe is immobile; therefore early symptoms of interveinal chlorosis in older leaves are most likely caused by Mg deficiency and in younger leaves are most likely caused by Fe deficiency. The translocation ability of each essential element is listed in Table 2 and is further discussed for each element in the section below.

4 Characteristics of Nutrient Disorders by Element

In this section, we provide characterization of nutrient disorder symptoms generally observed across species. However, specific symptoms may vary by species and cultivar.

4.1 Nitrogen (N)

Function. Nitrogen is a constituent of organic compounds and all proteins, amino acids, and nucleic acids.

Translocation. Nitrogen is a very mobile element in the plant. Nitrogen moves from older tissue to younger tissue. Plants uptake inorganic nitrogen in the form of nitrate (NO_3^-) and ammonium (NH_4^+) (Marschner 1995). Ammonium is readily

assimilated by plants to form organic compounds. Consequently, excessive ammonium can quickly accumulate in plant tissue and result in phytotoxicity, especially in cold months when growth is slow. In contrast, nitrate moves through the xylem and can be stored in the vacuoles of roots, shoots, and storage organs. Nitrate has to be reduced to ammonia to be incorporated as part of organic, which makes nitrate absorption a slower process compared with ammonium.

Visible Symptoms

Deficiency. Nitrogen deficiency symptoms begin with loss of color (light green or chlorosis) in the older leaves and size reduction of younger leaves (Jones 1965). Overall the plant is chlorotic and plant size and stem girth are smaller than normal (Barnes 2010; Barnes et al. 2012; Bennett 1993; Gibson et al. 2007; Winsor and Adams 1987). With prolonged deficiency, younger leaves also become chlorotic, while senescence and dehiscence of older leaves is accelerated (Hewitt 1984b). In some species (i.e., chrysanthemum, begonia, celosia, marigold, and pansy), the older leaves turn red, orange, or purple as a result of reduced chlorophyll content (Gibson et al. 2007; Hewitt 1984b; Laurie and Wagner 1940; Winsor and Adams 1987). Flowering is in general delayed and reduced in number and size (Barnes 2010; Laurie and Wagner 1940) (Fig. 2). The stem size is strongly reduced, affecting both cut and container-grown flowers (Laurie and Wagner 1940). An overall biomass reduction is observed across species (Barnes 2010).

Toxicity. Toxicity symptoms of N alone are not well defined. However, excessive vegetative growth and reduction of yields are observed when N levels are high. Ammonium, which is readily assimilated by plants, can result in toxicity. Ammonium toxicity symptoms include chlorosis or interveinal chlorosis of older leaves, and the margins of the leaves become scorched and rolled upward or inward (Gibson et al. 2007; Winsor and Adams 1987) (Fig. 3). Foliar and root biomass are significantly reduced with ammonium toxicity (Cox and Seeley 1984; Gaffney et al. 1982; Gibson et al. 2007; Winsor and Adams 1987). Roots are typically short, unbranched, and discolored.

Management

Nitrogen deficiency is easy to correct by applying nitrogen in the preferred form. Develop a nutrition program that matches the fertilizer rate to the optimum level of a specific crop and growth stage. Avoid using fertilizers with high ammonium concentration (>40%) in winter months. Ammonium toxicity is corrected by maintaining the growing media pH at > 5.5 (to avoid conversion of nitrate to ammonia) and by avoiding fertilizer with high concentration of ammonium and urea.

4.2 Potassium (K)

Function. Potassium builds up turgor and maintains osmotic potential in plants. Potassium also serves as a carrier across cell membranes and is involved in cell elongation, protein synthesis, enzyme activation, photosynthesis, and water uptake.



Fig. 2 Nitrogen deficiency symptoms in bacopa (*Bacopa monnieri*) (*top*) and New Guinea impatiens (*Impatiens hawkeri)* (*bottom*). Healthy plants (on the *left* of each photo) and nitrogen-deficient plant (*right* of each photo). Nitrogen deficiency symptoms: loss of green color in foliage (more accentuated in older leaves), reduction of leaf size, flowers, and stem girth. With prolonged deficiency, younger leaves also become chlorotic. Overall reduction of plant size and delayed flowering (P. Fisher © 2017. All Rights Reserved.)

Translocation. Potassium is not structurally bound in any plant component. It is mobile, and therefore it can be redistributed within plants. Potassium moves directly from the rhizosphere to the growing points and also translocates from older tissue to new tissue.

Visual Symptoms

Deficiency. Potassium deficiency symptoms begin with mild marginal or interveinal chlorosis, followed by leaf scorch (marginal necrosis or necrotic spots) of recently matured leaves (Barnes et al. 2012). In some cases, the leaf spots can be pronounced red or violet caused by accumulation of anthocyanin (Laurie and Wagner 1940). Consistently across species, as symptoms progress, older leaves become completely necrotic and symptoms progress bottom up. Dieback (terminal and lateral buds die) is observed in woody plants. Young plants are shorter and darker (dark green or blue green) than normal. In *Phalaenopsis* the tip or margin of the oldest leaves turns



Fig. 3 Ammonium phytotoxicity symptoms on vinca (*Vinca minor*). Ammonium toxicity symptoms include chlorosis or interveinal chlorosis of older leaves (*top*), and the margins of the leaves become scorched and rolled upward or inward (*bottom*). Foliar and root biomass are significantly reduced. Roots are typically short, unbranched, and discolored (R. Raudales © 2017. All Rights Reserved.)

yellow and then chlorosis progresses from the bottom up in the flowering stem (Wang 2007). As symptoms develop, the older leaves turn to bronze color and then the leaves die. K deficiency does not affect the number of leaves in *Phalaenopsis* (Fig. 4).

Toxicity. Potassium toxicity is rare in practice. Excess potassium in nutrient solutions or soils results in competition with other cations (e.g., Mg^{+2}). Cations are ions that have less electrons than protons, therefore give a positive charge (e.g., Ca^{+2}, Mg^{+2}, Mn^{+2}). However, the deficiency of competing cations is what results in abnormal plant growth and not a direct effect of potassium toxicity (Ulrich and Ohki 1965). In many cases, high levels of potassium can cause salt damage symptoms.

Management

Potassium is an element which is associated with cell processes and is a mobile element. Consequently, potassium deficiency symptoms are visible when the deficiency has progressed, and in many cases corrective actions to manage potassium deficiency are not effective (Ulrich and Ohki 1965; Wang 2007). When developing a



Fig. 4 Potassium deficiency symptoms on phalaenopsis (*Phalaenopsis* sp.). The tip or margin of the oldest leaves turns yellow and then chlorosis progresses from the bottom up in the flowering stem. As symptoms develop, the older leaves turn to a bronze color (*top*) and then the leaves die (*bottom*). The number of leaves is not affected by K deficiency (Y.T. Wang © 2017. All Rights Reserved.)

management strategy for K deficiency, it is important to identify whether the deficiency is caused by insufficient levels of K in the growing media or by imbalances of K with other nutrients. For example, a high ratio of N:K (2:1) or high concentration of sodium (>40 mg/L) inhibits K uptake. In general, potassium levels should be at similar levels to nitrogen. Supplemental fertilization of potassium at the right ratio may correct potassium deficiency. Deficiency caused by low levels of K in the growing media can be amended by supplementing with fertilizers.

4.3 Phosphorus (P)

Function. Phosphorus is an integral component of energy storage molecules, nucleic acids, and membranes.

Translocation. Phosphorus is a mobile element in the plant, moving from older tissue to younger tissue in response to low P levels.

Visual Symptoms

Deficiency. Early symptoms of P deficiency include slow emergence and growth, and the petioles are purple and foliage turns dark green. As the deficiency progresses, plants exhibit severe spindling and stunting and the foliage changes from dull green or bronze to purple or dark red (Fig. 5). Older leaves become deep green or red and

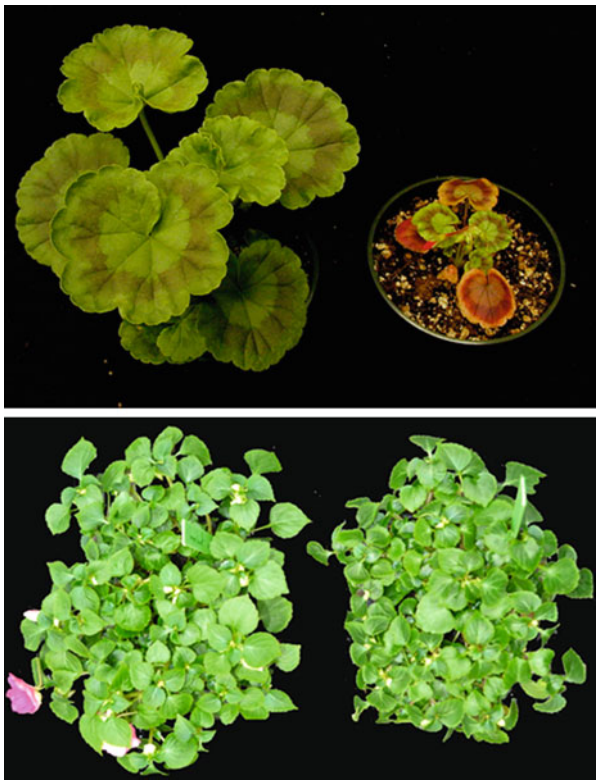


Fig. 5 Phosphorus deficiency symptoms in seed geraniums (*Pelargonium hortorum*, *top* photo) and impatiens (*Impatiens walleriana*, *bottom* photo). Healthy (*left* in both photos) and P deficient (*right* in both photos). Early symptoms of P deficiency include slow emergence and growth and petioles are purple. As the deficiency progresses, plants exhibit severe spindling and stunting and the foliage changes from dull green or bronze to purple. Older leaves become deep red and dull. In some cases, P deficiency symptoms are not distinctive and result in reduced growth and flowering (P. Fisher and R. Dickson © 2017. All Rights Reserved.)

become dull (Barnes et al. 2012; Epstein and Bloom 2005; Gibson et al. 2007; Hewitt 1984a). The leaves curl downward (Fig. 5). Flowering is significantly reduced.

Toxicity. Excessive concentrations of P do not cause direct damage to plants; however, high P concentrations can result in Cu or Zn deficiencies (Bingham 1965).

Management

P deficiencies result from insufficient P fertilization, slow growth (e.g., during cold weather), low B concentration (low B inhibits P uptake), high Fe, Zn or nitrate, or excessive leaching (Gibson et al. 2007). Corrective procedures include adjusting fertilization to match levels to the plant's needs.

Excess P in the root zone can be managed by increasing leaching and then using fertilizers low in P (e.g., 15-0-15).

4.4 Calcium (Ca)

Function. Calcium is a component of cell walls, maintains cell wall integrity, and regulates membrane permeability. Ca also enhances pollen development and germination and is involved in enzyme activation, cell mitosis, division, and elongation.

Translocation. Calcium is not mobile and is not redistributed after the initial absorption. Plants uptake Ca during transpiration; therefore Ca deficiencies generally appear when growth is slow (e.g., during cold weather, drought, high humidity, etc.).

Visual Symptoms

Deficiency. Calcium deficiency symptoms appear first on apical meristems of shoots and roots. The symptoms are known as "death of growing points" (Epstein and Bloom 2005; Hewitt 1984a). Young leaves become distorted, small, and chlorotic. Leaf margins and distorted areas might become necrotic as symptoms progress. The plant overall presents dieback symptoms. Dark spots are developed in some species. Flowers and fruit present "blossom end rot." Roots are short and thick and develop few root hairs (Barnes et al. 2011) and sometimes necrotic. Flowers and petals drop, and vase life of flowers is shortened. Flower size is smaller than usual.

Toxicity. Direct negative effects of excess of calcium are rare. However, excessive calcium concentration can reduce boron availability and result in B deficiency, especially at alkaline pHs when boron is less available. Calcium uptake can compete with other cations (e.g., Mg^{+}, K^{+}).

Management

Calcium deficiency is caused by insufficient Ca in the root zone (e.g., low Ca fertilizer or excessive leaching), slow growth (caused by low temperatures, high humidity, drought, etc.), or high concentrations of competing cations (e.g., Na^{+}, Mg^{+}) (Gibson et al. 2007) or total salts (Gislerod 1999). Ca reacts with phosphates or sulfates and forms insoluble compounds that plants cannot uptake resulting in Ca deficiency. This problem occurs when the elements are at high concentrations such as stock fertilizer solutions or high concentrations in the soil. Consequently, nutrient analysis of the growing media will provide information about the availability of Ca in the substrate. Management options include reducing leaching, balancing the cations, and supplementing Ca in the fertilization program. In addition, having a separate stock tank for calcium prevents formation of precipitates.

Excess calcium potentially comes in the water source, excessive lime application, or over fertilization. Corrective procedures include blending or changing the water source and revising the fertilization program to lower calcium levels or increase the cations that are deficient.

4.5 Magnesium (Mg)

Function. Magnesium is a component of the chlorophyll molecule and activates enzymes involved in photosynthesis, respiration, and DNA synthesis.

Translocation. Magnesium is a mobile element that is translocated from mature to young leaves.

Visual Symptoms

Deficiency. Early symptoms appear first in older leaves as interveinal chlorosis. Magnesium and iron are both important for chlorophyll formation; however Fe is immobile and Mg is mobile. Therefore, the symptoms can be distinguished by observing the location of the chlorosis during its early stages. Mg deficiency symptoms are not always as consistent across species. Overall, plants with Mg deficiency are smaller than healthy plants, and leaves turn chlorotic or present interveinal chlorosis. Older leaves turn reddish brown or reddish purple and veins remain green (Gibson et al. 2007; Hewitt 1984a; Laurie and Wagner 1940).

Toxicity. Magnesium phytotoxicity has not been reported. However, Mg can inhibit uptake of other cations (e.g., K^{+}).

Management

Mg deficiency can result from low Mg from the fertilizer program or high content of other cations (e.g., Ca^{+2}, Na^{+2}), dysfunctional roots, and slow growth. Magnesium sulfate (Epsom salts) or fertilizer blends with Mg are commonly used to correct Mg deficiency.

High levels of Mg in tissue can come from high Mg in the water source, excessive fertilizer, or low levels of other cations. Corrective procedures include adjusting the fertilizer program (lower Mg or increase other cations) or changing the water source.

4.6 Sulfur (S)

Function. Sulfur is a constituent of amino acids, coenzymes, and vitamins. Sulfur is also a structural component of aromatic compounds.

Translocation. Not easily mobilized, translocation varies by species.

Visual Symptoms

Deficiency. Sulfur deficiency symptoms are very similar to nitrogen (both are constituents of amino acids and proteins). Deficiency symptoms include general chlorosis, stunting, and reddish foliage (anthocyanin accumulation) (Barnes 2010; Hewitt 1984a). In some cases, the foliage is chlorotic and petioles are red (Dale et al. 1990). Root biomass is reduced, but the color is not altered. Sulfur is generally immobile; consequently, early symptoms appear in younger leaves. However, sulfur translocation varies by species and deficiency might appear in all leaves simultaneously (Taiz and Zeiger 2006).

Toxicity. Sulfur toxicity in floriculture crops is rarely observed in practice. High concentrations of sulfur inhibit nitrogen uptake and might result in N deficiency. As a consequence, S toxicity and deficiency could be easily confused.

Management

Sulfur deficiencies result from inadequate supply of sulfur in the nutrition program.

High sulfur comes from the fertilizers, acidifying agents (i.e., sulfuric acid, iron sulfate, elemental sulfur), or irrigation water; to correct the problem, discontinue application of sulfur. If water is the source of the problem, then blend with or change to water from another source.

4.7 Iron (Fe)

Function. Iron plays an important role in chlorophyll synthesis, nitrate reduction, and other enzymatic reactions.

Translocation. Iron is an immobile element.

Visual Symptoms

Deficiency. The visual symptoms of Fe deficiency are consistent across plants (Barnes et al. 2012; Gibson et al. 2007; Hewitt 1984a; Laurie and Wagner 1940). Deficiency symptoms start with interveinal chlorosis of young leaves, where the veins remain green and the tissue between the veins is chlorotic (Fig. 6). With prolonged deficiency or deficiency at the seedling stage, the whole leaf will turn chlorotic and dehiscence will occur (Fig. 6). Fe is immobile; hence deficiency symptoms appear in younger leaves. Mg deficiency symptoms are similar to Fe, but Mg symptoms first appear in older leaves.

Toxicity. Iron toxicity begins with a mild chlorosis followed by bronzing of leaves. Symptoms advance as necrotic spots or necrosis of whole leaves and death (Fig. 7).

Management

Fe is more soluble at lower pH (acidic) and less soluble at higher pH (basic). Therefore, plants which are "inefficient" at absorbing iron (e.g., petunias, pansies, snapdragons, etc.) tend to show iron deficiency symptoms when the pH is $\geq$ 6.2. In contrast, other species (e.g., seed geraniums, marigolds, lisianthus, etc.) are efficient at taking up Fe and Mn and tend to present Fe and Mn phytotoxicity when the pH is $<$6.0 (Argo and Fisher 2002). Corrective procedures for deficiency and phytotoxicity consist on matching the pH to the crop's requirement. If the pH is at the optimum level for the crop, then Fe should be applied as a drench or foliar spray to correct deficiencies.



Fig. 6 Iron deficiency symptoms in petunia (*Petunia* × hybrid): healthy leaf (*left*), interveinal chlorosis (*middle*), advanced chlorosis, and curling inward (*right*). Deficiency symptoms start with interveinal chlorosis of young leaves, where the veins remain green and the tissue between the veins is chlorotic. With prolonged deficiency or deficiency at the seedling stage, the whole leaf will turn chlorotic (P. Fisher © 2017. All Rights Reserved.)



Fig. 7 Iron toxicity in marigold (*Tagetes patula*). Iron toxicity appears as a mild chlorosis on the margins of fully expanding leaves, followed by bronzing or brown spotting of leaves, necrosis, and death (P. Fisher © 2017. All Rights Reserved.)

High iron comes from inadequate fertilization programs, foliar sprays of iron chelates, or the water source. Corrective actions include adjusting fertilizer programs with low Fe options. Irrigation water sources can be blended or alternated to avoid accumulation of Fe in the root zone.

4.8 Manganese (Mn)

Function. Manganese activates multiple enzymes in plant cells including ones involved in photosynthetic reactions.

Translocation. Immobile.

Visual Symptoms

Deficiency. Manganese deficiencies are characterized by interveinal chlorosis or chlorosis (similar to early stages of Fe and Mg deficiency) in either younger or older leaves depending on the plant species (Taiz and Zeiger 2006). Small necrotic spots also appear as symptoms progress. Acute deficiency results in total chlorosis of leaves. The size of plants is not affected until the symptoms are advanced.

Toxicity. Early symptoms of Mn toxicity include burning of leaf tips and margins, and flowering is inhibited (Frantz et al. 2006). Woody plants present internal bark necrosis and external bark damage. High concentrations of Mn are usually accompanied with Fe deficiency, caused by cation uptake competition. However, there are some crops that are very efficient in taking up Mn and Fe. For these "iron-efficient crops," Mn and Fe toxicity occurs simultaneously when the pH of the root zone is below 6.0 (Fig. 8) (Argo and Fisher 2002).



Fig. 8 Manganese and iron toxicity on geraniums (*Pelargonium* × *hortorum*) occur simultaneously when the pH of the growing media is below 6.2 (P. Fisher © 2017. All Rights Reserved.)

Management

Mn deficiency results from insufficient Mn in the fertilization program, high concentration of other cations (e.g., Ca, Na) in the water or nutrient solution, or high concentrations of Cu, Fe, and Zn which inhibit Mn uptake (Gibson et al. 2007). Corrective actions concentrate on balancing the nutrient program to match the crops' needs. A single foliar spray of manganese sulfate at a concentration of 40 mg/L is recommended for short-term remediation.

High Mn comes from high Mn in the fertilization program or water source. Adjust the fertilization program to lower Mn application and/or to increase pH of the growing media. Blend or change the water source if possible.

4.9 Zinc (Zn)

Function. Zinc is an enzyme activator and is involved in chlorophyll biosynthesis.

Translocation. Mobile.

Visual Symptoms

Deficiency. Zinc deficiency symptoms are very consistent across species (Barnes et al. 2012; Epstein and Bloom 2005; Hewitt 1984a; Laurie and Wagner 1940; Taiz and Zeiger 2006). Symptoms include chlorosis and short internodes, rosette growth habit, and small and distorted leaves (typically called "little leaf"). In some species, the older leaves present interveinal chlorosis. Zinc also causes flower abscission.

Toxicity: Zinc toxicity is very acute and results in mortality of tissue and eventually the whole plant.

Management

Zinc deficiencies result from low Zn in the fertilization program, or a pH above 6.4 at the root zone. Excesses of P or Cu inhibit Zn uptake (especially when Zn levels are low (Marschner 1995)). Corrective procedures include a single foliar spray or drench with zinc sulfate at 56 or 14 ppm, respectively (Bailey and Nelson 1998). Zinc can be very toxic; therefore, to reduce risk of plant damage test, apply it on a small portion of plants, avoid overapplication, and apply early during the day. Alternatively, fertilize with a mix that contains Zn and adjust the pH of the growing media if needed.

High Zn comes from fertilizers or the water source. Zinc toxicity is not easy to correct. Management options include stopping application of the element, leaching with clear water, and changing or blending water sources (if water sources are the problem). For micronutrients slightly raising the pH reduces availability, and using antagonistic elements (P and Cu) will reduce Zn uptake.

4.10 Copper (Cu)

Function. Copper is an enzyme component and activator.

Translocation. Immobile.

Visual Symptoms

Deficiency. Copper deficiency symptoms vary widely by species. Younger leaves can become dark green. Some plants present necrotic spots at the tips of leaves, interveinal chlorosis with green leaf tips, or inward curling of leaves. Some woody plants develop blisters and cracks on the bark. Some plants develop short internodes that results in rosetting. Some flowers develop a weak stem which cannot support the flowers (Hewitt 1984a). In a few instances, discoloration has been observed (Barnes 2010).

Toxicity. Copper toxicity results in evident growth reduction (height, branching, etc.) and chlorotic leaves. High concentration of Cu in the rhizosphere decreases Fe uptake which results in interveinal chlorosis.

Management

Copper deficiency is caused by inadequate fertilization or pH above 6.4 in the root zone. Corrective procedures include fertilizing with a mix containing Cu, a single drench of copper sulfate at 9 ppm (Bailey and Nelson 1998), or adjusting the growing media pH to 5.8.

High copper can come from fertilization or copper-based fungicides or increased availability at a low pH (<6.0). Corrective actions include discontinuing copper-based products. Slightly raising the pH reduces availability.

4.11 Boron (B)

Function. Boron is a component of cell walls and is involved in flower and seed development and cell division.

Translocation. Boron is immobile in the plant because it moves exclusively via xylem.

Visual Symptoms

Deficiency. Boron deficiency symptoms are difficult to diagnose visually, because the specific symptoms vary by crop and early symptoms are not always visible (Hewitt 1984a). In general, B deficiency results in degeneration of meristematic tissue causing distortion and necrosis of apical tips and buds. Boron is involved in cell division; therefore, the deficiency results in hard and brittle leaves. Additional symptoms include discoloration of the margin of older leaves, leaf dieback, corky leaves, cracked stems, short internodes, cracked midribs, and rosetting. Witch's broom is typically observed in boron-deficient plants because of bud mortality. Flowers are delayed, distorted, and drop early (Hewitt 1984a) and the total number of petals is reduced (Winsor and Adams 1987).

Toxicity. Boron toxicity is characterized by tip burning and then symptoms progress as reddish to brown margins of older leaves which spread to the rest of the leaf (Fig. 9). Necrotic spots may also develop along the margins of the leaves and finally the leaves may become completely necrotic and drop early. Flower number and quality are not affected (Hewitt 1984a; Winsor and Adams 1987).

Management

Boron deficiency can be corrected with drench applications of boron or including a fertilizer mix that contains boron. Boron moves only via xylem; therefore foliar sprays are only effective on the tissue sprayed because there is no reallocation of B to other tissues.

Boron toxicity can come from high concentrations of B in the irrigation water source or low pH in the growing media. If irrigation water is the source of the problem, then the recommendation is to alternate or blend the current water source with an alternative source. Boron in the soil can be leached with sufficient water and by increasing soil pH to reduce B availability (liming). Calcium, in the form of calcium nitrate, reduces B availability.

4.12 Molybdenum (Mo)

Function. Mo is a component of several enzymes including enzymes involved in the reduction of nitrate to ammonium.

Translocation. Mobile.

Visual Symptoms

Deficiency. Early deficiency symptoms appear as general interveinal chlorosis and necrosis in older leaves. Flowers are delayed and drop prematurely. Mo is



Fig. 9 Boron toxicity symptoms in heliotrope (*Heliotropium arborescens*, *top* photo) and rose (*Rosa* sp., *bottom* photo). Healthy heliotrope on the *left* compared with B toxicity on the *right*. B toxicity symptoms: early symptoms begin as tip burning, then symptoms progress as reddish to brown margins of older leaves, and finally the whole leaf turns bronze. Necrotic spots may also develop along the margins of the leaves and finally the leaves may become completely necrotic and drop early (P. Fisher and R.I. Cabrera © 2017. All Rights Reserved.)

associated with nitrate reduction; therefore, Mo deficiency may result in N deficiency if nitrate is a major N source. However, Mo deficiency is rare in floriculture crops and has only been reported in poinsettias as a distinctive and clearly marked yellow margin (Ecke et al. 2004; Gibson et al. 2007; Hewitt 1984a; Winsor and Adams 1987). Mealy cup sage and lilies exposed for weeks to a nutrient solution with very low Mo did not show any symptoms (Barnes et al. 2011, 2012).

Toxicity. Molybdenum toxicity is rarely observed in floriculture because Mo concentrations in water and soils tend to be low.

Management

Mo deficiency results from inadequate fertilization or a low substrate pH. Unlike most micronutrients, molybdenum is available at a wide pH range (5.4–10).

Availability of Mo decreases when the pH is <5.4. Corrective procedures include adjusting the pH of the growing media to ≥5.4, applying a single drench of sodium molybdate or a low continuous dose of fertilizer mixes with M or a single foliar application of sodium molybdate (57 mg/L Mo) or ammonium molybdate (81 mg/L Mo). As mentioned earlier, Mo deficiency is very rare in floriculture and has only been reported as problem in poinsettias.

Toxic concentrations of Mo come from high rate applications of fertilizers containing Mo. Management procedures include discontinuing fertilizers with Mo.

4.13 Chlorine (Cl)

Function. Chlorine is in the form of chloride in plants and is involved in water splitting in photosynthesis and cell division in leaves and roots.

Translocation. Mobile.

Visual Symptoms

Deficiency. Chloride deficiency symptoms begin with wilting of the leaf tips, followed by chlorosis and necrosis. Plants wilt during the day and recover at night or on cloudy days. Severe deficiency results in spindly and stunted plants. Roots appear stunted and thickened especially at the tip.

Toxicity. Early toxicity symptoms include burning of leaf tips and margins, leaf bronzing, and premature leaf abscission of older leaves. In general, excess chloride results in reduced leaf size and a slower growth rate.

Management

Chloride deficiency is rare because chloride ions are very soluble. Deficiency can be managed by supplementing with fertilizers containing Cl.

Toxicity of Cl comes from excess application of fertilizers or from the water source. Management of Cl toxicity begins with abundant leaching with clear water. If the water source is the cause of the problem, the water needs to be blended or changed. Another option is changing to a fertilizer source with no Cl.

4.14 Nickel (Ni)

Function. Nickel is a component of urease (an enzyme that catalyzes urea hydrolysis) required in N-fixing plants. Nickel is an element of concern mostly for field crops and is not known to be a problem in floriculture.

4.15 Sodium (Na)

Function. Sodium is an essential element only for C_4 and CAM photosynthetic-type plants (Brownell and Crossland 1972). Sodium is involved in carbon fixation and

regulates turgor pressure. Florist crops with CAM metabolism include orchids and bromeliads and C4 plants include monocot ornamental grasses.

Translocation: Sodium is a mobile element.

Visual Symptoms

Deficiency: Despite being an essential element in CAM plants, like bromeliads and orchids, Na deficiency symptoms in these crops have not been reported.

Toxicity. Sodium phytotoxicity symptoms begin with leaf tip and marginal chlorosis on the older leaves (Fig. 10) and root tip necrosis. As symptoms progress, the whole leaf becomes discolored followed by general necrosis. Overall plant biomass is reduced (Lee and Van Iersel 2008) and leaves become brittle. Stem quality of cut flowers is also affected (De Kreij and van Os 1989; De Kreij and van den Berg 1990). Potassium ion transporters do not discriminate between Na and K (Pardo and Quintero 2002). Therefore, K uptake is highly inhibited with high Na concentration. Na also competes with other cations (e.g., K^{+}, Ca^{+2}, Mg^{+2}) resulting in nutrient deficiencies.

Management

Sodium toxicity usually comes from the water source. Sodium levels above 50 mg/L in the water or soil can be a problem for many floriculture crops. Management of Na toxicity, similar to Cl, begins with abundant leaching with clear water. If the water source is the cause of the problem, the water needs to be blended or changed.



Fig. 10 Phytotoxicity in roses (*Rosa* sp.) caused by high concentration of sodium chloride (NaCl) in the root zone. Sodium phytotoxicity symptoms begin with leaf tip and marginal discoloration and brittle texture of the older leaves and root tip necrosis and then the whole leaf becomes discolored. Advanced symptoms may result in complete necrosis of the tissues (R.I. Cabrera © 2017. All Rights Reserved.)

5 Other Nutrient Disorders

Electrical conductivity (EC) is a measure of the ability of a solution to conduct electricity and is used in plant nutrition as an indicator of total concentration of salts in water, soilless-growing media, or soil (Mengel and Kirkby 2001). Plants vary in EC requirements by growth stage and genetics. At early stages, young plants are extremely sensitive to low nitrogen levels or high concentrations of salts (Gibson 2006; Styer and Koranski 1997). At advanced growth stages, chrysanthemums, poinsettias, and petunias require high concentration of nutrients (EC $>2.6 mS \cdot cm^{-1}$ by the Pour-Through method), whereas crops like zinnia, cyclamen, caladium, gerbera, begonias, and others grow better with lower EC levels (EC 1.0–2.6 $mS \cdot cm^{-1}$ by the Pour-Through method) (Whipker et al. 2011). Some plants, like snapdragon (*Antirrhinum majus* L.), are very tolerant to very high EC ($14 mS \cdot cm^{-1}$) (Carter and Grieve 2008). Low or high EC in the root zone can result in nutrient deficiencies and toxicities, respectively. High EC results in severe leaf discoloration (Fig. 11), root burning, and plant wilting.

5.1 pH

Most floriculture crops grow best within a pH of 5.6 and 6.4. Micronutrient deficiency or toxicity are likely to develop when plants are grown outside of the optimum pH (Argo and Fisher 2002; Fisher et al. 2003; Marschner 1995). Micronutrients (Fe, Mn, B, Cu, Zn) are more available at lower pH (acidic) and less available at higher pH (basic). Molybdenum is less available at pH $\leq$ 5.4. Plants which are "inefficient" absorbing iron (e.g., petunias, pansies, snapdragons, etc.) tend to show iron deficiency symptoms when the pH is $\geq$ 6.2. Other species (e.g., seed geraniums, marigolds, lisianthus, etc.) are efficient at taking up micronutrients and tend to present iron and manganese phytotoxicity when the pH is <6.0



Fig. 11 Phytotoxicity caused by high concentration of salts in the root zone in roses (*Rosa* sp.). Symptoms begin with a tip or marginal bleaching, bronzing, or desiccation and then the discoloration progresses to the remainder of the leaves (R.I. Cabrera © 2017. All Rights Reserved.)

(Argo and Fisher 2002; Smith et al. 2004). In conclusion, knowing the specific nutrient requirements of plants helps categorize potential problems based on growing conditions and practices.

6 General Management Guidelines

- Nutrient management begins with planning a nutrient program based on water quality, growing media properties, and crops' requirements (genetics and growth stage).
- Nutrient deficiencies in plants result from insufficient nutrient levels or imbalanced concentration of nutrients at the root zone. For example, high concentration of an element reduces uptake of other elements (e.g., B and Ca, cations). Micronutrients are less soluble with basic pH which may quickly result in deficiency in some crops. Other factors not directly related to nutrition such as plant diseases (Datnoff et al. 2007) or environmental conditions like temperature and humidity (Nelson 1998) may interfere with nutrient absorption (see ▶ Chap. 10, "Mineral Nutrition and Florists' Crops Diseases"). The diagnostician can provide an immediate on-site recommendation based on visual diagnosis. However, the recommendation should be adjusted when nutrient analysis results become available.
- Nutrient toxicities are not easy to correct. Management options include stopping application of the element, increased leaching with clear water, and changing or blending water sources (if water source is the problem). For micronutrients slightly raising the pH reduces availability, and the use of antagonistic elements decreases uptake.
- Foliar sprays can be used to correct micronutrient deficiencies. However, they have a greater risk of causing plant injury compared to drenches or fertigation (application via irrigation systems). To reduce the risk of injury, it is recommended to apply the foliar spray first to a small portion of plants, avoid overapplication, and apply early during the day (Gibson et al. 2007; Bailey and Nelson 1998).

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Insect Management for Disease Control in Florists' Crops

4

Raymond A. Cloyd

Abstract

Insect pests are common problems in greenhouse and field production systems associated with floriculture crops. Many insect pests cause direct feeding damage to crops; however, several cause indirect damage by vectoring pathogens such as fungi and viruses. The primary insect pests of floricultural crops that vector or transmit diseases are the western flower thrips (*Frankliniella occidentalis*), fungus gnats (*Bradysia* spp.), shore flies (*Scatella* spp.), green peach aphid (*Myzus persicae*), and sweetpotato whitefly (*Bemisia tabaci*). Because the tolerance level for insect pests that vector diseases is low, especially in regards to the western flower thrips due to the direct transmission of *Impatiens Necrotic Spot* virus, intensive pest management strategies need to be implemented. The pest management strategies used are generally associated with insecticide applications although extensive use of insecticides may lead to insecticide resistance. Therefore, in addition to the use of insecticides, and in order to effectively deal with insect vector populations, consideration should be given to cultural and sanitation practices and using biological control agents (natural enemies). However, these strategies must be effective in sustaining populations at very low levels in order to reduce the potential of disease transmission to floricultural crops.

Keywords

Insects • Vectors • Diseases • Pathogens • Viruses • Fungi • Tolerance • Insecticides • Plant protection

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© Springer International Publishing AG 2018

R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_3

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1 Introduction

Greenhouse- and field-grown floricultural crops are susceptible to an array of insect pests such as aphids, leafminers, mealybugs, thrips, and whiteflies. Most of these insect pests cause direct damage when feeding on plants; however, a number of insect pests are capable of transmitting or vectoring certain plant diseases such as fungi and viruses (Agrios 2005). These include the western flower thrips (*Frankliniella occidentalis*), fungus gnats (*Bradysia* spp.), shore flies (*Scatella* spp.), green peach aphid (*Myzus persicae*), and sweetpotato whitefly (*Bemisia tabaci*). The presence of insects that vector diseases may impact the types of crops grown. For example, the ability of the western flower thrips to transmit viruses resulted in the discontinued production of susceptible plant types such as gloxinia, *Sinningia speciosa* (Daughtrey et al. 1997). The exchange of plant material within greenhouses may result in the inadvertent movement of insect vectors. Furthermore, the adult stage of western flower thrips may migrate from field or vegetable crops, or weeds located outdoors, through unscreened greenhouses and threaten floricultural crops (Daughtrey et al. 1997). In addition, reports indicate that fungus gnats may be present in commercial bagged soilless growing media and rooted plant plugs (Cloyd and Zaborski 2004), which may allow fungus gnats to inadvertently enter greenhouses or be moved between greenhouses. Insects that vector diseases present a challenge in regards to management as the tolerance levels for these insect pests are extremely low (Parrella 1999).

The focus of this chapter will be to describe each insect pest and their associated disease interactions and then present information on the appropriate strategies that should be implemented to manage these insect pests in order to minimize problems regarding disease transmission.

2 Western Flower Thrips

Western flower thrips, *Frankliniella occidentalis*, is one of the most destructive insect pests of commercially grown floricultural greenhouse crops (Cloyd 2009; Kirk 2002; Robb and Parrella 1995). Western flower thrips adults are about 2.0 mm (0.078 in.) in length, slender, with two pairs of fringed (hairy) wings. The adults and larvae vary in color from yellow to brown (Fig. 1). The life cycle consists of an egg, two larval stages, two pupal stages, and an adult and takes 2–3 weeks to complete (Mound 1996; Tommasini and Maini 1995); however, development is contingent on ambient air temperature with the life cycle completed in 7–13 days at temperatures between 26 and 29 °C (79 and 84 °F). A new generation may occur every 20–30 days although phenology also depends on ambient air temperature (Gaum et al. 1994; Lublinkhof and Foster 1977). Females live approximately 45 days and can lay up to 300 eggs during their lifespan (Gerin et al. 1999).

There are a number of factors affiliated with the biology and life cycle that consequently contribute to western flower thrips being a major insect pest of greenhouse-grown floricultural crops, including: (1) high female reproductive capacity, (2) broad host range, (3) short life cycle, (4) feeding habit, (5) small size, (6) resistance to insecticides, and (7) ability to vector viruses (Jensen 2000). The ability of western flower thrips to damage plants is not only associated with direct damage to greenhouse-grown floricultural crops due to feeding on leaves and flowers, but also because western flower thrips can vector a number of tospoviruses that affect florists' crops, including *Impatiens Necrotic Spot* virus (INSV) (Allen and Broadbent 1986; Pappu et al. 2009).

Impatiens Necrotic Spot virus has a broad host range that includes many floricultural crops and common weeds. A number of different but common weeds may harbor the virus, such as, oxalis (*Oxalis* spp.), chickweed (*Stellaria media*), bittercress (*Barbarea vulgaris*), prostrate spurge (*Euphorbia supina*), and jewelweed



Fig. 1 Western flower thrips (*Frankliniella occidentalis*) larvae and adults feeding on chrysanthemum leaf (Raymond A. Cloyd; Kansas State University © 2017. All Rights Reserved.)

(*Impatiens capensis*) (Daughtrey et al. 1997). Symptoms commonly observed on infected plants include necrotic lesions and/or concentric lines and circular patterns with symptoms being expressed 1–2 weeks after inoculation depending on temperature and plant type (Agrios 2005). A number of plants and weeds may harbor symptomless infections or express symptoms that are not typical of the virus. Therefore, it is important to routinely test highly susceptible plants using commercially available serological methods. There is no cure once a plant has been infected with the virus and subsequently all infected plants should be immediately removed from the greenhouse and discarded. *Impatiens Necrotic Spot virus* is classified in the genus *Tospovirus* with a plant host range that overlaps with the *Tomato Spotted Wilt* virus, which is another tospovirus that is primarily associated with greenhouse-grown vegetable crops. However, plants may harbor infections of both viruses simultaneously (Daughtrey et al. 1997).

The western flower thrips is the primary vector of INSV although several other thrips species are reported to transmit the virus including *Frankliniella fusca*, *Frankliniella intonsa*, and *Frankliniella schultzei* (Sakurai et al. 2004). The virus is transmitted in a circulative manner, which means that the virus must circulate throughout the insect body before transmission occurs (DeAngelis et al. 1993; Gray and Banerjee 1999). Western flower thrips acquire the virus through their mouthparts when feeding on infected plants. The virus initially infects the insect gut where replication occurs. Afterward, the virus accumulates in the gut tissue and subsequently spreads internally within the insect. For transmission to occur, the virus must reach the salivary glands in the insect mouthparts (Nagata et al. 2002; Wijkamp et al. 1993). During the feeding process, the virus is secreted, along with saliva, into the new host plant and transmission occurs. In the virus acquisition phase, the virus is ingested by the larvae (1st and 2nd instar) and can be transmitted by late 2nd instar and/or adults during feeding (Moritz et al. 2004; Ullman et al. 1992). There is a latent period between acquisition and transmission of the virus during which the virus must infect gut cells, cross tissue barriers, and then reach the salivary glands so that transmission can occur (Whitfield et al. 2005). Adult western flower thrips are the primary vectors of INSV because they are very mobile. In addition, adults may feed on virus-infected plants and sustain midgut infections; however, adults that acquire the virus are unable to transmit it because the salivary glands do not become infected with the virus (Lindord 1932).

The larval stages of the western flower thrips obtain the virus by feeding on an infected host plant, with acquisition of the virus taking approximately 15–30 min (Moritz et al. 2004). The efficiency of acquiring the virus increases, based on longer feeding durations on infected plants. The ability to acquire and transmit the virus decreases when the virus is obtained at a later larval stage. Transmission of the virus occurs when adults feed, which generally requires 5 min to infect plants (van de Wetering et al. 1996); however, longer feeding times or durations may increase the efficiency of transmission. Once infected with the virus, western flower thrips remain infective throughout their approximate 45-day lifespan with the virus replicating (making copies of itself) inside the insect body (Sakimura 1962). Although the virus may replicate or multiply within the body of the western flower thrips, the

virus is not passed on to the offspring (there is no transovarial transmission). It is important to note that not all western flower thrips that possess the virus are able to serve as vectors or transmit the virus because a certain concentration (titer level) of virus must be present in order for transmission to occur (van de Wetering et al. 1996). Regardless, plants that are highly susceptible to both the insect vector and virus should be monitored regularly.

3 Fungus Gnats

Fungus gnat (*Bradysia* spp.) adults are approximately 3.0–4.0 mm (0.12–0.16 in.) in length, winged, with long legs and antennae. Fungus gnat adults live from 7 to 10 days and primarily aggregate near the growing medium surface. Females deposit 100–200 eggs into the cracks and crevices of the growing medium that hatch into white, translucent, legless larvae, which are about 6.0 mm (0.23 in.) in length (Fig. 2). Fungus gnat larvae possess a distinct black head capsule (Cloyd 2000; Ellisor 1934). The life cycle consists of an egg, four larval instars, pupa, and adult, which can be completed in 20–28 days, although development is contingent on growing medium temperature (Cloyd 2000; Wilkinson and Daugherty 1970). Larvae are located within the top 2.5–5.0 cm (1.0–2.0 in.) of the growing medium or inside plant tissue, such as the base or crown (Dennis 1978). Fungus gnat larvae feed on plant roots, such as root hairs, and organic matter in the upper 2.0 cm (0.78 in.) of the growing medium (Cloyd 2012; Kennedy 1971).

Fungus gnat larvae primarily cause direct plant damage by feeding on roots; however, their feeding may also predispose plants to attack from soil-borne plant pathogens by creating wounds that allow entry of certain soil-borne fungi (Fawzi and Kelly 1982; Gardiner et al. 1990; Leath and Newton 1969). In addition, larvae are capable of directly transmitting a number of fungal pathogens including *Pythium* spp., *Fusarium* spp., and *Verticillium* spp., from diseased to noninfected plants. Fungus gnat larvae can transmit *Fusarium avenaceum, Fusarium foetens, Fusarium*



Fig. 2 Fungus gnat (*Bradysia* spp.) larvae (Raymond A. Cloyd; Kansas State University © 2017. All Rights Reserved.)

oxysporum f. sp. *radicis-lycopersici, Thielaviopsis basicola*, and *Verticillium albo-atrum* (El-Hamalawi and Stanghellini 2005; Elmer 2008; Gillespie and Menzies 1993; Harris 1995; James et al. 1995). The oospores of certain *Pythium* spp. are able to survive passage through the digestive trait of *Bradysia impatiens* larvae. Furthermore, the larvae may ingest the propagules of *Pythium aphanidermatum* and macroconidia of *Fusarium avenaceum*, which may be subsequently introduced into young, healthy plants during feeding (El-Hamalawi and Stanghellini 2005; Jarvis et al. 1993). Fungus gnat larvae are capable of transmitting *Pythium* spp., although the extent of larval transmission is less of a factor under greenhouse conditions and may vary depending on specific *Pythium* species.

Fungus gnat adults may also carry, on their bodies, the aerial conida of certain foliar and soil-borne plant pathogenic fungi that produce aerial dispersal stages such as *Botrytis cinerea, Fusarium avenaceum, Fusarium acuminatum, Fusarium foetens, Thielaviopsis basicola, Verticillium dahliae,* and *Verticillium albo-atrum*, which may be transmitted to healthy plants (El-Hamalawi 2008; Elmer 2008; Gillespie and Menzies 1993; Harris 1995; James et al. 1995; Kalb and Millar 1986). However, adult fungus gnats are unlikely to vector *Pythium* spp., because any sexual and/or asexual reproductive structures are produced belowground as there are no aerial dispersal stages that are accessible to fungus gnat adults for acquisition because adults are active aboveground (Braun et al. 2010; Hyder et al. 2009).

4 Shore Flies

Shore fly (*Scatella* spp.) adults resemble houseflies and are 3.1 mm (0.125 in.) long, with black bodies (Fig. 3). Each forewing has at least five light-colored spots. Antennae and legs are short, and the head is small. Larvae are opaque, yellow-brown in color, with no black head capsule, and 6.3 mm (0.25 in.) in length. Shore fly adults are stronger fliers than fungus gnat adults. The life cycle



Fig. 3 Shore fly (*Scatella* spp.) adults (Raymond A. Cloyd; Kansas State University © 2017. All Rights Reserved.)

consists of an egg, three larval instars, pupa, and adult, which can be completed in 15–20 days although development is contingent on growing medium temperature (Cloyd 2012).

Shore flies, in general, cause minimal direct plant damage compared to fungus gnats because larvae do not feed on plant roots, although adults deposit fecal droplets (feces) on plant leaves, which may negatively affect aesthetics and thus crop marketability (Morton and Garcia Del Pino 2007; Van Epenhuijsen et al. 2001; Vänninen 2001). Furthermore, studies show that shore fly larvae and adults are capable of indirectly transmitting soil-borne plant pathogens, such as *Thielaviopsis basicola* (Stanghellini et al. 1999) and *Pythium aphanidermatum* (Goldberg and Stanghellini 1990; Goldberg et al. 1992; Hyder et al. 2009), as well as several other *Pythium* spp. (Hyder et al. 2009) by means of their feces. In addition, shore flies may acquire, both internally and externally, the macroconidia of *Fusarium avenaceum*, thus potentially transmitting the pathogen to healthy lisianthus (*Eustoma grandiflorum*) plants (El-Hamalawi and Stanghellini 2005).

5 Aphids and Whiteflies

The primary aphid and whitefly virus vectors encountered in floricultural crop production systems are the green peach aphid (*Myzus persicae*) and sweetpotato whitefly (*Bemisia tabaci*) (Cloyd 2012; Jones 2003). Aphids and whiteflies are phloem-feeding insects that not only reduce the aesthetic quality of floricultural crops (Olson and Oetting 1999) but also transmit viruses during the feeding process (Agrios 2005; Brown and Czosnek 2002; Jones 2003). However, the viruses vectored by aphids and whiteflies are generally less of a concern to greenhouse-grown floricultural crops compared to the viruses or diseases transmitted by western flower thrips, fungus gnats, and shore flies. Although there are a number of floricultural crops that are susceptible to certain viruses transmitted by aphids and whiteflies, these viruses are more of an issue with field- and greenhouse-grown vegetables, and tropical plants (Jones 2003; Polston and Anderson 1997; Wisler et al. 1998). Regardless, greenhouse producers should be aware of the problems associated with aphids and whiteflies as virus vectors.

5.1 Green Peach Aphid

Aphids in the field reproduce sexually by means of eggs laid on plants, whereas aphids in the greenhouse reproduce asexually, with unmated adult females producing live offspring that are all female (Cloyd 2012). Five nymphal stages with a mean reproductive period of 20 days, mean total longevity of 41 days, and mean fecundity of 75 offspring have been reported for the green peach aphid, *Myzus persicae* (MacGillivray and Anderson 1958). Adults are light-green, occasionally pink to orange, 1.8–2.1 mm (0.07–0.08 in.) in length, with long legs, antennae, and cornicles (two tube-like protuberances on the abdomen) that have black tips



Fig. 4 Green peach aphid (*Myzus persicae*) (Raymond A. Cloyd; Kansas State University © 2017. All Rights Reserved.)

(Fig. 4) (Dreistadt 2001). Green peach aphids tend to aggregate on the terminal growth of plants (Vehrs et al. 1992).

The host range of the green peach aphid includes many cultivated plants and weeds. Florists' crops affected include: carnation, chrysanthemum, gladiolus, rose, snapdragon, stock, and tulip. Green peach aphid transmits over 40 virus species (CABI 2015) that are damaging to florists' crops, including *Bean yellow mosaic* virus, *Carnation mottle* virus, *Cucumber mosaic* virus, *Lily symptomless* virus, and *Tulip breaking* virus. The green peach aphid vectors these viruses in a noncirculative (non-persistent) manner, which means the viruses are stylet-borne; consequently, the viruses do not enter the hemolymph (Pirone and Harris 1977).

5.2 Sweetpotato Whitefly

The life cycle of the sweetpotato whitefly (*Bemisia tabaci*) consists of an egg, nymphs, pupa (4th instar nymph), and adult. Development from egg to adult takes 14–40 days, depending on temperature and host plant. Adults are yellow with the wings held roof-like over their bodies (Fig. 5). Female whiteflies are about 1 mm (0.04 in.) in length with males being slightly smaller. Females live for 10–24 days during which time they can lay up to 250 eggs, depending on temperature and host plant (Dreistadt 2001; Cloyd 2012). Eggs, nymphs, and adults are located on the underside of plant leaves. Nymphs and adults possess piercing-sucking mouthparts that are used to feed on plant fluids in the phloem (Martin et al. 2000; Cloyd 2012).

The host range of the sweetpotato whitefly encompasses many vegetable, field, and ornamental crops, including chrysanthemum, lisianthus and poinsettia, and many weeds. The sweetpotato whitefly has been reported to transmit over 100 plant viruses (CABI 2016; Gilbertson et al. 2015). The whitefly transmits a number of begomoviruses damaging to ornamentals, including *Ageratum yellow vein* virus, *Rose leaf curl* virus, and *Tomato yellow leaf curl* virus. Begomoviruses are transmitted in a circulative and nonpropagative manner by the whitefly. Once the virus is acquired, then the whitefly remains viruliferous for life



Fig. 5 Sweetpotato whitefly (*Bemisia tabaci*) adults (Raymond A. Cloyd; Kansas State University © 2017. All Rights Reserved.)

(Brown and Czosnek 2002). As with western flower thrips, development of insecticide resistance is a major concern in regards to management of sweetpotato whitefly (Prabhaker et al. 1985).

6 Management of Insect Vectors

The primary means of minimizing disease spread is to limit vector acquisition of the pathogen or prevent the insects from transmitting diseases to susceptible floricultural crops. Therefore, implementing plant protection strategies such as sanitation and weed removal is important in alleviating problems with insects that vector diseases. For example, many weeds serve as a refuge for western flower thrips, aphids, and whiteflies. In addition, many weeds may serve as reservoirs for the viruses these insects vector (Daughtrey et al. 1997). Scouting is important in determining the population dynamics of western flower thrips, fungus gnats, shore flies, aphids, and whiteflies (Cloyd 2011). Insecticides can be used against these insect pests. However, problems may arise when using insecticides to control insect vectors including (1) development of resistance in pest populations, (2) resurgence of primary pests and/or unintentional promotion of secondary pests, and (3) elimination of natural enemies (Perring et al. 1999). The use of biological control may be an option for fungus gnats; however, biological control may not be feasible for use against western flower thrips, aphids, and whiteflies because these insects vector viruses. Below are strategies for each insect pest that are designed to reduce populations and consequently diminish the prospect of disease transmission.

6.1 Western Flower Thrips

Since western flower thrips vector viruses, the primary means of managing populations of the insect is by routinely applying insecticides. However, the continual use of

insecticides may result in the development of resistance (Espinosa et al. 2002; Gao et al. 2012; Jensen 2000). Therefore, a complete holistic plant protection strategy must be implemented in order to reduce western flower thrips numbers below damaging levels to avoid the prospect of viral transmission. First of all, always monitor populations using both yellow sticky cards and conducting routine visual inspections (Cloyd 2009) with intensive scouting efforts focused on those floricultural crops that are highly susceptible to western flower thrips and the viruses they transmit.

The removal of weeds from within the greenhouse and outdoors will eliminate refuge sites and reservoirs of the viruses vectored by western flower thrips. In addition, old stock plants maintained in greenhouses may serve as reservoirs for viruses (Daughtrey et al. 1997). The installation of insect screening, with a mesh or pore size of 150 μm, on greenhouse openings including vents and sidewalls may exclude and consequently reduce the numbers of adult western flower thrips entering into a greenhouse, thus alleviating problems associated with viral transmission (Bell and Baker 2000; Bethke and Paine 1991). An important operational procedure is to avoid having workers wear white, blue, or yellow-colored clothing (Daughtrey et al. 1997) as these colors are attractive to western flower thrips adults (Mateus and Mexia 1995). Mass-trapping, using yellow sticky cards or tape, may be an option in capturing western flower thrips adults at low densities; however, this strategy may not provide sufficient suppression of western flower thrips populations at high densities (Kawai and Kitamura 1987).

Trap plants are designed to concentrate insect pests into a localized area where they are subject to insecticides, biological control agents, or both (Hooper et al. 1999). Yellow flowers of both transvaal daisy (*Gerbera jamesonii*) and chrysanthemum (*Dendranthema grandiflora*) are attractive to western flower thrips adults (Blumthal et al. 2005; Buitenhuis and Shipp 2006). Trap plants may not be feasible under certain situations because they require space that would normally be used for plants associated with the main crop (Messelink et al. 2014). However, an option is to use cut flowers instead of whole plants, which would reduce the amount of production space needed. Trap plants cannot remain for extended periods of time in the greenhouse because plant age may impact their ability to attract and retain western flower thrips populations. Therefore, always remove flowering trap plants from the greenhouse before populations of western flower thrips build-up (de Jager et al. 1993). Some issues associated with using trap plants include (1) effectiveness of capturing western flower thrips adults will be greater when the main crop is not flowering (Mainali and Lim 2008); (2) using many trap plants may not be economically feasible for mass-trapping, especially when experiencing high populations of western flower thrips; and (3) the strategy is only effective against adults and not the larvae. Despite these issues, the placement of trap plants near greenhouse openings may be helpful in capturing western flower thrips adults that migrate into greenhouses from outdoors (Teulon et al. 1993).

Since western flower thrips can vector INSV, the tolerance level for this insect pest is "near zero" (Parrella and Jones 1987). Therefore, greenhouse producers regularly apply insecticides in order to suppress western flower thrips populations and consequently prevent floricultural crops from becoming infected with INSV

(Cloyd 2009; Reitz 2009). However, the intense selection pressure associated with routinely applying insecticides has resulted in resistance developing in certain western flower thrips populations (Herron and James 2005; Immaraju et al. 1992; Loughner et al. 2005; Zhao et al. 1995). When using insecticides to suppress western flower thrips populations, always rotate insecticides with different modes of action within a generation before switching to another mode of action (Bielza 2008; Cloyd 2010), which will mitigate or impede the possibility of resistance developing.

There are a number of biological control agents commercially available that may be used to regulate populations of the western flower thrips including the predatory mites *Neoseiulus cucumeris* and *Amblyseius swirskii*, and predatory bugs, *Orius* spp., (Cloyd 2009). However, implementing a biological control program against western flower thrips may not be feasible as the biological control agents may not sustain western flower thrips populations at low enough levels to prevent problems associated with transmission of INSV (Parrella 1999). The success of a biological control program would be contingent on making releases very early in the crop production cycle.

6.2 Fungus Gnats

The larval and adult stages of fungus gnats are capable of transmitting diseases, which means that the tolerance level for fungus gnat populations is very low, thus resulting in the need for implementing intensive plant protection practices. For example, a number of insecticides including insect growth regulators and microbial compounds are effective against the larval stages (Cloyd and Dickinson 2006; Hamlen and Mead 1979; Van Epenhuijsen et al. 2001). In addition, various biological control agents including a soil-dwelling predatory mite (*Stratiolaelaps scimitus*), rove beetle (*Dalotia coriaria*), and entomopathogenic nematode (*Steinernema feltiae*) are effective in suppressing or regulating fungus gnat larval populations when released/introduced early in the crop production cycle (Chambers et al. 1993; Cloyd and Chiasson 2007; Echegaray 2012; Gouge and Hague 1995; Harris et al. 1995; Ludwig and Oetting 2001).

Implementing proper sanitation and watering practices will alleviate problems with fungus gnats (Van Epenhuijsen et al. 2001). Furthermore, reducing moisture within the greenhouse will reduce or "eliminate" algae, which provide a conducive habitat for fungus gnats to breed and proliferate (Zack and Foote 1978). The installation of concrete flooring will reduce algae and consequently fungus gnat populations (Keates et al. 1989). Also, avoid overwatering plants in order to minimize keeping the growing medium too moist, which will prevent algae growth on the growing medium surface. Two additional strategies that can be implemented to manage fungus gnats include (1) placing yellow sticky tape in the middle of benches to capture adults and (2) installing a landscape or fabric barrier, which is a geotextile, nonbiodegradable material, underneath benches. The fabric will minimize problems with algae growth, thus reducing potential breeding sites for fungus gnats.

6.3 Shore Flies

The **management** strategies used to deal with shore flies is similar to those explained above for fungus gnats. For instance, reducing or eliminating algae within the greenhouse will substantially diminish problems with shore flies, which can be accomplished by using certain chemicals (e.g., disinfectants) (McHugh 1991). Hydrogen peroxide, for example, may lessen algae growth that will decrease populations of shore flies. However, there may be issues associated with phytotoxicity to certain plant types (Vänninen and Koskula 1998). Another option is placing large yellow sticky traps or tape above the crop canopy or near walls to capture shore fly adults (Morton and Garcia Del Pino 2007), which can significantly reduce the population of shore flies over time (Vänninen and Koskula 1996).

The use of biological control agents such as entomopathogenic nematodes (*Steinernema* spp. and/or *Heterorhabditis* spp.) has been evaluated under laboratory conditions to determine their ability to control shore flies. However, results have been inconsistent and efficacy may vary depending on greenhouse circumstances (Jacobson et al. 1999; Lindquist et al. 1994; Morton and Garcia Del Pino 2007; Vänninen and Koskula 2003). Moreover, soil-dwelling predatory mites, such as *Stratiolaelaps scimitus* and *Hypoaspis aculeifer*, are not effective in suppressing shore fly populations (Jacobson et al. 1999). The reason why biological control agents are not effective is that shore flies can survive under extremely moist conditions, even being completely submerged underwater (Eastin and Foote 1971), which is not conducive for the survival of many biological control agents.

6.4 Aphids and Whiteflies

The management strategies affiliated with aphids and whiteflies are similar and include: (1) scout or monitor weekly to avoid outbreaks (Cloyd 2012), (2) avoid overfertilizing plants with water-soluble nitrogen-based fertilizers as this increases attractiveness and may subsequently lead to higher populations of aphids and whiteflies (Bentz et al. 1995; Novak and Komor 2010; Petitt et al. 1994), (3) remove weeds from inside and outside the greenhouse as weeds can serve as a refuge or reservoir for the viruses transmitted by aphids and whiteflies, (4) screen greenhouse openings to prevent winged adults from migrating into the greenhouse, (5) apply contact or systemic insecticides, and/or (6) release biological control agents such as parasitoids or predators (Cloyd 2012; Perring et al. 1999). Since both aphid and whitefly females have high reproductive capacity (Dreistadt 2001), management strategies need to be implemented early in the production cycle to maintain populations below damaging levels and also avoid transmission of viruses (Cloyd 2011, 2012; Dreistadt 2001).

7 Conclusion

The management of insect vectors is the primary means of alleviating problems with a number of important diseases. Insect management involves implementing a variety of strategies, including scouting, sanitation, cultural control, insecticidal, and biological. Although, in most cases, complete "elimination" of any pest is not possible, what is most important is that these strategies keep populations of western flower thrips, fungus gnats, shore flies, aphids, and whiteflies at low enough levels in order to protect floricultural crops from diseases.

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Breeding for Disease Resistance in Florists' Crops

5

Zhanao Deng

Abstract

Utilization of disease-resistant cultivars is considered as the most cost-effective and environmentally sound approach to managing plant diseases including diseases of florists' crops. Improving disease resistance has been a main objective in a number of florists' crop breeding programs. The commonly used breeding approach has been classic hybridization followed by progeny screening for resistance to targeted diseases. Breeding efforts over the last several decades have resulted in the development and release of numerous cultivars with resistance to major diseases in anthurium, caladium, carnation, chrysanthemum, gerbera, gladiolus, hydrangea, lily, narcissus, rose, tulip, and other florists' crops. Considerable advances have been made to develop reliable disease resistance screening techniques, identify sources of genetic resistance, and understand the mode of inheritance for at least 24 disease resistance traits in these crops. At least six resistance gene loci in carnation, gerbera, and roses and up to 50 resistance QTL in carnation, gerbera, lily, rose, and tulip have been tagged with molecular markers. One disease-resistant carnation cultivar ("Karen Rouge") has been developed with the aid of molecular markers. The rose gene *Rdr1* conferring complete resistance to black spot disease has been cloned. It is expected that the availability of more molecular markers, the cloning of more resistance genes and QTL, the application of gene editing techniques, and adoption of other new biotechnological tools will increase the efficiency of breeding for disease resistance and allow the development of more cultivars with strong resistance to major diseases in florists' crops.

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R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_4

Keywords
Breeding • Disease resistance • Host resistance • Inheritance • Resistance breeding • Resistance gene • Resistance QTL • Resistance screening • Resistant cultivar • Resistant germplasm

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1 Introduction

The use of disease-resistant cultivars has been an essential tool for integrated management of severe, destructive diseases in many food crops. Disease-resistant cultivars have played a similar role in managing diseases of florists' crops (Daughtrey and Benson 2005). They have helped reduce application of pesticides, lower crop production costs on pesticides and labor, and ensure crop yield and quality. Other benefits from using disease-resistant cultivars may include easier management of crops, better control of soil-borne and virus diseases, reduced hazardous effects of pesticide applications to humans and the environment, better management of fungicide resistance in pathogen populations, and adoption of "softer" fungicides. Utilization of disease-resistant cultivars has been considered as the most cost-effective and environmentally sound approach to managing diseases in florists' crops (Debener 2009; Debener and Byrne 2014; Straathof et al. 1997b).

Improving disease resistance in florists' crops has been a main breeding objective in a number of public-funded plant breeding programs (Cadic and Widehem 2001; Debener 2009; Debener and Byrne 2014; Straathof et al. 1997b). Breeding efforts over the last several decades have resulted in the development of cultivars with resistance to major diseases in a number of florists' crops, including anthurium, caladium, carnation, chrysanthemum, gerbera, gladiolus, hydrangea, lily, narcissus, rose, and tulip. Some of the released cultivars have been widely accepted by growers as well as consumers.

The most widely used approach for breeding disease-resistant cultivars has been hybridization followed by rigorous progeny screening for disease resistance

(Debener 2009; Debener and Byrne 2014; Straathof et al. 1997b). The purpose of hybridization breeding between existing cultivars/breeding lines or between different species (interspecific hybridization) is to introgress and/or combine the underlying resistance genes into new genotypes that will eventually be released as new resistant cultivars. The minimum requirement for use of such a breeding strategy includes the availability of reliable disease resistance screening techniques and sources of genetic resistance. The level of resistance should be adequate to provide practical protection for commercial production, the sources of resistance be in breeding materials that are crossable with other cultivars, and the resistance can be readily transmitted from parent to progeny. Knowledge of the mode of inheritance for the targeted resistance traits can be particularly useful for selecting and pairing breeding parents and designing progeny screening schemes. The following section will provide a brief summary of the available information in these aspects for major diseases in 11 florists' crops.

Over the last 20 years, a number of biotechnological tools have been applied to florists' crops (Arens et al. 2014; Debener 2002, 2009; Debener and Byrne 2014; Yagi 2015). Application of these tools has created a significant amount of genetic and genomic resources for use in breeding for disease resistance. A variety of molecular markers have been used to characterize pathogen isolates and races, create genetic linkage maps of crop genomes, and tag genes or quantitative trait loci (QTL) underlying disease resistance in florists' crops. A number of QTL for various disease resistance traits in carnation, gerbera, lily, rose, and tulip have been associated with molecular markers. One new resistant carnation cultivar has been developed with the aid of molecular markers (Yagi et al. 2012b). It is expected that more molecular markers with high specificity for disease resistance traits will be developed more rapidly with the wide adoption of the genotyping by sequencing (GBS) strategy in florists' crops.

One important resistance gene (*R* gene) in rose has been molecularly cloned using a positional or map-based cloning strategy (Terefe-Ayana et al. 2011). The use of next-generation sequencing technologies has allowed the sequencing of the carnation genome (Yagi et al. 2013a) and soon the rose genome (Foucher et al. 2015). The genomes of some other florists' crops will likely be sequenced in the future. The availability of these genome as well as transcriptome sequences will change the paradigm for cloning other *R* genes in florists' crops. It is likely that more *R* genes will be isolated in the future using a reverse genetic approach rather than a forward genetic approach. The cloned *R* genes will be useful not only for improving disease resistance in the crops from which the genes are isolated, but also for related florists' crops.

A number of transgenic plants with increased resistance to fungal and viral pathogens have been created in florists' crops (Hammond 2006). Transgenic technologies have greatly expanded the pool of genes that can be used for improving disease resistance in florists' crops. Nevertheless, these transgenic plants contain "foreign" genes from unrelated plants, animals, and/or microbes. The genetic nature of these plants has created an almost insurmountable regulatory hurdle for research and commercialization of disease-resistant transgenics in

florists' and other crops. New technologies such as the CRISPR/Cas9 (clustered regularly interspaced short palindromic repeats/CRISPR-associated protein 9) will allow precise editing of target genes in florists' crops, resulting in genetically modified plants without foreign DNA (Xiong et al. 2015). It is anticipated that these plants or cultivars may be considered much more favorably by governmental agencies, and hopefully consumers as well, for production and consumption. Looking forward, it is very likely that the rapid development and application of biotechnological tools will increase the efficiency of breeding for disease resistance and allow the development and use of new florists' crop cultivars with greater and/or broader resistance to major diseases.

2 Breeding for Disease Resistance

2.1 Anthurium (*Anthurium* Schott)

2.1.1 Anthracnose or Spadix Rot (*Colletotrichum gloeosporioides* Penz.)

Anthracnose was once the most serious disease of anthurium in the high-rainfall areas in Hawaii (Aragaki et al. 1968).

Resistance screening. Aragaki et al. (1968) developed a procedure for screening anthurium for resistance to anthracnose. A spore suspension of the pathogen was sprayed onto anthurium spadices when the color change progressed to about half of the spadix length. The inoculated spadices were incubated in a moist chamber at 30 °C for 24 h. Disease severity on the spadices was scored 3 or 6 days post inoculation (dpi).

Sources of resistance and breeding. Cultivars 'Abe Pink,' 'DeWees,' 'Fukano,' 'Hayashi No. 1,' 'Hayashi No. 2,' 'Kansako No. 2,' and 'Marian Seefurth' were highly resistant to anthracnose (Aragaki et al. 1968). Using these cultivars as breeding parents, the University of Hawaii anthurium breeding program has developed more than 15 new anthracnose-resistant anthurium cultivars (Kamemoto and Kuehnle 1996). These cultivars have played a critical role in the successful management of anthracnose in commercial production of anthuriums.

2.1.2 Bacterial Blight [*Xanthomonas axonopodis* pv. *dieffenbachiae* (McCulloch & Pirone) Vauterin, Hoste, Kersters & Swings or *Xad*; Previously *X. campestris* pv. *dieffenbachiae*]

Xad has caused the most serious disease to the anthurium industry in Hawaii and throughout the world (Alvarez et al. 2006). Strains of *Xad* isolated from anthuriums fall into six serotypes (serotype 1–6) and have different capacities to hydrolyze starch (starch+ or starch−) (Alvarez et al. 2006; Lipp et al. 1992). *Xad* infection of anthurium plants consists of two phases, spreading inside leaves (foliar infection) and systemically throughout whole plants (systemic infection).

Resistance screening. A commonly used screening procedure has been spraying a *Xad* cell suspension onto anthurium leaves and stems (Aragaki et al. 1968; Norman

et al. 1999). Elibox and Umaharan (2008) inoculated anthurium leaf disks with *Xad* cell suspensions under vacuum, which resulted in uniform lesion development, reduced experimental errors, and increased cultivar differences in response to *Xad* inoculation. The time taken for the lesion to cover the leaf disks (TLC) correlated well with whole plant field resistance to foliar infection. This procedure is nondestructive to plants and may be applicable for screening large numbers of breeding lines for *Xad* resistance.

Sources of resistance and breeding. Anthurium cultivars developed for Hawaii's anthurium industry before 1980 were mainly selected for resistance to anthracnose and other desirable horticultural traits. Most of these cultivars were found to be susceptible to *Xad*. Cultivars with *A. antioquiense* in their background rarely showed systemic *Xad* infection. Crosses between *A. antioquiense* and *A. andraeanum* often resulted in *Xad*-tolerant cultivars. Twelve cultivars in Trinidad exhibited good levels of resistance (Anaïs et al. 2000). High levels of resistance were also observed in several pot anthurium cultivars (Norman et al. 1999). Cultivar 'Julia' showed no symptoms or only light symptoms after inoculation with four *Xad* isolates, thus possessing broad resistance. Eleven cultivars were highly resistant to foliar infection and two cultivars 'Bianco' and 'Honduras' were resistant to *Xad* during both the foliar and the systemic infection phases (Elibox and Umaharan 2008).

The University of Hawaii anthurium breeding program released several cultivars with good tolerance to *Xad*, including 'Blushing ARCS Hawaii,' 'Bride,' 'Kanapana,' and 'Tropic Ice' (Kamemoto and Kuehnle 1996). Improving anthurium resistance to *Xad* isolates in Guadeloupe was an important objective of the anthurium breeding program at the INRA Unité de recherches en productions végétales in France (Anaïs et al. 2000).

Modes of inheritance. Elibox and Umaharan (2007) investigated the genetic basis of anthurium resistance to systemic *Xad* infection by injecting the engineered *Xad* strain X4gfp (carrying the *green fluorescence protein* or *gfp* gene) into cut petiole stubs. Segregation data from 51 crosses suggested the presence of two recessive genes conferring resistance to systemic *Xad* infection. The authors named two genes as *A* and *B*. According to the proposed model, *A* is dominant to *a*, *B* is dominant to *b*, *aa* is epistatic to *B*-, *bb* is epistatic to *A*-, and the presence of *aa* or *bb*, singly or in combination, results in moderately resistant to resistant phenotypes. The inheritance of anthurium resistance to foliar infection by *Xad* seemed to be quantitative/polygenic with strong additive genetic effects and different from the mode of inheritance for resistance to systemic infection by *Xad* (Elibox and Umaharan 2010).

2.2 Caladium (*Caladium* × *hortulanum* Birdsey)

Florida growers produce over 95% of the caladium tubers used in the world. Diseases are the most important biotic factor limiting the tuber yield and quality of caladium cultivars.

2.2.1 Bacterial Blight or Xanthomonas Leaf Spot [*Xanthomonas axonopodis* pv. *dieffenbachiae* (McCulloch & Pirone) Vauterin, Hoste, Kersters & Swings or *Xad*; syn. *Xanthomonas campestris* pv. *dieffenbachiae* (McCulloch & Pirone) Dye, *Xanthomonas dieffenbachiae* (McCulloch & Pirone) Dowson]

Bacterial blight is the most common foliar disease of caladium (Seijo et al. 2010). Three *Xad* strains isolated from symptomatic caladium plants showed differential pathogenicity and virulence to caladium cultivars. Multiple races have been recognized in *Xad* causing bacterial blight on other aroids, but very little is known about the racial structure of *Xad* causing bacterial blight on caladiums.

Resistance screening. Seijo et al. (2010) inoculated caladium plants by spraying a *Xad* cell suspension (in 0.01 M $MgSO_4$ solution) onto both sides of mature leaves. The inoculated plants were enclosed in a plastic cover for 24 h and then grown in the greenhouse for 6 weeks. Disease severity was evaluated 3 and 6 weeks post inoculation (wpi) using the Horsfall-Barratt scale for area of leaf infection.

Sources of resistance. Five cultivars ('Candidum Junior,' 'Florida Red Ruffles,' 'Florida Sweetheart,' 'Mrs. Arno Nehrling,' and 'White Queen') were classified as being resistant to *Xad* (Seijo et al. 2010). Cultivars 'Carolyn Whorton' and 'Pink Symphony' showed resistance to bacterial blight in field evaluations under a naturally occurring disease epidemic (Seijo et al. 2010). 'White Queen' seemed to be different from other resistant cultivars as it exuded *Xad* from the lower surface of apparently healthy leaves. This may suggest quiescent infection in the leaves of 'White Queen.'

2.2.2 Fusarium Tuber Rot [*Fusarium solani* (Mart.) Saa.]

This is the most important disease that affects caladium tuber quality. Multiple *F. solani* isolates have been made from diseased caladium tubers (Goktepe et al. 2007). There was a considerable level of variation among isolates in virulence in causing tuber rot and a significant interaction between *Fusarium* isolates and caladium cultivars (Goktepe et al. 2007).

Resistance screening. The inoculation technique of Goktepe et al. (2007) has been commonly used in evaluating caladiums for resistance against *F. solani*. Highly virulent isolates were cultured on plates of carnation leaf agar until the carnation leaf pieces were covered with abundant sporodochia. Caladium tubers were surface-sterilized and cut into halves. Tuber halves were punctured in the center of the cut surface, and immediately two *F. solani*-colonized carnation leaf pieces were inserted into the hole. Inoculated tuber halves were incubated at 18 °C and ~100% relative humidity (RH) for 2 weeks. Disease severity was recorded on the basis of lesion size on tuber halves. Because of the observed cultivar × isolate interaction, two or more isolates need to be used in caladium cultivar evaluation or breeding line screening.

Sources of resistance. Five cultivars ('Aaron,' 'Candidum,' 'Florida Sweetheart,' 'Rosebud,' and 'White Christmas') showed high levels of resistance and two cultivars ('Red Flash' and 'White Wing') exhibited moderate levels of resistance to *F. solani* (Goktepe et al. 2007). Although 'Florida Sweetheart' was a progeny of two susceptible cultivars, it exhibited good resistance after inoculation. This may

suggest the involvement of multiple genes in the genetic control of *Fusarium* tuber rot resistance in caladium, and some susceptible cultivars may carry some resistance alleles that may be useful for developing new resistant cultivars. When these resistant cultivars were used as breeding parents, approximately 5–20% of their progeny expressed good levels of *Fusarium* tuber rot resistance (Z. Deng, unpublished).

2.2.3 Pythium Root Rot (*Pythium myriotylum* Drechs.)

Pythium root rot is one of the most damaging diseases of caladiums, able to reduce plant growth by 70% and tuber yield by 40% (Ridings and Hartman 1976). Young plants of susceptible caladium cultivars could lose as much as 90% of their roots and 85% of their leaves within 2–3 wpi after *P. myriotylum* inoculation (Deng et al. 2005a, b).

Resistance screening. Deng et al. (2005a) described a protocol for inoculating the root ball of caladium plants. *Pythium myriotylum* isolates were cultured on the acidified potato dextrose agar medium and then in V8 broth to produce mycelial mats. The mats were incubated at room temperature under fluorescent light to induce the production of oogonia, which were collected into a spore suspension. Five milliliters of the oogonium suspension were applied to the root ball of caladium plants. The inoculated plants were grown in the greenhouse at approximately 31 °C, and the root balls of inoculated plants were kept moist throughout the incubation period.

Sources of resistance. Seven caladium cultivars ('Apple Blossom,' 'Candidum,' 'Candidum Junior,' 'Etta Moore,' 'Florida Blizzard,' 'Freida Hemple,' and 'White Christmas') showed moderate levels of resistance (Deng et al. 2005a, b). 'Florida Blizzard' resulted from a cross between 'Aaron' and 'White Christmas' without Pythium root rot disease screening, yet it showed a level of Pythium root rot resistance comparable to that of 'White Christmas,' suggesting a good potential to breeding for new resistant cultivars by using the identified sources of resistance. In caladium breeding, *Pythium* screening was often postponed to year 5–8 after initial crossing when there were a sufficient number of plants available (Z. Deng, unpublished). A major difficulty in screening caladium breeding lines for root rot resistance has been the remarkable variation in root rot severity between replicates and experiments (Z. Deng, unpublished).

2.2.4 Root-Knot Nematodes [*Meloidogyne incognita* (Kofoid & White) *Chitwood*]

Severe root-knot nematode infection can stunt caladium plants and reduce caladium tuber yields.

Resistance screening. McSorley et al. (2004) and Dover et al. (2005) extracted nematode eggs from pepper plants, incubated the eggs in the laboratory to produce juveniles, and injected the juveniles into the soil around caladium plants grown in pots. Inoculated plants were assessed for severity of root galling 5 months post inoculation (mpi); nematode eggs and juveniles in the soil and caladium roots were extracted and counted.

Sources of resistance. Most of the 17 commercial caladium cultivars evaluated expressed resistance against root-knot nematodes and suppressed the infestation and/or multiplication of nematode juveniles in their roots (Dover et al. 2005; McSorley et al. 2004). Major commercial cultivars with nematode resistance include 'Candidum,' 'Candium Junior,' 'Fannie Munson,' 'Freida Hemple,' 'Postman Joyner,' and 'White Christmas.' Similar degrees of galling were observed on the roots of resistant and susceptible caladium cultivars. Root galling alone does not seem to be a reliable indicator of nematode resistance or susceptibility in caladium. Breeding for nematode resistance in caladium will likely need new screening methods such as molecular markers for identifying resistant breeding lines.

2.3 Carnation (*Dianthus caryophyllus* L.)

2.3.1 Carnation Bacterial Wilt [CBW; *Burkholderia caryophylli* (Burkholder) Yabuuchi, Kasako, Oyaizu, Yano, Hotta, Hashimoto, Ezaki, and Arakawa; Previously *Pseudomonas caryophylli*]

CBW has been one of the most damaging diseases affecting carnation production in Japan (Onozaki et al. 1999a, b). Control of CBW has been difficult because the pathogen infects the roots and colonizes the vascular system of carnation plants.

Resistance screening. A cut-root soaking method was described by Onozaki et al. (1999a) for inoculating carnation plants. Bacterial isolates were cultured in a potato sucrose liquid medium to produce inoculum. Rooted carnation cuttings were trimmed to remove tips of the roots and then dipped into the bacterial suspension and then planted in sterilized soil beds with a constant soil temperature at 31.5 °C. The number of wilted plants for each cultivar was recorded weekly for 13 wpi.

Sources of resistance and breeding. An old cultivar ('Durango') seemed to be immune to CBW. Onozaki et al. (1999a) identified three cultivars ('Wiko,' 'Nocto,' and 'Sandrosa') with partial resistance. 'Sandrosa' produced the resistant line (85-11) in later generations. Two species accessions *Dianthus capitatus* ssp. *andrzejowskianus* and *D. henteri* were almost completely resistant to CBW (Onozaki et al. 1999b). An effort was initiated in Japan in 1988 to introgress the resistance from *D. capitatus* ssp. *andrzejowskianus* into commercial cultivars. Crossing this wild accession with a susceptible carnation cultivar resulted in the resistant breeding line 'Carnation Nou No. 1.' The first resistant cultivar ('Karen Rouge') with flower characteristics comparable to other commercial cultivars was developed after seven other cultivars or breeding lines were used in crosses to improve flower characteristics of 'Carnation Nou No. 1' (Yagi et al. 2012b). The development of this resistant cultivar took six rounds of crossing and selection and more than 15 years (Yagi et al. 2012b).

Mode of inheritance. The resistance to CBW in *D. capitatus* ssp. *andrzejowskianus* and its progeny 'Carnation Nou No. 1' is a quantitative trait under the control of a major gene (Onozaki et al. 2004). Three QTL have been identified controlling the resistance. The *Cbw1* locus showed the largest effect,

accounting for 60.5% of the total phenotypic variance in CBW resistance (Onozaki et al. 2004). The *Cbw2* and *Cbw3* loci exhibited much smaller effects for CBW resistance (Yagi et al. 2006). The CBW resistance in 'Sandrosa' is also a quantitative trait. One major QTL (*Cbw4*) explained 63.0% of phenotypic variance for CBW resistance in a F_2 population of 'Sandrosa' (Yagi et al. 2012a).

Molecular markers. The *Cbw1* locus has been tagged with a specific marker STS-WG44 (Onozaki et al. 2004). Using this marker, Yagi et al. (2012b) selected the resistant breeding line 6ZA37-1, which subsequently was released as cultivar 'Karen Rouge.' This is the first disease-resistant cultivar selected with the aid of molecular markers in carnation breeding and probably in the breeding of florists' crops.

The *Cbw4* locus has been placed close to simple sequence repeat (SSR) markers CES1161 and ES2643. *Dianthus capitatus* ssp. *andrzejowskianus* and cultivar 'Karen Rouge' carry null alleles at these marker loci (Yagi et al. 2012a). The carnation linkage group B4 carrying the *Cbw4* from 'Sandrosa' and the linkage group A6 harboring the *Cbw1* from *D. capitatus* ssp. *andrzejowskianus* seem to be syntenic (Yagi et al. 2013b).

2.3.2 Fusarium Wilt (*Fusarium oxysporum* f. sp. *dianthi*)

Fusarium wilt is the most detrimental disease posing a constant threat to carnation crops worldwide. Eight races of *F. oxysporum* f. sp. *dianthi* have been identified (Garibaldi and Gullino 1987). Race 2 is the most common, widespread, and damaging in many carnation growing areas (Demmink et al. 1987). This race can cause up to 40% yield losses. Interactions are common between *F. oxysporum* f. sp. *dianthi* races and carnation cultivars and also between isolates of certain races and certain cultivars (Demmink et al. 1987).

Resistance screening. Carnation plants can be inoculated with *F. oxysporum* f. sp. *dianthi* either through the roots or the stem. Root inoculation can be achieved by growing rooted cuttings in infected soil or applying a conidial suspension (Demmink et al. 1987; Tramier et al. 1983). Subsequently, external (wilting) and internal symptom severity (vascular discoloration and degradation) of the inoculated plants can be evaluated (Demmink et al. 1987). Stem inoculation introduces the pathogen directly into the vascular system. This procedure can circumvent potential extravascular resistance in the roots and has been used to understand the resistance components in resistant carnation cultivars (Baayen et al. 1991).

Sources of resistance and breeding. Some carnation cultivars were resistant to six or seven races, and one cultivar 'Duca' was resistant to eight races. Tramier et al. (1983) indicated that 'Champagne' was highly resistant and several other cultivars ('Clair Avril,' 'Diano,' 'Duca,' 'Elsy,' 'Lanathena,' 'Lanakino') were as resistant to Fusarium wilt as 'Pallas,' which showed good field resistance and resisted natural inoculum but became susceptible when inoculated by root dipping or grown under low light intensities. 'Antares' and 'Palma' (a sport of 'Pallas') exhibited a high level of resistance. Cultivars 'Revada' and 'Novada' were resistant to races 1, 2, and 4 (Demmink et al. 1989). Cultivar 'Eveline' exhibited a high level of resistance; disease incidence of this cultivar remained below 3–10% by the end of the growing season (Ben-Yephet et al. 1993).

Complete resistance to race 2 of *F. oxysporum* f. sp. *dianthi* was found in 'Arbel' and 'Scarlette' (Ben-Yephet et al. 1997). Plants of these cultivars remained symptomless in field tests as well in net-house tests where conditions were very conducive for Fusarium wilt.

Extensive breeding efforts were made in France, Italy, the Netherlands, the UK, the USA, and other countries to develop new resistant carnation cultivars. A full range of standard and spray type cultivars with high levels of resistance have been released (Blanc 1983; Mitteau 1987). Fusarium wilt in carnation has been well controlled by the use of resistant cultivars (Ben-Yephet et al. 1996; Sparnaaij 1983; Sparnaaij and Demmink 1987). Sparnaaij (1983) recommended that screening carnation breeding populations for resistance to *Fusarium* be conducted in two stages: first a fairly mild screening of seedlings to eliminate the most susceptible ones, followed by a more rigorous screening after quality selection and the second stage is to be done on a clonal basis. Baayen et al. (1991) considered the predominant resistance mechanisms and the mode of inheritance and suggested the use of stem inoculation and selecting noncolonized carnation progeny as a quick and reliable selection method.

Modes of inheritance. The resistance to race 1 appeared to be complete and monogenically inherited (Demmink et al. 1989). The partial resistance to race 2 is polygenically inherited (Baayen et al. 1991). There seemed to be at least two resistance mechanisms involved: one for extravascular resistance and one for vascular resistance. The two mechanisms seemed to be inherited independently (Baayen et al. 1991). Tramier et al. (1987) indicated the existence of two types of resistance to race 2: one inversely affected by the inoculum level and the other one more stable and not influenced by the inoculum concentration. Scovel et al. (2001) proposed a different model for the inheritance of resistance to race 2 in breeding line 2217. In this model, two major genes (R_1 and R_2) control the resistance and they interact additively. Progeny with $R_1R_1R_2R_2$ or $R_1R_1R_2r_2$ genotype are resistant, while those with $R_1r_1r_2r_2$, $r_1r_1R_2R_2$, $r_1r_1R_2r_2$, and $r_1r_1r_2r_2$ genotypes are highly susceptible. The partial resistance to race 4 seemed to be polygenically inherited as well (Baayen et al. 1991; Demmink et al. 1989).

Molecular markers. A randomly amplified polymorphic DNA (RAPD) marker was identified for one of the two putative genes (*R2*) controlling the *Fusarium* resistance in breeding line 2217 (Scovel et al. 2001). This marker has been converted into a specific marker, which may be useful for monitoring the introgression of the *R2* locus into new carnation cultivars (Scovel et al. 2001).

2.4 Chrysanthemum (*Chrysanthemum* × *morifolium* Ramat.)

2.4.1 Chrysanthemum Stunt Viroid (CSVd)

CSVd is one of the most damaging diseases in chrysanthemum cultivation in Japan (Omori et al. 2009), Australia, and other production areas.

Resistance screening. Omori et al. (2009) and Matsushita et al. (2012) inoculated chrysanthemum plants by grafting them on CSVd-infected rootstock and conducted

RT-PCR, nested PCR, microtissue direct RT-PCR, and/or real-time RT-PCR to confirm the presence of the viroid and determine the viroid titer in the chrysanthemum scion's leaves. Nabeshima et al. (2012) inoculated CSVd directly into the shoot apical meristems (SAMs) of chrysanthemum by attaching CSVd-infected root tips to the SAMS in vitro.

Sources of resistance. Omori et al. (2009) showed that cultivar 'Utage' was resistant to CSVd. Some progeny of selfed 'Utage' exhibited even stronger resistance to CSVd. Matsushita et al. (2012) discovered a high level of resistance in an old cultivar 'Okayamaheiwa.' When 'Okayamaheiwa' was grafted between a CSVd-susceptible, infected rootstock and a CSVd-susceptible, noninoculated scion, the viroid moved through the phloem of 'Okayamaheiwa' into the susceptible scion but did not replicate in 'Okayamaheiwa.' Cultivar 'Sei no Issei' and 'Mari Kazaguruma' proved to be resistant to CSVd (Nabeshima et al. 2012).

Mode of inheritance. Approximately one eighth of the progeny of selfed 'Utage' showed resistance to CSVd (Omori et al. 2009). When 'Okayamaheiwa' was crossed with susceptible cultivars, about one eighth of their F_1 progeny was resistant to CSVd (Matsushita et al. 2012).

2.4.2 White Rust (*Puccinia horiana* Henn.)

White rust is one of the most serious fungal diseases in chrysanthemums, occurring widely and causing severe economic losses in the field production of chrysanthemum. There exist several physiological races in white rust pathogen populations.

Resistance screening. De Jong and Rademaker (1986) inoculated young chrysanthemum plants or seedlings by using susceptible, sporulating donor plants. Inoculated plants were maintained at 16–18 °C for disease development. Takatsu et al. (2000) reported an inoculation technique for in vitro grown chrysanthemum plantlets. The high humidity (90–100%) inside the culture vessel (plant box) and the temperature in the tissue culture room (20–25 °C) were suitable for white rust development. The degree of chrysanthemum susceptibility to white rust in vitro correlated well with the sporulation ratings of plants in the greenhouse.

Sources of resistance and breeding. Cultivars 'Bridesmaid,' 'Carillon,' 'Dollaroid,' 'Guilderland,' 'Lameet,' 'Renine,' 'Rewilo,' 'Roulette,' 'Ready,' and 'Smile' showed neither visible disease symptoms nor sporulation after *P. horiana* inoculation (De Jong and Rademaker 1986). Cultivars 'Keirin,' 'Oubai,' and 'PP213' exhibited partial resistance to white rust (Takatsu et al. 2000). New white rust-resistant cultivars have been released in Korea and other countries (Lim et al. 2009).

Mode of inheritance. De Jong and Rademaker (1986) analyzed the segregation of white rust resistance and susceptibility in multiple F_1 populations. Three segregation ratios were observed in various crosses, and all ratios suggest that a single dominant gene (*Ph*) controls resistance to white rust. The majority of resistant chrysanthemum cultivars carry one allele of the *Ph* gene (simplex), but breeding line 80,211 carries two *Ph* alleles (duplex).

2.5 Gerbera (*Gerbera jamesonii* Bolus ex Hook. f.)

2.5.1 Gray Mold (*Botrytis cinerea* Pers.:Fr.)

Gray mold infection mainly occurs postharvest on gerbera flowers. This disease has caused substantial losses in quality and quantity during gerbera production and transportation.

Resistance screening. Fu et al. (2015) sprayed gerbera flowers (capitulums) with a spore suspension of *B. cinerea* and incubated the flowers in a growth chamber for 5 days. Disease severity on the whole flower, ray florets, and disk florets were scored on a scale of 0 (no symptom) to 5 (completely blighted).

Sources of resistance. Gerbera genotype 'KK' showed partial resistance to gray mold (Fu et al. 2015).

Mode of inheritance. Gerbera resistance to gray mold seemed to be a quantitative trait controlled by multiple genes. Fu et al. (2015) detected multiple QTL with small effects for the *Botrytis* resistance in 'KK.' One of these resistance QTL is linked with the gerbera 2-pyrone synthase gene (*2-PS*). The product of *2-PS* synthesizes a putative precursor for two phytoalexins (gerberin and parasorboside) in gerbera. Silencing this gene in gerbera stopped the synthesis of gerberin and parasorboside and resulted in increased susceptibility to *B. cincerea* infection on disk florets (Koskela et al. 2011). SNP marker analysis based on the sequence polymorphisms in the *2-PS* gene may be useful for selecting gerbera lines resistant to gray mold (Fu et al. 2015).

2.5.2 Powdery Mildew [PM; *Podosphaera xanthii* (Synonym *P. fusca*; Formerly *Sphaerotheca xanthii*)]

PM occurs primarily on gerbera leaves but also on flower stalks. It has been one of the most common and destructive diseases in gerbera production. *Podosphaera xanthii* and *P. fusca* both were described as the causal agent of PM on gerbera (Granke et al. 2012; Kloos et al. 2005).

Resistance screening. Song and Deng (2013) inoculated young gerbera seedlings with *P. xanthii* in a settling tower. The inoculum was dislodged from sporulating plants of susceptible cultivars that were infected with naturally occurring *P. xanthii*. Kloos et al. (2005) described a procedure for inoculating gerbera leaf disks in vitro. Leaf disks cut from fresh detached leaves were placed on water agar containing benzimidazole in plastic dishes. Fresh spores on sporulating leaves were brushed directly onto the adaxial surface of leaf disks. Inoculated leaf disks were incubated in the dishes at 18 °C and low light for 14 days. PM severity (extent of sporulation) and incidence (number of disks showing infection) were recorded 5, 10, and 14 dpi.

Sources of resistance and breeding. Only a limited number of sources of PM resistance have been identified in commercial gerbera cultivars and breeding lines. The cut flower cultivar 'Terrafame' showed significantly less PM than 10 other cultivars evaluated, and 'Festival Semi-Double Orange' pot gerbera cultivar was moderately resistant to PM (Hausbeck 2004). In field and greenhouse trials in North

Carolina, USA, two gerbera lines (Plant 176 and 214) exhibited high levels of resistance to PM (Kloos et al. 2005). Two breeding lines UFGE 31-19 and UFGE 5-23 were found to be highly resistant to PM in Florida, USA. Progeny of UFGE 31-19 and UFGE 5-23 also showed good PM resistance when tested in several other sites in the USA (Song and Deng 2013). The PM resistance in UFGE 31-19 has been incorporated into several new gerbera cultivars via hybridization and phenotypic selection (Deng and Harbaugh 2010, 2013).

Mode of inheritance. The PM resistance in line 176 and 214 seemed to segregate in F_1, F_2, and backcross populations like a single dominant gene. Kloos et al. (2005) named this gene *Pmr1* (*p*owdery *m*ildew-*r*esistant). This gene exhibited significant variability in dominance or expressivity, especially in progeny where one parent of the crosses was highly susceptible to powdery mildew. Modifying genes were suspected that might have effects on the level of PM resistance in gerbera. The mode of inheritance for the PM resistance in UFGE 31-19 was described by Song and Deng (2013). Distribution of PM severity ratings among progeny of UFGE 31-19 was continuous but with two peaks, indicating that the PM resistance is a quantitative trait, likely controlled by major genes. Molecular marker and QTL analyses revealed two linked QTL, *Rpx1* and *Rpx2* (*r*esistance to *P. xanthii*), for the PM resistance. *Rpx1* and *Rpx* 2 could explain 56.5% and 14.6% of the phenotypic variance for PM resistance in the segregating population, respectively.

2.5.3 Phytophthora Wilt (*Phytophthora cryptogea* Pethybridge & Lafferty)

Phytophthora wilt can cause serious losses of gerbera plants in greenhouse and field production.

Resistance screening. Sparnaaij et al. (1975) cultured *P. cryptogea* isolates on a solid peat-oatmeal substrate and placed the ground substrate at the bottom of pots as inoculum. Gerbera plants with several leaves were transplanted into these pots and grown for approximately 3 months before healthy plants were counted.

Sources of resistance. One gerbera breeding line showed a good level of resistance to Phytophthora wilt with 84% of its plants remaining healthy 3 mpi (Sparnaaij et al. 1975).

Mode of inheritance. Resistance to *Phytophthora* in gerbera seems to be a quantitative trait with high heritability. Sparnaaij et al. (1975) showed that general combining ability (GCA) was the main component of total progeny variance and the genetic variation among gerbera lines in *Phytophthora* resistance was likely due to additive gene actions. The average narrow sense heritability for *Phytophthora* resistance was estimated at 0.81. A high level of resistance in the parental cultivars or clones would be most critical for producing resistant breeding lines and cultivars. A preferred way to test the breeding value of gerbera materials may be to run test-crosses with a standard susceptible cultivar. The ideal standard cultivar should be self-incompatible but highly cross-fertile so that test cross progeny could be readily produced (Sparnaaij et al. 1975).

2.6 Gladiolus (*Gladiolus* spp.)

2.6.1 Fusarium Yellowing and Corm Rot [*Fusarium oxysporum* f. sp. *gladioli* (Massey) Snyd. & Hans.]

Fusarium oxysporum f. sp. *gladioli* can cause corm and root rot and leaf yellowing in gladiolus. Fusarium corm rot is the most important disease in gladiolus production (Jones and Jenkins 1975). Two races of *F. oxysporum* f. sp. *gladioli* have been described (Roebroeck and Mes 1992), and more races may exist (Straathof et al. 1998).

Resistance screening. Several clonal tests have been described for screening gladiolus for *Fusarium* resistance. Löffler et al. (1997) standardized greenhouse conditions, inoculum preparation, and disease measurement for these tests. *Fusarium* was grown in an oatmeal-soil mixture and the mature cultures were ground and mixed with soil as inoculum. Gladiolus corms were planted in the infested soil and grown in a greenhouse at 18/14 °C (day/night). The relative shoot length (RL) and the average disease rating (ADR) of each cultivar were assessed 8–20 wpi. RL and ADR data from this procedure correlated well with each other (Löffler et al. 1997). In vitro tests using complete corms reduced the number of corms required by 90% (Löffler et al. 1997). Jones and Jenkins (1975) selected gladiolus seedlings in the fields heavily infested with *Fusarium*. Straathof et al. (1997a) described a modified method for screening gladiolus seedlings. A layer of *Fusarium*-infested soil was placed under noninfested soil, and gladiolus seeds were sown directly into the noninfested soil. Seedlings were harvested 30 wpi and the extent of plant decay was evaluated. This method detected variation in *Fusarium* resistance between and within breeding populations at the seedling level and made early selection feasible.

Sources of resistance. Partial resistance to *Fusarium* has been found in a number of commercial cultivars. Jones and Jenkins (1975) reported high to moderate partial resistance in 'Beverly Ann,' 'Fiat Lux,' 'Folies Bergere,' and 'Prince Bernhard.' High levels of resistance were found in 'Angelina,' 'Harlekyn,' 'Jessie M. Conner,' 'Roselind,' and 'Yellow Special' (Löffler et al. 1997; Straathof et al. 1998). High levels of resistance also exist in a number of species accessions, including *G. callianthus*, *G. dalenii*, and *G. garnierii*, and *G. gladanthera* (Jones and Jenkins 1975; Straathof et al. 1997b). The resistant *G. dalenii* genotypes can be particularly valuable for breeding resistance against *Fusarium*.

Developing *Fusarium*-resistant cultivars was a main objective in several gladiolus breeding programs in the USA and the Netherlands (Jones and Jenkins 1975; Straathof et al. 1997b; Wilfret 1981, 1986, 1993). Over a 10-year period, some 500,000 seedlings were screened in North Carolina in the fields heavily infested with *F. oxysporum*; and then about 1% of the seedlings that exhibited good commercial potential and some level of disease resistance were evaluated by artificial inoculation. Several selections including breeding lines 66-109-5 and 63-5-1 showed significantly better resistance than 'Prince Bernhart' and 'Folies Bergere' (Jones and Jenkins 1975). The *Fusarium* resistance of these lines seemed to be derived from the cultivar 'Snow Princess.' Gladiolus cultivars with partial resistance were released in Florida (Wilfret 1981, 1986, 1993). Highly resistant breeding lines were obtained

from interspecific crosses involving *G. dalenii* and other resistant species (Straathof et al. 1998).

Mode of inheritance. Straathof et al. (1997a) investigated the mode of inheritance for *Fusarium* resistance using progeny of 37 crosses between 8 gladiolus cultivars. The resistance of these progeny varied widely and the variation mainly fit an additive model of inheritance. Both GCA and specific combining ability (SCA) effects for *Fusarium* resistance were significant, but the former was much greater than the latter. In general, the *Fusarium* resistance level of parental cultivars determined at the clonal level can be used to predict the *Fusarium* resistance of their progeny at the population level. Some gladiolus breeding lines were resistant to *Fusarium* even when wounded, while others were resistant only when the corms were not wounded. The *Fusarium* resistance in gladiolus may be due to a surface barrier and/or chemical inhibition of fungal growth.

2.7 Hydrangea [*Hydrangea macrophylla* (Thunb.) Ser.]

2.7.1 Powdery Mildew (PM; *Erysiphe polygoni* DC.)

Erysiphe polygoni produces unattractive white mycelium on hydrangea leaf surfaces, causes extensive chlorosis or yellowing on leaves and defoliation, and reduces leaf size and shoot elongation.

Resistance screening. Resistance to powdery mildew in hydrangea cultivars has been assessed using pot-grown plants (Windham et al. 2011) or detached leaves (Li et al. 2009). Pot-grown plants were exposed to naturally occurring PM pressure, and the severity of PM on leaves was evaluated based on the percentage of foliage showing PM signs. Li et al. (2009) described a screening procedure based on detached leaves. Leaf disks cut out of fully expanded healthy leaves were placed on two layers of moistened filter paper in Petri dishes with the abaxial surface up. PM inoculum collected from naturally infected plants was applied to the leaf disks in a settling tower. Inoculated leaf disks were incubated in sealed Petri dishes at 21 °C with a continuous photoperiod before the leaf disks were examined for PM colony development.

Sources of resistance and breeding. There are four subspecies in *H. macrophylla*, and two of them are commercially grown. Windham et al. (2011) reported that cultivars 'Amagi Amacha,' 'Shirofuji,' and 'Veitchii' were the most resistant, while 'Diadem,' 'Komachi,' and 'Omacha' were highly to moderately resistant. These cultivars may represent different resources of PM resistance. 'Veitchii' is the only resistant cultivar that is a member of *H. macrophylla* ssp. *macrophylla*. Since 'Amagi Amacha,' 'Diadem,' and 'Omacha' produce large numbers of fertile flowers, they should be useful for improving PM resistance within *H. macrophylla* ssp. *serrata*, even for *H. macrophylla* ssp. *macrophylla*. Li et al. (2009) observed high frequencies of necrotic cells in the leaves of 'Veitchii,' and these cells contributed to the inhibition of fungal growth, colony development, and sporulation. When 'Veitchii' and 'Bailmer' were crossed, they produced a new resistant cultivar 'Blushing Bride' in the F_1 generation.

2.8 Lily (*Lilium* Hybrids)

2.8.1 Bulb Basal Rot (*Fusarium oxysporum* f. sp. *lilii* Imle)

Lily bulbs are used to produce cut flowers, pot plants, and garden plants. The production of lily bulbs is completed in three stages: induction of scale bulblets from scales, yearling bulbs, and commercial-sized bulbs. Bulb basal rot is one of the major problems in worldwide lily production, especially during the scale bulblet and yearling bulb stage (Straathof and Löffler 1994a). Specific interactions have been found between lily genotypes and *F. oxysporum* f. sp. *linii* isolates, suggesting the existence of physiological races in this pathogen (Löffler et al. 1995).

Resistance screening. Commercial bulbs, bulb scales, bulblets, and yearlings have been used in evaluating lily genotypes for *Fusarium* resistance. Bulb scales offer significant conveniences and advantages for resistance assays, especially when large numbers of genotypes are to be screened. Straathof and Löffler (1994a) demonstrated that disease severity score (DSS) values from scale tests correlated well with the average DSS over four lily developmental stages. The authors recommended use of the scale test for early selection and the scale bulblet test as a final check of selected bulbs for *Fusarium* resistance. Since only partial *Fusarium* resistance has been found in lily cultivars and species, the standardization of screening protocols and use of statistical analysis are critical to achieve high discrimination power and detect small differences among cultivars in resistance level. Straathof and Inggamer (1992) obtained the best discrimination of resistance differences among lily cultivars from using 1% *Fusarium* inoculum (mature, ground *Fusarium* culture mixed with soil at 1:100, w/w) and 8 weeks of incubation at 26 °C or 12 weeks of incubation at 18 °C.

Straathof and Löffler (1994a) described a standardized protocol to evaluate the resistance of lily cultivars to *Fusarium* at four developmental stages. Two highly virulent isolates (CPRO-Fol4 and CPRO-fol11) were incubated in an autoclaved oatmeal and soil mixture, and fully grown cultures were ground and mixed with nonsterile potting soil in a 1:100 ratio (1% inoculum concentration). Lily bulbs, scales, bulblets, or yearlings were planted into the infested soil and then placed in a temperature-controlled greenhouse at 18/14 °C and with 16-h day/8-h night. Lily bulbs, scales, bulblets, or yearlings were dug 6–22 weeks after planting and examined for tissue decay. Disease ratings were converted into DSS values according to a threshold model for ordered categorical data. Straathof and Löffler (1994b) described a seedling test for breeding lily for *Fusarium* resistance. Young seedlings were grown in the greenhouse for 19 weeks and then placed at 5 °C in dark for 9 weeks to produce bulblets. Bulblets of the seedlings as well as their parental cultivars were simultaneously transplanted into *Fusarium*-infested soil. After 5 weeks of incubation, all bulblets were harvested and rated visually according to an ordinal scale with five categories. Seedling bulblets with a disease rating 1 or 2 were selected. The selected progeny were then tested again at the clonal level when sufficient amounts of bulbs became available.

Sources of resistance and breeding. High levels of partial resistance have been found in a number of Asiatic cultivars, including ‘Connecticut King,’ ‘Mont Blanc,’

'Orlito,' 'Prominence,' 'Vonnie,' and 'Yellow Blaze' (Straathof and Löffler 1994a; Straathof and van Tuyl 1994). Löffler et al. (1995) tested the durability of the identified resistance by challenging the resistant cultivars with 31 isolates of *F. oxysporum* f. sp. *lilii*, f. sp. *gladioli*, and f. sp. *tulipae* from four countries. No major breakdown of the resistance was found. After five infection and re-isolation cycles on the resistant cultivars, the aggressiveness of the fungal isolates was not increased. These results suggest that the resistance in these cultivars will be effective for some time.

Generally bulbs of 'Connecticut King' were only slightly affected after *Fusarium* infection. This cultivar can serve as a very valuable source of *Fusarium* resistance for lily breeding. The high resistance of 'Connecticut King' was transferred to 'Orlito.' Resistant cultivar 'Mont Blanc' has been crossed with *L. longiflorum* cultivars including 'White Europe.' One of their hybrids was 'Loblanca.' A tetraploid version of 'Loblanca' has been used by breeding companies to produce commercial cultivars.

High levels of partial resistance also were found in several species accessions (Straathof and van Tuyl 1994). *Lilium dauricum* had the highest resistance among lily genotypes evaluated. *Lilium henryi* and *L. regale* carry resistance to *Fusarium* as well as to viruses and other fungi such as *Botrytis*. These species have been used in wide interspecific crosses with *L. longiflorum*. The resistance in 'Connecticut King' and other Asiatic cultivars can also be traced back to their ancestor species *L. dauricum*, *L. davidii*, and *L. tigrinum* (Straathof and van Tuyl 1994). These resistant species accessions can play an important role in improving lily resistance to *Fusarium*.

Developing new *Fusarium*-resistant cultivars has been a major goal for the lily breeding programs in the Netherlands (Straathof et al. 1993). Straathof and Löffler (1994b) assessed the possibility of obtaining resistant seedlings in 28 Asiatic hybrid lily populations derived from eight cultivars with different levels of *Fusarium* resistance. In the resistant by resistant crosses, an average of 34% of the seedlings could be selected as apparent resistant seedlings. Selection for *Fusarium* resistance at the seedling level appeared to be feasible, although some seedlings would likely be misclassified. The resistance of cultivar 'Hilde' seems to meet the minimal requirement for practical cultivation, and this level of resistance might be a reasonable selection criterion.

Mode of inheritance. *Fusarium* resistance in lily cultivars is a quantitative trait. The estimated heritability was high (0.90 or 0.94) (Straathof et al. 1996b). GCA, SCA, and maternal effects were significant in the 28 populations of lily seedlings described by Straathof and Löffler (1994b). The resistance level of progeny populations was highly correlated with the resistance level of their parental cultivars. Cross breeding can offer good potential for developing new resistant cultivars. At least one of the two parents in each cross should be highly resistant to *Fusarium*.

Marker-assisted selection. Although screening for *Fusarium* resistance at the seedling level is effective, it is labor-intensive and needs well-controlled conditions. Molecular marker-assisted selection is highly desirable for use in breeding lily for *Fusarium* resistance. The very large genome size (36 Gb) and highly heterozygous

genome structure of lilies have made it very challenging to develop molecular markers for desirable traits such as resistance to *Fusarium*. Van Heusden et al. (2002) identified four QTL (*QTL1*, *2*, *3*, and *4*) for *Fusarium* resistance. Shahin et al. (2011) detected two additional QTL for *Fusarium* resistance (*QTL5* and *6*). *QTL1* explained the largest percentage (about 24%) in the backcross population for *Fusarium* resistance variation; the expression of *QTL1* was consistent from year to year during a 4-year test. Work is under way to convert the existing molecular markers into more specific markers for use in lily breeding.

2.9 Narcissus

2.9.1 Basal Rot (*Fusarium oxysporum* Schl. f. sp. *narcissi* Snyder & Hansen)

Basal rot has been the most serious fungal disease in narcissus bulb production in the UK and other countries. Linfield (1997) reported the existence of significant variation in virulence among 18 *F. oxysporum* f. sp. *narcissi* isolates from seven countries.

Resistance screening. Several procedures have been used in artificial inoculation of narcissus bulbs and disease resistance assays. Bowes (1992) evaluated breeding progeny for basal rot resistance by growing carnation bulbs in pots containing a peat/grit compost that was infested with *F. oxysporum* f. sp. *narcissi* (Linfield and Price 1986). Bulb survival was assessed at harvest after one season's growth. Surviving bulbs were re-assessed for disease symptoms after storage. Carder and Grant (2002) reported a rapid in vitro scale inoculation technique for assessing basal rot resistance. Outermost fleshy white scales were excised from bulbs and placed on moistened tissue with the abaxial surface facing up. The abaxial epidermis was pierced with a sterile needle, drops of a *F. oxysporum* f. sp. *narcissi* spore suspension were placed on the wound sites, and lesion size was measured 7 and 14 dpi. Carder and Grant (2002) showed that the lesion size on inoculated scales correlated well with the whole-bulb basal rot resistance.

Sources of resistance. Many of the widely produced narcissus cultivars are highly susceptible to basal rot. Extensive efforts were made in the UK to identify resistance among commercial cultivars. Over a 6-year test, none of the bulbs of cultivar 'St. Keverne' rotted (Tompsett 1986). Moderate resistance was observed in 'Dulcimer,' 'Malvern City,' 'Rijnveld's Early Sensation,' 'Soleil d'Or,' and 'White Lion' (Tompsett 1986). In a 2-year test, 'Dawley,' 'Ice Follies,' and 'Paper White' did not show basal rot (Tompsett 1986). The yellow trumpet cultivar 'Royal Armour' appeared as resistant as 'St. Keverne.'

A breeding program with emphasis on utilization of the partial resistance of 'St. Keverne' and other cultivars was initiated at the Horticulture Research International facility in the UK in 1970s. Resistant cultivars 'Malvern City,' 'Rijnveld's Early Sensation,' and 'St. Keverne' proved to be good parents for use in resistance breeding (Bowes 1992). New useful cultivars were developed, and they displayed high resistance to basal rot (Bowes et al. 1992; Tompsett 1986).

Complete resistance has been found in *N. broussonetti*, *N. canaliculatus*, *N. elegans, N. jonquilla, N. serotinus, and N. viridiflorus* (Linfield 1986, 1992). A number of species hybrid cultivars, 'Buttercup,' 'Larkelly,' 'Nylon,' 'Parcpat,' 'Sugar Bush,' 'Tarlatan,' and 'Thalia,' showed resistance to basal rot (Linfield 1992). *Narcissus broussonetti* and *N. jonquilla* showed complete resistance when challenged with 18 *F. oxysporum* f. sp. *narcissi* isolates from seven countries (Linfield 1997). Some of these species accessions have been included as parents in narcissus breeding in the UK and other countries. *Narcissus jonquilla* was of particular interest because it was the only diploid accession with complete resistance and the resistance might be homozygous.

Modes of inheritance. Bowes (1992) and Bowes et al. (1992) inoculated more than 3500 bulbs from nearly 100 crosses between commercial cultivars and observed continuous variation in basal rot resistance among progeny from those crosses. This pattern of variation suggested a polygenic mode of inheritance for basal rot resistance, and progeny inherited resistance additively from both parents. Many progeny of self-pollinated *N. jonquilla* displayed complete or high levels of resistance. This species seemed to share a similar mode of inheritance with 'St. Keverne' (Carder and Grant 2002). Nicholson et al. (1989) concluded that the resistance in 'St. Keverne' involved a complex interaction between the accumulation of antifungal compounds following infection, cell wall modification, and the formation of lignituber-like structures around invading hyphae. Linfield (1997) observed a positive relationship between lectin content and apparent resistance in seven cultivars and species accessions including 'St. Keverne' and *N. jonquilla*.

2.10 Rose (*Rosa* spp.)

2.10.1 Black Spot [*Diplocarpon rosae* Wolf, Anamorph *Marssonina rosae* (Lib.) Died.]

Black spot is the most important and damaging disease of field-grown roses worldwide. Multiple pathogenic races of *D. rosae* have been observed in Belgium, Canada, Germany, the UK, and eastern USA (Whitaker et al. 2007a). A recent study of 15 *D. rosae* isolates from five countries revealed the existence of 11 distinct pathogenic races (Whitaker et al. 2010b). A larger total number of *D. rosae* races are expected to be found worldwide (Whitaker et al. 2010b).

Resistance screening. Rose resistance to black spot can be assessed on whole plants, detached leaves or leaflets, or leaf disks using naturally occurring disease pressure or cultured single-spore isolates. Correlation between resistance levels in whole plants and detached leaf assays is high; thus either method seems to be adequate for screening roses for black spot resistance. Considering the existence of multiple races in *D. rosae* and race-specific resistance in roses, it is highly desirable to inoculate detached leaves with characterized single-spore isolates of different races for determining the specificity and level of resistance in rose genotypes. Xu and Davidson (1998) described procedures for screening roses for partial resistance to black spot. Their measures of partial resistance included percentage leaf area with

symptoms (LAS), length of lesions (LL), and incubation period (IP) after artificial inoculation. Microscopic observations of hyphal morphology, location, and growth after inoculation have aided the classification of types and levels of rose resistance to black spot (Blechert and Debener 2005). Whitaker et al. (2007a) did not observe any specific clustering of eastern North American isolates with geographic origins and concluded that field screening for black spot resistance in multiple locations might not be necessary, but controlled inoculation with isolates representing known races would be highly desirable.

Sources of resistance and breeding. Partial black spot resistance has been identified in numerous rose cultivars. Eleven rose genotypes in Canada were partially resistant to black spot (Xu and Davidson 1998). Cultivars 'Applejack,' 'George Vancouver,' and 'Pink Gnome' showed moderate to high levels of resistance to black spot (Whitaker and Hokanson 2009; Whitaker et al. 2007b). In a 3-year systematic field evaluation in Texas, 16 cultivars showed good to high resistance (Byrne et al. 2010). The resistance in some cultivars can be effective to many races of *D. rosae*. Cultivars 'Hansa,' 'Mrs. Doreen Pike,' and 'Srivluv' (Baby Love™) were resistant to 10 races of the pathogen (Whitaker et al. 2010b).

The highest levels of black spot resistance have been found in wild rose species (Schulz et al. 2009). Eleven wild rose species accessions at Ahrensburg, Germany, were highly resistant to 13 isolates from 8 countries (Schulz et al. 2009). Rose species *R. wichurana*, *R. rugosa*, *R. multiflora, R. carolina, R. virginiana, R. laxa, and R. spinossisima* have contributed to the black resistance in rose cultivars (Byrne et al. 2010). In many cases, only a few generations are needed to transfer high levels of resistance from wild species into rose cultivars. A breeding line derived from interspecific crosses (91/100-5) resisted all five races of *D. rosae* in Germany and a broad mixture of isolates from several geographic locations (von Malek and Debener 1998).

Black spot resistance has been a main breeding objective in many rose breeding programs over the last 30 years. These breeding efforts have resulted in the release of numerous resistant cultivars. Field-testing of 400 rose cultivars in the USA indicated that 7% of the cultivars had high levels of black spot resistance (Byrne 2015; Byrne et al. 2010). The dominant black spot resistance in *R. multiflora* has been introgressed into both diploid and tetraploid rose breeding lines. Selection for black spot resistance in breeding programs generally is conducted in seedling beds or test fields on 1- or 2-year-old roses. Lühmann et al. (2010) recommended applying a conidial suspension of a broad spectrum of *D. rosae* races on young seedlings in year 2 in the greenhouse. This approach can reduce the number of susceptible rose genotypes; save space, labor, and costs; and improve breeding efficiency.

Mode of inheritance. There are both vertical resistance and horizontal resistance to black spot in roses (Debener and Byrne 2014). The former is conditioned by dominant resistance genes specific to races and the latter by multiple genes. Von Malek and Debener (1998) concluded that a single dominant gene (*Rdr1*) in duplex configuration controls the resistance in breeding line 91/100-5. This gene has been mapped to the telomeric end of rose chromosome 1 (Biber et al. 2010; Kaufmann

et al. 2003). *Rdr1* is active and confers black spot resistance in rose leaves and petals (Terefe-Ayana et al. 2011). Breeding line 91/100-5 also expresses resistance to *D. rosae* isolate DüA3. This resistance specificity is controlled by a single locus *Rdr2*, which is tightly linked with *Rdr1* (von Malek and Debener 1998). Whitaker et al. (2010a) demonstrated that another single locus, *Rdr3*, in tetraploid rose cultivars 'Applejack,' 'Folksinger,' and 'George Vancouver' confers complete resistance to race 8 of *D. rosae*. This locus is inherited independently from *Rdr1* and *Rdr2*.

The mode of inheritance for partial black spot resistance is less known. Such a type of resistance in roses can be also race-specific (Whitaker et al. 2007b). Significant GCA has been observed for partial black spot resistance (Whitaker and Hokanson 2009). Cultivar 'Pink Gnome' showed a high GCA for producing black spot-resistant progeny (Whitaker and Hokanson 2009).

Molecular markers. Both *Rdr1* and *Rdr3* loci have been tagged with molecular markers, which can be used to predict black spot resistance in rose progeny (Debener and Byrne 2014; Whitaker et al. 2010a). The use of molecular markers closely linked with *Rdr1* has enabled the molecular cloning of *Rdr1* (Terefe-Ayana et al. 2011). The gene encodes a Toll-interleukin-receptor nucleotide-binding site leucine-rich repeat (TIR-NB-LRR, or TNL) class protein. There are eight additional copies of TNL sequences at the *Rdr1* region, and five copies are expressed in rose leaves and petals. The cloned *Rdr1* will likely bring opportunities to introduce cisgenic black spot resistance in roses. Different roses may contain 5–20 copies of TNL sequences at the *Rdr1* locus (Terefe-Ayana et al. 2011). Further cloning, characterization, and transferring of these genes may change the paradigm for developing black spot-resistant rose cultivars in the future.

2.10.2 Downy Mildew (DM; *Peronospora sparsa* Berk.)

Downy mildew has increasingly become a problem for cut and garden roses. It causes red to dark spots on rose leaves, resulting in premature drops of leaves and complete defoliation. The pathogen *P. sparsa* can spread quickly among rose plants under optimal conditions.

Resistance screening. Roses can be inoculated by placing drops of a conidial suspension of *P. sparsa* on the lower side of young, fully expanded rose leaves, followed by incubating at 12–16 °C. Disease severity can be estimated at 10–14 dpi under a dissecting microscope on the basis of disease symptoms, lesion size, and density of conidophores (Schulz and Debener 2007). A serological detection method (ELISA) has been reported (Schulz and Debener 2007). This test has allowed precise quantification of the amount of *P. sparsa* in roses and may be used to replace visual disease screening and evaluation.

Sources of resistance. Thirteen rose accessions were found to be resistant to DM (Schulz and Debener 2007). Resistance is common in the *Caninae* section, which includes *R. agrestis*, *R. glutinosa*, *R. micrantha*, and *R. rugiginosa*. Resistance was also found in *R. corymbifera* 'Laxa,' *R. foliolosa*, *R. gallica officinalis*, *R. majalis*, *R. nitida*, and *R. rugose rubra*.

2.10.3 Powdery Mildew [PM; *Podosphaera pannosa* (Wallr.:Fr.) de Bary (syn. *Sphaerotheca pannosa*)]

Powdery mildew is the most common and severe disease in cut rose production. It has caused major yield losses and flower quality reduction on cut roses. Nearly 40% of the fungicides sprayed on cut roses were intended for controlling PM. This disease is also important on field-grown roses. There is a very high level of pathogen diversity in *P. pannosa*, with five races detected at two sites in the USA (Bender and Coyier 1984), eight races in northern German (Yan et al. 2006), and five races in China (Qiu et al. 2015). Five races were found in one location in German (Linde and Debener 2003).

Resistance screening. Rose leaves can be inoculated with powdery mildew by leaf-to-leaf contact, dusting or blowing dry conidia, or blowing conidia in a vacuum settling tower (Linde and Debener 2003). *Podosphaera pannosa* has been also successfully inoculated onto rose leaves using a spore suspension method (Yan et al. 2006). The key steps for using a spore suspension are quick preparation of the spore suspension, immediate application of the spore suspension onto rose leaves, and rapid evaporation of the water in inoculum droplets after dispersion on rose leaves (Yan et al. 2006).

Sources of resistance and breeding. Resistance to PM has been identified in cultivated and wild roses. Cultivar 'Yesterday' showed immunity to pathotype R-E (Hosseini Moghaddam et al. 2010, 2014). Rose breeding lines 91/100-5 and 95/13-90 did not show visible symptoms for all eight isolates tested (Linde and Debener 2003). Thirty-nine wild species accessions at Ahrensburg, Germany, showed PM resistance (Schulz et al. 2009). *Rosa wichuarana* resisted pathotypes R-P and R-E (Hosseini Moghaddam et al. 2010, 2014). Other wild species including *R. laevigata*, *R. longicuspis*, *R. luciae*, and *R. banksiae* were immune to PM (Qiu et al. 2015). Breeding efforts have been initiated in Belgium, Germany, and the Netherlands to develop new rose cultivars with PM resistance (Leus et al. 2007; Linde and Debener 2003; Yan et al. 2006). Linde and Debener (2003) indicated the need to simultaneously introduce several resistance genes to obtain durable resistance to the multiple races of *P. pannosa*.

Mode of inheritance. Both qualitative and quantitative PM resistance has been identified in roses. Linde and Debener (2003) showed that a single dominant gene (*Rpp1*) conditions the complete resistance to race 9 in the diploid rose line 97/9. Multiple QTL have been detected in various sources of PM resistance. Dugo et al. (2005) described two QTL (*Pm1* and *Pm2*) for PM resistance in *R. wichurana*, which is heterozygous at both loci. Linde et al. (2006) discovered 28 QTL for PM resistance in diploid breeding line 95/13-39 (resistant against race 9), of which three major QTL explained about 84% of the variation for PM resistance in the mapping population. Yan et al. (2006) observed relatively high broad-sense heritability (57–62%) for the PM resistance in tetraploid rose genotype P540 and P867. Hosseini Moghaddam et al. (2012) and Leus et al. (2015) detected nine QTL for the PM resistance in cultivar 'Yesterday' and *R. wichurana*.

Molecular markers. The *Rpp1* locus has been mapped to an interval of about 5.5 cM in the rose genome (linkage group 3) (Linde et al. 2006). A highly specific

marker has been developed for this gene. Xu et al. (2005, 2007) reported molecular markers for two PM resistance QTL in *R. roxburghii*.

2.11 Tulip (*Tulipa gesneriana* L. and Hybrids)

Tulips are one of the most important flower bulb crops in the world. About 85% of the world tulip bulbs is produced in the Netherlands, where approximately 10,000 ha of tulip fields produce one billion flowers and an export of two billion tulip bulbs (Straathof et al. 2002; van Tuyl and van Creij 2006).

2.11.1 Botrytis Blight [Gray Mold; *Botrytis tulipae* (Lib.) Lind.]

Botrytis tulipae can infect tulip bulbs, leaves, and flowers. Infection of tulip leaves early in the growing season can result in rapid destruction of leaves and severe reduction of bulb yields.

Resistance screening. Straathof et al. (2002) described an effective technique for screening tulips for resistance to Botrytis blight. Tulip plants were inoculated at the moment of flowering by rubbing leaves by hand, followed by spraying the leaves with a conidial suspension of *B. tulipae*. Inoculated plants were covered with plastic and incubated in a greenhouse at 18 °C. Disease severity was rated 8 dpi. The key steps of this technique were using plants at the moment of flowering for inoculation, removing the wax layer on tulip leaves, and providing continuous leaf wetness for 5 days after inoculation.

Sources of resistance: Absolute resistance has been found in *T. tarda* (Straathof et al. 2002). However, this species cannot be crossed with the commercial tulip cultivars. Partial resistance has been observed in cultivars 'Bellona' (with the minimal resistance level), 'Flair,' 'Generaal de Wet,' and 'Johann Strauss.' 'Flair' and 'Johann Strauss' have been crossed with commercial cultivars, and new hybrids have been obtained. Cultivar 'Princeps' showed a good level of disease resistance before flowering but susceptibility during and after flowering.

Mode of inheritance. Pang et al. (2006) observed a continuous distribution among progeny of 20 crosses in resistance to *Botrytis* and concluded that the resistance trait was of a quantitative nature.

2.11.2 Bulb Rot or Basal Rot (*Fusarium oxysporum* f. sp. *tulipae*)

Bulb rot is the most serious fungal disease in tulips. Rotted bulbs produce stunted plants with yellowing leaves, and plants frequently die before flowering. Crop losses often reach 30–50% in susceptible tulip cultivars.

Resistance screening. Reliable screening tests have been developed and standardized for tulip clonal materials and juvenile seedlings (van Eijk et al. 1978, 1979). Tulip bulbs were planted and grown in *Fusarium*-infested soil for an entire season (November through June). Temperature of the soil was carefully controlled between 12 °C and 22 °C to promote *Fusarium* infection at the proper time. After harvest, bulbs were examined, and the percentage of healthy clusters was used a criterion for disease resistance. To shorten this process, Tang et al. (2015) modified the test by

using bulbs in their normal storage period and incubating the bulbs in a *Fusarium*-infested substrate at 20–24 °C for 8 weeks. A spot inoculation was described recently (Tang et al. 2015). The most aggressive *Fusarium* isolate Tu67 was tagged with the *gfp* gene. Tulip bulbs were wounded by punctures and inoculated by dipping on a cushion soaked with a spore suspension of the engineered isolate. *Fusarium*-infected areas in the bulbs emitted green fluorescence, and these areas were captured by an imaging system and quantified using the *Fusarium* Screen Analysis Software.

Sources of resistance and breeding. Almost absolute resistance has been identified in cultivars 'Black Parrot,' 'Lucky Stride,' 'Mirjoran,' and 'Red Matador,' and moderate levels of resistance in 'Aristocrat,' 'Christmas Fire,' 'Mad. Lefeber,' and 'Rose Copland' (van Eijk et al. 1978, 1979). In progeny tests, 'Black Parrot,' 'Lucky Stride,' and 'Aristocrat' proved to be the most resistant cultivars and produced progeny with high levels of *Fusarium* resistance (van Eijk and Eikelboom 1983). Cultivars 'Kees Nelis,' 'Christmas Dream,' and 'Ile de France' also exhibited high to moderate levels of resistance (Tang et al. 2015). The resistance in some of these cultivars was inherited from old or older cultivars such as 'Centenaire,' 'Panorama,' 'Van der Eerden,' and 'Philippe de Comines' (van Eijk et al. 1985). High levels of *Fusarium* resistance have been also found in species and interspecific hybrids, such as *T. eichleri* hybrid 'Clare Benedict,' *T. eichleri* 'Excelsa,' *T. fosteriana* 'Mad. Lefeber,' *T. fosteriana* 'Princeps,' *T. kaufmanniana* 'Guiseppe Verdi,' and *T. vvedenskyi* (van Eijk et al. 1985). These sources of *Fusarium* resistance have been used in crosses, and new resistant cultivars, including 'Pink Impression' and 'Early Surprise,' and 'Furand,' 'Furanel,' 'Fusarino,' and 'Fusor,' have been developed (Straathof and Eikelboom 1997; van Eijk et al. 1985). Crossing with 'Don Quichotte' (resistant) has resulted in the development of 'Arisa' with resistance against bulb rot and tulip mild mottle mosaic virus (Tsuji et al. 2005).

Mode of inheritance. *Fusarium* resistance in tulip cultivars, such as 'Kees Nelis' and 'Cantata,' is a quantitative trait mainly based on additive gene action (van Eijk and Eikelboom 1983; Tang et al. 2015). GCA values were high and significant in reported tulip crosses. SCA values were low and its significance depended on tulip crosses. The *Fusarium* resistance of some progeny was higher than the resistant parent 'Kees Nelis.' Thus, transgressive segregation exits for *Fusarium* resistance in tulip.

Molecular markers: Six putative QTL have been identified for the *Fusarium* resistance in cultivars 'Kees Nelis' and 'Cantata' and tagged with SNP markers (Tang et al. 2015). Five of the QTL each explained 13.6–20.7% of the phenotypic resistance variation. Three QTL (*Fusarium2*, *Fusarium3*, and *Fusarium6*) were significant in all disease tests. Both the resistant parent 'Kees Nelis' and the susceptible parent 'Cantata' contributed resistance QTL to their progeny (Tang et al. 2015).

2.11.3 Mild Mottle Mosaic Disease (Tulip Mild Mottle Mosaic Virus, TMMMV)

TMMMV, transmitted by the obligate parasitic soil-inhabiting fungus *Olpidium brassicae*, has become a serious disease in some tulip production areas in Japan since 1979 (Morikawa et al. 2005).

Resistance screening. Tulip plants were exposed to the virus in the infected field for 2 years and then cultivated in containers with noninfected soil for 1 year. Peduncles were harvested from plants at flowering, and the presence of TMMMV was detected by tissue blot immunoassay (TBIA). The percentage of infected plants was used as the criterion for classifying disease resistance (Morikawa et al. 2005).

Sources of resistance. Morikawa et al. (2005) reported that 24 tulip cultivars could be considered highly resistant. Among these resistant cultivars are 'Arisa,' 'Ballade,' 'Ballerina,' 'Feu Superbe,' 'Gander,' 'Koiakane,' 'Juan,' 'Leo Visser,' 'Orange Emperor,' 'Princeps,' and 'Snowstar.'

2.11.4 Tulip Breaking Virus (TBV)

TBV is a serious problem in the production of tulip bulbs and flowers in many countries. The virus can cause a significant reduction in tulip bulb number, weight, and quality (Romanow et al. 1986).

Resistance screening. Reliable screening for TBV resistance has been performed at the clonal and seedling level by aphid or mechanical inoculation (Romanow et al. 1986, 1991). Aphid inoculation was achieved by placing viruliferous aphids on the leaves of tulip plants at flowering for 1 day. Tulip plants could be mechanically inoculated by pipetting droplets of virus inoculum on to tulip leaves followed by rubbing the leaves with carborundum powder by fingers. The inoculum was prepared by grinding TBV-infected leaf tissue in water. The inoculated plants were observed for flower breaking 1 year after inoculation. TBV titers in the plants were determined by ELISA.

Sources of resistance and breeding. Absolute resistance has been found in 'Cantata' and 'Princeps' (Eikelboom et al. 1992; Romanow et al. 1991; Straathof et al. 1996a). Cultivars 'Juan' and 'Mad. Lefeber' showed moderate levels of resistance. These four cultivars all are members of *T. fosteriana*. Cultivar 'Ad Rem' was the only representative of *T. gesneriana* tested that could be classified as TBV-resistant. This cultivar is a Darwin hybrid, resulting from a cross between 'Denbola' and 'Princeps.' The resistance in 'Ad Rem' should have come from 'Princeps.'

Mode of inheritance. TBV resistance in tulip is a quantitative trait. GCA effects explained the largest portion of the variation in tulip crosses (Straathof et al. 1996b). 'Cantana' and 'Princeps' showed significantly better GCA values than other cultivars, and they could be desirable parents for introducing TBV resistance from *T. fosteriana* into the *T. gesneriana* assortment.

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Environment Modification for Disease Management

6

Maria Lodovica Gullino and Angelo Garibaldi

Abstract

Florist's crops are often grown under protection and, at least in some countries, in sophisticated greenhouses which permit control of the environment. In the warmer climates, they are also grown outdoors, thus with more limitations in environmental control. Understanding the effects of environmental parameters on pathogens makes environmental manipulation for disease control feasible. In some cases, the manipulation of the environmental parameters by itself permits effective reduction of the incidence of several diseases. Frequently, such manipulation of environmental parameters permits reduction of further interventions. This chapter considers the manipulation of temperature, RH, soil moisture, pH, and light as well as the solutions that can be applied in the case of soilless cultivation.

Keywords

Temperature • Relative humidity • pH • Light • Soilless cultivation • Foliar diseases • Soil-borne diseases

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© Springer International Publishing AG 2018

R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_5

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1 Introduction

Florist's crops are often grown under protection and, at least in some countries, in sophisticated greenhouses which permit control of the environment (Castilla 2013). However, some crops, particularly in the warmer climates, are grown outdoors, thus with more limitation in environmental control.

This chapter will mostly deal with greenhouse crops, which are the ones most potentially influenced by modification of the environmental conditions. However, some of the approaches described can be applied, though at a lesser extent, to field grown crops. Understanding the effects of environmental parameters on pathogens makes environmental manipulation for disease control feasible. In some cases, the manipulation of the environmental parameters by itself permits effective reduction of the incidence of several diseases. Frequently, such manipulation of environmental parameters permits reduction of further interventions.

In this chapter the manipulation of temperature, RH, soil moisture, pH, and light will be considered as well as the solutions that can be applied in the case of soilless cultivation. The manipulation of mineral nutrition and the management of the irrigation water are not considered since they are already covered in other chapters.

2 Environmental Factors Favorable to Disease Development

The main environmental factors influencing plant growth and disease development are temperature (of both air and soil), relative humidity (RH), soil moisture, pH and mineral nutrition, dew, and, to a lesser extent, air movement and light (day length and intensity).

Pathogens often have well-defined temperature *optima* for infection, colonization, and disease development, with differences existing among pathogens as well as for the different phases of the infection process. Very often the most favorable temperature for a pathogen is very close to the temperature *optimum* for crop growth, so that little can be done by growers in terms of temperature manipulation. However, sometimes slight changes in temperature will result in a strong influence on disease development. This can happen, for instance, in the case of Verticillium wilt caused by *V. albo-atrum* and *V. dahliae:* temperatures higher than 25 °C permit reduction of disease severity in several crops. Growth and sporulation of *Botrytis cinerea*, the causal agent of gray mold, are inhibited by temperatures higher than 30 °C: increasing air temperature can help to reduce gray mold incidence in the case of crops tolerating temperatures higher than 30 °C (Trolinger and Strider 1985). The incidence of Fusarium wilt of carnation, caused by *Fusarium oxysporum* f. sp. *dianthi*,

can be reduced at air temperatures lower than 15 °C, while symptoms are severe at temperature higher than 20 °C (Fletcher and Martin 1972; Nelson 1961). Also soil or substrate temperature can influence disease development. *Fusarium oxysporum* f. sp. *dianthi* on carnation is less severe at temperatures of 21 °C or lower, and symptoms are absent at soil temperatures of 10-16 °C. Also Fusarium wilt of Paris daisy (*Argyranthemum frutescens)*, caused by *Fusarium oxysporum* f. sp. *chrysanthemi*, is most severe at soil temperatures higher than 20 °C (Angelo Garibaldi, unpublished).

Generally, warm air and soil temperature and high RH favor the development of bacterial wilts. In the case of bacterial blight of geranium, caused by *Xanthomonas hortorum* pv. *pelargonii* (formerly, *Xanthomonas campestris* pv. *pelargonii*), symptoms develop in 7 days at 27 °C and in 21 days at 16 °C; symptoms are enhanced between 21 and 23 °C, while they are suppressed at 10–15 °C or at 32–38 °C (Campbell 1985). While temperatures lower than 27 °C, at RH lower than 80%, reduce the severity of bacterial blight of chrysanthemum, incited by *Erwinia chrysanthemi* (Campbell 1985).

Air temperature has a direct effect on RH, which, in turn, has a strong influence on the development of foliar pathogens. Heating and ventilation inside a greenhouse permit the establishment of conditions less favorable to infection and sporulation. Diseases such as gray mold, downy mildew, rusts, many foliar spot diseases, and bacterial blight, all dependent upon deposition of water on the plant surface, can be easily controlled or, at least, reduced, by heating and/or ventilating. Although heating requires energy consumption, computerized monitoring systems permit adjustment of RH avoiding excessive use of heat and ventilation. It is advisable to maintain RH at 85% or below because RH at the leaf surface is likely to be 10% higher. Most fungal spores, except those of powdery mildews, can germinate at 95% RH.

Also the covering material (plastic or glass) might affect disease severity; for instance rust caused by *Uromyces caryophyllinus* and leaf spot by *Alternnaria dianthi* are more severe on carnation grown under plastic. On the contrary, rose powdery mildew, incited by *Sphaerotheca pannosa*, is more severe under glass. In all cases, the presence of higher RH values under plastic is responsible for such responses (Garibaldi et al. 1976).

Soil wetness can affect the development of some diseases, especially those caused by pathogens with a motile zoospore stage, such as *Olpidium*, *Pythium*, *Phytophthora*, etc. Epidemics of damping off caused by *Pythium* spp. and of basal rot caused by *Phytophthora* spp. are quite common at the nursery level for many ornamental crops, such as gerbera, rhododendron, and skimmia. In soils which are too wet also healthy roots do not function well because of low oxygenation, thus increasing the effect of root rot. Zoospore producing pathogens find the best situation for spreading in closed soilless systems and diseases caused by *Pythium* spp. and *Phytophthora* spp. are quite common (Gullino and Garibaldi 2007; Jarvis 1992; Stanghellini and Rasmussen 1994).

Wilt of gerbera, caused by *F. oxysporum* f. sp. *chrysanthemi*, is more severe on plants grown in soilless systems than in soil. Maintaining the pH of the nutrient

solutions at values higher than 6.0 reduces the spread of the inoculum of the causal agent (Garibaldi and Gullino 2012).

Although day length and light intensity do not have a direct effect on disease development, a combination of radiation with RH and other factors might affect the expression of certain diseases.

Alteration of the solar radiation inside the greenhouse can influence disease development, not only because of the effect on the spectral composition of the radiation with consequences on spore formation but mainly because plants grown at high density are more susceptible to diseases in the presence of reduced light levels (Louvet 1984). The modification of the spectral characteristics of greenhouse covers permits reduction of the incidence of foliar diseases and also protects crops from insects and insect-borne virus diseases (Raviv and Antignus 2004). A complete or partial absorption of solar UV radiation may interrupt the life cycle of fungal pathogens and/or alter the visual-based behavior of many insects and might play a role in pest and disease management. In the case of greenhouse grown roses, a supplementary radiation with low levels of UV suppresses the development of powdery mildew, incited by *Podosphaera pannosa* (Kobayashi et al. 2014).

The increase in temperature in the greenhouse, the prevention of agrochemical loss from rain, and the filtering of UV light of the plastic covering help to increase the efficiency of the fungicide treatments. However, an increased risk of phytotoxicity as a consequence of an increase in temperature should also be considered (Urban 1997).

It is also important to notice that some environmental parameters might also affect biocontrol agents or, more generally, the microorganisms that, at the phyllosphere or rhizosphere level, might play a role in the control of some pathogens.

3 Monitoring and Control of Environmental Parameters Under Greenhouse Conditions

In the case of greenhouse crops, growers have the unique possibility to exert some control over the environmental conditions in which crops are grown. Monitoring the environment and controlling it in order to avoid pests and diseases has long become an important component of greenhouse management: indeed, plant production under protection offers opportunities for specific control of climatic factors, both for optimization of plant growth and for integrated control of pathogens. In a closed system such a greenhouse, simple management practices can be adopted that are more difficult in an open field.

Disease control by specific climate management is most promising for pathogens which have critical climatic requirements for specific stages of development. Once the environmental parameters that influence pathogens are understood, they can be more precisely regulated. Monitoring is important, since it permits precise control of environmental parameters in order to reduce disease development using computerized systems, which also save energy and reduce production costs. Disease management is greatly enhanced by monitoring and controlling temperature, light, humidity,

water, ventilation, carbon dioxide, and crop nutrition. Of these factors, manipulating the interactions of temperature and humidity is probably the most important in the control of foliar diseases, while rhizosphere moisture and temperature are the most important for root and wilt diseases.

In the presence of high humidity, guttation can occur from leaves and pruning wounds. A repeated cycle of guttation and drying can lead to injury by phytotoxic salts that creates infection points for necrotrophic pathogens. Dew deposition in the greenhouse favorable for downy mildews, rusts, and *Botrytis* blight is common on cool nights following warm, humid days. Regulating day and night atmospheres is important for disease control and also helps in reducing the total amount of chemicals sprayed (Hausbeck and Moorman 1996).

Botrytis cinerea on fuchsia was managed with specific climate and/or ventilation management; in particular, controlled dehumidification combined with a drop of temperature in the morning, and with a supplementary direct ventilation to decrease air humidity within the canopy, reduced stem blight to one third and sporulation on necrotic leaves to half of that in the corresponding control (Friedrich et al. 2005). Avoiding temperatures conducive to *Phytophthora infestans* and reducing moisture reduced late blight on petunia (Becktell et al. 2005b), with a reduction in fungicide use (Becktell et al. 2005a). On rose, *Peronospora sparsa* is now much less important than in the past, due to the possibility of better control of greenhouse RH as well as the availability of effective chemicals (Garibaldi and Gullino, unpublished results)..

Controlling RH and lighting reduced powdery mildew development and increased the vase life of cut flowers (Mortensen and Gislerød 2005). Temperatures higher than the optimum (21 °C) for the disease reduced powdery mildew severity in poinsettia (Byrne et al. 2000).

In the case of anthracnose of azalea, caused by *Colletotrichum acutatum*, keeping leaf wetness duration lower than 12 h when temperatures exceed 15 °C helps in managing the disease, which can be serious on some cultivars (Bertetti et al. 2009) (Fig. 1).

Beside controlling temperature and RH in order to reduce foliar diseases, it is necessary to ensure that water is lost from the crop by the normal physiological processes and that evaporation occurs from all other surfaces of the greenhouse. This is achieved through heating and ventilation but at a considerable cost. Ausburger and Powell (1986) provided data on the cost of humidity management.

All heat saving techniques should be adopted, although in some cases, for example, using thermal screens can create longer periods of high RH which can increase disease risk. In some cases, e.g., for gray mold management, ventilation alone helps in minimizing the risks of epidemics by permitting good air movement.

Root-zone or soil heating can be accomplished with either floor or bench heating systems. Such a long-standing grower practice to improve plant growth can have a positive effect in reducing the severity of certain pathogens. Bottom heat, a traditional means of avoiding root rots incited by species of *Phytophthora*, *Pythium* and *Rhizoctonia*, is enhanced in cutting and seedling trays with upward air movement between the young plants.

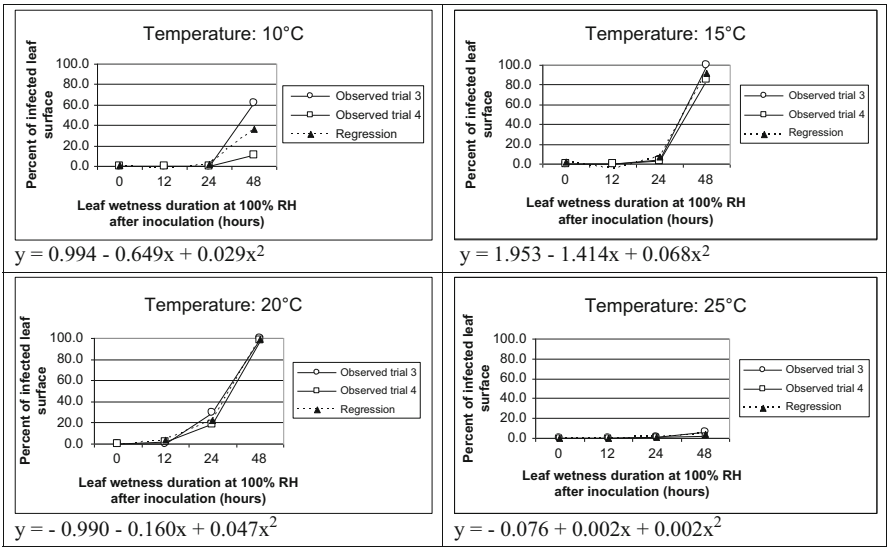


Fig. 1 Effect of leaf wetness duration on severity of anthracnose caused by *Colletotrichum acutatum* on *Rhododendron azalea* cv. Palestrina. For each temperature tested, data obtained showed similar variances with the Levene test. Thus, leaf wetness duration and severity can be related by using a single equation obtained by regression analysis as shown at the base of each graph. There were no symptoms on inoculated plants maintained at 5 °C and 30 °C (With permission from Bertetti et al. 2009)

Development of Fusarium root and crown rot incited by *Fusarium hostae* in container-grown hostas is affected by the type of wounding that occurs during propagation, container mix content, watering schedule, and temperature; peat or peat-bark container mix reduced disease incidence and severity, and disease was higher on plants growing in a dry-container mix and at moderate (20–25 °C) temperatures (Wang and Jeffers 2002).

The application of lime to the medium increases the pH and suppresses Fusarium wilts (Elmer 2012). This has been observed in the case of carnation (Engelhard 1979), chrysanthemum (Engelhard and Woltz 1973), and also on gerbera (Garibaldi, unpublished).

Altered greenhouse and bench design can improve air movement, thus reducing the risk of diseases. Through-the-bench air movement is, perhaps, the most neglected and simplest means of reducing seedling rots in tangled plant masses. A modest degree of spatial isolation, such as maintaining a distance of at least 10 m to fields where lilies were grown in the previous year, proved effective in reducing Botrytis leaf blight in lily. Such simple cultural practices were more effective than other cultural practices aimed at reducing survival of sclerotia (such as removal of crop residues and crop rotation) and was particularly effective when combined with the use of a relatively resistant cultivar (De Kraker et al. 2005). In the case of bulb crops, plant density can also consistently influence Fusarium wilt severity.

According to Price (1975a), since the different *formae speciales* of *Fusarium oxysporum* causing wilt on bulb crops have limited capability to grow in the soil, the spread of wilts to nearby bulbs occurs through the roots. Infection also occurs via wounds and bruises when the operations of digging, cleaning, and grading bulbs take place (Price 1975b), so that all practices which reduce contact among bulbs reduce wilt severity (Gullino 2012).

All factors reported above can be controlled to a greater or lesser extent according to how available sophisticated greenhouse structures and the equipment are.

Although light can affect spore germination for some pathogens, this has not been used often for the control of diseases. Shading is often used by growers when crops are wilting, but the effect of this practice is mostly on temperature rather than on light intensity. In the case of root rot of lavender, caused by *Phytophthora nicotianae* var. *parasitica* with a high level of rot incidence, a $\geq$50% reduction in rot incidence was achieved by growing plants under shade. Maintaining plants under shade provided sufficient root rot control for practical cultivation conditions (Minuto et al. 2001) (Table 1). The effect was linked to a reduced heating effect of black pots used by growers.

Proper management of RH, light intensity, and photoperiod has a significant impact on powdery mildew development on rhododendron. Particularly, RH was important both during spore germination and later during colony development, with disease development being adversely affected by reducing RH from 100 to 70 and 85%. Light intensity and photoperiod both had considerable effects on the induced resistance response of the host (Kenyon et al. 2002). Shading strongly increases the severity of powdery mildew, caused by *Golovinomyces cichoracearum* on aster (A. Garibaldi, unpublished).

4 Cultural Techniques, Including Soilless Media

Under greenhouses, growers have powerful management tools for production, many of which could significantly affect the epidemiology or severity of some diseases. The use of hydroponic, soilless cultures and artificial substrates have been gaining popularity for a number of years, particularly for high value crops such as rose, carnation, gerbera, basil, and lettuce. Soilless cultivation permits starting a production cycle completely free of pathogens. It also permits eradication of pathogens present in the recirculating nutrient solutions (Van Os et al. 2012). Although such cultural systems are ultimately intended to reduce production costs and maximize profits, precise environment and nutrition controls pushing plants to new limits of growth and productivity can generate chronic stress conditions, difficult to measure, but apparently conducive to diseases caused by pathogens such as *Penicillium* spp. or *Pythium* spp. The spread of diseases in soilless cultivation systems can be solved by adopting proper disinfection methods for the recirculating solution, such as slow sand filtration. Moreover, preventative methods to make plants less susceptible to diseases as well as the use of diagnostic tools permit a fully integrated approach in such systems (Van Os et al. 2012).

Table 1 Effectiveness of different growing conditions of lavender on root rot, caused by *Phytophthora nicotianae* var., expressed as percent of dead plants at two dates, June 26 and August 21, 1996 (With permission from Minuto et al. 2001)

Number and site[a]	Pot, volume (l)	Substrate[b]	Substrate inoculation	Fungicide treatment	Percentage of dead plants at June 26 and August 21	
1. S	Clay, 2	L	+	–	33.8	62.0
2. S	Clay, 2	HC	+	–	15.2	27.0
3. S	Clay, 2	HP	+	–	27.5	46.3
4. S	Clay, 2	L	+	+	26.3	46.4
5. S	Clay, 2	HC	+	+	19.3	31.3
6. S	Clay, 2	HP	+	+	14.7	23.3
7. S	Clay, 2	L	–	–	2.0	3.3
8. S	Clay, 2	HC	–	–	0.0	0.0
9. S	Clay, 2	HP	–	–	0.0	1.3
10. S	Clay, 2	L	–	+	3.8	3.8
11. S	Clay, 2	HC	–	+	2.0	2.0
12. S	Clay, 2	HP	–	+	1.9	1.9
13. S	Plastic, 2	L	+	–	22.1	55.2
14. S	Plastic, 2	HC	+	–	27.5	54.5
15. S	Plastic, 2	HP	+	–	27.4	46.3
16. S	Plastic, 2	L	+	+	18.3	39.7
17. S	Plastic, 2	HC	+	+	42.2	63.4
18. S	Plastic, 2	HP	+	+	29.1	44.2
19. S	Plastic, 2	L	–	–	7.4	18.9
20. S	Plastic, 2	HC	–	–	0.0	2.0
21. S	Plastic, 2	HP	–	–	1.3	3.4
22. S	Plastic, 2	L	–	+	7.2	12.2
23. S	Plastic, 2	HC	–	+	1.4	2.7
24. S	Plastic, 2	HP	–	+	0.7	0.7
25. O	Clay, 2	L	+	–	22.4	48.4
26. O	Clay, 2	HC	+	–	18.4	55.0
27. O	Clay, 2	HP	+	–	59.9	85.8
28. O	Clay, 2	L	+	+	44.3	83.7
29. O	Clay, 2	HC	+	+	74.1	90.8
30. O	Clay, 2	HP	+	+	65.4	93.3
31. O	Clay, 2	L	–	–	3.5	16.7
32. O	Clay, 2	HC	–	–	0.0	23.0
33. O	Clay, 2	HP	–	–	0.7	20.5
34. O	Clay, 2	L	–	+	9.1	31.7
35. O	Clay, 2	HC	–	+	1.4	50.1
36. O	Plastic, 2	HP	–	+	9.2	49.8
37. O	Plastic, 2	L	+	–	14.6	34.2

(*continued*)

Table 1 (continued)

Number and site[a]	Pot, volume (l)	Substrate[b]	Substrate inoculation	Fungicide treatment	Percentage of dead plants at June 26 and August 21	
38. O	Plastic, 2	HC	+	–	36.3	65.7
39. O	Plastic, 2	HP	+	–	39.4	64.4
40. O	Plastic, 2	L	+	+	16.3	58.0
41. O	Plastic, 2	HC	+	+	26.1	60.8
42. O	Plastic, 2	HP	+	+	55.0	85.7
43. O	Plastic, 2	L	–	–	5.5	75.2
44. O	Plastic, 2	HC	–	–	0.0	55.1
45. O	Plastic, 2	HP	–	–	0.0	71.8
46. O	Plastic, 2	L	–	+	5.1	63.4
47. O	Plastic, 2	HC	–	+	2.6	78.1
48. O	Plastic, 2	HP	–	+	0.0	72.5

[a]*S* shaded, *O* open field
[b]*L* light, with perlite, *HC* heavy, with clay, *HP* heavy, with pomix

Closed recirculating soilless systems represent an interesting environment for exploiting innovative disease management options. For instance, increasing the value of electrical conductivity of the nutrient solution, use of potassium silicate amendments, and the combination of both have proven effective against a number of foliar and soil-borne diseases, such as powdery mildews, downy mildews, several leaf spots, and Fusarium wilts (Gullino et al. 2015). Silicon provided at least partial control of powdery mildews of greenhouse crops as well as of brown patch (*Rhizoctonia solani*), dollar spot (*Sclerotinia homeocarpa*), and gray leaf spot (*Magnaporthe grisea*) on turfgrass (Bélanger et al. 1995; Brecht et al. 2004; Uriarte et al. 2004). When associated with a higher level of electrical conductivity, potassium silicate provides very interesting results. Under these conditions, in addition to the deposition of amorphous silica in the cell wall, there could be an increase in the production of lignin due to the presence of NaCl that could contribute to limiting the penetration of the pathogen within the plant cell, especially in the presence of foliar pathogens (Gullino et al. 2015). The effects of electrical conductivity to induce resistance against *Fusarium oxysporum* f. sp. *cyclaminis* on cyclamen have been documented by Elmer (2002); plants of cyclamen inoculated with the pathogen and treated with chloride salts showed a better growth and fewer wilt symptoms. The possibility of applying silicon as well as of manipulating the electrical conductivity to ornamental crops grown hydroponically is interesting because this type of cultivation is becoming more important and there is a scarcity of registered fungicides (Garibaldi et al. 2012).

Soilless systems also permit better manipulation of the microbiological conditions through microbial optimization with the application of microorganisms able to colonize the rooting system of plants grown under a strictly controlled environment.

Table 2 Effect of the application of selected antagonistic fungal strains into different closed soilless growing systems on the suppression of gerbera root rot incited by *Phytophthora cryptogea* (with permission from Garibaldi et al. 2003)

Treatment				Percentage of diseased plants at							
Soilless system	Slow sand filtration	Addition of antagonists	Inoculation of *P. cryptogea*	1/29		3/16		4/17		5/23	
Closed	Yes	No	Yes	0.0	a[a]	0.0	a	5.6	ab	11.1	b
Open	No	No	No	0.0	a	3.2	ab	5.3	ab	12.6	b
Closed	Yes	*Fusaria*	Yes	0.0	a	0.0	a	0.0	a	1.9	a
Closed	No	No	Yes	0.0	a	7.6	b	9.1	b	22.8	c
Closed	Yes	*Trichoderma*	Yes	0.0	a	0.0	a	0.0	a	2.1	a
Closed	UV	No	Yes	0.0	a	0.0	a	0.0	a	9.0	b

[a]In each column means followed by the same letter do not significantly differ according to Duncan's Multiple Range Test ($P = 0.05$)

Slow sand filtration combined with the application of different antagonistic strains of *Fusarium* spp. and *Trichoderma* spp. proved effective against *Phytophthora cryptogea* in gerbera (Tables 2, 3 and 4) (Garibaldi et al. 2003).

Suppressiveness of reused substrates adopted in soilless cultivation represents a very interesting feature, with many possible practical applications (Clematis et al. 2009). Antagonistic microorganisms, isolated from soilless systems and better adapted to this cultural system, have already been selected and proved effective against Fusarium wilt of rocket (Srinivasan et al. 2009), while strains of *Trichoderma* spp. isolated from soilless systems controlled *Pythium ultimum* on cucumber (Liu et al. 2009). This new development of biological control could also be interesting in the case of ornamentals.

Although interesting for field crops and also for vegetables, the widespread use of organic amendments for disease control is still not being practiced in the case of ornamental crops, due to many factors such as the type of amendments, the lack of standardization, the inconsistency in their efficacy, and the complexity of their use. A gap between good results observed in the laboratory and greenhouse compared to the few promising results in the field is still relevant today, as mechanisms of action are largely unknown and risk for ornamental production is too high compared to other disease control strategies.

5 Substrate and Compost Suppressiveness

Certain soil-borne diseases do not occur or are less severe in some soils that are referred to as suppressive. In many cases, suppressiveness had a microbiological origin that was destroyed by steaming. Some bacteria and/or fungi indigenous in these soils have been shown to be responsible for suppression of Fusarium wilt pathogens. The most efficient were identified as *Pseudomonas* and *Alcaligene*s in the USA and as *Fusarium* spp. in France and Italy. Antagonistic *Fusarium* spp., isolated from the rhizosphere of carnation

Table 3 Effect of the application of selected antagonistic fungal strains into different closed soilless growing systems on the production of flowers per healthy gerbera plant (With permission from Garibaldi et al. 2003)

Treatment				Number of flowers per healthy plant at											
Soilless system	Slow sand filtration	Addition of antagonists	Inoculation of *P. cryptogea*	1/23		2/5		3/1		3/30		4/23		5/10	
Closed	Yes	No	Yes	1.5	a[a]	2.5	a	4.2	a	6.9	a	15.9	ab	17.8	b
Open	No	No	No	1.5	a	2.4	a	4.2	a	7.5	a	12.9	ab	14.8	b
Closed	Yes	*Fusaria*	Yes	1.4	a	2.3	a	4.7	a	7.3	a	10.0	ab	11.8	c
Closed	No	No	Yes	1.2	a	2.1	a	3.6	a	5.7	a	8.4	b	9.4	c
Closed	Yes	*Trichoderma*	Yes	1.7	a	2.7	a	4.9	a	7.6	a	10.5	ab	11.9	c
Closed	UV	No	Yes	1.2	a	2.5	a	8.6	a	14.7	a	22.5	a	28.8	a

[a]In each column means followed by the same letter do not significantly differ according to Duncan's Multiple Range Test ($P = 0.05$)

Table 4 Effect of the application of selected antagonistic fungal strains into different closed soilless growing systems on the production of gerbera flowers/m^2 (From Garibaldi et al. 2003)

Treatment				Number of flowers/m^2 at											
Soilless system	Slow sand filtration	Addition of antagonists	Inoculation of *P. cryptogea*	1/10		2/5		3/1		3/30		4/23		5/10	
Closed	Yes	No	Yes	3.5	a[a]	12.1	a	19.1	A	27.9	a	35.1	a	40.7	a
Open	No	No	No	2.7	a	11.0	a	17.3	A	28.1	a	39.9	a	47.4	a
Closed	Yes	*Fusaria*	Yes	4.2	a	10.8	a	20.4	A	30.3	a	40.8	a	47.8	a
Closed	No	No	Yes	2.6	a	9.0	a	12.3	b	16.6	b	21.5	b	24.0	b
Closed	Yes	*Trichoderma*	Yes	3.3	a	12.3	a	19.5	a	28.2	a	37.4	a	42.0	a
Closed	UV	No	Yes	2.6	a	10.7	a	16.4	a	25.4	a	37.5	a	45.8	a

[a]In each column means followed by the same letter do not significantly differ according to Duncan's Multiple Range Test ($P = 0.05$)

plants grown in suppressive soils, did show high rhizosphere competence as compared with saprophytic nonantagonistic *Fusarium* spp. isolated from the same soils. When applied to soil and substrates they control Fusarium wilts on crops such as tomato, basil, carnation, cyclamen, and bulb crops (Gullino and Garibaldi 2007). Strains of *Trichoderma viride* showed good antagonistic activity against Fusarium wilts and have been used in chrysanthemum. Soils suppressive to *Rhizoctonia solani* are correlated with the presence of large amounts of *Trichoderma* spp. (Chet 1987).

Suppressiveness has also been found for substrates used in floriculture; in this case often microbiological and physical causes co-exist (Pugliese et al. 2015). Sphagnum peat mixes can naturally suppress diseases caused by soil-borne pathogens, but within a few weeks after potting they become conducive to diseases (Hoitink and Boehm 1999). Bacteria and fungi contribute to the suppression of root rots and wilts in peat mixes (Tahvonen 1993). Light peat decomposes in pots during production and the disease suppression effect is lost; amendment of decomposed peat mixes with microorganisms should consider the decomposition state (Hoitink and Locke 2012). Mixing light peat with more decomposed peat, at a 1:1 ratio, provides professional sphagnum potting mixes capable of providing, through suppressiveness, control of soil-borne pathogens for most greenhouse crops (Hoitink and Boehm 1999).

In the case of substrates, when hardwood bark (composted or not) is used, improved plant growth is generally observed, especially in potted plants. Suppressiveness and improved vigor of plants in such bark substrates result from the physical characteristics of bark composts and from higher levels of antagonists supported by these composts (Hoitink and Boehm 1999). Such peat mixes also support well the introduction of biocontrol agents or the addition of composts (Hoitink and Locke 2012).

During the past two decades various composted organic wastes have partially replaced peat in container media used for the production of ornamentals. Recycling of these wastes has been adopted for economic and production reasons. The cost of composts can be lower than that of peat. Production costs may also be decreased because some of the compost-amended media, and particularly those amended with composted bark, suppress major soil-borne plant pathogens, thus reducing plant losses and avoiding the use of fungicides (Hoitink and Boehm 1999). Additional research is showing that the use of composted materials can suppress soil-borne pathogens (Noble and Coventry 2005; Termorshuizen et al. 2006). To improve the consistency of disease control using composts, biological control agents (BCAs) have been added to compost amendments; composts can provide a food base for BCAs of soil-borne pathogens. Results indicate that mixtures of bacterial and fungal BCAs are more effective than single BCAs in inducing suppression of *Rhizoctonia* and *Pythium*. Compost amendments could, therefore, be used in flower nurseries as partial substitutes for peat substrates in the production of ornamentals with consequent advantages in terms of money and production. Growing mixes fortified with *Trichoderma hamatum* 382 controlled Botrytis blight on geranium (Olson and Benson 2006) and begonia (Horst et al. 2005). The same BCA added to potting mixes controlled Fusarium wilt of cyclamen (Hoitink and Locke 2012).

Although composts may not control diseases to a level that will replace the use of fungicides, their integration into current disease management practices may reduce the use of fungicides. This has been shown for instance in the case of suppression of dollar spot (*Sclerotinia homeocarpa*) on turfgrass (Boulter et al. 2002a, b). Increasing the use of compost as a potting substrate for plants would contribute to the recycling of wastes and to reducing the use of nonrenewable fertilizers. Compost is also interesting as a peat substitute, in particular after recent increasing concern about the environmental impact of peat extraction and the damage to peat lands natural habitats by the horticulture industry (Silva et al. 2007). The suppressive capacity of compost against soil-borne pathogens has been demonstrated in several studies leading to greater production efficiency and reduced nontarget effects (Garibaldi 1988; Hadar 2011; Hoitink and Boehm 1999; Hoitink and Fahy 1986; Noble and Coventry 2005). Compost was demonstrated to be the most suppressive material, with more than 50% of cases showing effective disease control, compared to other amendments such as crop residues and peat (Bonanomi et al. 2007). In container experiments using soil or sand, compost derived from green wastes and/or dairy cow manure generally showed a suppressive effect on *Pythium* species and *Rhizoctonia solani,* but the results did not necessarily translate into the field (Noble and Coventry 2005). Compost suppressiveness was dependent on the type of wastes used. Low rates of compost in growing media are generally indicated in order to avoid negative growth effects and phytotoxicity caused by high pH and electrical conductivity and other phytotoxic compounds present in composts (Sullivan and Miller 2001). However, it is generally necessary to include at least 20% v/v of compost in containers in order to observe a suppressive effect. Cases of increase of disease severity caused by composts used in containers have also been reported. A 50% spruce bark compost increased black root rot caused by *Thielaviopsis basicola* in poinsettias and Fusarium wilt of cyclamen, compared to a peat substrate (Krebs 1990). Highly saline composts were reported to enhance Pythium and Phytophthora diseases, while composts with higher nitrogen or ammonium content enhance Fusarium wilts (Hoitink et al. 2001). Among soil-borne pathogens, *Rhizoctonia solani* is considered to be the most difficult one to control with compost (Bonanomi et al. 2007; Scheuerell et al. 2005). Variability also depends on the pathosystem (Termorshuizen et al. 2006). Success or failure of compost for disease control depends on the nature of the raw materials from which the compost was prepared, on the composting process used, and on the maturity and quality of the compost (Termorshuizen et al. 2006). Control of soil-borne diseases with organic amendments must be viewed not as a stand-alone management approach but rather as part of a systems approach where several aspects of the impact of crop production practices on resident soil microbial communities are addressed. New approaches to monitor how microbial community structures in soil change as a result of organic amendment may lead to a better understanding of which changes in microbial communities are responsible for conferring the disease suppressive effects. This may eventually lead to improved and more reliable disease control resulting from organic amendment of soil, sand, or peat, both in container crops in greenhouses and in the field.

6 Concluding Remarks

Monitoring the environment and controlling it to keep plants disease-free has become a fundamental part of greenhouse management. Once the environmental parameters that influence pathogens and biocontrol organisms are understood, they can be more precisely regulated. Because of differences in climate, some of these strategies can be more easily and effectively applied in countries where greenhouses are more sophisticated. For instance, in Europe the structures most adapted to environmental control are located in the northern countries: under such conditions the adoption of proper techniques very often permits reduction of the severity of most foliar diseases.

Acknowledgments This chapter reports results of research funded under the European Union's Horizon 2020 Research and Innovation Programme under grant agreement no. 634179.

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Fungicides and Biocontrols for Management of Florists' Crops Diseases

7

Cristi L. Palmer and Ely Vea

Abstract

Florists' crops are prone to numerous diseases caused by fungi, oomycetes, bacteria, nematodes, viruses, viroids, phytoplasmas, and abiotic agents. Since consumers prefer and typically require high quality aesthetics for florists' crops, any marring due to pathogens will greatly reduce crop value if not render it nonsalable. Continued grower livelihood requires successful management of pathogens using all available components of integrated disease management. In this chapter, we will briefly describe the current development pathway for chemical and biological tools to manage diseases, guidelines for use including considerations for resistance management, and application technology. We will highlight current fungicides and biopesticides registered in the United States to ameliorate diseases of florists' crops.

Keywords

Fungicides • Bactericides • Nematicides • Chemicals • Biologicals • Rotations • Tank mixes • Disease management

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© Springer International Publishing AG 2018

R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_6

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1 Introduction

Environmental horticulture crops (EHCs) are one of the highest value-per-acre specialty crop industries in the United States (US) farm sector at $20.34 billion in US Gross Domestic Product (GDP) (Hodges et al. 2015). Broadly defined, EHCs are plants grown for placement into residential and commercial landscapes, interiorscapes, arboreta, parks, sports fields, and recreational areas. These plants are utilized and valued for numerous nonedible attributes, including removal of pollutants from air and water, CO_2 sequestration, reduction of urban heat islands and soil erosion, increased property values, pollinator services, recreational and relaxation activities, and aesthetic qualities. Florists' crops are a subset of environmental horticulture crops typically although not exclusively sold through florist shops. The types of plants include cut flowers, cut foliage, indoor houseplants such as tropical flowering or foliage plants, and special occasion flowering potted plants such as azaleas and poinsettias.

Florists' crops are prone to numerous diseases caused by fungi, oomycetes, bacteria, nematodes, viruses, viroids, phytoplasmas, and abiotic agents (Horst 2001). Since consumers prefer and typically require high quality aesthetics for florists' crops, any marring due to foliar pathogens will greatly reduce crop value if not render a crop nonsalable. Stem and root pathogens can reduce crop growth or cause wilting or plant death. Continued grower livelihood requires successful management of pathogens using all available components of integrated disease management (scouting, record keeping, disease/pathogen identification, prevention, sanitation, plant culture, biological and chemical tools) and applying them effectively based on understanding pathogen biology, genetics, host-interactions, and life cycles.

Over the last 100 years or so, understanding of common pathogens has increased substantially and has laid the foundation for studying newly arising pathogens. While much of the research in the beginning of the twentieth century focused on developing basic biological and epidemiological knowledge for fungal and bacterial plant pathogens, professors and graduate students also began more in depth investigations into methods and tools to mitigate diseases; some of the first graduate student stipends provided came from grower donations for nursery stock and tree diseases (Whetzel 1945).

Discovery of tools to manage disease-causing organisms prior to the twentieth century occurred often by happenstance. For example, the discovery of Bordeaux mixture as a fungicide came about by grape farmers applying the liquid to vines to

discourage thieves from taking ripened grapes. Sprayed vines had less downy mildew (Gianessi and Reigner 2005; Morton and Staub 2008). Early disease management tools tended to be inorganic molecules such as elemental sulfur dust and copper sulfate and were applied primarily to high value food crops (Russell 2005) rather than florists' crops or row crops grown over large acreages. From the beginning of the twentieth century through World War II, major changes occurred for managing plant diseases and pests. Growers shifted from mixing their own concoctions to purchasing products manufactured by companies (registrants) who registered the materials with the US government (Davis 2014). Over time, the focus on fungicides and bactericides for specialty food crops shifted with row crop agriculture driving the discovery and development of new active ingredients since the mid-twentieth century. A notable exception was the active ingredient dodemorph that was commercialized and introduced in 1965 specifically for controlling rose powdery mildew (Russell 2005).

The sections below cover contemporary processes and perspectives for discovering new active ingredients, developing them into viable products, and registering them within the United States. Since plant pathology and the tools to manage florists' crop diseases continually evolve, references to historical perspectives are included to show changing viewpoints over time.

2 Discovery, Development, and Registration

2.1 Active Ingredient Discovery and Early Development

Discovery of new active ingredients whether chemical, biological, or genetic focuses on screening prospective tools for their activity against certain key model pathogens. Ideas for prospective tools arise from many different areas. For example, new chemicals can arise from studying biopesticide-pathogen interactions or other microbial interspecies interactions and isolating the molecule(s) associated with pathogen inhibition or mortality (Erjavec et al. 2012). The strobilurin class of fungicides arose from this strategy with the subsequent design of molecules that possessed a similar structure to the original molecule but with slightly different properties (Balba 2007; Bartlett et al. 2002). Chemicals may also be discovered by understanding key biological processes within the pathogen that are distinct from pathways in mammals or plants and designing molecules that interfere with that process. It is estimated that more than 150,000 synthesized molecules are screened for a single new registered active ingredient (Phillips McDougall 2016). New biologically based active ingredients are discovered in similar ways to new chemicals. These discoveries can be based on verbal histories or lore or on keen observations of natural situations. New biopesticides are also revealed by isolating and identifying endophytes from plants and soils that inhibit pathogen growth in lab studies.

Once a new active ingredient is discovered, a key step before continuing on the development pathway is determining whether it can economically be manufactured. For a new biological active ingredient, it might involve developing a suitable large-scale

fermentation process. For microbial or plant extracts, this might be whether the organism can be grown quickly, efficiently, and still consistently produce a quality extract containing the active moiety. For chemistry, this could involve determining whether synthesis of the molecule can be scaled up economically.

At the same time as determining production methodologies, a new active ingredient will be screened on a broader array of pathogens to learn the breadth and limitations of its effectiveness. These studies initially occur in the laboratory, then in the greenhouse, and subsequently in small plots in the field with well-studied pathogen-host systems.

These early stages of development may be accompanied by a battery of studies to examine stability and degradation of the active ingredient under different conditions (moisture, light, temperature) and its degradation pathways (photolysis, hydrolysis, microbial, plant metabolism, mammalian metabolism, secondary metabolites, etc.). Further characterization of its target site and mode of action occur as well as deposition and movement within plant tissues. In the United States, chemically based active ingredients tend to have broader requirements for toxicology and ecotoxicology studies than biopesticides (US EPA 2016c).

In addition to characterizing an active ingredient's physicochemical characteristics, beginning steps occur to create a formulation to deliver it appropriately, at the very least for the small plot field tests. Further refinements occur through late stage development. Considerations may include buffering at a certain pH to maintain optimal stability, including the best balance of surfactants to keep the active solubilized or in solution so it does not precipitate out, and delivering a formulation that is easy to use and does not have negative characteristics like the propensity to clog sprayer nozzles in the case of liquid products or like undue amounts of dust as granular based products are dispensed and applied.

Early development is also the period when initial acute toxicology studies are conducted for mammalian oral, inhalation, and dermal toxicity. If there are any concerns raised during early development, additional research activities for that active ingredient are dropped and it is no longer considered a viable candidate.

2.2 Product Development: Efficacy and Crop Safety

When an active ingredient passes early screening, large scale field trials commence examining performance under different climatic conditions and weather patterns. Researchers collect data on a wider range of pathogens and crops. Most of these research activities occur on food and fiber crops. However, during the late stages of product development when the registrant is confident of commercialization, research activities are expanded to include environmental horticulture crops and their diseases.

In studying the efficacy of a new product for a specific pathogen, a series of different rates (rate range) is used to find the optimal concentration using different application methodologies. For active ingredients with limited plant mobility, foliar applications will typically be screened for foliar and floral diseases only.

Applications for trunk or stem diseases may also include painting. For materials with plant systemic activity, soil applications may be screened in addition to foliar to determine efficacy level and length of residual activity. The length of residual control is assessed by stopping applications and then continuing to rate efficacy until it is clear the treatment is no longer effective. Experiments are established to screen for preventative activity (what happens when it is applied prior to being challenged with a pathogen) or curative activity (what happens when it is applied as the pathogen is first causing infection or soon after).

In addition to examining a product by itself, it may be screened as part of a programmatic approach. It will be tested in rotation or as a tank mix with other active ingredients. This will determine whether the actives together provide the same, enhanced, or diminished activity. Tank mix compatibility may be included in the product label.

Products are also screened for whether they impact plant growth and development. The triazole fungicides, for example, are known to have growth regulator effects on certain crops. Assessing growth habit, chlorosis, necrosis, flower bud abortion, floral color and number, or other unusual occurrences after application on multiple crops contributes to how registrants write product labels.

Continued screening of new late-stage development active ingredients and currently registered products ensures that end users have the most up-to-date information regarding options for managing diseases.

2.3 Product Development: Toxicology

Simultaneously to large scale field testing, in depth toxicology and ecotoxicology studies begin. Acute testing occurs for the active ingredient formulated as a manufacturing use product (only used to make end use formulations) and as proposed end use formulations. These studies are designed to determine the concentration at which 50% of the tested population will have a negative reaction after a single exposure. The "six pack" is acute oral toxicity, acute dermal toxicity, acute inhalation toxicity, primary eye irritation, primary dermal irritation, and dermal sensitization (US EPA 2016b). The results from these six studies will classify the formulated product label into caution, warning, or danger signal words. Information on acute neurotoxicity is required, and delayed neurotoxicity data may be required.

Subchronic testing examines concentrations below those found during the acute testing to screen for impacts with repeated ingestion or contact with the active ingredient. These studies include 90 day oral, 21/28 or 90 day dermal, and 90 day neurotoxicity (US EPA 2016c). Depending on the outcomes of the acute studies, 90 day inhalation and 28 day delayed neurotoxicity may be required. Chronic testing includes long-term feeding studies to examine lethality and carcinogenicity. Developmental toxicity, reproduction/fertility, and mutagenicity testing are required to examine potential sublethal impacts. Mammalian metabolism and immunotoxicity studies further characterize active ingredients for how they break down and are excreted and whether they impact the immune system. Toxicity to birds, fish, aquatic

invertebrates, and honey bees is examined as is phytotoxicity for terrestrial and aquatic plants.

Worker safety is paramount. It is required to determine whether applicators will be exposed to the product during application and whether workers who come in contact with the plants after application will be exposed. The types of studies include dermal and inhalation exposure, dislodgeable foliar residues, soil residue dissipation, and how the product will be used. For example, if the product will be formulated as a time-release aerosol which can be set to release after the applicator leaves the greenhouse range, the applicator will have little exposure.

In addition to the degradation studies listed above in Sect. 2.1, additional environmental fate studies are conducted to determine the likelihood an active ingredient will move out of the treated area and/or persist. These include leaching through and adsorption/desorption in soil, terrestrial dissipation studies, and monitoring ground water if an active ingredient and/or its metabolites may be likely to leach into ground water. For example, a highly water soluble active ingredient with little capacity to bond to organic matter in the soil might require ground water monitoring.

Please note that the above are highlights of US EPA toxicology and ecotoxicology studies, and anyone considering registering a new biological or chemical active ingredient should consult with the US EPA or other relevant national agency for full requirements and tiered assessment guidelines.

2.4 Product Registration in the United States

Registration of products in the United States today is very different from what is was at the turn of the twentieth century. Up through the early 1900s, products were sold based on the reputation of the manufacturer or were created by the end user from recipes without any federal or state system to vet and register them. Toxicology as a scientific discipline was in its infancy and focused solely on acute mortality rather than potentially subtle chronic impacts (Davis 2014). The primary factors for a successful product at that time were whether it was efficacious and whether the level of plant injury was acceptable, and these were solely determined by the marketplace.

Throughout the twentieth century, toxicology and risk assessment developed and expanded to address not only acute and chronic human concerns but also impacts on the environment. Since initial experiments into the toxicology of DDT during the 1930s and 1940s which focused almost exclusively on acute human mortality, toxicology and assessing risk from the use of tools to manage pests, diseases and weeds has evolved (Davis 2014) to include environmental and product chemistry; avian, aquatic, and mammalian toxicology; endocrine disruption; and other nontarget impacts as described previously. In the United States, these studies must be conducted under Good Laboratory Practices (GLP) and are reviewed by the US EPA and collaborating regulatory bodies such as California Department of Pesticide Regulation and international agencies when products are considered for joint review

Table 1 Increasing cost to discover, develop, and register new chemical tools (1980–2014)

Year	Cost of discovery and development (US$ M)	2014 value (US$ M)	Expenditure increase (US$ M)
1980	$23	$66	$220
1995	$152	$236	$50
2000	$184	$253	$33
2008	$256	$281	$5
2014	$286	$286	–

(Phillips McDougall 2016); http://www.usinflationcalculator.com/

and registration by multiple countries at the same time. Under joint review, the regulatory bodies divide the submitted registration package for simultaneous, collaborative evaluation. US EPA carefully considers the hazards and risks based on the submitted data package (US EPA 2016a). If US EPA finds the active ingredient and its formulated product(s) meet a predetermined level of concern, it approves the registration. If the product does not meet the level of concern, US EPA will recommend mitigation language be added to the product label (such as additional protective gear for applicators or a longer re-entry interval) or fewer uses for the final label (such as only greenhouse and not outdoor applications) prior to approving the registration.

From 1980 to 2014, the cost to discover, develop, and register a single new chemical crop protection tool increased from US $23 million to US $286 million (Phillips McDougall 2016). This is amortized across all the molecules screened. When converting this to US dollar value in 2014, expenditures for a single active ingredient have increased to US $220 million since the 1980s (Table 1), an indication of the enhanced testing requirements globally to ensure product safety and performance with the heightened awareness of potential subacute, chronic impacts of these tools on nontarget organisms.

3 Practical Considerations

Once a product is registered and commercialized, growers may revise disease control programs to include the new active ingredient. The characteristics of the new product will determine how best it can be incorporated. A product that has curative activity (can stop disease progression) can be combined with scouting for application as soon as disease symptoms are observed. A product with preventive activity (must be present prior to the pathogen) will be applied when environmental conditions are suitable for disease development without obvious plant symptoms. Reading the product label, technical literature from registrants and research bulletins from extension personnel will provide guidance on use. This section covers some practical considerations on product labels, application technology, and resistance management.

3.1 Labels and Use Directions

Based on all the research activities over a 10–12 year span, registrants write product labels. These labels contain vital information to maintain worker safety (applicators and handlers), safeguard consumers (preharvest intervals for any edible crops on the label and re-entry intervals for anyone touching the plants without protective gear), prevent unintended impacts on the environment, and provide optimal efficacy (US EPA 2016b). The label does have many parts with the worker safety and environmental considerations at the beginning. The use directions focus on how to apply the product. Rotational or tank mix recommendations may be included, but of particular note are the maximum number of applications prior to rotating to a different mode of action. The FRAC code is prominently displayed on most fungicide labels to aid growers in selecting tools of different modes of action.

Application methods can include foliar sprays, soil drench, sprench (a heavy foliar spray with the intent to apply product to the soil also), soil injection, and other potential methods such as irrigation line injection, ebb and flow benches, dip application, or painting. Routine calibration of equipment and checking delivery are critical for ensuring the intended amount of product is being applied. Calibration of the applicator is just as important. An applicator spraying foliage who moves slower will potentially deliver more active ingredient per area than an applicator with a faster pace, assuming the same volume output from the application equipment (Wilen 2011; Anon 2016).

3.2 Application Technology

Application equipment is designed based on the product formulation and targeted plant part (foliage, trunk/stem, roots). Products may be formulated as liquids (soluble concentrates, flowable/suspension concentrates, emulsifiable concentrates, microemulsions, oil dispersions, microencapsulated particles) or as solids (wettable powders, water dispersible granulars, granulars). Additional formulation types and the international coding system were published by CropLife International (Anon. 2008). With the exception of granulars, aerosols, and other ready-to-use preparations, formulations are designed to be diluted with water and applied using sprayers, liquid fertilizer lines, and soil or tree injection equipment. Aerosols typically are self-contained application equipment that may deliver the active ingredient in a carrier other than water.

3.2.1 Sprayers

There are various types of sprayers available but they fall into three groups: high volume hydraulic sprayer, targeted low-volume sprayers, and ultra-low volume sprayers.

High volume sprayers deliver volumes up to and beyond 1870 L per hectare (200 gal per acre), typically delivering large droplets, but droplet size can vary considerably with nozzles and pressure (70–200 μm). Hydraulic sprayers build

pressure through a mechanical or hand-operated pump and can range from low to high pressure. Nozzles create different droplet sizes and spray patterns. Most product labels are written based on volume levels needed to achieve good coverage with high volume sprayers. Plugs and young plants may need 93–234 L per hectare (10–25 gal per acre), while larger plants in production might need up to 1870 L per hectare (200 gal per acre). Mature shrubs and trees in the landscape may require 3741 L per hectare (400 gal per acre) or more for adequate coverage, but it is exceedingly rare for an entire hectare (or acre) to be planted with trees and shrubs requiring simultaneous treatment for pathogen management. While high volume sprayers can be mechanized and be used to treat large areas, some can be used for smaller areas or spot sprays.

Targeted low-volume sprayers deliver the same active ingredient load per area but in higher concentrations with smaller droplets and less volume. These sprayers may deliver 47–421 L per hectare (5–45 gal per acre). With appropriate nozzles hydraulic sprayers can be used. Electrostatic sprayers fit into this category. Electrostatic sprayers apply a charge to the solution that improves coverage by causing the spray droplets and plants to attract each other. The droplet size for electrostatic sprayers is between 15 and 25 μm.

Ultra-low volume sprayers deliver product in very little water. The droplet size is typically 50–60 μm. Foggers, coldfoggers, and aerosols fit in this category.

3.2.2 Granular Applicators

There are two common types of granular product spreaders: rotary and drop. The rotary spreader has a spinning disk (manual or mechanical) which distributes granules to the front and sides. In the drop spreader, granules drop through open holes in the bottom of the hopper. These types of spreaders tend to be used for outdoor applications for plants grown in the field. For indoor applications, granular products may be incorporated into the soil mix prior to planting, or, for certain spot applications, granulars may be applied using a shaker can.

3.3 Resistance Management

The Fungicide Resistance Action Committee (FRAC) classifies chemically and biologically active ingredients by mode of action (www.frac.info). Each mode of action group member has the same site of activity and is assigned a FRAC code which appears prominently on the US label. If a pathogen population develops resistance to one member, all members of that group will not be as effective on the same population. To delay development of resistance, it is recommended that applicators rotate to different modes of action. Labels for most products containing active ingredients with single site modes of action require rotation to a different FRAC group after two or three consecutive applications. Tank mixing two or more active ingredients from different FRAC groups is another strategy to preserve effective control over time. Knowing which actives are effective for different fungal stages leads to combinations that can interfere with multiple stages such as

preventing cell membrane formation (FRAC 3) combined with reduced mitochondrial respiration (FRAC 11). Often registrants will offer premix formulations containing two actives which target different sites. Even then, most labels will require rotation to other FRAC groups after multiple consecutive applications of single site mode of action materials.

4 Products Currently Registered in the United States

Products in the marketplace continually shift as new tools are discovered and older products discontinued. The complement of choices may vary depending on marketplace and prevalence of disease. In the United States during 2016, 22 different mode of action classes are registered for managing pathogens on environmental horticulture plants. These products are classified by the types of pathogens they manage. For example, fungicides manage fungi (ascomycetes, basidiomycetes) and water molds (oomycetes) and bactericides manage bacteria. However, some tools, particularly broad spectrum active ingredients with general biocide activity, may manage diseases across pathogen types.

4.1 Fungicides

Fungi and oomycetes constitute the largest number of plant pathogens that cause more than 85% of plant diseases and are responsible for most diseases of environmental horticulture plants. They damage plants by killing cells and/or causing plant stress. They are typically multicellular organisms that produce tiny thread-like filaments called hyphae. Most pathogenic fungi produce spores which serve to reproduce and disseminate them. Management of diseases caused by fungi and oomycetes commonly requires use of fungicides that are discussed below.

4.1.1 Inorganics

Sulfur was the first fungicide known (Williams and Cooper 2004; Horsfall 1956) and was the earliest fungicide registered in the United States for application to florists' crops (Table 2). It is still used today by commercial growers and homeowners.

Sulfur belongs to a group of contact fungicides that are considered protectants or preventive fungicides. They remain on the plant surface and do not penetrate into the plant. They inhibit the fungi on the plant surface, usually spore germination, so the fungus will not be able to infect the plant. Contact fungicides affect multiple sites in fungi so resistance to these is not a major concern. They are used with resistance-prone systemic active ingredients as a part of fungicide resistance programs if multiple applications of fungicides are needed for season long disease control.

Copper-based compounds. See information under Sect. 4.2.

Bicarbonates – Several potassium bicarbonate products have been registered to control powdery mildew and other fungal diseases of environmental horticulture

Table 2 Active ingredients currently registered in the United States for managing diseases of environmental horticulture crops[a]

Active ingredient[b]	Product trade name(s)[c]	Product type[d]	Year of first US registration for environmental horticulture crops[e]	FRAC code[f]	MOA code	Chemical group
Ametoctradin + dimethomorph	Orvego	Designed chemical	2012	45 + 40	C8: QoI + F5: CAA fungicides	Triazolo-pyrimidylamine + cinnamic acid amide
Azoxystrobin	Heritage	Designed chemical	1998	11	C3: QoI	Methoxy-acrylates
Azoxystrobin + benzovindiflupyr	Mural	Designed chemical	2015	11 + 7	C3: QoI +	Methoxy-acrylates +
Azoxystrobin + difenoconazole	Alibi Flora	Designed chemical	2015[i]	11 + 3	C3: QoI + G1: DMI (SBI class 1)	Methoxy-acrylates + triazole
Bacillus amyloliquifaciens Strain D747 (aka B. subtilis CX-9032)	Double Nickel, Triathlon BA	Microbial	2011	44	F6: Microbial	Microbial disrupters of pathogen cell membranes
Bacillus subtilis strain QST 713	Cease, Rhapsody, Serenade Optimum, etc.	Microbial	2014	44	F6: Microbial	Microbial disrupters of pathogen cell membranes
Bacillus subtilis var *amyloliquefaciens* strain FZB24	Taegro	Microbial	2000	44	F6: Microbial	Microbial disrupters of pathogen cell membranes

(*continued*)

Table 2 (continued)

Active ingredient[b]	Product trade name(s)[c]	Product type[d]	Year of first US registration for environmental horticulture crops[e]	FRAC code[f]	MOA code	Chemical group
Bacillus subtilis strain IAB/BSO3	Mildore	Microbial	2015	44	F6: Microbial	Microbial disrupters of pathogen cell membranes
Bacillus subtilis GB03	Companion	Microbial	2008	44	F6: Microbial	Microbial disrupters of pathogen cell membranes
Captan	Captan	Designed chemical	1973	M4	Multi-site: phthalimide	Phthalimide
Chlorothalonil	Concorde, Daconil, Echo, etc.	Designed chemical	1974 (conifers) 1985	M5	Multi-site: chloronitrile (phthalonitrile)	Chloronitrile (phthalonitrile)
Chlorothalonil + zinc	Daconil Zn	Designed + Natural Chemical	1992 (conifers) 1997	M5	Multi-site: chloronitrile (phthalonitrile) +	Chloronitrile (phthalonitrile) +
Cinnamaldehyde	Cinnacure	Natural chemical	1999	NC		
Coniothyrium minitans	Contans	Microbial	2002			
Copper diammonia diacetate complets	Copper-Count-N	Natural chemical	1997	M1	Multi-site	Inorganic
Copper hydroxide	Champ, Champion, CuPRO, Kentan, Kocide, etc.	Natural c-hemical	1983	M1	Multi-site	Inorganic

Copper hydroxide + copper oxychloride	Badge SC, Badge *X2*	Natural chemical	2005	M1	Multi-site	Inorganic
Copper octanoate	Camelot O	Natural chemical	1997	M1	Multi-site	Inorganic
Copper sulfate	Basicop, Cuprofix	Natural chemical	1975[g]	M1	Multi-site	Inorganic
Copper sulfate pentahydrate	Phyton	Natural chemical	1991	M1	Multi-site	Inorganic
Cuprous oxide	Nordox	Natural chemical	2000	M1	Multi-site	Inorganic
Cyazofamid	Segway O	Designed chemical	2007	21	C4: QiI	Methoxy-acrylate
Cyprodinil + fludioxonil	Palladium	Designed chemical	2010	9 + 12	D1: AP fungicides + E2: PP fungicides	Aniline-pyrimidine + phenylpyrrole
Dicloran	Botran	Designed chemical	1997	14	F3: AH fungicides	Aromatic hydrocarbons
Didecyl dimethyl ammonium chloride	Kleengrow	Natural chemical	2007	NC		
Dimethomorph	Stature	Designed chemical	2002	40	F5: CAA fungicides	Cinnamic acid amimde
Etridiazole	Terrazole, Truban	Designed chemical	1972	14	F3: AH fungicides	1,2,4 thiadizole
Extract of *Reynoutria sachalinensis*	Regalia	Plant extract	2000	P5	P5: plant extract	Complex mixture, ethanol extract
Fenamidone	Fenstop	Designed chemical	2004	11	C3: QoI	Imidazolinone
Fenarimol	Rubigan	Designed Chemical	1990[g]	3	G1: DMI (SBI class 1)	Pyrimidine
Fenhexamid	Decree	Designed chemical	1999	17	G3: hydroxyanilide (SBI class III)	Hydroxyanilide
Fludioxonil	Medallion	Designed chemical	1997	12	E2: PP fungicides	Phenylpyrrole

(*continued*)

Table 2 (continued)

Active ingredient[b]	Product trade name(s)[c]	Product type[d]	Year of first US registration for environmental horticulture crops[e]	FRAC code[f]	MOA code	Chemical group
Fluopicolide	Adorn	Designed chemical	2008	43	B5: benzamide	Pyridinylmethyl-benzamide
Fluoxastrobin	Disarm, Fame	Designed chemical	2007	11	C3 QoI	Dihydro-dioxazine
Flutolanil	Contrast, Prostar	Designed chemical	1999	7	C2: SDHI	Phenyl-benzamide
Fosetyl Al	Aliette	Designed chemical	1983	33	Unknown: phosphonate	Ethyl phosphonate
Gliocladum catelunatum strain J446	Prestop	Microbial	2006	NC		
Gliocladium virens GL21	SoilGard	Microbial	1990	NC		
Hydrogen dioxide	ZeroTol	Natural chemical	1998[i]	NC		
Hydrogen dioxide + peroxyacetic acid	ZeroTol 2.0	Natural chemical	2014	NC		
Iprodione	Chipco 26019, etc.	Designed chemical	1986	2	E3: dicarboximide	Dicarboximide
Mancozeb	Dithane, Fore Protect, Pentathlon, etc.	Designed chemical	1965[g]	M3	Multi-site: dithiocarbamates	Dithiocarbamates and relatives
Mandipropamid	Micora	Designed chemical	2011	40	F5: CAA fungicides	Mandelic acid amide
Mefenoxam (metalaxyl-M)	Subdue, Fenox	Designed chemical	1996	4	A1 Phenyl amide	Acyl alanine
Metconazole	Tourney	Designed chemical	2007	3	G1: DMI (SBI class I)	Triazole

Mineral oil	JMS Stylet Oil, SuffOil-X, etc.	Natural chemical	1992	NC		
Myclobutanil	Eagle, etc.	Designed chemical	2003	3	G1: DMI (SBI class I)	Triazole
Hydrophobic neem oil extract	Triact 70, Trilogy, etc.	Plant extract	1997	18B		
Oxathiapiprolin	Segovis	Designed chemical	2015	U15		
Paraffinic oil	Sunspray UltraFine oil, Ultra Pure Oil, etc.	Natural chemical	1989	NC		
PCNB	Terraclor	Designed chemical	1971[h]	14		
Phosphorus acid salts	Agrifos, Alude, Fosphite, Magellan, OxiPhos, Phos Fungicide, Rampart, Vital, etc.	Natural chemical	2002	33	Unknown: phosphonate *(literature indicates induced resistance and direct toxicity)*	Phosphonate
Piperalin	Pipron	Designed chemical	1972	5	G2: amines (morpholines) SBI Class 2	Piperidine
Polyoxin D zinc salt	Affirm, Veranda	Microbial extract	2005	19	H4: Polyoxins	Polyoxins
Potassium bicarbonate	Armicarb, Kaligreen, Milstop	Natural chemical	1997	NC		
Potassium salts of fatty acids	M-Pede, Ringer Aphid Mite Attack, Safer Soap, etc.	Natural chemical	1980	NC		

(*continued*)

Table 2 (continued)

Active ingredient[b]	Product trade name(s)[c]	Product type[d]	Year of first US registration for environmental horticulture crops[e]	FRAC code[f]	MOA code	Chemical group
Potassium silicate	Sil-Matrix, Carbom Defense	Natural chemical	2006	NC		
Propamocarb	Banol, Proplant	Designed chemical	1987	28	F4: carbamates	Carbamates
Propiconazole	Banner Maxx, ProPensity	Designed chemical	1995 (elm, oak) 2005	3	G1: DMI (SBI class I)	Imidazole
Pyraclostrobin	Insignia, Empress Intrinsic	Designed chemical	2006	11	C3: QoI	Methoxy-carbamate
Pyraclostrobin + boscalid	Pageant Intrinsic	Designed chemical	2008	11 + 7	C3: QoI + C2: SDHI	Methoxy-carbamate + pyridine-carboxamide
Pyraclostrobin + fluxapyroxad	Orkestra Intrinsic	Designed chemical	2016	11 + 7	C3: QoI + C2: SDHI	Methoxy-carbamate + pyrazole-carboxamide
Streptomyces griseoviridis K61	Mycostop	Microbial	1993	NC		
Streptomyces lydicus	Actinovate	Microbial	2004	NC		
Streptomycin sulfate	Agrimycin, Firewall	Microbial extract	1961	25	D4: glucopyranosyl antibiotic	Glucopyranosyl antibiotic

Sulfur	Microthiol Disperss, Bonide Micronized Sulfur, Miller's Garden Sulphur, etc.	Natural chemical	1966[g] (1948 was listed on EPA for first label, but 1966 was earliest scanned posted label)	M2	Multi-site	Inorganic
Tebuconazole	Torque, Tebuconazole SC T&O Fungicide	Designed chemical	2002	3	G1: DMI (SBI class I)	Triazole
Thiophanate methyl	3336, OHP 6672, etc.	Designed chemical	1983	1	B1: MBC	Thiophanate
Triadimefon	Bayleton, Strike	Designed chemical	1990	3	G1: DMI (SBI class I)	Triazole
Trichoderma asperelloides strain JM41R	Tricho Plus Biofungicide	Microbial	2015	NC		
Trichoderma hamatum strain 382	Incept	Microbial	2010	NC		
Trichoderma harzianum	PlantShield, RootShield	Microbial	1995	NC		
Trichoderma harzianum + Trichoderma virens	RootShield Plus	Microbial	2012	NC		
Trifloxystrobin	Compass O	Designed chemical	1999	11	C3: QoI	Oximino-acetate
Triflumizole	Terraguard	Designed chemical	1991	3	G1: DMI (SBI class I)	Imidazole

(*continued*)

Table 2 (continued)

Active ingredient[b]	Product trade name(s)[c]	Product type[d]	Year of first US registration for environmental horticulture crops[e]	FRAC code[f]	MOA code	Chemical group
Triticonazole	Trinity	Designed chemical	2013	3	G1: DMI (SBI class I)	Triazole
Ziram	Ziram	Designed chemical	1981[g]	M3	Multi-site: dithiocarbamates and relatives	Dithiocarbamates and relatives

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[b]Mixtures of two or more active ingredients are only listed if one of the active ingredients is not available in the United States as a stand-alone product
[c]Trade names are listed for products registered in the United States for use on environmental horticulture crops as of 2016. While this information is included to aid end users, the names may shift over time and multiple formulations may exist. Additional trade names may be available
[d]These categories represent generalized groupings of active ingredients. Natural chemical refers to those active ingredients existing in nature which can be formulated into products with minimal human intervention. Designed chemical refers to active ingredients discovered to be active through high-throughput screening or chemically transformed natural molecules. The latter are known as biologics in the pharmaceutical industry and are represented by the strobilurin class of chemistry in agriculture
[e]This is the first year the active ingredient was registered with US Environmental Protection Agency for environmental horticulture uses. The active ingredient may have been registered for edible crop or other uses in prior years
[f]These are the mode of action group codes assigned by the Fungicide Resistance Action Committee. Full descriptions and listings can be found at www.frac.info
[g]Earliest year legible label available with environmental horticulture uses at the US EPA Pesticide Product Label System (http://ofmpub.epa.gov/apex/pesticides/f?p=PPLS:1)
[h]No longer registered
[i]Currently registered but not available commercially

plants. They mainly inhibit fungal mycelial development. Its mode of action is linked with osmotic pressure, pH, and specific bicarbonate/carbonate ion effects. Like sulfur, the bicarbonates belong to a group of contact fungicides that are considered protectants or preventive fungicides.

Phosphonic acid and derivatives – This broad category of phosphonate fungicides includes several products that are registered to control mainly oomycete diseases including seedling blights, damping-off, root rots, foliar blights, and downy mildews.

Phosphonate fungicides have systemic activity and application methods including foliar spray, soil drench, soil incorporation, basal bark application, bare root dip, etc. They are recommended for application prior to disease development for optimum control. Their mode of action has not been established, but both a direct effect by inhibiting the oxidative phosphorylation or other metabolic processes in the fungi and an indirect effect by stimulating the plant's natural defense response against pathogen attack have been suggested. Risk for phosphonate resistance is considered lower than single-site activity fungicides.

Silicon – Silicon-based fungicides like potassium silicate are used for preventative control of fungal diseases of environmental horticulture plants. Silicon belongs to a group of contact fungicides that are considered protectants or preventive fungicides. Silica accumulation in cell walls is believed to act as a physical barrier to pathogen infection resulting in plant disease resistance.

4.1.2 Dithiocarbamates

The dithiocarbamates (thiram, zineb, maneb, mancozeb, etc.), first introduced in 1942 with thiram, are the most widely used group of organic fungicides. This group represented a major improvement over the previously used inorganic fungicides in that they were more active, less phytotoxic, and easier to prepare by the user. Of these active ingredients, the most widely used is mancozeb, a broad spectrum protectant fungicide which was first registered on environmental horticulture plants in 1965. The dithiocarbamates are considered contact, multi-site activity fungicides, inhibiting -SH groups in amino acids, proteins, and enzymes. Thus, the risk for resistance is lower than single-site activity fungicides.

4.1.3 Substituted Aromatics

This group of preventative contact fungicides includes three active ingredients that are registered on environmental horticulture plants. Chlorothalonil is one of the most widely used broad spectrum fungicide in turf and ornamentals. The other two, etridiazole and pentachloronitrobenzene or PCNB, control mainly soil-borne diseases caused by several pathogens that include *Phytophthora, Pythium, Rhizoctonia*, etc. These fungicides are considered multi-site activity fungicides, inhibiting amino acids, proteins, and enzymes by combining with amino and thiol groups. Thus, the risk for resistance is considered lower than single-site activity fungicides.

4.1.4 Pthalimides

This group of fungicides (captan, captafol, folpet) was introduced for plant disease control in the 1950s and early 1960s. Captan is currently registered for use on environmental horticulture plants. The pthalimides are considered contact, multi-site activity fungicides, inhibiting a host of processes in the cell, including enzyme function, amino acid synthesis, cellular metabolism, etc. Thus, the risk for resistance is considered lower than single-site activity fungicides.

4.1.5 Benzimidazoles

This group of fungicides was introduced for plant disease control in the 1960s and early 1970s. This group includes benomyl, carbendazim, thiabendazole, thiophanate-methyl, etc. Thiophanate-methyl is currently registered on environmental horticulture plants. The benzimidazoles are contact fungicides that possess unique properties not seen before in the protectant fungicides. These included low use rates, broad spectrum, and systemicity with post-infection action that allowed for extended spray interval. They interfere with fungal mitosis (normal cell division), so they affect the growth of fungi. They affect fungi at a very specific site in the metabolic pathway, inhibiting DNA synthesis (nuclear division) and, therefore, resistance to these fungicides develops very easily. See recommendations for resistance management from product labels and Sect. 3.3 for managing fungicide resistance.

4.1.6 Sterol Inhibitors

Sterol inhibitors, other than the triazoles (see Sect. 4.1.8), include hydroxyanilides (fenhexamid, etc.), imidazoles (imazalil, prochloraz, triflumizole, etc.), morpholines (tridemorph, etc.), piperazine (triforine), piperidines (piperalin, etc.), pyrimidines (fenarimol, etc.), and others. Products in these groups that are registered on florists' crops include fenhexamid, imazalil, triflumizole, triforine, piperalin, and fenarimol.

4.1.7 Dicarboximides

This is a group of agricultural fungicides, including iprodione, procymidone, and vinclozolin, that have excellent protectant activity on diseases caused by *Botrytis, Monilinia,* and *Sclerotinia.* Iprodione is registered on florists' crops. Dicarboximides inhibit DNA and RNA synthesis, cell division, and cellular metabolism. Resistance to dicarboximides has been well documented in several crops; hence, recommendations for avoiding fungicide resistance should be practiced. See recommendations for resistance management from product labels and Sect. 3.3 for managing fungicide resistance.

4.1.8 Triazoles

Triazoles are the largest class of fungicides. They are highly effective against many different fungal diseases, especially powdery mildews, rusts, and many leaf-spotting fungi. Triadimefon was the first triazole to be registered on florists' crops in 1990.

Since then, other triazoles were labeled including metconazole, myclobutanil, propiconazole, tebuconazole, and triticonazole.

The triazoles belong to a group of demethylation inhibitors (DMI) that inhibit one specific enzyme, C14-demethylase, which plays a role in sterol production. Sterols, such as ergosterol, are needed for membrane structure and function, making them essential for the development of functional cell walls. Therefore, these fungicides result in abnormal fungal growth and eventually death. They are locally systemically mobile in plant tissues, readily taken up by leaves and moves within the leaf. Triazoles may be applied preventively or as early-infection treatments. They affect fungi at a very specific site in the metabolic pathway; hence, resistance to these fungicides develops very easily. Therefore, recommendations for avoiding fungicide resistance should be practiced. These include avoiding repeated use, rotation with products with a different mode of action, preventative application, and applying rates within the labelled range.

4.1.9 Phenylamides

The phenylamide fungicides, including metalaxyl, mefenoxam (metalaxyl-M), benalaxyl, and kiralaxyl, brought a completely new level of control to the Oomycetes through their systemic properties by offering protection to the plants as seed treatments and soil or foliar applications. Mefenoxam is registered for use on florist' crops. The phenylamides are specific inhibitors of ribosomal RNA polymerases in the target fungi. Resistance to phenylamides develops very easily and has been well documented in several crops; hence, recommendations for avoiding fungicide resistance should be practiced. See recommendations for resistance management from product labels and Sect. 3.3 for managing fungicide resistance.

4.1.10 Strobilurins

The strobilurin fungicides, commonly referred to as Q_oI fungicides, have a broad spectrum of activity, are highly efficacious, and are suitable for a wide range of crops. The first active ingredient azoxystrobin was registered on florists' crops in 1998. Since then, additional Q_oI fungicides have been developed, with several now commercially available or expected to be available soon. These include fenamidone, fluoxastrobin, pyraclostrobin, and trifloxystrobin. In addition, several products contain strobilurins formulated with other fungicides to broaden the activity spectrum and slow down development of resistance. These products include azoxystrobin + benzovindiflupyr, azoxystrobin + difenoconazole, pyraclostrobin + boscalid, and pyraclostrobin + fluxapyroxad.

The strobilurin fungicides exhibit translaminar movement, and thus the fungicide can be found on both leaf surfaces even if only one leaf surface was treated. Some active ingredients in this group, e.g., azoxystrobin, also move systemically in the plant's vascular system. They kill germinating spores, and thus they are excellent as preventive fungicides. Although some products in this group provide curative activity, the best use of Q_oI fungicides is to apply them before infection takes

place. All Q_oI fungicides share a common biochemical mode of action: they all interfere with energy production in the fungal cell by blocking electron transfer at the site of quinol oxidation (the Qo site) in the cytochrome bc_1 complex, preventing ATP formation. They affect fungi at a very specific site in the metabolic pathway and, therefore, resistance to these fungicides develops very easily. See recommendations for resistance management from product labels and Sect. 3.3 for managing fungicide resistance.

4.1.11 Biofungicides

Biological fungicides (biofungicides) are composed of beneficial microorganisms, including specialized bacteria (*Bacillus, Pseudomonas*, and *Streptomyces*, etc.) and fungi (*Gliocladium* and *Trichoderma*, etc.) that suppress soilborne and foliar plant pathogens. Many of these microorganisms are found naturally occurring in soils. Researchers have isolated specific strains which have been formulated with additives to enhance their performance and storage. There are a number of ways in which biofungicides work, including direct competition or exclusion; antibiosis, predation, or parasitism; induced resistance; and plant growth promotion. Thus, the risk for resistance is considered lower than chemical fungicides.

Products containing biological control agents that are currently registered for disease management of florists' crops include *Bacillus subtilis*, *Coniothyrium minitans*, *Gliocladum catelunatum*, *Gliocladum virens*, *Streptomyces griseoviridis*, *Streptomyces lydicus*, *Trichoderma asperelloides*, *Trichoderma hamatum*, *Trichoderma harzianum*, and *Trichoderma harzianum* + *Trichoderma virens*. These are environmentally friendly integrated pest management (IPM) tools with great potential, but, currently, they do not adequately control ornamental plant diseases on their own in most cases.

4.2 Bactericides

Bacteria are single cell microscopic organisms. Bacteria are ubiquitous in and around plants. Only a small portion are plant pathogenic, but they can be difficult to manage. Several groups of products used to manage bacterial diseases of florists' crops are discussed below.

4.2.1 Inorganics

Copper-based compounds – There are several different copper-based fungicides registered for use in environmental horticulture plants. They have broad-spectrum activity, acting on bacteria as well as fungi. These products include copper diammonia diacetate complex, copper hydroxide, copper hydroxide + copper oxychloride, copper octanoate, copper sulfate, copper sulfate pentahydrate, and cuprous oxide. They are considered contact, multi-site activity bactericides/fungicides, with activity resulting from

nonspecific denaturation of proteins and enzymes. Thus, the risk for resistance is considered lower than single-site activity fungicides.

4.2.2 Antibiotics

Streptomycin – Streptomycin belongs to a group of bactericidal antibiotics including kasugamycin, oxytetracycline, and terramycin used in agriculture. Streptomycin was discovered and developed in the 1940s to be the first effective treatment for tuberculosis. Products based on streptomycin sulfate were registered in 1961 to control bacterial and fungal diseases of florists' crops. They are used as preventive treatments and can be applied as foliar and blossom spray, soil drench, pre-plant dip, etc. Streptomycin interferes with amino acid synthesis. Resistance to streptomycin sulfate has developed in some agricultural crops, and hence recommendations for avoiding fungicide resistance should be practiced. See recommendations for resistance management from product labels and Sect. 3.3 for managing antibiotic resistance.

4.2.3 Biocides

Several active ingredients with general biocide activity are registered for bacterial control either as disinfectants of tools and hard surfaces or as applications on plants for preventative and early curative control. These include quaternary ammonium compounds like didecyl dimethyl ammonium chloride (KleenGrow) and oxidizing agents like hydrogen dioxide + hydrogen peroxide (ZeroTol 2.0) and hydrogen peroxide + peroxyacetic acid (SaniDate 12.0).

4.3 Nematicides

Nematodes are small, multicellular wormlike animals. Depending on the type, they feed on roots, bulbs, stems, leaves, or seeds. The majority of plant parasitic nematodes live in the soil and damage plants by feeding in large numbers on the roots, impairing the plant's ability to take up water and nutrients. Although nematodes rarely kill plants, they can drastically reduce plant growth and quality (Table 3).

Management of nematode diseases is primarily preventative. Using healthy nursery stock is the best way to avoid soilborne nematode diseases. Most nematicides used to control soil nematodes are very toxic and can only be applied by certified applicators. Soil fumigants recommended for use in florists' crops before planting include 1,3-dichloropropene and sodium methyldithiocarbamate. These cannot be used in greenhouses or other enclosed areas. These fumigants are generally recommended only as a last resort when other management strategies have not been successful or are not available. Ethoprop is registered as a soil treatment for field nursery stock only. One biological nematicide, *Myrothecium verrucaria*, is registered as a soil treatment for florists' crops. For foliar nematodes, products are not readily available.

Table 3 Activity, use sites, and re-entry interval of active ingredients registered in the United States for environmental horticulture crops during 2016[a]

Active ingredient	Product trade names in the United States[b]	FRAC code[c]	Registered use sites in the United States (2016)[d]	REI	Bacteria	Botrytis	Downy mildew	Leaf spots anthracnose	Nonoomycete root rots	Phytophthora	Powdery mildew	Pythium	Rusts
Ametoctradin + dimethomorph	Orvego	45 + 40	G, I, L, N, S	12 h			X			X			
Azoxystrobin	Heritage	11	G, L, N, S	4 h		X	X	X	X	X	X	X	X
Azoxystrobin + benzovindiflupyr	Mural	11 + 7	G, L, N, S	12 h		X	X	X	X	X	X	X	X
Azoxystrobin + difenoconazole	Alibi Flora	11 + 3	G, L, N, S	12 h		X		X			X		X
Bacillus subtilis	Cease, Companion, DPZ, Rhapsody, Serenade Optimum, etc.	44	G, I, N, S	4 h	X	X	X	X	X	X	X	X	X
Captan	Captan	M4	G	48 h		X		X	X	X		X	
Chlorothalonil	Concorde, Daconil, Echo, etc.	M5	G, L, N, S	12 h		X		X		X	X		X
Chlorothalonil + propiconazole	Concert II	M5 + 3	N	12 h		X		X			X		X
Chlorothalonil + thiophanate methyl	Consyst, Spectro	M5 + 1	G, I, N	12 h		X		X	X		X		X
Chlorothalonil + zinc	Daconil Zn	M5	N	12 h		X		X		X	X		X
Cinnamaldehyde	Cinnacure	NC	G, N	4 h							X		
Copper ammonium complex	Copper Count-N, etc.	M1	G, I, N, S	12 h	X	X		X			X		
Copper hydroxide	Champ, Champion, CuPRO, Kentan, Kocide, etc.	M1	G, I, N, S	48 h	X	X	X	X		X	X		

Copper hydroxide + copper oxychloride	Badge SC, Badge *X2*	M1	G, N, S	48 h	X	X		X		X	X		
Copper hydroxide + mancozeb	Junction	M1 + M3	G, N	48 h	X	X	X	X			X		X
Copper octanoate	Camelot O	M1	G, I, N, S	4 h	X	X		X			X		X
Copper sulfate	Basicop, Cuprofix	M1	G, N, S	48 h	X	X		X			X		
Copper sulfate pentahydrate	Phyton	M1	G, I, N	24 h	X	X	X	X			X		X
Cuprous oxide	Nordox	M1	G, I, N, S	24 h		X		X			X		
Cyazofamid	Segway O	21	G, N	12 h			X			X		X	
Cyprodinil + fludioxonil	Palladium	9 + 12	G, L, N, S	12 h		X		X	X		X		
Dicloran	Botran	14	G, N	12 h		X		X					
Didecyl dimethyl ammonium chloride	Kleengrow	NC	G	48 h	X	X	X	X	X	X	X	X	
Dimethomorph	Stature SC	40	G, L, N	12 h			X			X			
EBDC	Fore, Pentathlon, etc.	M3	G, N	24 h						X			
Etridiazole	Terrazole, Truban	14	G, N	12 h					X	X		X	
Etridiazole + thiophanate methyl	Banrot	14 + 1	G	12 h					X	X		X	
Extract of *Reynoutria sachalinensis*	Regalia	P5	G, I, L, N, S	4 h	X	X	X	X			X		
Fenamidone	Fenstop	11	G	12 h			X			X		X	
Fenarimol	Rubigan	3	G, N	12 h							X		
Fenhexamid	Decree	17	G, N	12 h		X		X					
Fludioxonil	Medallion	12	G, I, L, N, S	12 h		X		X	X				

(*continued*)

Table 3 (continued)

Active ingredient	Product trade names in the United States[b]	FRAC code[c]	Registered use sites in the United States (2016)[d]	REI	Bacteria	Botrytis	Downy mildew	Leaf spots anthracnose	Nonoomycete root rots	Phytophthora	Powdery mildew	Pythium	Rusts
Fludioxonil + mefenoxam	Hurricane[e]	12 + 4	G, I, N, S	48 h					X	X		X	
Fluopicolide	Adorn	43	G, L, N, S	12 h			X			X		X	
Fluoxastrobin	Disarm, Fame	11	G, I, N, S	12 h		X	X	X	X	X	X		X
Flutolanil	Contrast, Prostar	7	G, N, S	12 h					X				X
Fosetyl Al	Aliette	33	G, N	12 h	X		X			X		X	
Gliocladum catelunatum strain J446	Prestop	NC	G, I, N, S	0 h		X		X	X	X		X	
Gliocladium virens	SoilGard	NC	G, I, N	0 h					X	X		X	
Hydrogen dioxide	ZeroTol	NC	G, I, N	0 h	X	X		X		X	X	X	
Iprodione	Chipco 26019, etc.	2	G, N	12 h		X		X	X				
Iprodione + thiophanate methyl	26/36	2 + 1	G, N	12 h		X		X	X				
Mancozeb	Dithane, Protect, Pentathlon, etc.	M3	G, N	24 h	X	X	X	X		X		X	X
Mancozeb + myclobutanil	Clevis[e]	M3 + 3	G, N	24 h		X		X			X		X
Mancozeb + thiophanate methyl	Zyban	M3 + 1	G, N	24 h		X		X			X		X

Mandipropamid	Micora	40	G, L, N, S	4 h			X			X			
Mefenoxam	Subdue, Fenox	4	G, L, N, S	0 h or 48 h			X			X		X	
Metconazole	Tourney	3	N	0 h or 12 h		X		X	X		X		X
Mineral oil	JMS Stylet Oil, SuffOil-X, etc.	NC	G, N	4 h		X		X			X		X
Myclobutanil	Eagle, etc.	3	G, N	24 h				X			X		X
Neem oil extract	Triact 70, Trilogy, etc.	18B	G, I, L, N, S	4 h		X	X	X			X		X
Oxathiapiprolin	Segovis	U15	G, L, N, S	4 h			X			X			
PCNB	Terraclor	14	G, I, N, S	12 h					X				
Phosphorus acid salts	Alude, Fosphite, Magellan, Rampart, Vital, etc.	33	G, N	4 h	X		X			X	X	X	
Piperalin	Pipron	5	G	12 h							X		
Polyoxin D zinc salt	Affirm, Veranda	19	G, L, N, S	4 h		X	X	X	X		X		
Potassium bicarbonate	Milstop	NC	G, I, L, N, S	1 h		X		X			X		X
Potassium salts of fatty acids	M-Pede	NC	G	12 h							X		
Potassium silicate	Sil-Matrix	NC	G, N	4 h							X		
Propamocarb	Banol, Proplant	28	G, N	24 h						X		X	
Propiconazole	Banner Maxx, ProPensity	3	N	12 h		X		X			X		X

(*continued*)

Table 3 (continued)

Active ingredient	Product trade names in the United States[b]	FRAC code[c]	Registered use sites in the United States (2016)[d]	REI	Bacteria	Botrytis	Downy mildew	Leaf spots anthracnose	Nonoomycete root rots	Phytophthora	Powdery mildew	Pythium	Rusts
Pyraclostrobin	Insignia, Empress Intrinsic	11	G, I, L, N, S	12 h		X	X	X	X	X	X	X	X
Pyraclostrobin + boscalid	Pageant Intrinsic	11 + 7	G, I, L, N, S	12 h		X	X	X	X	X	X	X	X
Pyraclostrobin + fluxapyroxad	Orkestra Intrinsic	11 + 7	G, I, L, N, S	12 h		X	X	X	X	X	X	X	X
Streptomyces griseoviridis	Mycostop	NC	G, N	4 h		X		X	X	X		X	
Streptomyces lydicus	Actinovate	NC	G, I, L, N, S	1 h	X	X	X	X	X	X	X	X	
Streptomycin sulfate	Agrimycin, Firewall	25	N	12 h	X								
Sulfur	Microthiol Dispersss, etc.	M2	G, N	24 h		X		X			X		X
Tebuconazole	Torque, Tebuconazole SC T&O Fungicide	3	N	12 h		X		X			X		X
Thiophanate methyl	3336, OHP 6672, etc.	1	G, I, L, N, S	12 h		X		X	X		X		X

Triadimefon	Bayleton, Strike	3	G, N	12 h		X		X			X		X
Trichoderma harzianum	PlantShield, RootShield	NC	G, I, N, S	0 h					X	X	X	X	
Trichoderma harzianum + *Trichoderma virens*	RootShield Plus	NC	G, I, N, S	0 h or 4 h					X	X		X	
Trifloxystrobin	Compass O	11	G, I, L, N, S	12 h		X	X	X	X	X	X		X
Trifloxystrobin + triadimefon	Strike Plus	11 + 3	G, N	12 h		X	X	X			X		X
Triflumizole	Terraguard	3	G, I, N, S	12 h		X		X	X		X		X
Triticonazole	Trinity	3	G, I, L, N, S	12 h		X		X	X		X		
Ziram	Ziram	M3	G, N	48 h		X		X			X		

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[b]Trade names are listed for products registered in the United States for use on environmental horticulture crops as of 2016. While this information is included to aid end users, the names may shift over time and multiple formulations may exist. Additional trade names may be available

[c]These are the mode of action group codes assigned by the Fungicide Resistance Action Committee. Full descriptions and listings can be found at www.frac.info

[d]Registered Use Sites: *G* greenhouse, *L* lath house, *I* indoors/interiorscape, *N* nursery, *R* landscape, *S* shade house

[e]Currently registered but not available commercially

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Soil/Media Disinfestation for Management of Florists' Crops Diseases

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Abstract

In-ground and pot-based ornamental producers face many challenges related to management of soilborne pests. Ornamental crops are susceptible to a wide range of soilborne pathogens that are capable of surviving for long periods of time on plant debris or on weed hosts. Some of these pathogens have broad host ranges, making it difficult to use crop rotation as a management tool. The number of different species and varieties of ornamentals that may be grown in a small area is often very high, which makes control measures for individual plant pathogens impractical. The availability of commercially desirable varieties that have resistance to specific diseases is limited or unknown for many of the crops grown in pots and in-ground; therefore, broad-spectrum control measures are more feasible, both technically and economically. For many years, both potting soil and field soil were fumigated with methyl bromide, a cost-effective, broad-spectrum biocide, which controlled soilborne fungal and bacterial plant pathogens, plant-parasitic nematodes, and weeds. The loss of this versatile compound, due to its negative impact on the ozone layer, has led to the development of new soil disinfestation approaches as well as renewed interest in improving technologies used in the past. The tools that are currently available for soil

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R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_7

disinfestation include fumigants, nonfumigant soil applied chemicals, steam, solarization, and anaerobic soil disinfestation.

Keywords

Soilborne plant pathogens • Nematodes • Weeds • Fumigation • Steam • Solarization • Anaerobic soil disinfestation

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1 Introduction

The California floriculture industry is the largest in the USA with a 2014 value of $1.05 billion. Florida follows closely with a 2014 value of $890 million in wholesale value. In both states, the industry consists of bedding and garden plants and potted and in-ground cut flowers (CAS 2013; USDA 2015). In 2014, the covered area used for floriculture production in California was 145 million sq. ft. and open ground was 10,844 acres (USDA 2015). For Florida, in 2014, there were approximately 299 million sq. ft. under cover and 5,200 acres in open ground production (USDA 2015).

The production of cut flowers in open fields in Florida has continued a steady decline, with less than $3 million generated from this industry in 2014. Florida production of potted foliage plants, valued at $420 million, represents 72% of the US total. Florida contributes 79% of the cut greens produced in the USA and this commodity is valued at $58 million (USDA 2015). Florida also produces approximately 95% of the world caladium supply (Fig. 1), tubers from which are used for propagative material for landscape and potted plants (Deng et al. 2005). The California cut flower industry has an annual impact of $10.3 billion on the state's economy contributing nearly $28.2 million on a daily basis to the state (CCFC 2008). California supplies about 76% of the cut flowers sold in the USA at a wholesale value of $261 million in 2012 (USDA 2015). California has 5,000 acres of land used for commercial cut flower production of which 4,000 acres are under open-field production (USDA 2007).



Fig. 1 Florida caladium production accounts for approximately 95% of the world's supply of caladium tubers (E. Rosskopf. USDA.)

2 Pests Controlled by Soil Disinfestation

Both potted ornamentals and open field flowers suffer from soilborne diseases such as Fusarium and Verticillium wilts, damping off and root rots caused by *Pythium* spp., and crown and root rots caused by *Phytophthora* spp. Nematodes such as *Meloidogyne* spp. (Fig. 2) also cause significant reductions in flower yield. Many cut flower varieties of sunflower, for example, are highly susceptible to infection by root-knot and other nematodes and to the diseases southern blight, bacterial wilt, and Fusarium stalk rot. Calla lilies, in particular, have problems with root rots caused by *Pythium* and *Phytophthora* spp. as well as soft rot of rhizomes due to *Erwinia* or *Pectobacterium* spp. (Dreistadt 2001). Similarly, caladium tubers suffer from *Pythium* spp. and root-knot nematodes, which severely limit both yield and quality. It also succumbs to foliar infection by *Xanthomonas axonopodis* pv. *dieffenbachiae* causing bacterial blight (Seijo et al. 2010). These pathogens may all be present in the same production area (Fig. 3). Although there has been progress in terms of breeding for resistance in many crops, it is rare to have a single variety that has desirable horticultural characteristics as well as multiple disease resistance genes (Kokalis-Burelle and Rosskopf 2013; also refer to ▶ Chap. 5, "Breeding for Disease Resistance

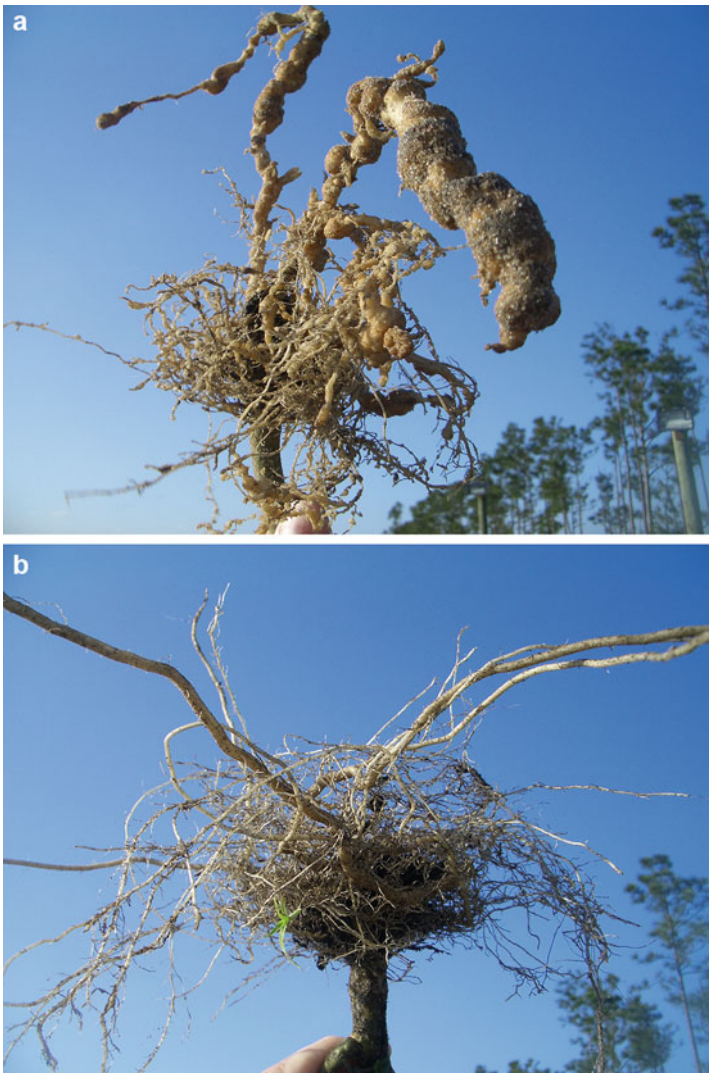


Fig. 2 Roots from an untreated area with significant root galling caused by *Meloidogyne arenaria* (**a**) compared to roots collected from a fumigated field (**b**) (E. Rosskopf. USDA.)

in Florists' Crops"). Weeds, including volunteer bulbs and tubers from previous crops, nutsedge (*Cyperus* spp.), mallow (*Malva* spp.), clover (*Trifolium repens*), geranium (*Geranium carolinianum*), and bluegrass (*Poa* spp.) are major problems in cut flowers (USEPA 2013). If weeds are not effectively controlled in these systems, they can contribute to the survival and reproduction of other pests, such as nematodes (Kokalis-Burelle and Rosskopf 2012; Kokalis-Burelle et al. 2012). These soilborne pests, and many others, have traditionally been controlled using soil



Fig. 3 Impact of the complex of *Pythium* infection and root-knot nematode infestation by *Meloidogyne arenaria* in a caladium field where the fumigant injection was incomplete (E. Rosskopf. USDA.)

disinfestation via chemical fumigation with methyl bromide, which has been phased-out for the majority of agricultural uses in the USA. Without this broad-spectrum biocide, potting media and soil in ornamental production systems must be treated in other ways to minimize the impact of soilborne pests.

3 Nursery Potting Media

3.1 Soilless Media Components

Many large nurseries mix their own potting media or have it custom blended and then delivered by the truckload. Components of substrates include coir, perlite, peat moss, vermiculite, sawdust, sand, biochar, and bark. Gypsum and dolomitic limestone are also frequently added to supply calcium and/or magnesium and to maintain a suitable substrate pH. Compost, either produced on-site or off, is used in some mixes, and more recently, by-products of other agricultural processes such as nut or rice hulls are included in the mix. Recently, the use of composted algae has been investigated as an alternative substrate component that also provides fertilization (Fig. 4) (Albano et al. 2010). Fertilizer, mycorrhizae, or wetting agents may be added, but this is generally done after the media base formula is mixed.

Whether media is custom blended and delivered to the nursery, or blended at the nursery, it is important to test or assay the organic substrates (e.g., coir, peat moss,



Fig. 4 Increased plant vigor resulting from the application of (*left*-to-*right*) composted algae with fertilizer compared to commercial peat-based substrate with fertilizer, composted algae without fertilizer, and commercial peat-based substrate without fertilizer (J. Albano © 2017. All Rights Reserved.)

bark) for pests and disinfest if needed prior to blending. Then the substrates are blended as soon as possible after disinfestation.

After blending, the mix should be placed on concrete pads or other clean areas located away from trash and other piles that are used for holding plant debris. If the media is held for more than 2 weeks, it is helpful to use tarps or other coverings to minimize contamination from windblown weed seeds and fungal spores.

3.2 Soil as a Potting Media Component

In general, soil should be a minor (<10%) component of a potting mix. This is due to the fact that soil physical properties vary depending on where it is from, and these properties affect drainage and root growth. Additionally, mineral soil is heavier than soilless mixes and will increase labor and shipping costs (Hanan 1997). Where soil is used as a component of potting media, it should be disinfested to reduce pathogens, nematodes, and weed seeds and propagules.

3.3 Substrate Disinfestation Methods

3.3.1 Chemical

The primary method of chemical disinfection of potting media substrates is currently the use of dazomet (Agrian 2015c; Fritz and Dimcock 2005). There are important

safety restrictions when using this material so the label must be followed carefully. This dry, powder-like formulation is thoroughly mixed into the moist substrate(s) using a cement mixer, front-end loader, or similar equipment. Once distributed, the media can be piled up to 1 m high, preferably on a clean solid surface such as a concrete pad or on polyethylene plastic sheet. The pile is then covered for at least 7 days as the dazomet degrades into the active fumigant, methyl isothiocyanate. The cover is removed and the soil allowed to vent undisturbed for 7 days before using the mix for potting. Because dazomet does not degrade to the active fumigant unless exposed to moisture, it is important the pile be thoroughly irrigated after treatment application. The soil temperature must be above 6 ° C during the entire fumigation period.

An alternative to piles for treatment application to media is the use of 30 cm tall frames. These frames are easily constructed using lumber and wooden or metal stakes. If possible, the frame should be lined with polyethylene plastic sheeting. Dazomet is then uniformly applied to the media surface and mechanically incorporated using a rototiller or similar equipment. The treated media is watered and tarped as above. Although this method requires more space, it more uniformly wets the media, increasing fumigant effectiveness.

3.3.2 Steam

Heating with steam (Fig. 5) is very effective and has the advantage over chemical treatment in that the soil can be used immediately after cooling and is not subject to the regulatory restrictions associated with fumigation. Although pure steam at sea



Fig. 5 Soil cart used for steaming potting mix (S. Fennimore © 2017. All Rights Reserved.)

level is at 100 °C/212 °F, the temperature at which steam is used to treat soil is usually about 82 °C/180 °F because of air that is present in the steam or in the soil being treated. If air is mixed with steam, the temperature of the steam-air mixture can be closely controlled, depending on the ratio of air to steam. Steam may be used to treat stationary and moving soils (Baker and Roistacher 1957).

Many plant pathogens are killed by short exposures to high temperatures; however, experience has shown that the soil temperatures should be maintained for approximately 30 min to ensure the soil is thoroughly disinfested. Most plant pathogens can be killed by temperatures of 60 °C for 30 min; however, some viruses (as well as weed seeds) may survive this treatment (Koike and Wilen 2009). It has been demonstrated that some diseases, such as *Rhizoctonia* damping-off, are much less severe in soil that has been treated at 60 °C/140 °F rather than at 82 °C/180 °F due to mortality of beneficial thermophilic microorganisms at temperatures above 60 °C/140 °F. To maximize control and reduce energy costs, growers or potting media suppliers may need to experiment to find the best temperature and length of time to treat the media. A good starting point is 60 °C/140 °F for 30 min.

Steam from a boiler can be efficiently blended with bulk soil using a cement mixer (see Miller et al. 2014). When this type of system is used it is not necessary to introduce air into the steam because a large amount of air is present in the mixer and the temperature can be controlled by simply regulating the flow of steam.

Stationary systems such as soil vaults or tarp-covered piles require the use of air blowers. If circulating fans can be placed within or external to the vault and the steam can be introduced into the recirculating air, then blowers are not necessary. Nevertheless, it is important to leave space between the vaults and check temperatures throughout the vault to ensure that there is good circulation of steamed air. Another method to ensure that the media is uniformly heated is to combine steam heating with a vacuum system to draw the steam through the media. In this system, perforated pipes are placed 40–45 cm below the media to be treated and attached to vacuum pumps (Hanan 1997).

Steam heating of soil or potting media is also an effective method to kill weed seeds. In this process, steam is mixed with air and injected into tarp covered, wetted soil mix to heat it to 82 °C/180 °F for 30 min (Elmore and Wilen 2000; Baker and Roistacher 1957). Length of time and temperature are critical if weed seeds are to be controlled (Vidotto et al. 2013), and temperature probes must be placed so that the internal temperature is verified. Moist soil conducts heat more readily than dry soil; therefore, the media should be thoroughly moist prior to injecting the steam to facilitate even heat distribution. Also, weed seed are more easily killed when imbibed with moisture.

3.3.3 Double-Tent System for Solarization

A “double-tent” setup can be used to heat media (Fig. 6). This method has been demonstrated to reach at least 60 °C/140 °F for 60 continuous min and 70 °C/158 °F for 30 continuous min (CDFA 2002). In this system, media at or near field water holding capacity is placed in polyethylene planting bags or in small piles (not more than 30 cm high) placed on pallets or wire mesh screen on a layer of polyethylene

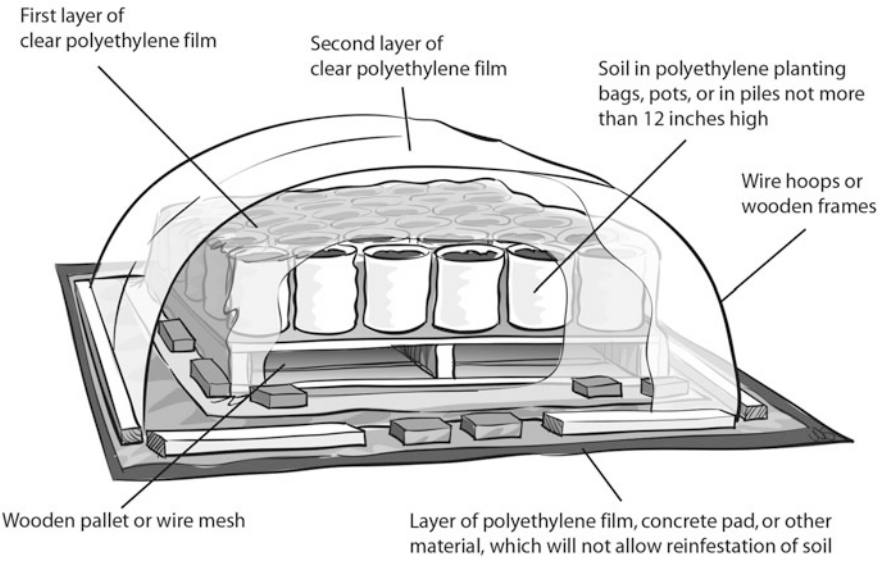


Fig. 6 "Double tent" system for using solarization to disinfest containers and planting mix. Illustration by William Suckow (Reprinted from Stapleton JJ, Wilen CA, Molinar RH. 2008). Pest Notes: Soil Solarization for Gardens & Landscapes. Oakland: Univ. Calif. Div. Agric. Nat. Res. Publ. 74145 (Reprinted with permission from the UC Statewide IPM Program)

film, concrete pad, or other impervious material. An additional layer of clear polyethylene film is then suspended over the first layer to create a still air chamber over the soil to be treated. This approach has been successfully used to treat potting mix for weed control (Prather et al. 2002). This system works best in full sun. Temperature probes should be placed at the bottom center of the pile or bag to monitor that the effective temperatures are reached.

Note that any fertilizer or mycorrhizae should be added after the media has cooled. For many slow-release fertilizer formulations, nutrient release is controlled by temperature and moisture. High temperatures, created by steaming or solarization, will cause the fertilizer to be released immediately. Similarly, mycorrhizae will be killed at high temperatures.

3.3.4 Compost as Disease Suppressive Media

Compost may be a minor component of conventional potting mixes but is commonly used in organic mixes. Organic materials should be thoroughly composted prior to use and should constitute only 20–30% of the mix (Rynk 1992). Composted animal manures and vegetable waste added to peat have resulted in the suppression of several plant diseases (Pugliesi et al. 2015). Disease suppression is highly dependent upon the inputs and chemical composition of the compost. Diseases caused by *Pythium* and *Phytophthora* spp. may be exacerbated when composts have high salinity (Hoitink and Fahy 1986). Colonization of the compost by beneficial

organisms, such as *Trichoderma* spp. (Horst et al. 2005), may be the mechanism of disease suppression, and the addition of microbial inoculants could increase disease suppression. Unfortunately, there is a great deal of variability associated with the use of compost for disease suppression and it is not a common practice in the commercial conventional floriculture industry.

An important note on integrated pest management relative to media disinfestation is that reuse of containers is a common practice and disinfesting media without sanitizing used pots will result in the reintroduction of pathogens at planting (Parke et al. 2008).

4 Soil Disinfestation

Soil disinfestation in open-field and shade house production is performed to control multiple pests such as soilborne pathogens and weed seeds but also to kill flower propagules (bulbs, rhizomes, tubers) from the previous crop. Fumigation with methyl bromide was the principal treatment for control of many soilborne plant pathogenic fungi, bacteria, weeds, nematodes, and insects in soil for field-grown cut flowers. Its loss presents a particularly difficult challenge for in-ground ornamental producers (Rosskopf et al. 2005, 2010b; Schneider et al. 2003). Cut flowers are produced using very high-density plantings, which necessitates full-field soil disinfestation in order to sufficiently treat the ground. Materials with low vapor pressure do not move through soil in the manner that methyl bromide does with reduced efficacy as a result. This also limits the efficacy of applications around fixed infrastructure such as shade and glass house posts (Figs. 7 and 8). Buffer zones of up to 400 m or more in sensitive areas near hospitals or schools make use of fumigants combined with chloropicrin for cut flowers challenging as flower production often occurs on small acreages and/or in coastal areas near cities where land rental values are high. Regulatory restrictions on fumigant use create a need for safe fumigants and nonfumigant alternatives to disinfest soils for flower production.

4.1 Soil Fumigants

Fumigants are chemicals that volatilize when applied to the soil, and the vapors diffuse through the soil to contact the target pest. The overall effectiveness of any fumigant is determined by the dosage delivered to the pest and is a function of the concentration and time of exposure. This is referred to as the CT factor (concentration/time factor). Other parameters that are important to the success or failure of fumigation are soil temperature, soil moisture, soil texture, organic-matter content, application method, and surface seal. The objective of a fumigant application is to reach the critical concentration, come in contact with the soil pest, control it, and then the fumigant must degrade to prevent nontarget exposure to people and other plantings in the vicinity of the field and to ensure safety to the crop when it is planted in the days or weeks following fumigation.



Fig. 7 Soil disinfestation is hampered by permanent infrastructure such as posts for shade cloth and glass (E. Rosskopf. USDA.)

Optimum soil temperatures for application of most soil fumigants is between 18° and 24 °C/75 °F at a depth of 15 cm. Cool soil temperature slows volatilization, which results in slow fumigant dispersal and poor pest control. At low soil temperatures, or in excessively wet soil, fumigants break down slowly which may result in persistent fumigant concentrations that can linger and injure the crop if planted before the fumigant concentration falls to safe levels. If soil temperatures are too high, the fumigant may diffuse too rapidly or escape before reaching an adequate concentration for good pest control.

Fumigants present significant hazards to workers and the environment and must be handled carefully. They are classified, with a few exceptions, as restricted use pesticides in the USA and, as such, must be applied only by certified fumigant applicators (Blecker and Thomas 2012; Noling 2013). Most shank applications are conducted using nitrogen pressurization to push the materials through specialized tubing (Fig. 9) that is attached to a shank that runs through the soil (Fig. 10). As the fumigant is injected below the soil surface, a polyethylene tarp is applied over the soil to prevent the fumigant from dissipating too quickly (Fig. 11).

Fumigants can be applied by shank or by chemigation through the drip irrigation system (Fig. 12) (Ajwa et al. 2002). Whether the fumigants are applied by chemigation or shank application, they will benefit from the use of impermeable films such as "totally impermeable film" (TIF) (Fennimore and Ajwa 2011). The TIF



Fig. 8 Areas in which fumigants or disinfestation practices are difficult to use are often treated using many chemical spot-injections, commonly known as "stinging." This is useful with chemicals that have lower vapor pressure than methyl bromide (E. Rosskopf. USDA.)

blocks fumigant emissions and retains the fumigant in the soil at higher concentrations for longer periods than standard films which results in better pest control. Some fumigant labels only allow for application under high barrier films such as TIF (Agrian 2015d).

The most useful soil fumigant, in the absence of methyl bromide, for field grown flowers in CA, is chloropicrin (Agrian 2015a). Although registered for use, metam sodium and metam potassium (Agrian 2015b) and dazomet are not very effective for controlling many soilborne pathogens, including *Verticillium* and *Fusarium oxysporum*. Two more recently registered fumigants, allyl isothiocyanate (Agrian 2015e) and dimethyl disulfide (Agrian 2015d), are available for use in the USA (FL) and are more feasible for use in floriculture in many cases than are 1,3-dichloropropene and chloropicrin due to fewer regulatory restrictions (Table 1).

4.1.1 Chloropicrin

Chloropicrin must be injected into soil, and it is very effective for controlling many plant pathogenic fungi. Chloropicrin is often combined with 1,3-dicloropropene (1,3-D) in various mixtures such as 56.6% chloropicrin, 37.1% 1,3-D (Table 1) depending upon the target pests in the soil. When chloropicrin is used alone, a water



Fig. 9 Fumigants are injected into soil through a series of specialized tubes that are pressurized using nitrogen gas. Different fumigants are delivered with different sizes of tubing and flow meters in order to achieve precise application amounts and effective distribution of material (E. Rosskopf. USDA.)

seal may be used to confine the gas; however, chloropicrin has a very objectionable smell and is irritating to the eyes (it is commonly known as tear gas) and, if not effectively confined, it may drift to inhabited areas. This is a restricted use material in the USA and requires a permit from the county agricultural commissioner to be purchased and/or applied (Koike and Wilen 2009).

Numerous studies have been conducted in the USA (CA) on the use of chloropicrin and 1,3-D mixed with chloropicrin. In studies using inoculum bags buried at multiple depths, Telone C35 (63.4% 1,3-D, 34.7% chloropicrin) was extremely effective in killing propagules of *Pythium* and *Verticillium* but not very effective for control of *Fusarium* or *Phytophthora* (Cabrera et al. 2015). In a series of calla lily trials aimed at control of Pythium and Phytophthora rots of the rhizomes, drip application of chloropicrin and combinations of 1,3-D and chloropicrin resulted in the greatest yields and reduced incidence of the disease (Gerik et al. 2006). In similar work, drip application of chloropicrin reduced the incidence of *Sclerotinia sclerotiorum* on liatris, although metam sodium provided a greater level of control (Gerik 2005). Combinations of fumigants that include chloropicrin generally provide improved disease control. The use of 1,3-D is heavily regulated, and restrictions on its use may prevent implementation in some areas (Carpenter et al. 2001).



Fig. 10 Green and blue tubing, seen here, runs from the fumigant cylinder, through a series of flow meters, down to the shanks where the gas is injected into the soil. These shanks have small plates on the back of the shanks to close the cut in the soil into which the gas was injected (E. Rosskopf. USDA.)

4.1.2 Dimethyl Disulfide (DMDS)

The volatile sulfur compound dimethyl disulfide (DMDS) has been tested for control of several nematodes including potato cyst (*Globodera* spp.) and root-knot (*Meloidogyne* spp.) nematodes, as well as diseases caused by *Fusarium*, *Pythium*, and *Phytophthora*. Greenhouse experiments were conducted by Coosemans (2005) against potato cyst and root-knot nematodes on potato and tomato. Five rates of DMDS were tested and compared with other nematicides including methyl bromide and aldicarb, reducing both potato cyst and root-knot nematode populations, for increasing plant growth and saprophytic nematode populations.

Although more work has been done with this fumigant in the control of pathogens of vegetables, cut flower field trials were conducted in FL as early as 2004 to assess the efficacy of DMDS for control of the root-knot nematode *M. arenaria* and Pythium root rot on cockscomb (*Celosia argentea*). DMDS and DMDS plus chloropicrin provided excellent nematode and disease control (Church et al. 2004, although it was more effective in eliminating *Pythium* from soil than it was for *Fusarium*. In caladium (*Caladium* x *hortulanum*), DMDS was as effective in controlling *M. arenaria* as methyl bromide. Root condition was better and gall ratings were lower in the DMDS treatment than for several 1,3-D treatments. Control of *Pythium* root rot in caladium by fumigation was highly dependent upon interaction between fumigant and caladium cultivar, with “Sweetheart” combined with DMDS having the lowest pathogen



Fig. 11 After injection of the gas into the soil, the fumigated area is immediately covered by a plastic tarp. Each tarp is glued to the previous one as the gas is injected in each pass. The glue in this photo is *red* and the worker is making sure that it is sealing properly (S. Fennimore © 2017. All Rights Reserved.)

recovery from roots (Fig. 13) (Kokalis-Burelle et al. 2010). In trials conducted in CA cut flowers, DMDS did not provide consistent control of *Pythium* or *Fusarium* (Gerik 2005).

4.1.3 Allyl Isothiocyanate

The use of *Brassica* species as soil-incorporated green manures has been studied extensively for their disease-management effects. In the early 1990s, Kirkegaards et al. (1993) introduced the idea of using *Brassica* species for biofumigation in which the break-down products of brassicas were identified as isothiocyanates. Recently, a formulated biofumigant containing allyl isothiocyanate was registered in the USA. This product is labeled for use in ornamental production in FL, and registration is pending in CA. Several trials have been conducted with this material in cut flowers in FL, and it has provided root-knot nematode control equivalent to methyl bromide in snapdragon, lupin, larkspur, iris, sunflower, celosia, zinnia, and Queen Anne's lace. Control of *Sclerotium rolfsii* was also equivalent to methyl bromide (Rosskopf et al. 2014). Weed control with this compound has been excellent for grass weeds, but it has not provided control of Carolina geranium (*Geranium carolinianum*).



Fig. 12 Fumigants can be applied through drip-irrigation lines, also referred to as "chemigation." The *lines* are pressurized using nitrogen gas and the fumigant is injected into a port which flows into a static mixer and is pushed out through the drip tape (H. Ajwa © 2017. All Rights Reserved.)

4.2 Steam

Steam has been used for disinfesting soil since the 1800s. This approach was commonly used in cut flower production in the USA prior to the development of fumigants. In many South American countries and the Netherlands, steam continues to be used for soil disinfestation for flower production. The loss of methyl bromide resulted in a resurgence of interest in this approach. Typically, "sheet steaming" was used in which steam is applied to the soil surface, under a heat resistant tarp. The heat moves from the soil surface down (Runia 1984). Injection of steam, either through a fixed drain tile system (Fig. 14), spike application system (Fig. 15), or mobile unit (Figs. 16 and 17), is more efficient. There continues to be significant improvement in the efficiency and, therefore, cost of steam application (Fennimore et al. 2014).

Work on steam to control soilborne pathogens including nematodes was conducted by Van Loenen et al. (2003). They studied the effects of steam on survival structures of several important fungal pathogens, weeds, and the potato cyst nematodes *Globodera rostochiensis* and *G. pallida* in field soil. Using aerated steam at temperatures ranging from 40 °C to 80 °C they found that steaming at 50 °C or 60 °C (122 °F or 140 °F) for 3 min resulted in 100% kill of all pathogens and weeds including nematodes.

Steam has recently been shown to be effective for broad-spectrum nematode and fungal plant pathogen control in field soil (Rosskopf et al. 2010a; Kokalis-Burelle et al. 2014). In cut-flower field trials conducted in Florida, root-knot nematode

Table 1 Fumigants labeled for use in floriculture in the USA and relative effectiveness against soil pests (Modified from Elmore et al. 2007)

Common name	Trade name	Broadcast rates	Nematodes	Fungi	Weeds	Comments
1,3-dicloropropene	Telone II	9–12 gal/A 84–112 L/ha 1 gal = 10.1 lb	Excellent	Poor	Fair	Effective against nematodes and insects
1,3-dicloropropene plus chloropicrin	Pic-Clor 60	0–48.6 gal/A 0–454 L/ha 1 gal = 12.1 lb	Excellent	Very good	Fair	Most effective against nematodes, fungi, and insects
Chloropicrin	Tri-Clor	15–30 gal/A 140–280 L/ha 1 gal = 13.76 lb	Fair	Excellent	Fair	Effective for control of fungi and insects
Dazomet	Basamid G	218–525 lb/A 244–588 kg/ha	Good	Good	Good	Dry powder incorporated into soil or watered into soil
Metam potassium	K-Pam Sectagon K	30–60 gal/A 280–561 L/ha 1 gal = 5.8 lb	Good	Good	Good	Water soluble liquid forms MITC, the active fumigant
Metam sodium	Vapam Sectagon	37.5–75 gal/A 351–702 L/ha 1 gal = 4.26 lb	Good	Good	Good	Same as metam potassium
Allyl isothiocyanate[a]	Dominus	20–40 gal/A 187–374 L/ha 1 gal = 8.5 lb	Excellent	Good	Fair	Not registered in CA
Dimethyl disulfide[a]	Paladin	35–51.3 gal/A 328–480 L/ha 1 gal = 8.85 lb	Excellent	Good	Fair	Not registered in CA

[a]Modified from Elmoredeng



Fig. 13 Cultivars or varieties of the same species often have different levels of susceptibility to pathogens. Some caladium cultivars are resistant to both root-knot and Pythium root rot, caused in this case by *Meloidogyne floridensis* and *Pythium* spp., respectively (N. Kokalis-Burelle. USDA.)

(*M. arenaria*) populations in steam treated soil were equivalent to methyl bromide treatment and lower than those found in solarized soil. Total nematode numbers, including free-living nematodes, were lower after steam treatment compared with solarization and methyl bromide.

In an effort to better understand the dynamic effects of steaming soil on a variety of soil organisms, McSorley et al. (2006) investigated the effects of steam on nematode and microbial populations in sand and muck soils, two important soil types in Florida ornamental crop production. Steamed or nonsteamed soil of each type was inoculated with 2,000 eggs of *M. incognita* per pot. A soil type × steam treatment interaction occurred, with root-knot nematodes suppressed in nontreated sand but not in steamed sand nor in any (steamed or nonsteamed) muck soil. A variety of organisms were monitored in both soils including free-living nematodes (bacterivores, fungivores, omnivores, and predators), enchytraeids, Collembola, mites, nematode-trapping fungi, egg-parasitic fungi, *Pasteuria* spp., rhizosphere fungi including *Fusarium* and *Rhizoctonia*, and a variety of rhizosphere bacteria including Gram positive bacteria, fluorescent pseudomonads, and siderophore producers. Most of these organisms did not show population patterns consistent with the biological suppression of root-knot nematodes observed in the nonsteamed sand. For example, *Pasteuria* and other Gram positive bacteria were more abundant in soils that had been steamed; however, more inoculated root-knot nematodes survived in steamed soils as well.



Fig. 14 Steam can be delivered to the field using a fixed boiler system that is attached to buried drain tile. The drain tile is attached to a manifold (seen here) that is connected to the boiler through heat-resistant hose (S. Fennimore © 2017. All Rights Reserved.)

Population trends of predatory nematodes were most consistent with the suppression of root-knot nematodes observed in nontreated sand.

Finally, steam has some very practical applications in controlling nematode pests for certification requirements in some ornamental crops. Nurseries in Hawaii must follow strict certification requirements to export potted plants. *Rotylenchulus reniformis*, reniform nematode, must be disinfested from mined volcanic cinder, which is utilized as a potting medium by nematode certified nurseries. Cabos et al. (2012) investigated two steam delivery systems for efficacy in disinfesting cinder of *R. reniformis,* a low capacity system consisting of a portable steam generator connected to a cart with a treatment volume of 1.68 m^3 and a large capacity system consisting of a modified dump truck bed with a treatment volume of 24.5 m^3 connected to a boiler. Cinder inoculated with *R. reniformis*-infested roots of *Ipomoea batatas* were buried in various locations and depths in the cinder contained in the two steaming systems. Once the steam was evenly distributed, both systems were successful at eradicating all live *R. reniformis* (Cabos et al. 2012). Many logistical issues remain for steam application and before steam can be adopted on a large scale,



Fig. 15 Steam from a fixed boiler is applied through heat-resistant hose with spikes which are inserted through the plastic into the bed area (S. Fennimore © 2017. All Rights Reserved.)



Fig. 16 Numerous commercial mobile steam units are available that mix soil with steam as it moves through the field (S. Fennimore © 2017. All Rights Reserved.)



Fig. 17 Large-scale application of steaming requires mobile units with access to water and fuel (S. Fennimore © 2017. All Rights Reserved.)

these would need to be resolved. Also the expense of steam application is dependent on energy costs and may limit adoption of steam as an alternative soil treatment in the short term. Nonetheless, this may be a practical approach for the treatment of areas in which chemical fumigant use is restricted.

4.3 Solarization

Soil solarization is a process where sunlight is used to heat moist soil to control various pest organisms (Elmore et al. 1997; Katan et al. 1987). Soil solarization is accomplished by using a clear, thin polyethylene covering on the soil during a period of high radiation, with little wind or cloud cover. This condition is most common during the summer (late May to late August) in the central valley regions of California and may be present in May or more likely in late August or early September, in the coastal regions. Solarization can be used in Florida for the production of fall crops, with solarization conducted from May–October. Similar timing would be expected at similar latitudes.

Some soil-borne pathogens are rather easily controlled when there are enough heat units (temperature and time). The amount of control depends upon the organism and the heat units. Many organisms including weeds, soilborne pathogens, and nematodes are killed by soil temperatures in excess of 50 °C/122 °F. Because soilborne pathogens and nematodes may be found throughout the soil profile,

pests below 30 cm will not be controlled as well as pests near the surface where temperatures are hotter. Because weeds mostly emerge from the shallow surface layers, weeds are generally better controlled than nematodes which must be controlled throughout the soil profile. Soil solarization may control nematodes in some cases, particularly in the upper 30 cm of soil in areas of high solar output, light and medium textured soil, soil moisture near field capacity, shallow root depth of previous crop, and limited acreage. Nematodes are also mobile in the soil and thus certain species may escape the thermal killing by movement into cool soils, as do earthworms.

The killing with soil solarization is predominantly by heat induced membrane disruption, though suppression of some organisms may be caused by certain gases formed as the organisms are killed. See Elmore et al. (1997) for details on species of soil pathogens and nematodes controlled with solarization. Keep in mind that these species will not be controlled as well in coastal California under cool foggy conditions as in hot inland valleys.

Most weeds are controlled with 4–6 weeks of solarization in the central valley region of California (Elmore et al. 1997). All winter annual weeds, except some clover species, are controlled. The biennial weed bristly oxtongue (*Picris echioides*) is also controlled. Some perennial weeds can be controlled in the Central Valley area, such as Bermudagrass (*Cynodon dactylon*), Johnsongrass (*Sorghum halepense*), and yellow buttercup (*Ranunculus acris*). Many summer annual weeds are also controlled with the exception of sweet clovers (*Melilotus* spp.). If the treatment is marginal, then common purslane (*Portulaca oleracea*) will not be controlled or will only be partially controlled. Weeds that are only partially controlled include yellow (*Cyperus esculentus*) and purple nutsedge (*C. rotundus*). Field bindweed (*Convolvulus arvensis*) is controlled at the surface (0–10 cm), but it will regrow from deeper rhizomes. In coastal regions of California, the control of weeds is often spotty, depending upon local conditions (Elmore et al. 1997).

The most critical time of high radiation is during the first week of cover. If the weather is warm, sunny, and calm, control is generally good. Fields tarped during cloudy, foggy, or windy conditions will often result in poor control. Common purslane, if present, seems to be a good indicator species. If the conditions are correct, common purslane is controlled and indicates that the solarization procedure has worked.

A great deal of data is available on the effects of soil solarization on nematodes. In general, solarization proves to be more effective for reducing nematode populations when applied during the hottest months in tropical or subtropical growing regions. Early work on solarization using clear or gas-impermeable plastic was conducted in Florida to evaluate combinations of solarization with soil fumigants, herbicide, or cabbage residue (Chellemi et al. 1997). Solarization plastic was applied to vegetable planting beds and left in place for 40–55 days. After the solarization period the plastic was painted white and used for tomato production. At the end of the trial, the density of *Paratrichodorus minor* and *Criconemella* spp. populations were reduced in solarized plots. No differences in populations of *Helicotylenchus* spp. were

observed between plots receiving solarization and plots fumigated with methyl bromide + chloropicrin.

In laboratory studies, *Meloidogyne incognita* eggs or J2 were incubated in test tubes containing sand:peat mix and immersed in a water bath heated to 38 °C, 39 °C, 40 °C, 41 °C, 42 °C, 43 °C, 44 °C, and 45 °C for a series of time intervals. Controls were maintained at 22 °C. Nematodes surviving or hatching were collected after 3 weeks of incubation. Regression analyses between percent survival or egg hatch and hours of heat treatment were performed for each temperature. Effect of temperature on nematode survival was not determined by heat units, and oscillating temperature between cool and warm did not interfere with the nematode suppressive effect of the heat treatment. In field studies, solarization for 6 weeks during the summers of 2003 and 2004 in Florida accumulated heat exposure times in the top 15 cm of soil that surpassed levels required to kill *M. incognita* as determined in the water bath experiments. Although near zero *M. incognita* were detected right after solarization, the nematode population densities increased after a cycle of a susceptible pepper crop (Wang and McSorley 2008).

Solarization has also been studied for control of nematodes and pathogens in multiple ornamental species. In a study conducted in west-central FL using 3–4 weeks of solarization implemented in early fall, incidence of Phytophthora blight of Madagascar periwinkle (*Catharanthus roseus*), caused by *P. nicotianae*, was significantly reduced (McGovern et al. 2000). In a similar study, solarization conducted during the fall using a double layer of clear plastic mulch was consistently effective in reducing disease in impatiens (*Impatiens x wallerana*) caused by *Rhizoctonia solani* and *Pythium* spp. (McGovern et al. 2002). The severity of root galling caused by root-knot nematodes was reduced by solarization, as were the populations of several other plant parasitic nematodes. Control of Pythium damping off on snapdragon (*Antirrhinum majus*) was tested in one season using a 4-week solarization period and a 6-week period in the second season (McSorley et al. 2009). The snapdragon plant mortality in the first year was equivalent to the number lost in the untreated control, but after the 6-week period, the incidence was equivalent to that found in methyl bromide-treated plots. Neither of the solarization treatments resulted in an increase in marketable yield over the nontreated control.

One of the advantages of soil solarization is its compatibility with other disease management inputs. In the vegetable study mentioned above, the severity of root galling, caused by *Meloidogyne* spp., was lower when soil solarization was combined with 1,3-dichloropropene + chloropicrin applied under a gas-impermeable film (Chellemi et al. 1997).

In many other countries, an approach referred to as biosolarization is used. For example, a combination of soil solarization and raw or pelletized poultry manure amendments was evaluated for the control of root-knot nematode (*Meloidogyne incognita*) in carnation (*Dianthus caryophyllus*) grown in beds under plastic-covered greenhouse conditions in southern Spain. Soil solarization alone did not provide sufficient control of root-knot nematode, because the carnation growing season in this region only partly coincides with the most effective period for

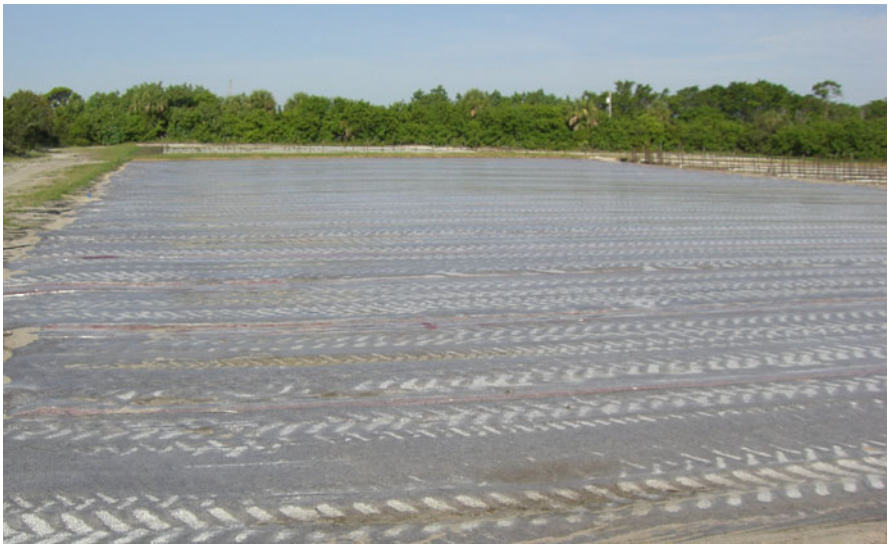


Fig. 18 Full-field solarization using solid tarp requires that tarps are glued together in a manner similar to that used for chemical fumigation (E. Rosskopf. USDA.)

solarization, resulting in an insufficient duration of treatment. The combination of soil solarization and raw or pelletized poultry manure was slightly less effective than chemical fumigation for control of this pathogen but crop yields after 9 months were similar. However, the higher root gall indices observed after 9 months, in comparison with chemically fumigated plots, indicated the need for a reapplication of the organic manure at the start of each successive growing season (Melero-Vara et al. 2012).

The results of this study (Melero-Vara et al. 2012) as well as those reported by Wang and McSorley (2008) indicated above point to one of the weaknesses of the use of solarization that nematode suppression is short-lived under solarization in comparison to fumigation in many cases, and the control does not result in increased crop yields. Solarization in FL is particularly unreliable when there are heavy summer storms (Wang et al. 2004). Full-field tarping for solarization (Fig. 18), which is commonly used in some locations during hot and dry conditions (Katan et al. 1987), is risky in FL due to the potential for loss of the plastic, or for water to pool in wheel ruts (Figs. 19, 20, and 21), or ineffectiveness of solarization at field edges (Grinstein et al. 1995) where inadequate heating can occur, resulting in poor control of weeds and diseases.

One approach to overcoming the expense of steaming and the variability of control associated with solarization is to combine these. In trials conducted in CA on calla lily, steam combined with solarization resulted in greater suppression than either method alone (Rainbolt et al. 2013).



Fig. 19 Utilization of solid-tarp solarization is hampered by the need to treat soil during the rainy season, which can result in water pooling in tractor tire tracks, which reduces soil heating in these areas (E. Rosskopf. USDA.)



Fig. 20 When water pools on *top* of the solarization plastic, the reduction in soil heating can lead to the survival and rapid spread of weeds (E. Rosskopf. USDA.)



Fig. 21 The effects of reduced soil heating from solarization when water pooled in tire tracks under the solarization can be seen in the subsequent crop; in this case, in increased incidence of Pythium damping off in snapdragon (E. Rosskopf. USDA.)

4.4 Anaerobic Soil Disinfestation

Anaerobic soil disinfestation (ASD), a nonchemical alternative to MB, was developed in Japan (Momma 2008) and the Netherlands (Blok et al. 2000) to control soilborne pathogens and nematodes in strawberries and vegetables. ASD integrates principles behind solarization and soil saturation to control nematodes and pathogens in situations where neither is effective or feasible when applied alone (Rosskopf et al. 2015). Anaerobic soil conditions are created during the ASD process when a readily available carbon-source is incorporated into topsoil (Figs. 22 and 23), covered with a plastic tarp and then irrigated to field capacity. The tarp is left in place to maintain soil moisture above field capacity and to sustain anaerobic conditions. Anaerobic decomposers are then able to respire using the added carbon, which results in the build-up of anaerobic by-products that are toxic to pathogens (Katase et al. 2009). These products are degraded rapidly once the tarp is removed or holes are punched through the tarp for planting. Studies were conducted over the past 4 years to optimize ASD for use in California strawberry and Florida vegetable and ornamental production systems. Overall, ASD was effective in suppressing



Fig. 22 Application of rice bran as the carbon source for anaerobic soil disinfestation in California (J. Muramoto © 2017. All Rights Reserved.)

Verticillium dahliae in soils and resulted in 85–100% of the marketable fruit yield observed with fumigated controls in coastal California strawberries when 20 t ha^{-1} of rice bran was pre-plant incorporated and 7.5–10 cm of irrigation was applied in sandy-loam to clay-loam soils (Shennan et al. 2011). In the cooler conditions of the central coast of California, however, ASD may not provide effective control of many weed species (Fennimore et al. 2013). To ensure consistency of pest suppression across varying locations, effects of soil temperatures and treatment length, and the mechanisms of pest suppression by ASD are being further studied. Integration of ASD with other nonfumigant approaches may also have promise. For example, a combination of ASD and mustard seed meal application is currently being tested (Shennan and Muramoto unpublished). Given the fact that much of the flower production in California is near the coast and that ASD does not provide effective weed control in coastal California conditions, control of volunteer flower bulbs and weeds in CA flower fields with ASD will likely be problematic (Fennimore et al. 2013).

In FL, ASD has proven very effective in the field for parasitic nematode control in specialty crops including cut-flowers (Rosskopf et al. 2010b; Butler et al. 2012a, b). The anaerobic conditions are lethal to nematodes and other soilborne pests and pathogens. Experiments were done in Florida to test the use of summer cover crops which could be grown in fields and then incorporated into soil as the organic component of ASD treatments. Several legumes and grasses including pearl millet (*Pennisetum glaucum*), sorghum-sudangrass (*Sorghum bicolor x S. bicolor* var.



Fig. 23 Incorporation of rice bran and bed formation conducted in a single pass in preparation for anaerobic soil disinfestation in CA (J. Muramoto © 2017. All Rights Reserved.)



Fig. 24 On-farm application of molasses as the carbon source for anaerobic soil disinfestation in FL (E. Rosskopf. USDA.)

sudanese), cowpea (*Vigna unguiculata*), and sunn hemp (*Crotalaria juncea*) were grown and then incorporated into soil. These were then compared to molasses, a standard ASD amendment, and a nonamended control. Root galling by the root-knot nematode *M. incognita* was low among all cover crop treatments except pearl millet, which had the highest levels of galling (Butler et al. 2012a). This was most likely due to the other crops tested as amendments (sorghum-sudangrass, cowpea, and sunn hemp) being nonhosts or poor hosts of *M. incognita* compared to pearl millet, which has been reported as a good host (McSorley 1999; McSorley et al. 1994).

Although the generation of organic acids is the most plausible control mechanism for plant-parasitic nematodes during ASD treatment (Katase et al. 2009), the generation of other by-products during amendment decomposition or shifts in microbial populations which favor naturally occurring biocontrol organisms may also play a role (McBride et al. 2000). Further field studies which evaluated ASD combined with soil solarization as an alternative to methyl bromide fumigation for plant parasitic nematode and soil-borne pathogen control defined in more detail the inputs necessary to make the system effective for nematode control (Butler et al. 2012b). In cut flowers in particular, ASD utilizing molasses (Fig. 24) as the labile carbon source, addition of composted broiler litter, soil saturation, and covering with a clear, gas-impermeable film resulted in cut flower yields that were equivalent to or greater than methyl bromide for the first crop (Rosskopf et al. 2010c. Delphinium and *Dianthus* yields under ASD exceeded those produced using steam and were equivalent to methyl bromide, but snapdragons were better yielding with methyl bromide than from any other soil treatment.

5 Summary

A significant amount of progress has been made in the discovery and development of alternatives to methyl bromide for soil disinfestation. Several of the methods discussed here have proven efficacy against a wide range of soilborne pests associated with production of ornamental crops, but the most consistent approaches still include the application of chemical fumigants. For the treatment of potting mixes, there are limited options and the use of steam and dazomet provide the most consistent results. California cut flower growers are likely to continue using chloropicrin in areas where fumigation is allowed, while implementing steam disinfestation in sensitive sites. The FL in-ground industry will continue to depend on the development of new chemical fumigants as the use of 1,3-D and chloropicrin is allowed in limited production areas. Significant advances in steam technology may increase its application on a larger scale in the future, but it is likely that future approaches to soil disinfestation will require the use of integrated approaches such as those used in ASD. Significant advances continue to be made on this and other nonfumigant approaches that will increase feasibility and implementation on a wider scale.

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Sanitation for Management of Florists' Crops Diseases

9

Warren E. Copes

Abstract

Sanitation involves efforts aimed to prevent introduction of pathogen propagules into a production facility, remove sources of propagules from active production areas to substantially slow disease development, and eliminate pathogen propagules from future production areas. Sanitation includes many practices such as using disease-free certified seed and propagative material, maintaining weed-free zones around production areas, use of screens to block entry of insects, use of double doors and foot paths, cleaning and sanitizing production areas between crops, sanitizing tools and equipment, and early removal of diseased plant material. General sanitation should be a matter of routine good agricultural practices. Because it is so important to do a thorough job, key sanitation practices should be selected that control the pathogens that routinely affect plants at a facility and those that are prevalent and would severely impact production if introduced. Good records are an important tool to select which pathosystems are problems, to determine cost benefit decisions, and to select subject matter for worker training modules. The information in this chapter represents an overview of methods known to be effective.

Keywords

Sanitation • Sanitize • Disinfect • Disinfectant • Disinfest • Disinfestant • Cleanliness

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R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_9

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1 Introduction

The plant pathogens that cause plant diseases include fungi, oomycetes, bacteria, viruses, and nematodes. These organisms are spread as various types of propagules (mycelia, conidia, ascospores, basidiospores, chlamydospores, sclerotia, zoospores, sporangia, oospores, bacterial cells, virus particles, viroid particles, nematodes, and nematode eggs). The propagules can be in an active growing state, which is easier to kill, or in a resting state, which can be difficult to kill. Even though the vast majority of propagules have no ability to move under their own power, plant pathogens are very adept at passively taking advantage of the mobility of other creatures and the forces of nature to move short and long distances. Propagules are often produced in large numbers and may be carried by air currents, water, insects, animals, humans, equipment, or contaminated plant tissue. For the most part, the only possibility of detecting their movement is by seeing plants with symptoms that may contain pathogen structures. The biology and epidemiology of pathogens are extremely diverse. In the context of plant production, not understanding these processes can cause devastating financial losses.

For disease to occur, three main components are needed: a susceptible plant, a virulent pathogen, and a favorable environment; this represents the classic disease triangle. Sanitation targets the second component of the disease triangle, by engaging in a direct assault on the pathogen. Sanitation can be performed at multiple production stages to achieve several goals: preventing introduction of pathogen propagules into a production facility, removing sources of propagules from active production areas to substantially slow disease development, and eliminating pathogen propagules from future production areas. Sanitation practices involve generating clean plant material (e.g., plant indexing, chemical or heat treatment); sanitizing

production areas, tools, and equipment (e.g., discarding infected plant tissues, washing, and the use of disinfestants, fungicides, and bactericides); and using barriers to restrict entry to specific parts of a facility (e.g., screening of fan and vent openings, protective doorways, positive pressure houses, weed-free zones, distance, wash stations, and work flow patterns). Sanitation is a proven disease management method in ornamental plant production (Daughtrey and Benson 2005) and can be one of the most reliable control measures available because it can completely or nearly eliminate the pathogen at strategic points. Economically, it is unreasonable to apply all these procedures to all production areas, so a strategy is required that identifies critical production points that are responsive to select control practices (see Sect. 13).

The information in this chapter represents an overview of methods known to be effective. Where they exist, specific disease control recommendations that include sanitation will be given in individual florists' crop chapters. Although sanitation is perceived as simple control methods, information that fully addresses all the complexities is not always available and that will be evident in the presentation of some topics.

2 Disinfestants

Disinfestants are biocides that kill microorganisms on the surface of living and dead tissues and on inert materials, such as wood products, galvanized metal benches, plastic containers, etc. Safety and environmental regulations pertain to these products just as to other pesticides. Recommendations listed on disinfestant labels should be followed. The disinfestants commonly used in plant production are listed in Tables 1–8. Each product has advantages and disadvantages that affect their suitability for particular microorganisms, uses, and environmental conditions. Relevant usage and efficacy information are given in Tables 1–8 based on available data. The following section will focus on factors affecting the efficacy of disinfestants.

A common misconception is that disinfestants are broad spectrum sterilants; that kill all microorganisms present. Microorganisms vary in sensitivity to any given chemical, and as a result different disinfestants and/or rates may be needed to properly inactivate or kill propagules of a specific pathogen genus or species. This information is not broadly documented but has been clearly demonstrated (Koponen et al. 1992; Mebalds et al. 1996, 1997; Nichols and Jodon 1972). Mebalds et al. (1997) reported that the lethal activity of sodium hypochlorite varied from 0.2% to 5.25% for viruses, 1% to 10% for fungi, and from 10% to 12.5% for bacteria. Additionally, microorganisms can vary in their sensitivity to disinfestants due to environmental conditions (e.g., temperature, pH, dissolved oxygen). Microorganisms generally absorb disinfestants rapidly between 20 °C (68 °F) and 30 °C (86 °F). Use disinfestant recommendations developed for specific microorganisms, when available. The term "inactivate" is commonly used in scientific literature because the term addresses uncertainties in measuring recovery and viability of microorganisms and in dealing with different organisms' habits, such as viruses

Table 1 Summary information for chlorine releasing disinfestants

Classification	Halogen releasing, oxidizing agents	Percent a.i.	8.25 for bleach products (sodium hypochlorite as example)
Active ingredient (a.i.)	Sodium hypochlorite, calcium hypochlorite, or chlorine gas (chlorine releasing agents, CRA); chlorine dioxide (has different properties than the CRAs)	Rate range	7% bleach solution (8.25% NaOCl) (=1 part bleach : 14 parts water; as example)
Trade name	Chlorox® and many bleach products (sodium hypochlorite as example) Chlorine gas is a different product Chlorine dioxide is a different product	Contact time	10–15 min

pH range of good activity		Water hardness activity[a]	
Hypochlorite and Cl^- gas:	5.0–7.0	Hypochlorite and Cl^- gas:	++
Chlorine dioxide:	2.0–10.0	Chlorine dioxide:	–
Corrosiveness[b]	Residual activity[c]	Phytotoxicity[d]	
Hypochlorite and Cl^- gas: ++	–	Hypochlorites and Cl^- gas:	+
Chlorine dioxide: +	–	Chlorine dioxide:	±

Human concerns	Volatile, irritating to skin and eyes
User sites	Greenhouses, pots, and benches need specified contact time of 10 min; dip pruners for 10 s and allow to dry (one source says to not rinse pruners, another says to rinse tools like pruners because residue may cause phytotoxicity on sensitive plant species); irrigation systems (flooded floors, flooded benches, recycled water systems, capillary mats, humidification, and misting systems)

Comments	Use product labeled for greenhouse use. Mix fresh sodium hypochlorite solutions every 2 h because efficacy will drop as chlorine gas is lost at the liquid surface. Chemical decomposition of a.i. increases above pH 7.0, above 15.6 °C (60 °F), in the presence of inorganic (e.g., nitrogen, potassium, iron, manganese, sulfur, arsenic) and organic compounds, and due to the exposure to sunlight. The microorganism's response rate to inactivation diminishes as the temperature drops moderately below 20 °C (68 °F) and rapidly above 37.8 °C (100 °F). At higher concentrations bleach can be sporicidal. The active moiety of hypochlorite and Cl^- gas is hypochlorous acid that has activity by electrophilic substitution (chlorination) and oxidation. The active moiety of chlorine dioxide is dissolved chlorine dioxide gas that has activity by oxidation. Chlorine dioxide reacts slower with many inorganic and organic compounds, is less corrosive, and has smaller changes in activity due to temperature than hypochlorous acid					
Pathogens controlled[e]	Fungi	Vegetative bacteria	Bacterial spores	Enveloped viruses	Nonenveloped viruses	Algae
Level of control	+	+	+	+	+	+

[a]Water hardness reactivity with a disinfestant resulting in loss of sanitizing activity as categorized by symbols: (++) high demand, (+) moderate demand, (±) weak demand, (−) no measurable demand, (NIF) no information found

[b]Corrosiveness as categorized by symbols: (++) highly corrosive, (+) moderately corrosive, (±) somewhat corrosive, (−) weakly corrosive, (NIF) no information found

[c]Residual activity as categorized by symbols: (+) hours of residual activity after the surface dries, (±) short-lived residual activity after the surface dries, (−) no residual activity after the surface dries, (NIF) no information found

[d]Phytotoxicity as categorized by symbols: (++) high risk of phytotoxicity, (+) potential to be phytotoxic, (±) phytotoxicity unlikely but possible, (−) infrequently phytotoxic, (NIF) no information found

[e]Susceptibility of microorganisms to chemical disinfestants as categorized by symbols: (++) highly effective, (+) effective, (±) limited or variable, (−) no measurable activity, (NIF) no information found

Table 2 Summary information for peroxygen compounds

Classification	Peroxygen, oxidizing agents			Percent a.i.	27.1% and 2% (ZerTol® 2.0 as example)	
Active ingredient (a.i.)	Hydrogen dioxide (=hydrogen peroxide), and peroxyacetic acid (ZerTol® 2.0 as example)			Rate range	3.9–19.5 ml/L (0.5–2.5 fl oz/gal) Rate depends on use, so read label (ZerTol® 2.0 as example)	
Trade name	ZerTol® 2.0, Sanitdate, Oxidate®, X3™			Contact time	1–10 min	
pH range of good activity		4–6.5	Water hardness activity[a]		NIF	
Corrosiveness[b]	+	Residual activity[c]	±	Phytotoxicity[d]	±	
Human concerns	Concentrated form can cause irreversible eye damage, skin irritant, potentially fatal if swallowed					
User sites	Benches, work areas, pots, flats, trays, cutting tools, foot bath mats, evaporative coolers, irrigation systems (flooded floors, flooded benches, recycled water systems, capillary mats, humidification and misting systems), soil or media drench, plant material (foliage, cuttings, stock, seed) seed beds, soil treatment					
Comments	Can be used in chemigation as it is compatible with macronutrients (e.g., nitrogen, phosphorus, potassium); however, not recommended with chelated micronutrients (e.g., copper, manganese, zinc). High organic and biological loads can place demand on the product. Peroxyacetic acid contains acetic acid that can lower water pH if water has a low buffering capacity (alkalinity). Never tank mix with pesticides. Slightly corrosive to aluminum					
Pathogens controlled[e]	Fungi	Vegetative bacteria	Bacterial spores	Enveloped viruses	Nonenveloped viruses	Algae
Level of control	+	+	+[f]	+	±	++

[a]Water hardness reactivity with a disinfestant resulting in loss of sanitizing activity as categorized by symbols: (++) high demand, (+) moderate demand, (±) weak demand, (−) no measurable demand, (NIF) no information found

[b]Corrosiveness as categorized by symbols: (++) highly corrosive, (+) moderately corrosive, (±) somewhat corrosive, (−) weakly corrosive, (NIF) no information found

[c]Residual activity as categorized by symbols: (+) hours of residual activity after the surface dries, (±) short-lived residual activity after the surface dries, (−) no residual activity after the surface dries, (NIF) no information found

[d]Phytotoxicity as categorized by symbols: (++) high risk of phytotoxicity, (+) potential to be phytotoxic, (±) phytotoxicity unlikely but possible, (−) infrequently phytotoxic, (NIF) no information found

[e]Susceptibility of microorganisms to chemical disinfestants as categorized by symbols: (++) highly effective, (+) effective, (±) limited or variable, (−) no measurable activity, (NIF) no information found

[f]Hydrogen peroxide combined with a strong oxidizing agent, such as peracetic acid, is sporicidal

Table 3 Summary information for quaternary ammonium disinfestants

Classification	Quaternary ammonium compounds (QAC), cationic surfactants			Percent a.i.	7.5% (KleenGrow as an example)
Active ingredient (a.i.)	Didecyl dimethyl ammonium chloride (KleenGrow as an example), many different QAC active ingredients are available			Rate range	(KleenGrow as an example) 7.8 ml/L (1.0 fl oz/gal) nonporous surfaces. Range of 0.23–15.6 ml/L (0.03–2.0 fl oz/gal) depending on use, so read label
Trade name	Consan Triple Action 20, Formula 409 Orange Cleaner Degreaser, Formula 409 Antibacterial All Purpose Cleaner, Green-Shield®, KleenGrow®, Lysol Disinfectant Antibacterial Kitchen Cleaner, MAQUAT 128-MT, Physan 20®, Simple Green d Pro 3, others			Contact time	10–15 min
pH range of good activity		Good: >7 Best: 9 to 10 Poor: <3.5	Water hardness activity[a]		+ Third generation QACs, such as Green Shield, and earlier have reduced efficacy at the upper range, greater than 200 mg/L calcium carbonate ± Newer (4th generation) QACs maintain activity in the upper range
Corrosiveness[b]	±	Residual activity[c]	+	Phytotoxicity[d]	±
Human concerns	Corrosive, eye and skin irritant that can cause burning and tissue damage depending on product. Harmful if swallowed				
User sites	Nonporous surfaces: packing facility, equipment, greenhouse walls, trucks, trailers, transportation equipment, plastic containers, benches, walkways, tools, plant, irrigation lines				

(*continued*)

Table 3 (continued)

Comments	Later generation QAC, like KleenGrow, are more germicidal, less foaming, and more tolerant to organic loads. Similar, but not equal, efficacy between commercial QAC products. Plant application is label specific. Poor activity on porous materials, such as wood benches and in presence of soap					
Pathogens controlled[e]	Fungi	Vegetative bacteria	Bacterial spores	Enveloped viruses	Nonenveloped viruses	Algae
Level of control	+	++	–	±	–	+

[a]Water hardness reactivity with a disinfestant resulting in loss of sanitizing activity as categorized by symbols: (++) high demand, (+) moderate demand, (±) weak demand, (−) no measurable demand, (NIF) no information found

[b]Corrosiveness as categorized by symbols: (++) highly corrosive, (+) moderately corrosive, (±) somewhat corrosive, (−) weakly corrosive, (NIF) no information found

[c]Residual activity as categorized by symbols: (+) hours of residual activity after the surface dries, (±) short-lived residual activity after the surface dries, (−) no residual activity after the surface dries, (NIF) no information found

[d]Phytotoxicity as categorized by symbols: (++) high risk of phytotoxicity, (+) potential to be phytotoxic, (±) phytotoxicity unlikely but possible, (−) infrequently phytotoxic, (NIF) no information found

[e]Susceptibility of microorganisms to chemical disinfestants as categorized by symbols: (++) highly effective, (+) effective, (±) limited or variable, (−) no measurable activity, (NIF) no information found

Table 4 Summary information for alcohol disinfestants

Classification	Alcohols			Percent a.i.	70–90% ethanol, >90% less effective 60–95% ispropanol	
Active ingredient (a.i.)	Ethanol or isopropanol			Rate range	Product used directly	
Trade name	Many names			Exposure time	5–10 min; tools 10 s for select pathogens	
pH range of good activity		NIF[a]	Water hardness activity[a]		NIF	
Corrosiveness[b]	–	Residual activity[c]	–	Phytotoxicity[d]	NIF	
Human concerns	Irritating to injured skin					
User sites	Propagation tools, e.g., knives and pruning tools, allow to dry, do not rinse					
Comments	Flammable, evaporates quickly, greater than 90% ethanol is less effective because water content is too low for required activity. Limited activity in presence of high concentrations of organic matter. Ineffective against some viruses on metal tools. Can damage rubber and plastic. Phytotoxic to plants					
Pathogens controlled[e]	Fungi	Vegetative bacteria	Bacterial spores	Enveloped viruses	Nonenveloped viruses	Algae
Level of control, Ethanol	+	++	–	+	+	NIF
Level of control, Isopropanol	+	++	–	+	–	NIF

[a]Water hardness reactivity with a disinfestant resulting in loss of sanitizing activity as categorized by symbols: (++) high demand, (+) moderate demand, (±) weak demand, (−) no measurable demand, (NIF) no information found

[b]Corrosiveness as categorized by symbols: (++) highly corrosive, (+) moderately corrosive, (±) somewhat corrosive, (−) weakly corrosive, (NIF) no information found

[c]Residual activity as categorized by symbols: (+) hours of residual activity after the surface dries, (±) short-lived residual activity after the surface dries, (−) no residual activity after the surface dries, (NIF) no information found

[d]Phytotoxicity as categorized by symbols: (++) high risk of phytotoxicity, (+) potential to be phytotoxic, (±) phytotoxicity unlikely but possible, (−) infrequently phytotoxic, (NIF) no information found

[e]Susceptibility of microorganisms to chemical disinfestants as categorized by symbols: (++) highly effective, (+) effective, (±) limited or variable, (−) no measurable activity, (NIF) no information found

Table 5 Summary information for potassium peroxymonosulfate plus sodium chloride

Classification	Oxidizing agents			Percent a.i.	21.41% plus 1.5%	
Active ingredient (a.i.)	Potassium peroxymonosulfate plus sodium chloride			Rate range	1:100 dilution [2.7 ml/L (1.3 fl oz/gal)] 1:50 dilution if not precleaned	
Trade name	Virkon-S®			Exposure time	10 min	
pH range of good activity		NIF	Water hardness activity[a]		+	
Corrosiveness[b]	–	Residual activity[c]	–	Phytotoxicity[d]	+	
Human concerns	Concentrated form is corrosive; causes irreversible eye damage and skin burn. 1% solutions are nonirritating to the eye and skin					
User sites	Inanimate environmental surfaces, such as floors, walls, glasshouse structures, ventilation equipment, other equipment, tools, containers, flats, trays, evaporative pads, vehicles					
Comments	Not approved in California for use against plant pathogens. Efficacy of 1% solution was determined in 400 ppm hard water and 5% organic matter. Solutions are stable for 7 days. Ventilate buildings					
Pathogens controlled[e]	Fungi	Vegetative bacteria	Bacterial spores	Enveloped viruses	Nonenveloped viruses	Algae
Level of control	+	+	+	+	±	+

[a]Water hardness reactivity with a disinfestant resulting in loss of sanitizing activity as categorized by symbols: (++) high demand, (+) moderate demand, (±) weak demand, (−) no measurable demand, (NIF) no information found

[b]Corrosiveness as categorized by symbols: (++) highly corrosive, (+) moderately corrosive, (±) somewhat corrosive, (−) weakly corrosive, (NIF) no information found

[c]Residual activity as categorized by symbols: (+) hours of residual activity after the surface dries, (±) short-lived residual activity after the surface dries, (−) no residual activity after the surface dries, (NIF) no information found

[d]Phytotoxicity as categorized by symbols: (++) high risk of phytotoxicity, (+) potential to be phytotoxic, (±) phytotoxicity unlikely but possible, (−) infrequently phytotoxic, (NIF) no information found

[e]Susceptibility of microorganisms to chemical disinfestants as categorized by symbols: (++) highly effective, (+) effective, (±) limited or variable, (−) no measurable activity, (NIF) no information found

Table 6 Summary information for phenolic disinfestants

Classification	Phenols			Percent a.i.	See product label	
Active ingredient (a.i.)	o-phenylphenol			Rate range	See product label	
Trade name	Several products			Exposure time	10 min	
pH range of good activity		NIF	Water hardness activity[a]		–	
Corrosiveness[b]	NIF	Residual activity[c]	±	Phytotoxicity[d]	NIF	
Human concerns	Eye and skin irritant. Penetrates latex gloves					
User sites	Equipment, containers, flats, benches, walkways					
Comments	Has activity in hard water and in presence of organic matter. Good activity in presence of soaps, commonly formulated with soap to increase penetration ability. A 2% concentration is highly toxic to animals, especially cats. Household products: Lysol (contains phenol plus quaternary ammonium compound)					
Pathogens controlled[e]	Fungi	Vegetative bacteria	Bacterial spores	Enveloped viruses	Nonenveloped viruses	Algae
Level of control	+	++	–	±[f]	–	NIF

[a]Water hardness reactivity with a disinfestant resulting in loss of sanitizing activity as categorized by symbols: (++) high demand, (+) moderate demand, (±) weak demand, (−) no measurable demand, (NIF) no information found

[b]Corrosiveness as categorized by symbols: (++) highly corrosive, (+) moderately corrosive, (±) somewhat corrosive, (−) weakly corrosive, (NIF) no information found

[c]Residual activity as categorized by symbols: (+) hours of residual activity after the surface dries, (±) short-lived residual activity after the surface dries, (−) no residual activity after the surface dries, (NIF) no information found

[d]Phytotoxicity as categorized by symbols: (++) high risk of phytotoxicity, (+) potential to be phytotoxic, (±) phytotoxicity unlikely but possible, (−) infrequently phytotoxic, (NIF) no information found

[e]Susceptibility of microorganisms to chemical disinfestants as categorized by symbols: (++) highly effective, (+) effective, (±) limited or variable, (−) no measurable activity, (NIF) no information found

[f]Varies with composition of the disinfestant

Table 7 Summary information for aldehyde disinfestants

Classification	Aldehydes			Percent a.i.	50%	
Active ingredient (a.i.)	Glutaraldehyde			Rate range	2% (w/v)	
Trade name	Glutex GS1, Ucarcide			Exposure time	10–30 min	
pH range of good activity		Best above 7	Water hardness activity[a]		–	
Corrosiveness[b]	–	Residual activity[c]	±	Phytotoxicity[d]	NIF	
Human concerns	Highly irritating, may cause severe irritation with corneal injury which may result in permanent impairment of vision, even blindness. Chemical burns may occur. Brief skin contact may cause burns. Toxic to humans and animals with contact or inhalation, potentially carcinogenic					
User sites	Glutex GS1 can be used in farm animal and poultry housing facilities and on industrial equipment and buildings. Ucarcide can be used as an active ingredient in commercial household products					
Comments	Noncorrosive to metals, rubber, plastic, and cement. Glutaraldehyde is more efficacious in the presence of organic matter, soap, and hard water than formaldehyde; however, it should be applied to precleaned surfaces. Can be diluted with available water without concern of water hardness. Application can be by fogging, atomizing, mopping, or spraying.					
Pathogens controlled[e]	Fungi	Vegetative bacteria	Bacterial spores	Enveloped viruses	Nonenveloped viruses	Algae
Level of control, Glutaraldehyde	+	++	–	++	+	NIF
Level of control, Formaldehyde	+	++	±[f]	++	+	NIF

[a]Water hardness reactivity with a disinfestant resulting in loss of sanitizing activity as categorized by symbols: (++) high demand, (+) moderate demand, (±) weak demand, (−) no measurable demand, (NIF) no information found

[b]Corrosiveness as categorized by symbols: (++) highly corrosive, (+) moderately corrosive, (±) somewhat corrosive, (−) weakly corrosive, (NIF) no information found

[c]Residual activity as categorized by symbols: (+) hours of residual activity after the surface dries, (±) short-lived residual activity after the surface dries, (−) no residual activity after the surface dries, (NIF) no information found

[d]Phytotoxicity as categorized by symbols: (++) high risk of phytotoxicity, (+) potential to be phytotoxic, (±) phytotoxicity unlikely but possible, (−) infrequently phytotoxic, (NIF) no information found

[e]Susceptibility of microorganisms to chemical disinfestants as categorized by symbols: (++) highly effective, (+) effective, (±) limited or variable, (−) no measurable activity, (NIF) no information found

[f]Formaldehyde is sporicidal, glutaraldehyde is not

Table 8 Summary information for ozone disinfestants

Classification	Oxidizing agent			Percent a.i.	1% (10,000 mg/L)	
Active ingredient (a.i.)	Ozone			Rate range	0.4–5 mg/L (ppm)	
Trade name	Many product names			Exposure time	4–20 min	
pH range of good activity	Best: 4–6 Good: 6–8.5		Water hardness activity[a]		–	
Corrosiveness[b]	++	Residual activity[c]	–	Phytotoxicity[d]	±, unlikely from treated water, but possible from escaped ozone near the generator	
Human concerns	Off-gases are extremely irritating and possibly toxic, so off-gases must be captured and destroyed to prevent worker exposure					
User sites	Irrigation systems (flooded floors, flooded benches, recycled water systems, capillary mats)					
Comments	Ozone generally has higher germicidal action than other disinfestants and results in increased dissolved oxygen concentration in the receiving water stream. Must be generated on site. Equipment and operating costs are high. Excess ozone must be continuously monitored and recycled within the system or destroyed. High demand occurs with high levels of suspended solids, biochemical oxygen demand, chemical oxygen demand, and organic carbon. Efficiency is reduced with increased water temperature due to slower mass transfer in water					
Pathogens controlled[e]	Fungi	Vegetative bacteria	Bacterial spores	Enveloped viruses	Nonenveloped viruses	Algae
Level of control, Glutaraldehyde	+	++	+	++	+	NIF

[a]Water hardness reactivity with a disinfestant resulting in loss of sanitizing activity as categorized by symbols: (++) high demand, (+) moderate demand, (±) weak demand, (−) no measurable demand, (NIF) no information found

[b]Corrosiveness as categorized by symbols: (++) highly corrosive, (+) moderately corrosive, (±) somewhat corrosive, (−) weakly corrosive, (NIF) no information found

[c]Residual activity as categorized by symbols: (+) hours of residual activity after the surface dries, (±) short-lived residual activity after the surface dries, (−) no residual activity after the surface dries, (NIF) no information found

[d]Phytotoxicity as categorized by symbols: (++) high risk of phytotoxicity, (+) potential to be phytotoxic, (±) phytotoxicity unlikely but possible, (−) infrequently phytotoxic, (NIF) no information found

[e]Susceptibility of microorganisms to chemical disinfestants as categorized by symbols: (++) highly effective, (+) effective, (±) limited or variable, (−) no measurable activity, (NIF) no information found

that only propagate in living host cells. The general term "kill" will be used in place of "inactivate" in this chapter.

Before applying any disinfestant, it is important to remove soil, organic matter, and plant parts from equipment, production surfaces, and tools for two reasons. First, the disinfestant is weakened when it reacts with soil and organic matter. This results in a reduction in the active ingredient (a.i.) and too little a.i. remaining to kill microorganisms, which is called a demand load. Second, the soil and organic matter can physically shield microorganisms from contact with a disinfestant.

Disinfestants can lose activity due to several reasons. Chemical decomposition of a.i. can occur in seconds to minutes in response to water characteristics (e.g., pH, concentrations of mineral and organic compounds, water hardness, temperature) and reactivity to production surfaces (metal, plastic, wood). These same factors reduce longevity of the a.i. when mixing disinfestant solutions used for soaking materials (such as containers), for spray application, and for treating irrigation water. Loss of a.i. can occur over a period of hours due to some environmental conditions (e.g., wind, sunlight). This information is presented in Tables 1–8 and varies from specific to general based on available research data. In a number of cases, additional research is needed to better define some of the knowledge gaps.

Howard et al. (2007) reported several interesting comparisons between disinfestants, even though no statistical analyses were presented, which included differences in pathogen sensitivities to disinfestants, differences between disinfestants in microbial lethality, corrosiveness and plant toxicity, and differences in efficacy when applied to different material surfaces. Several plant pathologists have demonstrated differences in disinfestant efficacy due to the material surface being treated (Copes and Hendrix 1996; Copes 2004; Koponen et al. 1992; Nichols and Jodon 1972). For example, the percent *Botrytis* conidia killed was nearly equal when spraying bleach on plastic, metal, and pressure-treated wood surfaces but less effective on untreated wood (Table 9) (Copes 2004). The quaternary ammonium compound (QAC) had the highest activity on metals, slightly less on plastics, and was ineffective on untreated wood. Hydrogen dioxide was ineffective at the preventative rate but uniformly effective on plastics, metals, and pressure-treated wood at the curative rate except on untreated wood. However, a total kill of *Botrytis* conidia on production surfaces required higher than label rates for the QAC and hydrogen dioxide, especially on untreated wood. Toro et al. (2012) found *Fusarium* spp. inoculum could be eliminated from wooden stakes with high pressure washing and several sanitizing agents, including NaOCl (Fig. 1).

While most of this information focuses on loss of disinfestant activity, pathogens can also develop resistance to these chemistries. Resistance to many classes of disinfestants has been well documented in bacteria, fungi, and viruses and can be intrinsic as a natural chromosomal property or acquired through mutation or acquisition of plasmids or transposons (McDonnell and Russell 1999). Resistance has been studied with human pathogens, food contaminants, food yeasts, industrial fouling, and water line biofilms but to the authors knowledge has not been documented with plant pathogens (Araújo et al. 2011; McDonnell and Russell 1999). If needed, consult a private or university plant disease clinic to have isolates

Table 9 Effect of disinfestant label rate and substrate on mortality of *Botrytis cinerea* calculated from a lethal dose response curve (Copes 2004)

	Chlorox[a]	Green Shield[b]		ZeroTol[c]	
	1 part to 10	3.6 ml/L (1 Tbs/gal)	4.9 ml/L (1 tsp/quart)	7.9 ml/L (1 fl oz/gal)	19.8 ml/L (2.5 fl oz/gal)
Substrate	Mortality (%)				
Polyethylene fabric	98[d]	65	70	2	75
Plastic container	99	75	80	6	75
Galvanized metal	99	75	90	8	75
Stainless steel	98	85	90	2	75
Treated wood	97	65	65	20	75
Nontreated wood	90	5	8	1	35

[a]Bleach [6% sodium hypochlorite (The Chlorox Co., Oakland, CA)]
[b]Green Shield [10% n-Alkyl(60%C14, 30%C16, 5%C12, 5%C18 dimethyl benzyl ammonium chloride and 10% n-Alkyl(68%C12, 32%C14) dimethyl ethylbenzyl ammonium chloride] (Whitmire Micro-Gen Research Laboratories, St. Louis, MO). The 3.6 ml/L (1Tbl product/gal) is labeled for greenhouse benches, containers, and irrigation water. The 4.9 ml/L (1 tsp product/qt) is labeled for cutting tools and greenhouse glass
[c]ZeroTol [30% hydrogen dioxide (Biosafe Systems, Glastonbury, CT); Older formulation that is no longer sold]. The 7.9 ml/L (1 fl oz product/gal) is labeled for greenhouse benches, containers, and irrigation water. The 19.8 ml/L (2.5 fl oz product/gal) is labeled for cutting tools and greenhouse glass
[d]Lethal dose values (with a variable unit range: 1–10 in 1 unit increments, 15–90 in 5 unit increments, and 91–100 in 1 unit increments) from statistical output not shown in Copes (2004)



Fig. 1 Contaminated wooden stakes that have not been cleaned and sterilized could serve as a source for pathogen spread when used in production (R.J. McGovern © 2017. All Rights Reserved.)

tested. If chemical resistance is suspected, a disinfestant of a different chemical class can be used.

When submerging products, such as clean containers free of organic matter, in a disinfestant solution, it is critical to also dislodge all air bubbles or air pockets by

shaking and tilting the containers so that air bubbles escape to the surface. Air pockets prevent the disinfestant solution from contacting that part of the container, as a result the containers are not being uniformly treated. For example, stacked containers can be orientated with the large opening up, the stack swirled and tilted to dislodge air bubbles, and a heavy screen laid on top to keep containers submerged.

In this chapter, disinfestants will be referred to by the *common chemical name* when referring to them collectively or generally and by trade names when referring to a specific product. *Chemical nomenclature* will not be used in this chapter. Bleach will be referred to as NaOCl, which is a chemical abbreviation for sodium hypochlorite the active ingredient (a.i.) in bleach, and rates will be referred to as percent a.i. of sodium hypochlorite. This is to avoid confusion caused by using percent bleach, since bleach products contain different percents of NaOCl.

In both general and scientific literature, the terms "disinfectant" and "disinfestant" will be found referencing the same products and the same chemical activities. The term "disinfestant" specifically means a product is inactivating pathogens only on the surface of organisms and materials, which more accurately reflects how these products work, and will be used exclusively in this chapter.

3 Surfaces of Tools, Equipment, and Structures

Washing and applying disinfestants are the main tools used to disinfest hard surfaces. Sunlight can also contribute to the inactivation of some pathogen propagules, particularly on structures and floors exposed to sunlight for an extended period (Copes 2015).

3.1 Tools

Tools become contaminated by contact with infested rooting media, soil, or plant material. Any tool (mechanical pruners, trowels, shovels, fertilizer dispersers, motorized shears, tagging machines, etc.) that may have been exposed to pathogens should be cleaned prior to moving to another site or block of plants. Contamination of cutting tools should be a constant concern, since they easily acquire and carry pathogen propagules from infested or diseased plants to healthy plants. Shovels and trowels should be sanitized also to prevent carrying pathogens into a noncontaminated area.

The steps in sanitizing tools are (1) washing or brushing surfaces free of soil, media, and plant tissue; (2) removing films and sap with a cleanser; and (3) dipping tools in the disinfestant. It is commonly recommended that tools be dipped for 2 min.

Bleach, QACs, peroxygen compounds, and alcohols are common disinfestants used for treating tools. Contaminated pruners dipped in a 0.525% NaOCl for 2 s killed *Erwinia amylovora* from infected apple stems, and for 1 min killed tobacco mosaic virus (TMV) from infected petunia (Keil and van der Zwet 1967;

Lewandowski et al. 2010). To minimize corrosiveness caused by bleach, all metal parts of the pruners need to be washed and coated with oil daily following bleach treatment. Alternative less corrosive choices include alcohol (isopropyl or ethanol) and QACs (Bennett et al. 2011). Some pathogens are harder to eliminate from tools. Immersing metal tools for 15 min in 0.525% a.i. NaOCl was consistently more effective than five other disinfestants at inactivating potato spindle tuber viroid (Olivier et al. 2015); however, none of the treatments eliminated the viroid. Dipping pruners in 20% wt/vol of nonfat dry milk plus 1% Tween 20 was as effective as 0.525% a.i. NaOCl in killing TMV obtained from petunia, but again, neither treatment totally prevented virus transmission to petunia on contaminated tools (Lewandowski et al. 2010). Immersion time influenced the rate required for both bleach and QAC to kill potato virus Y (PVY) particles on pruner blades (Wintermantel 2011). PVY particles were more consistently eliminated from pruners with a 2 s immersion in 0.525% NaOCl than a 15 s immersion in 1% QAC.

3.2 Plant Containers

Reuse of plant containers is risky, because pathogens could have produced aggregates of propagules, including difficult to kill survival structures that could tightly adhere to the container (Fig. 2). Pieces of potting mix or plant debris attached to the container can further protect pathogens from chemical disinfestants. Using new containers provides the surest way to avoid this type of contamination. Placing disposable plastic inserts into reused styrofoam transplant trays will also prevent pathogen transmission.

If soiled containers are to be reused, containers should be thoroughly washed to remove all soil, organic media, and plant tissue, which requires labor. Cleaned containers should be submerged in disinfestant for 10–30 min or treated with steam at 65.6 °C (150 °F) to 71.1 °C (160 °F) for 45–60 min. Removing debris by washing followed by soaking in a disinfectant was done in all of the following studies. Chase (2012) found QAC (KleenGrow) to be more effective at killing *Thielaviopsis basicola* spores than bleach, another QAC (GreenShield), or hydrogen peroxide (Zerotol). Warfield and Konczal (2003) killed *T. basicola* on plug trays either by spraying trays with a 1–50 ratio of ZeroTol to water solution or by submerging plug trays in 0.525% NaOCl for 10 min. *Thielaviopsis basicola* spores infesting flats used for growing tobacco seedlings were killed with steam (Chase and Daughtrey 2013; Choudhary et al. 2006). Spray application of NaOCl at 0.525% consistently reduced microconidial densities of *F. oxysporum* f. sp. *callistephi* on styrofoam to an undetectable level (Gilbert et al. 2007). Soaking plastic irrigation stakes in NaOCl or a QAC reduced populations of *F. oxysporum* f. sp. *radicis-lycopersici* to an undetectable level (Toro et al. 2012). McGovern et al. (1993) found *F. oxysporum* f. sp. *radicis-lycopersici* was reduced to an undetectable level from styrofoam trays with steam treatment (71.1 °C (160 °F) for 45 min) but not with QAC or bleach.



Fig. 2 Clean and soiled containers are stored together and pose an unnecessary risk for pathogen dissemination (W.E. Copes. USDA.)

3.3 Equipment (Conveyers, Trailers, Transplanters, etc.)

Preventing spread of pathogen propagules on large equipment to a new area where a susceptible crop is being grown is also critical. This applies to diverse pieces of equipment, such as automated seeding machines, conveyers, irrigation pipes, sprayers, tractors, trailers, and transplanters. Factors to consider include the likelihood that equipment is contaminated with a pathogen, the potential for crop loss, the rate of spread from the contaminated area, and the difficulty of eradicating the introduced pathogen. With seeders and transplanters, cleaning may be needed when plant types are changed. With conveyors, trailers, carts, and racks, cleaning and disinfesting equipment should be planned as part of the plant transfer activity after handling plants that have a history of disease and prior to handling plants prone to the same disease.

General sanitation recommendations follow, although more specific efforts may be required with certain pathogens including quarantine status pathogens. General guidelines may be available from the manufacturer, extension publications, national regulatory agencies such as the USDA Animal and Plant Health Inspection Service (APHIS), or international regulatory groups (see Sect. 6). Specific guidelines for quarantine status pathogens would be mandated by local, regional, national, or international regulatory agencies. Cleaning and sanitizing may be divided into six

steps: (1) dry removal of gross contamination and solids, (2) wet washing and solvent washing, (3) rinsing, (4) drying, (5) disinfesting, and (6) machine maintenance (Anon 2013). Decontamination of important pathogens should only be done where the contamination occurred to reduce the possibility of unintentionally spreading disease to off-site locations.

1. Use brushes, scrapers, gloved hands, and/or low-pressure air sprayers to remove organic and inorganic material adhering to equipment surfaces and filling crevices (Anon 2013; Gordon and Koike 2015). High-pressurized air should not be used for cleaning because of the increased potential over low-pressure air for disseminating fine particles outside of the cleaning area. Remove unattached and loose equipment and parts, such as car mats, and any equipment or supplies that may be sitting in compartments. Flooring, panels, and the under carriage of vehicles should be thoroughly checked and disinfested.
2. Proper cleaning and washing of surfaces are critical for fully exposing pathogen propagules to the activity of disinfestants. Wet washing involves scrubbing with a soap or detergent solution. Wash from the top downward, using a hose with normal water pressure, about 30–50 psi. High-pressure water streams should not be used, because the process could produce aerosols containing pathogen propagules. Allow 10 min of soaking to allow encrusted dirt to soak up the detergent, during which time other parts of the vehicle or equipment could be cleaned. Water and detergents will remove many of the pathogen propagules, finer organic and inorganic material, and films. Use an appropriate solvent to remove sticky and oily materials adhering to equipment surfaces. If wash water needs to be collected, see the paragraph on sanitizing stations.
3. Rinsing of vehicles, equipment, or machines should be done from the top down.
4. Drying reduces potential for dilution of disinfestants. Allow at least 5 min for a vehicle or piece of equipment to drip dry, after which the remaining water will not cause significant dilution of disinfestants.
5. Select a disinfestant that has proven activity against the pathogen being targeted and is not corrosive to equipment (Tables 1–8). Quaternary ammonium compounds have been used frequently because they are less corrosive (Bennett et al. 2011). Check product labels for necessary contact time and whether an adjuvant may improve activity. Reapplication of multiple light applications is required to maintain "shiny" wet surfaces for 10 min or the specified contact time, as merely damping surfaces is not adequate (Anon 2014). Surfaces should be thoroughly wetted and crevices flushed using a low-pressure sprayer. Porous and rough surfaces will require more disinfestant than smooth surfaces. A heavy application of disinfestant that runs off and puddles is not effective. All removed components and parts should be scrubbed with a disinfestant. The inside of vehicles (e.g., dashboard, steering wheel, handbrake, gear shift, and seats), and operator controller stations, should be wiped thoroughly with an appropriate disinfestant. Wheels, wheel wells, and underside of vehicles should be sprayed with a disinfestant.

6. Equipment maintenance will depend on the equipment and product used. Many disinfestants are corrosive to rubber gaskets and metallic parts in pumps and pressure washers. Disinfestants should be rinsed from the equipment as soon as the recommended contact time has passed. Normally disinfestants will include directions for equipment cleaning as part of the label instructions. If this information is not present, equipment should be flushed liberally with fresh water followed by warm water containing light mineral oil or liquid detergent (Anon. 2014). Additionally, consider lubricating bearings, connectors, and gears as appropriate for the equipment. At this time, all removed parts can be reassembled on the equipment or vehicle.

A sanitizing station is a containment area. A sanitizing station inside a facility should be set up on an impermeable floor. If the sanitizing operation is adjacent to plant production areas, a wall of plastic sheeting should be hung either from a temporary wall frame of wood or metal tubing or from overhead structures. Depending on the pathogen, water should be directed to flow into a drainage system or sump well for later treatment and removal if needed or contained in a barrier and mopped into containers for later treatment and removal. Drums or plastic containers can be used to contain spent cleaning and disinfestant fluids. The area needs to be cleaned and disinfested after equipment treatment has been completed.

An outside sanitizing station should be separated from normal vehicle traffic flow and plant production areas. The cleaning and disinfesting procedures should be done at the site where contamination by diseased crops occurred. Sanitizing stations should consist of an impermeable layer, such as a layer of plywood covered with thicker than 2 mil plastic sheet; a berm [e.g., 10.2 × 10.2 cm (4 × 4 in.) wood, sand tubes, sand bags, etc.]; framing materials to build the containment structure; sump pump and power supply; and drums or plastic totes to contain spent cleaning and disinfesting fluids.

3.4 Production and Greenhouse Structures

Sanitation begins outside the greenhouse. All weeds around production areas should be removed to prevent refuges for pathogens and insects that can transmit them (Fig. 3) (Hilje et al. 2001). Areas of grass can be planted for a 3.1 m (10 ft) distance around a greenhouse. Insect screening should be used to cover vent and fan openings to exclude insect vectors (Snehi et al. 2015). Screen mesh sizes should be selected to optimize insect exclusion while allowing adequate airflow (Bell and Baker 2000). Additionally, plastic sheets and screening are available that maintain high transmission of visible light while blocking portions of the ultraviolet spectrum, which interferes with the ability of insects to orient and find plant hosts (Hilje et al. 2001; Hochmuth and Sprenkel 2015). Insects can easily enter through the opening of hinged doors. A simple double door entrance with a small entryway can be used. An air-lock double door further allows a positive pressure balance between outside doors and pressure balance into the greenhouse facility (Hochmuth and Sprenkel 2015).



Fig. 3 Aquatic weeds in a recycled containment basin can serve as potential hosts and sources for pathogen disseminated in irrigation water (R.J. McGovern © 2017. All Rights Reserved.)

Foot baths should be located at access doors and used to prevent potential pathogen-infested soil and debris from being carried inside (Buonassisi et al. 2013; Dvorak 2008; Gullino et al. 2015). Foot baths vary from depressions in walkways to use of shoe covers that can be immersed in tubs. Prior to using a foot bath, rinse and wipe debris from shoes then immerse them in the solution for 30 s to 3 min depending on the disinfestant label. Disinfestant solutions in foot baths should be changed daily, since many products, such as QACs and peroxy compounds, are less effective when plant matter or soil is present. Phenols react less with organic matter.

Greenhouse interiors must be routinely sanitized, since some pathogens can survive for years in soil and over a month on metal, rubber, and wood surfaces (Buonassisi et al. 2013; Norman and Strandberg 2012). At least annually, wash upper greenhouse structures, walls, benches, and floors and treat with a disinfestant. For algal problems select disinfestants such as peroxy compounds, QACs, and bleach. QACs were very effective against fungal pathogens on most greenhouse surfaces, other than some synthetic materials, like polyethylene (Bennett et al. 2011; Mebalds et al. 1997). To sanitize greenhouse sections, remove all plants and organic and inorganic debris from benches and floors. This is best done routinely during production operations.

If soil floors exist under bench production, cover soil with gravel so no soil is exposed, because soil can harbor pathogens, shore flies, and fungus gnats. Areas with gravel can be first covered with ground fabric. Drips from condensation on the roof, from leaks in the roof, and from water lines can generate enough splash force to collect and carry pathogen propagules from the soil onto crops on benches or from container to container; therefore, the occurrence of drips should be minimized. Install enough hose-end holders for convenient storing of hose nozzles to keep them off the ground and uncontaminated. Hose nozzles and watering wands should never come in to contact with the floor and should be cleaned and allowed to air dry between uses.

Sanitation practices are easier to implement if pots and trays are set on top of benches. Due to the cost of benches, some businesses set pots and propagation trays

directly on polyethylene or gravel floors. In such cases, sanitation is more difficult, but still can be done. Organic matter should be removed because it can serve as a habitat and food source for pathogens (Copes 2015). The bulk of organic matter can be removed with a broom or blower. Brooms should be sanitized afterwards to prevent pathogen spread to other areas of the operation. Blower direction needs to be considered so pathogens are not blown out of the house into surrounding areas of the operation. Cleaning the floor and lower walls with soap and/or water further removes small particles of organic matter. Spray application of a disinfestant on the floor and walls further reduces persistence of pathogen populations on those surfaces.

In the case of high tunnel greenhouses and some shade houses, plants are grown directly in soil. A number of plant pathogens naturally survive in the soil environment. This paragraph provides a short summary of information that is covered in more depth elsewhere (Ajwa et al. 2003; El-Shami 1990; El-Shami et al. 1990a, b; Garmendia and Goicoechea 2011; Gupta et al. 2005; Paulitz and Bélanger 2001; Tanaka et al. 2003; Tello and Bello 2002; Walker and Thompson 1950). Populations of soil-borne plant pathogens can be controlled between crops using heat, fungicides, and/or fumigants. Soil is heated by injecting aerated steam into air spaces within the soil that is covered with plastic or enclosed in a container or by soil solarization that is an option in warmer climates with regular sunlight. Success in eliminating a pathogen population in soil will be affected by the pathogen's tolerance to heat, the maximum temperature attained, the length of time heat is maintained, and the ability to avoid recontamination of the treated area from surrounding soil. A general guideline to eliminate common pathogens in soil is temperatures of 60 °C (140 °F) to 70 °C (158 °F) for 30–60 min, although not all pathogens will be killed at those settings. Attaining those temperatures by soil solarization will require covering the soil with a double layer of transparent plastic. A single layer of plastic will generate lower temperatures and require durations of 4–6 weeks. An advantage of heat is plants can be planted immediately after temperatures drop. Fungicides, such as fosetyl-Al, iprodione, mefenoxam, quintozene, thiophanate-methyl, and triflumizole, can be applied to soil to provide a broad or specific control of fungal and oomycete pathogens. Biocontrol products that contain various organisms, such as *Gliocladium virens*, *Streptomyces griseoviridis*, or *Trichoderma* spp., can suppress certain pathogen populations. Soil fumigants, such as methyl bromide and/or chloropicrin, are restricted use materials that might be considered for moderate-to-severe pathogen infestations.

4 People

Workers who contact plants can transmit pathogens. Pathogens can stick to tools, equipment, shoes, clothes, and skin, including hands. Where ever a worker goes next, they have potential to spread pathogen propagules. Workers, managers, and visitors should be instructed to avoid casually touching plants and benches. Employees should receive in-house training to teach them how pathogens can be

accidentally spread by common production activities. Supervisors should reinforce that training to help make it common practice.

Washing hands is a good basic practice between tasks or stations. Contaminated hands of workers were found to be a major means of spreading *Fusarium oxysporum* f. sp. *radicis-lycopersici* within tomato transplant facilities (Menzies and Jarvis 1994). Each worker should have a single defined task and purpose when entering a production area, such as watering, scouting for diseases and arthropods, collecting cuttings, or moving plants. Before switching to another task, workers should clean up properly at a wash station.

The primary function of soaps and detergents is to remove organic matter, soil, grease, and various contaminants, including pathogens. The use of hot water and scrubbing helps remove contaminants. Use of latex, nitrile, or vinyl gloves provides a smoother surface from which spores can more easily be washed than from skin. Gloves also protect hands from harsh detergents, disinfestants, and other chemicals. Gloves add an additional cost that can be justified in high risk activities. Cost of over-clothing may only be justifiable for high risk diseases.

Spores of many pathogens are produced in a gelatinous substance that adheres to surfaces, including clothing as people walk through production areas. Spores of *Colletotrichum acutatum*, in numbers sufficient to cause disease on leatherleaf ferns, were recovered from cotton denim fabric; the spores stayed viable for up to 4 weeks (Norman and Strandberg 1997). If possible, workers should wear clean clothes to work each day or wear over-clothing such as aprons that are cleaned by the business. For serious disease problems, such as *Fusarium* wilt at tomato transplant production facilities, over-clothing (aprons, lab coats, Tyvek suits, boot covers) should be worn in high risk areas so clothing can be removed before working in other blocks. Over-clothing adds a level of protection that can be treated with a disinfestant or removed and bagged before leaving areas (McGovern 2015). Citric and acetic acids are safe to use on clothing and skin, yet can damage the envelope of acid-sensitive viruses (Anon 2014).

People should avoid wearing bright colored clothing, such as yellow and blue that can inadvertently attract and disseminate insect vectors (Snehi et al. 2015).

5 Seeds, Tubers, Corms, Rhizomes, Stolons

When possible, seeds, tubers, corms, rhizomes, and stolons should be obtained from areas of the world where the pathogen of concern is not present or from producers that use disease-free certification programs. Literature is available describing sanitation methods, including disinfestants, thermotherapy (dry or moist heat), electrotherapy, pesticides (fungicides, bactericides, and oomyceticides), biological controls, micropropagation techniques, and plant indexing (Ascough et al. 2009; Brand 2006; Chan et al. 1996; Geneve et al. 1997; Gilad and Borochov 1993; Liu et al. 2014; Maene and Debergh 1986; Munkvold 2009; Paulitz and Bélanger 2001). Companies that sell or generate plant selections from these sources often have developed proprietary sanitation protocols they implement at a commercial scale.

6 Plant Certification

The international seed and plant trade businesses have been affected by phytosanitary regulations that are changing faster than the supportive science (Munkvold 2009). Sanitary and phytosanitary (SPS) regulations of imported goods does impact trade and is viewed as a security concern for maintenance of the national plant health of countries. Pathogen identification technologies of increasing sophistication are allowing better standardization of internationally acceptable methods by relying on various biological and molecular methods, such as lateral flow devises (LFD), enzyme-link immunosorbent assay (ELISA), and traditional nested and quantitative PCR (Djalali Farahani-Kofoet et al. 2012; Linfield 1994; Miller et al. 2009; Sastry 2013). Inevitably, new genetic platforms, such as DNA microarrays and sequencing (DNA barcoding), and combinations of serological, nano, and nucleic acid detection technologies will further increase identification capabilities and improve the plant/seed certification process.

Seed health tests have primarily been focused on agronomic crops; such tests have been developed for over 100 pathogens. Eventually, this process will be applied to ornamental crops. Providing good seed sanitation does not exclude the possibility of latter introduction through growing media, local sources in the production area, or irrigation. This occurred with *Pythium ultimum* on seed-propagated geranium (Hausbeck et al. 1989).

The European and Mediterranean Plant Protection Organization (EPPO) has been developing schemes for certification of pathogen-free ornamentals, based on the principle of "filiation" in which nuclear stock plants of the highest health status are established and maintained, from which certified plants can be propagated under strictly controlled conditions (McNamara et al. 1996). Several certification and classification schemes have been accepted as standards for the Euro-Mediterranean region, particularly for bulb crops. In cases where filiation is not practical or necessary, a crop may be grown under near-normal conditions (e.g., in the field) then subjected to inspections where progeny are officially segregated for flower production or further propagation (McNamara et al. 1996). Dahlia crops in the Netherlands receive a visual inspection for viruses in the field, followed by serological testing for tomato spotted wilt tospovirus (TSWV) performed by the Flowerbulb Inspection Service, the Netherlands (Schadewijk et al. 1996). Growers are allowed to remove infected tubers from their stock. The scheme has led to a rapid decline of TSWV in Dutch dahlia crops.

Some plant health regulatory organizations include the National Plant Protection Organization (NPPO), the European and Mediterranean Plant Protection Organization (EPPO), the North American Plant Protection Organization (NAPPO), National Agricultural Biosecurity System (NABS) in India, and the International Plant Protection Convention (IPPC) (McNamara et al. 1996; Miller et al. 2009; Rishi 2009). Several organizations have been involved in internationally standardizing seed health testing methods: International Seed Testing Association (ISTA), International Seed Health Initiative (ISHI), International Seed Federation (ISF), and in the United States, the National Seed Health System (NSHS) (Munkvold 2009). Action is taken

through treaties involving the IPPC and the Food and Agriculture Organization (FAO) of the United Nations that operates under international standards for phytosanitary measures (ISPM) and includes at least 170 countries. Human resources to provide expertise in pathogen and disease identification include organizations such as the International Plant Diagnostic Network (IPDN); Plant Health Australia; the regulatory agency of the United States Department of Agriculture (USDA) Animal and Plant Health Inspection Service (USDA APHIS); the USDA APHIS Center for Plant Health Science Technology (CPHST); the National Plant Diagnostic Network (NPDN) in the United States; Agricultural Development and Advisory Service (ADAS) and CABI in the United Kingdom; the Global Plant Clinic (GPC) a consortium of CABI Bioscience; the National Reference Laboratories (NRLs) in the European Union; the Taiwan Agricultural Research Institute (TARI); New Partnership for Africa's Development (NEPAD); National Certification System for Tissue Culture Raised Plants (NCS-TCP), IITA Ibadan, and the Biosciences eastern and central Africa (BecA); and the Kenya Plant Health Inspectorate Services (KEPHIS), the African Union (AU), Inter-African Phytosanitary Council (IAPSC), the Center for Phytosanitary Excellence (COPE), and the Standards and Trade Development Facility (STDF) of eastern and southern Africa (Miller et al. 2009; Rishi 2009). Certification programs are being used by some companies and grower groups, such as Naktuinbouw Elite Ornamental Crops (http://www.naktuinbouw eu/node/192).

7 Stem and Leaf Cuttings

Stem and leaf tissue used for propagation can be contaminated with various pathogens. The preferable means of obtaining clean propagation plant material is to start with healthy plants and to maintain plant health with an integrated program for disease control. Stock plants should be maintained in separate greenhouses and fields at a distance from production facilities.

If disease-free stock plants are not available, several strategies may be considered to minimize pathogen introduction into propagation facilities. The strategy used will depend on crop production schedules and the biological realities of the pathogen. Three strategies are: (1) Start stock plants using micro-propagation or plant indexing techniques. (2) Plant material can be collected during periods when the pathogen is inactive. This would require knowing if defined infection periods occur and the length of latent periods, which is the time from infection to appearance of symptoms. In this scenario, new infections are unlikely and current infections have already developed symptoms. As a result noninfected healthy plant tissue can be visually identified and collected. (3) An appropriate pesticide (e.g., fungicide) can be applied to inhibit disease development by reducing development and spread of propagules prior to collecting cuttings. The later is more likely with fungal pathogens.

The latter two techniques do not guarantee pathogen-free propagative material. If cuttings are collected from field grown plants, caution is needed to reduce the possibility of pathogens being carried to the plant by rain splash. Stem cuttings should



Fig. 4 Peat-based media stored on a cement floor, but left uncovered where air-borne spores can contaminate the media (R.J. McGovern © 2017. All Rights Reserved.)

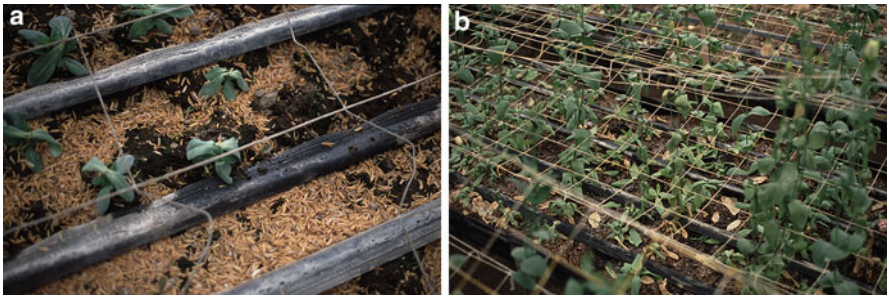


Fig. 5 (**a** and **b**) *F. oxysporum* f.sp. *eustomae* was diagnosed as the cause of seedling blight and possibly was spread because of inadequate soil fumigation and inadequate sanitation practices (R.J. McGovern © 2017. All Rights Reserved.)

be collected only from the upper portion of plants, above the height where rain splash is likely to carry pathogen propagules. Larger rain drops have been shown to splatter upward of 0.9 m (3 ft), carrying pathogens such as *Phytophthora* spp., *Pythium* spp., and *Thielaviopsis* spp. Potentially this also relates to greenhouses with dirt floors under benches. Do not neglect to use clean equipments, tools, and media (Figs. 4 and 5).

8 Plants

More disinfestants are being labeled for direct application on plants, yet limited information is available to advise growers of preferred practices (Cayanan et al. 2008, 2009; Celar et al. 2007; Copes 2009). Currently, hydrogen dioxide plus peroxyacetic acids and quaternary ammonium compounds (QAC) are two products labeled for direct application on plants in the USA. All disinfestants can cause phytotoxicity if applied at a high enough rate or at close intervals; therefore, it is important to follow label directions and test apply the material on a small number of plants before full scale application.

9 Spatial Arrangement of Plant Stock

Spatial separation of plants and worker activities can be used to restrict spread of pathogens. But this type of control requires forethought. Plant selections from different sources can be physically separated in different greenhouses or fields. The reason is to limit exposure of all plants to a pathogen present on one source of plants. This approach is commonly recommended for stock plants used as a source of cuttings. Spatial separation in a large bay may not be adequate, especially when propagules can be easily air dispersed across the bay.

10 Discarded Plant Material

Diseased plant material can become a major source of pathogen spread once propagules are produced on or in the tissue. Diseased plant material should be collected and removed as quickly as possible, at least daily, from production areas. Common recommendations are to place plant tissue in a covered container and remove the container from a production area, at least daily, and dispose of all discarded plant material. It is the author's belief that a container should have a properly but loose fitting lid, so the lid can be removed gently and infected plant tissue set, not dumped, in the container. While tight lids are more securely attached, more force is required to remove the lid which could momentarily produce a suction that aids dispersal of air-borne pathogens.

Discarded plant tissue should be enclosed in plastic bags or a closed container and removed from production areas to a distant site. The discard site should be as far from production plants as possible (Fig. 6). If it must be discarded near the production site, it minimally needs to be downhill out of the direct path of storm water flows, downwind of the production area, and preferably processed in compost piles. Use of barriers, such as tall hedges, in some cases could further diminish the impact of wind dispersal, by absorbing or diverting direct wind flow. Caution should be exercised relative to overall land topography and wind flow, as wind may rise over barriers and flow directly down on the discard pile to increase dispersal.

Discarded plant material can be a significant source of pathogen inoculum. Serious disease problems have occurred when high numbers of air-borne spores have been blown from discarded plant material near production areas onto a crop. Spore-containing water may flow downhill from discard piles and move through production blocks, causing serious plant losses.

In some cases, removal of diseased plant tissue has produced substantial reduction in disease. The incidence of viral diseases [Narcissus latent, macluravirus (NLV); Tobacco rattle, tobravirus (TRV); Cucumber mosaic, cucumovirus (CMV); and an unidentified corky pit virus-like agent (UCPVA)] was reduced by removing infected Gladiolus plants in the Netherlands (Asjes et al. 1997). In contrast, conidia of *Colletotrichum acutatum* remained infective for up to 49 days in leaf debris from the leatherleaf fern on the soil surface in commercial ferneries and up to 31 weeks under dry soil conditions (Norman and Strandberg 1997, 2012). In the case of

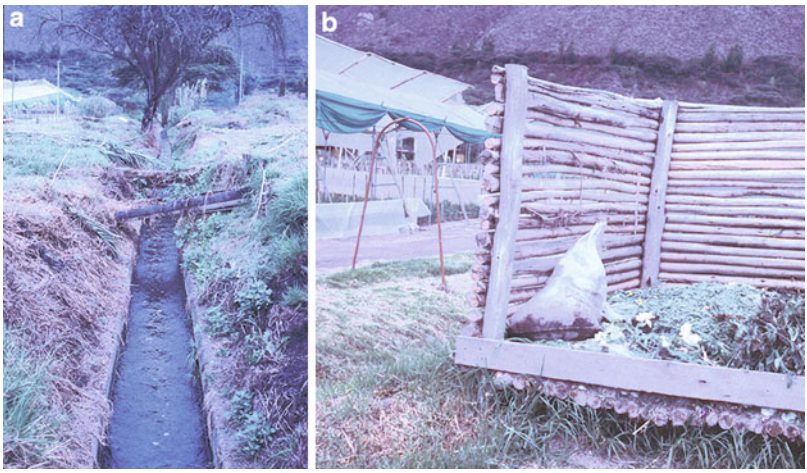


Fig. 6 (**a** and **b**) Diseased plants discarded into a plant refuge pile, which can now serve as a source of wind-blown spores (R.J. McGovern © 2017. All Rights Reserved.)

Botrytis blight, removal of diseased plant material alone will not reduce disease without use of cultural controls and applications of fungicides; however, not collecting diseased tissue could increase disease pressure dramatically (Hausbeck and Moorman 1996).

11 Irrigation Water

Selecting a water treatment system requires consideration of equipment, installation and operation costs, maintenance requirements, source(s) of water, quality of water chemistry, and maximum daily water usage. The main components of a water treatment system include the electronic control system, filtration system, preinjection system (dependent on water quality), and disinfestant injection system. Multiple equipment and disinfestant options are available. The primary disinfestants are chlorination (chlorine gas, electro chemical activated (ECA) water, bleach); chlorine dioxide; ozone; and peroxygen compounds (hydrogen dioxide = hydrogen peroxide (H_2O_2), peroxyacetic acid). Physical disinfestation techniques are ultraviolet (UV) light, heat, and filtration. The critical factors for selection of disinfestants have been thoroughly covered by Fisher (2014). Chlorine gas is the most commonly used disinfestant because it is effective and has the lowest cost. However, each system has advantages and disadvantages that should be considered. Multiple decisions are involved in choosing a water disinfestation system, and several web-based sites address the topic to assist growers: (http://watereducationalliance.org/; Irrigation Pathogen and Water Quality Webinar Series-http://www.irrigation-pathogens.ppws.vt.edu/webinar/ index.php).

Water treatment to eliminate pathogens involves exposure to a disinfestant at a certain rate for a minimum time. Chlorination will be used as an example. A rate of 1–2 ppm free chlorine is commonly recommended to kill zoospores of *Phytophthora* spp. in irrigation water (Cayanan et al. 2009; Hong et al. 2003), and this is less than the 2.5 ppm free chlorine considered safe for application on most ornamental plants (Cayanan et al. 2008; Skimina 1992). However, 12–14 ppm free chlorine with 6–10 min exposures was required to killed *Fusarium oxysporum* and *Rhizoctonia solani*, which is a level toxic to ornamental plants (Cayanan et al. 2009). However, hydrogen peroxide plus peroxyacetic acid may be an alternative in this application, because a rate below the label rate that is safe to most ornamental plants killed *Fusarium foetens* conidia, the cause of begonia wilt (Elmer 2008). This highlights the fact that disinfestant selection sometimes depends on the current problem (Floyd 2008).

Characteristics of the water source(s) is an important consideration in considering water treatment (Hong and Moorman 2005). Irrigation water drawn from capped deep wells and from municipal treated drinking water generally is considered to be free of plant pathogens, therefore should not require treatment (Copes et al. 2015; Hong and Moorman 2005). However, unusual cases have been documented. For example, a plant-indexing propagation operation found municipal water to be the source of *Pythium* spp. at the facility. Irrigation water drawn from natural water ways and from recycling water basins potentially contain plant pathogens, such as plant pathogenic *Erwinia* spp., *Fusarium* spp., *Pythium* spp., *Phytopythium* spp., and *Phytophthora* spp., that may require treatment (Bush et al. 2003; Del Castillo Múnera and Hausbeck 2015; Elmer 2008; Ghimire et al. 2011; Choudhary et al. 2016; Moorman et al. 2002; Parke and Grunwald 2012). Even with these seemingly risky water sources, water treatment is only required when pathogens capable of causing crop loss are being dispersed in irrigation water.

Recent research results have shown that detection of *Pythium* spp., *Phytopythium* spp., and *Phytophthora* spp. in irrigation water does not confirm a plant pathogen problem (Copes et al. 2015; Hong and Moorman 2005; Choudhary et al. 2016; Loyd et al. 2014; Parke et al. 2014; Zappia et al. 2014). Unfortunately, these results provide an additional burden for plant producers, because isolates need to be identified to species by qualified plant disease clinics and associated with past or current plant disease problems, or a high potential for disease to determine whether water treatment is required. Current knowledge demonstrates that certain species can be considered as highly pathogenic and their presence at any level is of serious concern, while many other species cause crop loss less commonly. For example, *Phytophthora nicotianae and P. tropicalis* are considered to be high risk pathogens to herbaceous plants, thus water treatment should be implemented (Hong et al. 2014). *Phytophthora citrophthora*, *P. pini*, *P. cactorum*, *P. drechsleri*, *P. megasperma*, *P. palmivora*, *P. cryptogea*, and *P. taxon drechsleri III* are considered to be moderate risk pathogens to herbaceous plants, thus water treatment may not be required unless disease problems exist or have been a problem (Hong et al. 2014). Many other *Phytophthora* spp. are considered to be of minor risk and water treatment would not be necessary unless an active disease problem was identified. Fungal and bacterial pathogens should be evaluated similarly. In Florida,

populations of *E. chrysanthemi* and *E. carotovora* subsp. *carotovora* that are pathogenic on ornamental plants in the Araceae family were shown to increase in a recycling containment basin without water treatment, thus water treatment would be needed (Norman et al. 2003).

If an outdoor recycling containment basin (= pond) is used, the system can be designed to promote the natural decline of *Phytophthora* zoospores. The main objective is to prevent the pathogen from reaching irrigation pump inlets (Ghimire et al. 2011; Hong 2011). The flow of irrigation runoff can be slowed and sedimentation promoted, which will reduce the diversity and level of pathogens reaching pump intakes for distribution in irrigation water. In a single pond system, run-off should be channeled to a single entrance point that is furthest from the pump uptake. One way to achieve this is to dig a J-shaped canal that routes water along the side of the reservoir to the opposite end from the pump house; in addition, several mounds of gravel laid perpendicular to the water path can be added in the canal to create intermittently slowed water travel that further facilitates settling of pathogen propagules (Hong 2011). A multiple pond system that includes a sedimentation pond and multiple catchment ponds will increase the travel distance and time for zoospores to settle out and increase the possibility of zoospore mortality (Hong 2011; Zhang et al. 2015b). The size of the basin system relative to daily water gallonage received will affect sedimentation capability. It is assumed that a similar decline would occur with both *Pythium* spp. and *Phytopythium* spp. Nonmotile spores of most fungi, such as *Fusarium* spp., would only be carried by currents, and as such should be susceptible to settling out when water flow is slowed and the distance increased between inflow and water uptake ports. In addition, water pH has been shown to adjust from an alkaline to a slightly acidic level after passage through multiple ponds.

Water conditions in a pond can also contribute to mortality of zoospores (Kong et al. 2012a, b; Kong and Hong 2014). Water pH levels in recycling containment ponds that were monitored 24-h per day in the mid- to south-eastern USA were found to be alkaline the majority of the time. Average pH levels only present part of the picture, as water basins in those same nurseries typically experience a vertical stratification of temperature, pH, and dissolved oxygen from May to October, even in ponds no deeper than 1 m (3.3 ft) (Zhang et al. 2015a–c). The stratification patterns in nursery ponds in other parts of the country have not been monitored yet. In Maryland and Virginia, USA, temperature, percent dissolved oxygen, and pH level are higher in the upper layer, during these stratification periods. Additionally, pH levels diurnally fluctuate several pH units in ponds, being lower and possibly acidic by early morning and being higher and possibly alkaline by early evening. A similar diurnal fluctuation occurs with dissolved oxygen. These fluctuations occur due to the biological activity of algae and other microorganisms. Water drawn from the lower stratification, especially during early day hours, will be most suitable for chlorination without acidification treatments on most days.

It is not known if the same principles apply to containment tanks in greenhouses or if the relative water exchange is too high to retain a resident body of water that cushions or buffers against rapid changes.

12 Host Resistance

Plants that are highly and even moderately disease resistant may develop few if any disease symptoms. Durable plant resistance is often the preferred means of disease control when available. However, moderately tolerant cultivars can be carriers of pathogens without obvious symptoms. Pathogens that affect multiple crops may persist on tolerant plants and, as a result, be a possible source of the pathogen. This possibility needs to be considered when control expectations are not being achieved.

13 Integrated Disease Management

Integrated disease management (IDM) and the systems approach refers to coordinating multiple strategies, such as utilizing disease tolerant plant selections, sanitation, cultural, biological and chemical controls, and identifying key control points, to address disease concerns. Ornamental plant production has the unique challenge of producing a large number of plant selections at a single production site. IDM should be considered at two scales, integrating multiple controls to target a single disease and integrating controls at the operational scale against multiple diseases. The operational needs, resources, and production goals of the business must be considered when designing disease control procedures that allow production of high quality plants for the least input and cost outlay. Labor efforts should be planned so tasks can be implemented in a coordinated effort on multiple crops. A three step approach to selecting controls is suggested below:

1. Identify the major and common pathogens that infect the plants grown at a production facility, as these will be the target of management. Major pathogens are those that pose a threat of serious crop loss and/or regulatory involvement. Common pathogens are those that are recurring within an operation. Be diligent in inspecting new crops, because pathogens imported from other facilities may become new problems requiring intensive control efforts. A list of relevant pathogens may be available from university and private crop advisers and plant suppliers. Current literature and internet resources may be helpful but need to be evaluated as to whether the pathogens are relevant to your area. Good record keeping within a facility can aid in identifying recurring problems.
2. Identify which disease control practices are recommended for the major and common diseases pertinent to the operation and select relevant controls.
3. List and schedule the control practices on a general calendar flow chart for all crops. The purpose is to lay out a schematic diagram for organizing and consolidating labor and equipment needs in an economical approach. Determine possible scheduling conflicts so practices can be prioritized and alternative schedules and solutions written into the operation plan. If labor scheduling conflicts or excessive costs are evident, the schematic becomes an accounting tool to help prioritize and select the most valuable practices

based on crop value and potential crop losses at the facility. When new disease problems occur, new control measures may need to be introduced into the plan.

14 Record Keeping

Records are an important tool for identifying and prioritizing the importance of tasks. Records can be kept in a notebook, in a computer spread sheet, or an online storage site. Images are easily taken with cell phones and can be stored in any of these filing systems. Record the date, crop, crop age or stage, location in the facility, disease symptoms and their progress over time, diagnosis, who made the diagnosis and whether it is by field recognition of symptoms or pathogen identification, name and rate of chemicals applied, total area and crops treated, and an estimation of percent crop loss (number of plants and their value per container). Records can serve as a reference for comparing current to past problems. Records are an important tool for helping to decide what control efforts may be of high, moderate, or low value. Good records can help you save money.

Acknowledgments I thank Melanie Putnam, Oregon State University, and Alan Henn, Mississippi State University, for reviewing the manuscript. This review on sanitation was supported through the US Department of Agriculture, Agricultural Research Service, Thad Cochran Southern Horticultural Laboratory, Southern Horticultural Research Unit project number 6404-21430-001-00D.
Disclaimer "Mention of trade names or commercial products in this chapter is solely for the purpose of providing specific information and does not imply recommendation or endorsement by the U S Department of Agriculture."

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Mineral Nutrition and Florists' Crops Diseases

10

Lawrence E. Datnoff and Wade H. Elmer

Abstract

The health of any ornamental crop is a direct function of the nutritional status of the plant. Disease susceptibility or resistance is often governed by the speed in which the plants can directly react to infection by plant pathogen and/or indirectly by their ability to tolerate predisposing stresses such as drought.

Information is presented herein on the role of essential minerals N, P, K, Ca, Mg, S, Cl, Fe, Mn, Cu, Zn, B, Mo, and Ni along with beneficial elements Si and Al for their effects on suppressing diseases in ornamental crops.

Keywords

Plant health • Host defense • Fertilizers • Disease management

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R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_10

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1 Introduction

The maintenance of plant health and disease suppression in florists' crops is a function of the plant's nutritional status (Datnoff et al. 2007). A proper and balanced nutrition can often govern the fine line between host susceptibility and plant disease resistance. It has been observed that sufficient nutrition can also delay the onset of pathogenesis beyond a critical threshold where the plant's marketability is not compromised by disease. Complete and balanced nutrition should always be the first line of defense against biotic stresses especially plant diseases. However, one critical obstacle in providing proper plant nutrition is the lack of information on optimal nutritional requirements for the vast array of different florists' crops. Many ornamental plants will vary in their nutritional requirements and each nutrient can affect disease susceptibility differently. For example, the nitrogen sufficiency range for cut flowers is 3.1–4.7, while the range for herbaceous perennials is 2.2–3.2 (Barker and Bryson 2007). As a consequence, each ornamental plant's nutritional requirements need to be thoroughly understood on a "case by case" basis. Unfortunately, detailed information on the fertility requirements of specific ornamental crops is often lacking. Furthermore, the fertilizer requirements necessary to maximize plant health when a pathogen is present may differ from those required when the pathogen is absent (Elmer and Datnoff 2014).

In many cases, the nutrient amounts needed for plant disease suppression may far exceed a healthy plant's normal nutritional requirement for that nutrient, suggesting that many nutrients may function in multiple defense and/or physiological mechanisms for disease suppression. Chemical interactions with the soil, pH, and/or with specific communities of microorganisms can, in turn, influence the development of disease. For example, the form of nitrogen can have striking effects on plant disease through root-mediated changes in pH, microbial profile in the rhizosphere, and alterations in the availability and function of micronutrients. For example, although Ca only composes approximately 0.5% of the dry weight of most plants, it is routinely applied in great quantities to potting mixes and field soil to affect soil pH and to suppress certain plant diseases.

Plant disease resistance mechanisms that are affected by nutrition include those metabolic pathways that lead to the production of lignin, phenol, phytoalexins, and other defense-related compounds. Many of these pathways utilize enzymes that require B, Cu, Mn, Mo, and Zn as cofactors or activators. Other elements like K and Cl affect disease through osmotic relations, water cycling, and root exudation, which, in turn, influence beneficial microbes. Indirect mechanisms include effects on nitrification, soil pH, and chemical transformation of micronutrients like Mn. Although each nutrient's effect on plant disease will be reviewed, nutrition

must be viewed holistically since all nutrients affect the uptake and function of other nutrients and can ultimately increase or decrease plant disease development. A detailed list of ornamental plant diseases suppressed by macro-, micro-, and beneficial nutrients is summarized in Table 1.

2 Nitrogen

The fourth most abundant element in plants after hydrogen, carbon, and oxygen is nitrogen (Huber and Thompson 2007). Nitrogen is a component of amino acids, enzymes, hormones, phenolics, phytoalexins, and proteins, all of which can have direct effects on plant growth and disease development. Although most growers are familiar with symptoms of N deficiency, most do not recognize the role that N form may play in enhancing or suppressing disease. Nitrogen can be applied as a salt of the oxidized anion NO_3^- or as the reduced cation NH_4^+. Most ornamentals can use either form, but due to phytotoxicity associated with NH_4-N, especially with seedlings, most growers apply N in the NO_3-N form. However, in ornamentals, excess nitrate can increase Pythium diseases by increasing salt stress and increase Botrytis blight by promoting green and succulent tissue (Gladstone and Moorman 1989).

Once absorbed by the plant, NO_3-N and NH_4-N are metabolized differently and can have opposite effects on diseases. This, in turn, has led to many of the conflicting reports found in the literature regarding the role of N and plant disease, which may be due to a failure to recognize and/or report the N form. For example, increasing N suppressed bacterial leaf spots on philodendron and bacterial blight on Schefflera (Haygood et al. 1982; Chase 1990) but increased bacterial leaf spot and cutting rot on chrysanthemum caused by *Pseudomonas cichorii* and *Erwinia chrysanthemi*, respectively (Jones et al. 1985; McGovern et al. 1985). However, when the form of N is known, some general rules can be made for certain diseases. For example, Fusarium wilts and root rots of carnation, chrysanthemum, cyclamen, and gladiolus are usually less severe when the N is applied as NO_3-N and more severe when applied as NH_4-N (Huber and Thompson 2007). The opposite is true for Verticillium wilt and Thielaviopsis diseases. Studies have shown that applying ammonium sulfate provided good suppression of Verticillium wilt on eggplant, maple, and snapdragon. In addition, growers should pay attention to the ion that accompanies N. Depending on the plant and the disease, the companion ion (Ca, K, Cl, and SO_4) may either enhance or decrease disease protection.

3 Phosphorus

Phosphorus plays a vital role in cell division and energy transfer, as well as a regulatory role in the transport of sugars and starches within the plant (Prabhu et al. 2007). The examples where P has been shown to be effective in suppressing plant disease in ornamental plants are few. For example, P has been shown to suppress root rot in geranium caused by *Pythium ultimum* (Gladstone and Moorman

Table 1 Some differential responses to macro-, micro-, and beneficial nutrients in some ornamental host-pathogen interactions

Nutrient	Hosts	Diseases	Pathogens	Effect	Reference(s)
Nitrogen					
Ammonium	Syngonium	Blight	*Xanthomonas* sp.	Decrease	Chase (1989)
	Chrysanthemum	Cutting rot	*Erwinia chrysanthemi*	Increase	McGovern et al. (1985)
	Chrysanthemum	Leaf spot	*Pseudomonas cichorii*	Increase	Jones et al. (1985)
	Schefflera	Leaf spot	*Xanthomonas hederae*	Decrease	Chase (1990)
	Pansy	Root rot	*Thielaviopsis basicola*	Decrease	Copes and Hendrix (1996)
Nitrate	Carnation	Root rot	*Gibberella zeae*	Increase	Stack et al. (1986)
	Chrysanthemum	Wilt	*Fusarium oxysporum*	Decrease	Woltz and Engelhard (1973)
	Cyclamen	Wilt	*Fusarium oxysporum*	Decrease	Elmer and Daughtrey (2012)
	Geranium	Root rot	*Pythium* sp.	Increase	Gladstone and Moorman (1989)
	Gladiolus	Corm rot	*Fusarium oxysporum*	Decrease	Engelhard and Woltz (1978)
Phosphorus	Geranium	Root rot	*Pythium ultimum*	Increase	Gladstone and Moorman (1989)
	Gladiolus	Yellows	*Fusarium oxysporum* f. sp. *gladioli*	Decrease	Prabhu et al. (2007)
Potassium	Aster	Wilt	*Phialophora asteris*	Increase	Burge and Isaac (1977)
	Carnation	Bacterial wilt	*Pseudomonas caryophylli*	Decrease	Nelson and Laurie (1951)
		Stem rot	*Gibberella zeae*	Increase	Stack (1989)
		Wilt	*Fusarium* sp.	Decrease	Gasiorkiewicz (1960)
	Pelargonium	Stem rot	*Xanthomonas pelargonii*	Decrease	Kivilaan and Scheffer (1958)
	Philodendron	Leaf blight	*Erwinia chrysanthemi*	Increase	Haygood et al. (1982)
	Snapdragon	Wilt	*Verticillium dahlia*	Decrease	Dutta and Isaac (1979)

Calcium	Rose	Gray mold	*Botrytis cinerea*	Decrease	Starkey and Pedersen (1997)
	Vinca	Root rot	*Phytophthora parasitica*	Decrease	Von Broembsen and Deacon (1997)
Magnesium	Calendula	Seedling disease	*Pythium aphanidermatum*	Increase	Gill (1972)
	Carnation	Wilt	*Fusarium oxysporum*	Decrease	Lyakh (1986)
Manganese	Chrysanthemum	Wilt	*Fusarium oxysporum*	Decrease	Malini (1961)
Chloride	Cyclamen	Fusarium wilt	*Fusarium oxysporum* f. sp. *cyclaminis*	Decrease	Elmer (2002)
Nickel	Daylily	Rust	*Puccinia hemerocallidis*	Decrease	Reilly et al. (2005)
Silicon	Gerbera daisy	Powdery mildew	*Podosphaera fusca*	No effect	Moyer et al. (2008)
	Morning glory	Anthracnose	*Colletotrichum gloeosporioides*	Decrease	Kunoh and Ishizaki (1975)
	Paper daisies	Anthracnose	*Colletotrichum gloeosporioides*	Decrease	Muir et al. (2001)
	Rose	Powdery mildew	*Sphaerotheca pannosa*	Decrease	Voogt (1992), Shetty et al. (2012)
	Sunflower	Powdery mildew	*Golovinomyces cichoracearum*	Decrease	Kamenidou (2005)
	Tigergrass	Leaf spot	*Exserohilum rostratum*	Decrease	Brunings (2008)
	Zinnia	Powdery mildew	*Golovinomyces cichoracearum*	Decrease	Kamenidou (2005) (unpublished data WH Elmer)
Aluminum	Vinca	Damping off	*Phytophthora parasitica*	Decrease	Benson (1993)

1989) and yellows in gladiolus caused by *Fusarium oxysporum* f. sp. *gladioli* (Prabhu et al. 2007). Most general reports on P and plant disease in other cropping systems suggest that increasing phosphorus above that which is necessary for proper growth may be associated with an increase in plant disease development. In fact, those cases where P reduced disease may have been in situations where the element was deficient. Cases where the disease became worse following P application might have been in soils where excess P decreased the availability of other elements which, in turn, increased the plant's susceptibility. In most soils, maximum availability of P would be expected in the slightly acid to neutral pH range. In acid soils, P reacts with Al, Fe, and Mn to form insoluble products, making P less available. In alkaline soils, P reacts with Ca and reduces P availability. Therefore, maintenance of an appropriate soil pH is very important to maximize availability of P and plant health.

Products containing phosphorous acid (H_3PO_3), and its salts (phosphonate, phosphate, or phosphonic acid), have been developed and marketed to control plant diseases caused by *Phytophthora* and *Pythium* (Brunings et al. 2012). Phosphorous acid has a mixed mode of action involving direct toxicity to the plant pathogen along with enhancing the plant's defense response. These products do not provide any appreciable P nutrition to the plant, and their mode of action is not through normal pathways associated with P metabolism. Similarly, monopotassium phosphate (MKP) has been marketed as a foliar spray to induce local and systemic protection against some foliar pathogens in several crops.

The role of beneficial mycorrhizal fungi in P nutrition and disease development also deserves attention (Elmer and Datnoff 2014). Mycorrhizae form a symbiotic relationship with the plant's roots. These fungi improve plant health and may reduce disease damage. Many ornamentals have mycorrhizal associations that are likely interrupted during propagation cycles. There may be a need to alter the P applications since mycorrhizal infection can be inhibited by increased P availability. Commercial applications of mycorrhizae are available and may have value in ornamental production for management of soilborne disease. Their use may allow other nutrients to be more available since added P could lead to the precipitation of other elements. Much research is still needed to identify the P demands of ornamentals that respond to supplemental applications of mycorrhizae.

4 Potassium

Potassium is found in large quantities in plant tissues and may exceed nitrogen levels for certain plants. Potassium improves water and nutrient uptake, increases cellulose and protein content, and enhances and regulates at least 60 different plant growth enzymes (Prabhu et al. 2007). Many diseases can be increased or decreased on the basis of K nutrition alone or when combined with other elements. Unfortunately, no patterns have emerged with K and plant disease that would allow for any specific generalizations. For example, investigators have noted that applying K suppressed bacterial diseases on carnation and geranium (Nelson and Laurie 1951; Kivilaan and Scheffer 1958) but increased bacterial leaf blight on philodendron (Haygood

et al. 1982). Similar observations have been made for fungal pathogens attacking ornamental plants where K would increase or decrease diseases (Menzies and Colhoun 1976). Many times these discrepancies are a result of investigators not mentioning the form of K. Potassium is most often applied as KCl, K_2SO_4, or KNO_3 and the form can be of great importance in disease management. The accompanying ion can often dictate the response of a plant to K, and this factor may explain the large number of discrepancies regarding K and plant disease. Although studies on ornamentals are rare, there are a number of reports on other crops that show that the positive response to K fertilization was only associated with the KCl form and not any other K forms (Elmer 2007). Another important principle of N, P, and K nutrition is that providing more of these fertilizers than what is required by the plant can aggravate disease caused by species of *Pythium*. When Pythium root rot is a persistent problem, growers should consider a balanced nutrient solution containing relatively low levels of N, P, and K and proportionally higher levels of Ca and K (Gladstone and Moorman 1989).

5 Calcium and Magnesium

Calcium and Mg are the second and third most abundant essential nutrients, respectively, in a plant (Rahman and Punja 2007). Calcium concentrations in plants are usually twice that of Mg. Calcium is extremely important in normal cell growth where it forms Ca pectate in the middle lamellae for cell wall stabilization. Calcium also functions in many enzymatic reactions involved in defense mechanisms. Calcium is commonly applied as $CaCl_2$, $CaSO_4$, or $Ca(NO_3)_2$. The role of Ca in the management of plant disease has received much attention, but examples for ornamentals are few.

Many previous studies investigating the role of $Ca(NO_3)_2$ credited all of the plant disease suppression to the NO_3 and failed to recognize the Ca ion. Unfortunately, most studies do not examine different sources of Ca and its companion ion to demonstrate the contribution of Ca to plant disease control. A study on roses found foliar applications of $CaCl_2$ reduced the incidence of gray mold caused by *Botrytis cinerea* (Starkey and Pedersen 1997). The same study found that increasing Ca concentrations in the soil improved the shelf life of the flower. Calcium may also influence the spread of *Phytophthora* spp. in water. Vinca plants that were flood irrigated and then infected with *Phytophthora* spp. were found to be healthier when the complete fertilizer solution was amended with $Ca(NO_3)_2$ (Von Broembsen and Deacon 1997). Other studies demonstrated that Ca applied as either $CaCl_2$ or $Ca(NO_3)_2$ in water or in Ca-free soluble fertilizer solutions suppressed the release and the motility of the swimming zoospore (Rahman and Punja 2007). These results demonstrated that there is an important need to understand more about how Ca amendments interfere with *Phytophthora* in recirculating irrigation systems.

Magnesium is usually applied as $MgSO_4$ or $MgCl_2$ and has been associated with both increased disease and disease suppression of plants (Jones and Huber 2007). Of 46 studies investigating the role of Mg on plant disease, 22 found that Mg decreased

disease, 18 found that Mg increased disease, and 6 found that there was little or no difference. Since the data are so conflicting, no real patterns can be discerned regarding the effect of Mg on plant diseases. It may be that many of these studies corrected a Mg deficiency and the result was a healthier plant with more vigor and disease resistance. When Mg was applied in excess for normal growth, a nutritional imbalance developed that promoted plant stress and more disease. For example, the addition of extra Mg to the potting soil was associated with increased damping off of calendula (Gill 1972). On the other hand, applications of Mg suppressed Fusarium wilt of carnations when grown on calcareous soils (Lyakh 1986). The latter study was probably a result of the added Mg correcting a deficiency due to the high Ca concentration.

6 Chlorine and Sulfur

In the past, Cl applications were thought to have little value in improving plant growth because Cl was thought to be highly available in soils and not essential. However, Cl has been routinely applied as a companion ion for NH_4-N, K, and Ca fertilizers (Elmer 2007). The contribution of Cl has long been underestimated in crop production and plant health. Reports are still erroneously assigning the effect of Cl to the accompanying cation. For example, while it is well documented that proper K nutrition will suppress some plant diseases, many subsequent studies that examined different forms of K found that the ameliorating effects on disease were restricted to KCl amendments suggesting that Cl, not K, was the active ion.

Nutritionally, Cl is regarded as a micronutrient, yet, as in the case of Ca, benefits are achieved with rates that far exceed the plant's nutritional requirements. High rates of Cl salts have marked effects on inhibiting soil nitrification, enhancing availability of Mn and other micronutrients, and on increasing beneficial microorganisms. As an element, Cl^- is the only inorganic anion that is not structurally bound to a metabolite. Its major role is to serve as a charge-balancing ion. When a cell absorbs Cl^-, it accumulates in the cell vacuole and lowers the cell water potential below that of the medium surrounding the cell. Water then flows into the cell and increases hydrostatic cell pressure so it maintains a pressure that exceeds the force exerted by the plasmalemma. The cells remain turgid and are able to grow even when drought conditions prevail. Chloride ions also alter the quantity and quality of organic solutes that are exuded into the rhizosphere, thus reducing the germination of and infection by root pathogens. Another major role of Cl in disease suppression comes from its effect on increasing the availability of Mn, which has direct effects on host resistance. Monocots tend to be more tolerant of high amounts of Cl, but there is no information on how ornamental grasses would respond to Cl applications. Cyclamen plants inoculated with *Fusarium oxysporum* f. sp. *cyclaminis* and exposed to Cl salts were larger and had fewer symptoms of disease than plants that received no Cl (Elmer 2002). Any studies with Cl must ensure that salt levels do not build up and predispose plants to root infection by *Pythium*.

Sulfur (S) is an essential element and a component of proteins (Haneklaus et al. 2007). This element also is probably the oldest recorded fungicide known (Agrios 2005). Sulfur has been used for over 100 years to suppress powdery mildew, certain rusts, and leaf blights such as black spot of rose. However, the significance of soil-applied S on plant disease only recently became evident when S deficiency symptoms were noted following the drastic decrease of SO_2 emissions from coal-burning facilities. Most growers do not consider S nutrition a component in their fertility programs. When this element is used, it is frequently applied as $NH_4(SO_4)_2$, Ca_2SO_4, or $MgSO_4$. These salts, however, are almost exclusively applied with the aim of supplying the cation. When lowering the soil pH, elemental S and $AlSO_4$ are often used. Currently, there is very little information available on how sulfur nutrition affects ornamental diseases. However, on field crops, several studies have shown that proper sulfur nutrition can induce resistance to plant pathogens through its effect on the function and availability of S-containing amino acids. Cysteine, methionine, and glutathione play roles in protein synthesis of many plant defense products. As air quality improves and atmospheric S decreases further, growers may need to be more attentive to S nutrition.

7 Effect of Micronutrients on Diseases

The metals iron (Fe), manganese (Mn), zinc (Zn), copper (Cu), molybdenum (Mo), and boron (B) have diverse but essential roles in plants, functioning as cofactors or activators of enzyme systems. Many of these enzyme systems play pivotal roles in disease resistance in the production of defense barriers (Datnoff et al. 2007). In general, the concentrations that correct visual deficiency symptoms in plants are often far below the levels needed to ensure proper health and defense against disease. Most micronutrients become less available as the pH rises so growers should be aware that although crops that favor alkaline soils may not show deficiencies, they may be more susceptible to attack from plant pathogens. One quick method to correct aboveground deficiency symptoms and boost resistance to foliar diseases is to apply a foliar application of the micronutrient needed. However, since micronutrients are not translocated basipetally to the roots, a foliar application would not be very effective for suppressing a root-infecting pathogen. Although most ornamental growers lump micronutrient deficiencies all together, future research may allow for prescribed applications to optimize and balance micronutrient fertility to increase protection from disease.

Proper Fe nutrition not only improves plant development and health, this metal indirectly affects disease in the rhizosphere where its availability may limit the growth of pathogens (Expert 2007). One of the major situations in which Fe influences plant disease is through siderophores. Siderophores are low molecular weight molecules (<1500 Da) that possess a high affinity for Fe^{3+}. They specifically capture Fe^{3+} ions and transport them back into the microorganism that produces them. So, certain microorganisms have the ability to acquire Fe and rob the potential plant pathogen of Fe in the rhizosphere or even within the plant. However, the

siderophores can also release their Fe^{3+} ions to iron-deficient plants. Therefore, microbial siderophores present in soils may increase Fe availability on the root surface, depending on their different chemical, physical, and absorptive characteristics. Mechanisms involved in these processes have been poorly defined, and no research to date has been conducted using ornamental-pathogen systems as a model.

Although a rich body of information exists on the role of Mn nutrition on plant disease (Elmer and Datnoff 2014), very little research has been conducted using ornamentals. Manganese affects the production of many host defense products such as lignin, tannins, and phytoalexins. Manganese functions primarily as an activator of enzymes, including dehydrogenases, tranferases, hydroxylases, and decarboxylases. Mn is an activator of several enzymes in the shikimic acid pathway for production of important secondary metabolites that lead to the production of phenolics, cyanogenic glycosides, and the deposition of ligneous defense barriers. More directly, Mn can reduce growth of the pathogen, which may explain why these organisms evolved mechanisms for oxidizing Mn to unavailable forms. Growers need to be aware that Mn is most available to plants at a soil pH levels of 5.0–6.5 and is unavailable when above 7.8. Mn-transforming microbes play a major role in this soil availability.

Zinc nutrition is essential to the metabolism associated with important plant defense pathways against fungal and bacterial pathogens, and although examples are found only for vegetable and field crops, they may have relevance to ornamentals (Elmer and Datnoff 2014). Examples of bacterial and fungal species affected by Zn include *Xanthomonas*, *Pseudomonas*, *Rhizoctonia*, *Puccinia*, *Fusarium*, and *Bipolaris*. Zinc plays an important role in the disease resistance signaling proteins and protects plant cells from toxic oxygen radicals. Zinc also influences fungal growth and secondary metabolism by indirectly altering host susceptibility. The availability of zinc also affects fungal pathogens and modulates the accumulation of citric acid, stimulates glucose uptake, and pigment and antibiotic production by microbes (Duffy 2007).

Copper activates many enzyme systems involved in plant defense (Elmer and Datnoff 2014). Copper affects plant disease by three known mechanisms. Direct toxicity as a bactericide or fungicide is the most common one. However, the effects of Cu on plant resistance and pathogen virulence can also operate simultaneously. For example, Cu enhances production of polyphenol oxidases that are involved in plant defense against infection. In addition, the element is also involved in the production of antimicrobial compounds and general disease resistance. Copper has been identified as a micronutrient for some plant pathogens, and the limited availability of this element can result in the pathogen becoming avirulent.

Molybdenum is essential to higher plants and plays a role in activating the nitrate reductase enzyme (Elmer and Datnoff 2014). Molybdenum has been shown to denature the protein coat of viruses and disrupt zoospore production, but only in vegetable production systems. Manipulating Mo nutrition to date has not been demonstrated to be effective for managing plant diseases.

Boron plays a crucial role in plant defense by improving cell wall integrity and enhancing phenol metabolism (Elmer and Datnoff 2014). Studies on field crops have

found that minor B deficiencies can lead to increased plant disease susceptibility. Examples of pathogens negatively affected by B include species of *Fusarium*, *Leveillula*, *Plasmodiophora*, *Puccinia*, and *Verticillium*.

Nickel (Ni) has been regarded as an essential element due to its direct role in activating urease in plants, but Ni fertility has received little, if any, attention among ornamental growers. Nickel is absorbed as Ni^{+2} in minute quantities. However, on hydrangea and pyracantha, Ni deficiency has been observed and was thought to occur when other micronutrients were applied in excessive quantities. Applications of Ni as a foliar spray have been associated with reductions in rust on daylilies (Graham et al. 1985; Reilly et al. 2005). The mechanisms for this reduction in disease are not known, but the metal may boost defense mechanisms, induce resistance to disease, and directly inhibit the pathogen. The widespread use of Ni in ornamental disease management is yet to be explored.

8 Effect of Beneficial Elements on Diseases

Although not all elements are viewed as essential, a growing number of experiments have shown that beneficial elements can suppress a number of plant diseases. The ameliorating effects of these elements are frequently only realized when the plant is under an abiotic or biotic stress such as a plant disease. These findings suggest that these nutrients probably play a role in host defense mechanisms.

Although silicon (Si) is the second most abundant element in the earth's crust, many soils are known to be low or limiting in this element and include some of the following soil orders: Entisols, Histosols, Inceptisols, Oxisols and Ultisols (Tubana and Heckman 2015). Soilless mixes would also be deemed to be low in plant available Si. Silicon is root absorbed as monosilicic acid ($Si(OH)_4$), and plants are known to accumulate this element in great quantities (between 1% and 10% dry weight). Consequently, all plants contain Si and some plants in quantities that may exceed a number of essential mineral nutrients. However, Si was largely absent from plant experiments or used as a crop amendment until the early twentieth century since visible deficiency symptoms were lacking. Nowadays, silicon is known to dramatically decrease the intensity of a number of plant diseases, which include damping off, leaf blights, leaf spots, galls, powdery mildews, root rots, rusts, and wilts (Datnoff and Rodrigues 2015a). Although research on some ornamentals has begun, most ornamental plants have not been thoroughly assessed for the role of Si in suppressing plant disease. Of the ornamentals examined to date, morning glory, paper daisy, rose, tigergrass, and zinnia have all shown improved plant protection following silicon application (Brunings 2008; Locke et al. 2006; Rodrigues et al. 2015a). While the underlying mechanisms that govern disease protection are still not completely understood, research has shown that Si can form a mechanical barrier through polymerization below the cuticle and in the cell walls (Rodrigues et al. 2015b). This was the first proposed hypothesis to explain how this element reduced disease development. New insights, however, have shown that silicon's effect on plant resistance to a number of plant diseases may also occur through

mediated host plant resistance mechanisms against pathogen infection. On the basis of research conducted to date, the following facts about silicon in relation to plant disease suppression are known: A minimum silicon concentration is needed to suppress a plant disease; once that level has been obtained, plant disease suppression increases proportionally as the silicon concentration increases in plant tissues; silicon supply to the plant must be continuous or the disease-suppressing effects will be reduced or nonexistent; silicon will influence many components of host resistance, which include the incubation period, latent period, lesion number, and lesion size; silicon may confer resistance to susceptible and partially resistant plants at almost the same level as complete genetic resistance; only when applying silicon to the roots will this element change plant responses to pathogen infections at both the physiological and molecular levels; and silicon may suppress plant disease as effectively as fungicides (Datnoff and Rodrigues 2015b). Given the low Si content found in most soilless horticultural media and a number of soil orders, it would seem prudent to consider Si amendments as a simple, inexpensive method to reduce plant disease development.

Recent research has shown that aluminum (Al) is similarly taken up in plants at low levels and may be associated with plant health. It has been a long-held practice to lower soil pH with $AlSO_4$, which is accompanied by reductions in many plant diseases caused by species of the soilborne plant pathogens *Verticillium* and *Thielaviopsis*. However, evidence may suggest that the Al^{+} ion is partially responsible for suppression of disease through its effect on the germination of fungal spores in soil. Applications of different forms of Al to peat soils at pH 4 and 6 were followed by reductions in the densities of *Phytophthora parasitica* infecting vinca (Benson 1993). Given the problems ornamental growers face with *Phytophthora* diseases in peat-based media, additional research on Al amendments is definitely warranted and may lead to another simple, environmentally safe management strategy for disease control.

9 Summary

Mineral nutrition clearly has marked effects against plant diseases (Datnoff et al. 2007). These mineral elements often serve as the first line of defense against disease. A few examples have been highlighted where manipulating the mineral nutrition can reduce disease in ornamental plants (Kamenidou 2005). However, the fractionation of the data and disarray of concepts have hindered a working plan for using nutrition as a current disease management practice. Experimental studies to date have only focused on one plant, one element, and one disease in isolation of other variables and provide only "snapshots" of information. This dearth of experimental data on ornamentals, combined with the gigantic number of ornamental plant species whose fertility requirements differ widely from each other, makes the development of this disease management strategy a daunting task. Growers are encouraged to consider their fertility regimes based on the aforementioned information and make adjustments first on a small scale and then expand as the results

justify. Researchers are encouraged to explore an ornamental's horticultural and disease response to a wide array of elements applied in many combinations. The role that each element plays in disease must be viewed in the greater context of its numerous interactions with other elements, the host, soil medium, and with beneficial and pathogenic microorganisms.

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Postharvest Disease Management

11

Anastasios I. Darras

Abstract

Pre- and postharvest disease control for ornamental plants is mainly provided via fungicide or bactericide application. However, disease control with conventional chemical compounds carries the risk of resistance development by new pathogen races. Additionally, there is increasing public concern over fungicide usage in terms of human and environmental risk. For this reason, and over the past 20 years, researchers developed novel postharvest disease management strategies for cut flowers and other ornamentals. For example, the generally recognized as safe host defense inducers may provide an alternative solution to socially and environmentally less desirable control using conventional fungicides. There are also various biological agents and microorganisms that affect disease development via antagonism and, in many cases, help in integrated disease management (IDM) strategies. However, most of those biotic and/or abiotic agents have not yet been put into practice by growers, who merely rely on chemical control. The current chapter offers an overview on postharvest disease management of various pathogens infecting ornamental plants and cut flowers.

Keywords

Ornamentals • Cut flowers • Postharvest pathogens • Vase solutions • Chemical control • IPM • Elicitors

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R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_11

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1 Introduction

The majority of postharvest diseases lead to permanent loss of a plant's ornamental value. Although this is not relevant to the major issue of food safety, it is relevant to significant economic losses for growers and wholesalers (Darras et al. 2004). Postharvest diseases of ornamental plants and cut flowers destroy approximately 10–30% of the total crop yield produced worldwide (Dole and Wilkins 2004). Postharvest diseases usually cause these losses by reducing the quality or shortening the shelf life of ornamentals or the vase life of cut flowers. The fungi and bacteria causing postharvest diseases usually attack healthy, living tissue, which they disintegrate and rot. Often, however, other fungi and bacteria follow them and live saprophytically on the tissues already killed and macerated by the former pathogens (Agrios 1997).

Before applying treatments to control postharvest decay, it is crucial to recognize the concept of postharvest disease development. Postharvest phase is related to products that are ready for consumption (fruits and vegetables) or ready to be used (ornamentals) by consumers. Therefore, postharvest disease implies to the damaging of the final product at that phase. Apparently, the infection process starts in the greenhouse during cultivation, but disease symptoms develop postharvest. These symptoms are revealed as decay of the root system, as damage on leaves or on flowers, or as premature wilting and dieback of cut flowers held in water.

The current chapter reviews key conventional and novel disease control strategies successfully used during the last 20 years for the management of the most common postharvest diseases of ornamental plants and cut flowers.

2 Most Common Postharvest Pathogens Infecting Ornamental Plants and Cut Flowers

Postharvest diseases are caused by a relatively small number of *Ascomycetes* and Fungi Imperfecti and by few species of *Basidiomycetes* and bacteria (Agrios 1997). The bacteria are of the genera *Dickeya*, *Pectobacterium*, *Xanthomonas*, and

Pseudomonas. Microorganisms that develop and infect after harvest express weak pathogenic ability and are saprophytic in nature, although they infect via natural openings and wounds found on the mature plant's epidermis when conditions are favorable (Agrios 1997).

Postharvest diseases develop on plant tissue during storage and/or at transportation or after the consumer's purchase. During this period, the floricultural products may or may not show disease symptoms of infections that started in the greenhouse during production (i.e., latent infection). For example, *B. cinerea* conidia germinate on flower petals in water droplets and penetrate the epidermis with or without infection structures (Jarvis 1977; Salinas et al. 1989). Then, infection becomes invisible/latent until harvest, and symptoms develop slowly during storage at low temperatures and at high relative humidity (Elad 1988; Darras et al. 2006a). Infection is observed as either gray mold or as hypersensitive flecking on the host (Salinas et al. 1989; Salinas and Verhoeff 1995; Darras et al. 2006a). This latent period may last for up to 6 weeks depending on postharvest conditions, tissue maturity, and host resistance (Jarvis 1977).

2.1 Gray Mold (*Botrytis cinerea*)

2.1.1 Morphology, Geographic Occurrence, and Impact

B. cinerea Pers. belongs to the class *Deuteromycetes* and the phylum *Ascomycota*. The disease caused by *B. cinerea* is called gray mold or *Botrytis* blight. *B. cinerea* mycelium has the typical characteristics of the phylum *Ascomycota* (Jarvis 1977). Extension from the germinated spore occurs at the hyphal apex and growth of the hyphae increases over time. The conidiophores are tall, dark colored, and irregularly or dichotomously branched. The conidia are hyaline or pigmented, ellipsoid to obovoid – globoid – and usually continuous, with 1–3 septa (Ellis and Walter 1974). Conidia germinate in nutrient solutions and less readily in water to usually form 1 to 5 germ tubes (Jarvis 1977). The fungus is pathogenic to most of the cultivated ornamental potted plants and cut flowers. In the Netherlands, substantial quantities of *B. cinerea*-infected rose, *B. cinerea-infected* gerbera, and *B. cinerea*-infected freesia flowers are rejected by sellers in the UK at certain times of the year (Darras et al. 2004). Such rejections result in economic losses to both growers and wholesalers and make professional collaboration problematic.

2.1.2 Infection Process, Symptoms, and Host Range

Infection by *B. cinerea* occurs when a single conidium germinates, forms a germ tube, and penetrates an epidermal cell (Darras, et al. 2006a). Fungal hyphae then colonize adjacent cells resulting in visible lesions (flecks or specks) (Darras et al. 2006a). Gray mold on gerbera and freesia flowers is observed as small necrotic, dark-brown lesions, "spots." Similar symptoms developed in the laboratory under controlled conditions following artificial inoculation of gerbera or freesia inflorescences at temperatures ranging from 4 to 25 °C (Darras et al. 2006a; Salinas and Verhoeff 1995). *B. cinerea* infects rose (*Rosa hybrida*) flowers and produces necrotic

spots or blister-like patches on petal surfaces (Pie and de Leeuw 1991; Williamson et al. 1995). Infection has been described by Elad (1988) as restricted, brown, volcano-shaped lesions. *B. cinerea* damages phylloclades of *Ruscus* (*Ruscus aculeatus*) by causing small, dark, water-soaked necrotic lesions encircled by a faint halo. These lesions later become brown without growing in size (Elad et al. 1993). *B. cinerea* is also pathogenic to Geraldton waxflower (*Chamelaucium uncinatum*), an Australian native plant which holds a high ornamental and commercial value (Joyce 1993; Tomas et al. 1995). Geraldton waxflower sprigs showed increased abscission of flowers from their pedicels after inoculation with *B. cinerea*. Symptoms of gray mold on lisianthus are reported as crown rot, damping off, stem cankers, and stem, leaf, and flower blights. Gray mold also can cause postharvest deterioration of flowers (Wegulo and Vilchez 2007). Botrytis blight can be a very destructive disease for growers of cut liliums (*L. tigrinum*, *L. speciosum* var. *rubrum*, *L. regale*); by gathering information on genotypic resistance and making it available to growers, resistant and superior cultivars can be grown for commercial purposes or in private or public gardens, resulting in reduced losses from the disease and less reliance on fungicide applications (Daughtrey and Bridgen 2013). In a series of field trials from 2008 to 2011, the resistance of *Lilium* spp. against *B. elliptica* infection was evaluated (Daughtrey and Bridgen 2013). The most susceptible cultivars (70–94% spotted or scorched leaves) were Chianti, Connecticut King, Côte d'Azur, and Gironde and for *L. tigrinum* the cvs. Pink, Menorca, Red Alert, Royal Fantasy, Sweet Kiss, Vermeer, and White Heaven. In 2009, *Lilium* spp. plants with less than 21% symptomatic leaves were those of cvs. African Queen, Casa Blanca, Cobra, Conca d'Or, Le Rêve, Mozart Zanlazart, Sorbonne, and Time Out. In 2011, the most severely affected (70–100% symptomatic leaves) *Lilium* spp. plants were those of cvs. Chianti, Connecticut King, Cote d'Azur, Gironde, and Red Alert (Daughtrey and Bridgen 2013).

B. cinerea disease symptoms on geranium (*Pelargonium zonale*) plants have been described by Strider (1985) as flower blight, leaf blight, and stem rot. *Botrytis* blight, of scented geranium (*Pelargonium* sp.), caused by *B. cinerea*, is one on the most destructive diseases affecting geranium production causing stem, leaf, and flower blight. The disease symptoms are characterized by gray, fuzzy sporulating lesions commonly observed under humid conditions. Martinez et al. (2008) reported the infection of *Pelargonium* x *hortorum*, *Euphorbia pulcherrima*, *Lantana camara*, *Lonicera japonica*, *Hydrangea macrophylla*, *and Cyclamen persicum* by *B. cinerea.*

2.1.3 Biology and Epidemiology

Infection of gerbera (*Gerbera jamesonii*), freesia (*Freesia hybrida*), and rose (*Rosa hybrida*) plants occurs inside the glasshouse during crop cultivation, but symptoms develop during storage or transportation after passing through a latent period (Darras et al. 2004; Elad 1988; Harkema et al. 2013; Salinas and Verhoeff 1995). Favorable and/or fluctuations in temperature and relative humidity (RH) after harvest result in rapid disease development (Darras et al. 2006a; Harkema et al. 2013; Salinas et al. 1989; Williamson et al. 1995). For example, infection of *Fuchsia hybrida* inflorescences after artificial inoculation appeared in less than 24 h at 12 °C and

80–90% RH. Even at 5 °C, disease symptoms were evident in a saturated atmosphere (ca. 100% RH) within the first 24 h of incubation. Moreover, clear differences in *Botrytis* damage were observed between wet and dry long-term transport of cut roses of vars. Red Naomi, Aqua, and Avalanche at low temperatures and at high RHs (Harkema et al. 2013). *Botrytis* developed during dry or wet transport, but infection was significantly lower in dry transport conditions. These findings showed that dry transport of roses has a significant positive effect on product quality with special focus on *Botrytis* development (Harkema et al. 2013).

2.2 *Alternaria* Species

2.2.1 Morphology, Geographic Occurrence, and Impact

Alternaria spp. belong to the Fungi Imperfecti, from class *Hyphomycetes*, from order *Moniliales*, and from family *Dematiaceae* (Rotem 1994). Some species of *Alternaria* are the asexual anamorphs of the ascomycete *Pleospora*, while others are speculated to be anamorphs of *Leptosphaeria*.

Alternaria species are mainly saprophytes. However, some species have acquired pathogenic capacities collectively causing disease over a broad host range (Rotem 1994; Thomma 2003). Alternaria diseases are quite common on ornamental crops, including landscape plants and cut flowers. The most common species of *Alternaria* found is *A. alternata* (infecting vinca, dahlia, gerbera, hibiscus, and geranium) (Simmons 1992; Thomma 2003). In most other cases, the species of *Alternaria* have not been identified. Spores of *Alternaria* spp. are dark brown to black and appear in black masses on leaf and petal spots. These are generally spread by watersplash or air movement. In some plants, such as zinnia (*Zinnia* sp.), the infection originates from contaminated seeds (Rotem 1994).

2.2.2 Infection Process, Symptoms, and Host Range

In general, *Alternaria* species are foliar pathogens that cause a relatively slow destruction of host tissues through the reduction of photosynthetic potential. Infection leads to the formation of necrotic lesions, which sometimes have a target-like appearance due to growth interruptions caused by unfavorable conditions and diurnal cycles (Rotem 1994; Thomma 2003). The fungus resides in the center of the lesion, which is surrounded by an un-invaded chlorotic halo, a symptom that is commonly observed for the infection process of necrotrophic pathogens. This zone is created by the diffusion of fungal metabolites like toxins (Agarwal et al. 1997; Rotem 1994; Tewari 1983). Members of the genus *Alternaria* frequently cause quiescent infections in which the fungus enters the tissue and remains dormant until conditions favor infection development (Rotem 1994).

A. alternata caused leaf spots on different geranium species (e.g., *Pelargonium domesticum*, *P. peltatum*, and *P. scented*). The symptoms were brown, irregular spots with increasing diameters (Furukawa and Kishi 2001). These spots turned yellow until they covered the whole leaf area. Similar spots occurred on the flower petals. On petunia, the disease was characterized by small spots which were initially water

soaked (Agarwal and Gupta 1984). These spots turned reddish brown or black, were roughly circular, and reached 3–4 mm in diameter. Their centers were frequently tan to white.

Alternaria leaf spots on impatiens were small (less than 1 mm in diameter), initially water soaked that turned reddish brown with tan centers (Wolkan and Grego 2004). They reached 3–4 mm in diameter and were round. Spots frequently merged to affect most parts of the leaf.

A. dianthi and *A. dianthicola* each cause diseases on carnation and pinks that are recognized by the gray-brown leaf and petal spots with purple margins. Black spore masses were sometimes observed in those spots (Arbeláez 1987).

Infection of *Zinnia acerosa* plants in Dallas, TX, field plots appeared as small, brown flower spots, which enlarged until they covered whole petals, causing conspicuous flower blighting (Colbaugh et al. 2001). Microscopic examination of lesions from infected flower blossoms indicated the presence of short-beaked, cylindrical spores near the smaller lesions on flower petals. Isolation from symptomatic flower petals consistently yielded cultures of an *Alternaria* sp. strain with long chains of conidia.

2.2.3 Biology and Epidemiology

In general, *Alternaria* species are favored by wet and warm conditions. Conidia germinate in just 35–45 min when temperatures range between 28 and 30 °C (Rotem 1994). Tissue penetration and infection occur in 12 h at 10 °C, 8 h at 15 °C, and 3 h at 22 °C (Rotem 1994). Epidemiological studies on safflower (*Carthamus tinctorius* L.) plants infected by *A. carthami* showed that the percentage of leaf and flower area with infection symptoms was greatest at 25 °C, while the frequency of stomatal penetration was greatest at 30 °C (MacRae et al. 1984). Additionally, a long period of wetness (i.e., >6 h) was needed for symptoms to develop. Safflower plants that were kept wet for less than 6 h did not develop symptoms.

2.3 Bacterial Species

Bacteria are invariably present whenever fleshy plant tissues are rotting in the field or in storage (Agrios 1997). Bacterial rot of plants is characterized by soft and watery tissue; cellular debris also frequently oozes from infected cracks. In many soft rots, the bacteria involved are not plant pathogens but rather live saprophytically as secondary parasites (i.e., in tissues already killed by pathogens and environmental causes or in weakened senescent tissues that are unable to resist microbial attack) (Agrios 1997). This is basically the case for harvested ornamental material. Usually, natural defense responses of harvested products are low and sharply decrease. During this period, bacteria may grow and macerate mature and susceptible tissues. However, some bacteria may attack and infect living tissues and cause soft rots in the field or in storage. The most common bacterial genera causing postharvest rots of ornamentals are *Dickeya dadantii* (syn. *E. chrysanthemi*), *Pectobacterium*

carotovorum spp. *carotovorum* (syn. *E. carotovora* pv. *carotovora*), *Xanthomonas campestris*, and *Pseudomonas fluorescens*.

2.3.1 *Dickeya and Pectobacterium* Species

Dickeya and Pectobacterium and closely related bacteria are straight rods, 0.5–1.0 by 1.0–3.0 μm, and are motile by means of several to many peritrichous flagella (Agrios 1997). These bacteria are the only plant pathogenic bacteria that are facultative anaerobes. Some do not produce pectic enzymes and cause necrotic or wilt diseases (the "amylovora" group), whereas the "carotovora" group has strong pectinolytic activity and causes soft rots (Agrios 1997). Soft rot caused by *Pectobacterium carotovorum* spp. *carotovorum* has a significant negative impact on the flower bulb industry (van Doorn et al. 2011). Bulbous ornamentals such as *Hyacinthus*, *Dahlia*, *Iris*, *Muscari*, *Freesia*, and *Zantedeschia* are susceptible to soft rot infections that damage plant tissue and decrease flower production. In Malaysia, kalanchoe (*Kalanchoe gastonis-bonnieri*) can be infected by *P. carotovorum* spp. *carotovorum* during cultivation in the greenhouse or during postharvest storage or transportation (Golkhandan et al. 2013). Recent experiments by Gracia-Garza et al. (2004) showed the effects of phosphorous addition to soilless mixes or to nutrient solutions on soft rot incidence caused by *P. carotovorum* spp. *carotovorum* on calla lilies (*Zantedeschia* spp.). Soft rot incidence increased by up to 51% when a soilless mix was amended with superphosphate in comparison to the unamended soilless mix. In contrast, addition of phosphorous in the nutrient solution met the phosphorous needs of the plant without enhancing the soft rot. The plant height, fresh mass, and number of flowers per plant were greater in calla lilies irrigated with nutrient solution containing phosphorous than no phosphorous treatments (Gracia-Garza et al. 2004).

2.3.2 *Xanthomonas* Species

The cells are straight rods, 0.4–1.0 by 1.2–3.0 μm, and are motile by means of a polar flagellum (Agrios 1997). Growth on agar media is usually yellow, and most are slow growing. All species are plant pathogens and are found only in association with plants or plant materials (Agrios 1997). Different *Xanthomonas* species have produced large-scale crop damages and losses in the past. For example, the bacterium *X. campestris* pv. *dieffenbachiae* wiped out the whole anthurium (*Anthurium andraeanum*) crop production in the French Antilles in the 1980s (Anais et al. 2000). In 1972, Hayward described the disease caused by *X. dieffenbachiae*, on cv. Kansako Red of *A. andraeanum*, as angular, pale brown, necrotic spots, 1–3 mm with marked chlorotic halos. Scales of bacterial exudate occurred on the under surface of older leaves, while on younger leaves the scales were less obvious, and the lesions were dark brown to black and more extensive (Hayward 1972). The pathogen *X. campestris* was found to be pathogenic on *Syngonium* plants (*Syngonium podophyllum*), and the symptoms of the disease were distinct from those infected by *X. campestris* pv. *syngonii* (Dickey and Zumoff 1987).

2.3.3 *Pseudomonas* Species

The pseudomonads are straight to curved rods, 0.5–1.0 by 1.5–4 μm. They are motile by means of one or many polar flagella (Agrios 1997). Most pathogenic *Pseudomonas* species infect plants. *P. fluorescens* produces yellow-green, diffusible, fluorescent pigments on a low-iron medium. Temperature and relative humidity both play important roles in *Pseudomonas* disease development. For example, the number and size of lesions formed on chrysanthemum and pelargonium leaves and stems after *P. cichorii* infection increased with increases in temperature from 16 to 28 °C but were greatly inhibited at temperatures >28 °C (Jones et al. 1984). Lesions on leaves of both plant species tested constantly expanded under high-moisture conditions. Different isolates of *P. cichorii* were found to be pathogenic to geranium (*P. hortorum*) (Engelhard et al. 1983), different magnolia species (*M. grandiflora*, *M. macrophylla*, *M. soulangeana*, and *M. tripetala*) (Mullen and Cobb 1984), chrysanthemum (*Dendranthema grandiflora*) (Jones et al. 1990), and schefflera (*Schefflera arboricola*) (Chase and Brunk 1984). In greenhouse-grown impatiens, *P. syringae* infections were recorded as large, water-soaked, necrotic lesions on leaves (Cooksey and Koike 1990). However, no reactions occurred after inoculation of *Begonia* × *semperflorens-cultorum*, *Dieffenbachia maculata*, or *Calendula officinalis* with the pathogen.

2.3.4 Stem Blockage of Cut Flower Stems by Bacteria

Bacterial occlusions can cause interruption of water uptake and wilting problems in cut flowers. A high number of bacteria were detected using light microscopy at the cut surface of dahlia flowers (*Dahlia variabilis*) placed in water for a number of days (van Doorn 1997). Ultrastructural investigations of cut roses also showed that a population of bacteria at the cut surface was responsible for vascular occlusions (van Doorn et al. 1991). For most cut flower species, bacterial populations in holding solutions are relatively small at the grower's storage but increase during handling at the auctions and retailers (Hoogerwerf and van Doorn 1992).

Microorganisms found inside the xylem of flower stems or growing in water or in holding solutions resulted in a decrease in hydraulic conductivity of the stems, especially in the basal stem segment (van Doorn and De Witte 1991). The development of occlusions in the stems of rose cut flowers was correlated with the increasing numbers of microorganisms in stems (Florack et al. 1996). Postharvest increase in stem flow resistance was dependent on the presence of microorganisms in cv. Forever Yours roses (Marousky 1969). High bacterial counts in stems were also correlated with vascular occlusion in the petioles of *Adiantum raddianum* fern fronds (van Doorn et al. 1991). Not only bacteria but also yeasts and filamentous fungi can lead to vascular blockage (Put and Clerkx 1988). For example, in carnation (*D. caryophyllus*), increasing numbers of bacteria and yeasts in the vase solutions resulted in a significant decrease in flower vase life (van Doorn et al. 1995; Zagory and Reid 1986). Among those microorganisms that negatively affected vase life of carnations, *Pseudomonas* bacteria were the most devastating. Moreover, solution turbidity attributed to bacterial growth in the vase was negatively correlated with both the vase life ($P < 0.01$; $r^2 = -0.48$) and the vase solution usage ($P = 0.05$; $r^2 = -0.65$) of cut cv. Baccara roses (Pompodakis et al. 2004).

Reduction of the vase life of cv. Sonia rose was found after inoculations of 10^6 cfu/mL with *Bacillus subtilis*, *Enterobacter agglomerans*, *P. fluorescens*, or *P. putida* (Put and Jansen 1989). The vase life of "white sim" carnations was also reduced when the vase water was inoculated with 5×10^8 cfu/mL suspensions that contained a mixed bacterial population of *Pseudomonas* spp., *Acinetobacter calcoaceticus*, and *Alcaligenes* sp. (van Doorn et al. 1995). Numbers of bacteria in the vase water of gerbera flowers that showed a curvature of more than 90° were found to be 10^6 and 10^8 cfu/ml in the cvs. Liesbeth and Mickey, respectively (van Doorn et al. 1994). Bacterial counts reached a maximum of 10^7 cfu/ml after a few days of vase life in roses, carnations, tulips, and chrysanthemums (van Doorn 1997). When cut cv. Sonia roses were put in vases for 1–4 days, the number of bacteria in the basal 5 cm of the stems was linearly correlated with the number of bacteria in the vase solution, indicating that bacteria in the flower stems contaminated the vase solution (van Doorn and De Witte 1991). Bacteria found in the vase water belonged to the *Achromobacter*, *Alcaligenes*, *Bacillus*, *Escherichia*, *Flavobacterium*, *Micrococcus*, and *Pseudomonas* genera. In the vase water in which rose stems were held, the predominant bacterium found was *Pseudomonas*, while *Enterobacter* was a minor accompanying genus (Florack et al. 1996).

The effect of bacteria on vase life is mainly physical (van Doorn 1997). Hydraulic conductivity of rose stems placed in water containing either 5×10^9 or 2×10^7 cfu/ml of either living or dead bacteria declined rapidly (De Witte and van Doorn 1992). Occlusion by bacteria does not depend on their physiological activity or any defense response of the plant (van Doorn 1997). Bacterial extracellular polysaccharides and globular proteins can also result in vascular blockage (De Witte and van Doom 1992). When bacterial growth was eliminated, vascular occlusion was still apparent due to the produced extracellular polysaccharides (van Doorn 1997). Vascular blockage was also found in the basal end of the stems of cut roses when the extracellular polysaccharides from *P. cepacia* were added to sterile water (De Witte and van Doorn 1992). A thick layer of material consisting of bacteria with associated extracellular polysaccharides was often found to cover the xylem at the cut surface after a few days in the vase (van Doorn 1997).

Microorganisms through their pectic enzymes may also block the xylem cells, inducing numerous loose vessel fragments (Put and Rombouts 1989). When pectolytic enzymes, such as pectate lyase produced by *P. fluorescens* and polygalacturonase produced by *Kluyveromyces fragilis*, were added to vase water of "Sonia" roses, water relations were disturbed. This may be due to enzymatic degradation of the structures of xylem vessels (Put and Rombouts 1989).

3 Management

In most cases, management of postharvest diseases should be applied before harvest in the greenhouse or in the field (Darras et al. 2006a, 2007). This is crucial as pathogens complete their life cycles in the soil or the growing media. Inoculum loads are always present inside the greenhouses and may infect host tissue when

conditions are favorable. Preharvest infections may become latent and develop postharvest in a compatible host-pathogen interaction (Agrios 1997; Darras et al. 2006b). For that reason, sources of infection should be eliminated, and preventive management has to be applied by means of fungicide treatments. However, caution should be given as common postharvest pathogens such as *B. cinerea*, *Penicillium* spp., and *Alternaria* spp. may develop fungicide resistance. Careful processing of the final product is also critical as it minimizes mechanical damage and significantly reduces subsequent wastage due to postharvest fungal or bacterial infection.

3.1 Chemical Agents (Fungicides/Bactericides)

Various fungicides are used for the pre- and postharvest control of pathogens (Table 1). The best-known chemical classes for *B. cinerea* control include the chlorobenzenes, benzimidazoles, and dicarboximides (Panagiotarou and Chrisaugi 1998). Grinstein et al. (1997) tested pyrimethanil and prochloraz + Zn + folpet against *B. cinerea* infecting rose flower stems. Prochloraz + Zn + folpet controlled infection by increasing plant cover density. In contrast, pyrimethanil + Zn + folpet was better in preventing *B. cinerea* disease when rose petals were exposed to vapors, and no direct contact with the fungicide was used. It was concluded that *Botrytis* blight was delayed when cut rose flower bunches were wrapped in packing paper strips or cellophane bags and previously sprayed with pyrimethanil (Grinstein et al. 1997).

In field evaluations, the fungicides hexaconazole, carbendazim, propineb, chlorothalonil, mancozeb, and the biotic agents *P. fluorescens*, neem oil and garlic clove extracts were tested against *A. alternata* infecting chrysanthemum plants (Kumar et al. 2011). Hexaconazole applied at 0.1% was the most effective against the disease. Chrysanthemum plants treated with hexaconazole showed the lowest disease index (i.e., 4.49) compared to the control plants followed by those treated with chlorothalonil at 0.2% and mancozeb at 0.2%. Furthermore, plants treated with hexaconazole recorded the highest yield of 76.25 quintals/ha (Kumar et al. 2011).

According to Nagrale et al. (2012), preventive sprays of gerbera plants grown in the greenhouse with a range of recommended fungicides for *A. alternata* were more effective compared to curative applications. Preventive sprays of 0.6% Bordeaux mixture, 0.1% tricyclazole, and 0.1% iprodione + carbendazim reduced *A. alternata* disease by up to 95.85%, 96.59%, and 95.88%, respectively, compared to the untreated control plants (Nagrale et al. 2012).

Preplant copper-based dips of calla lily (*Zantedeschia elliottiana*) rhizomes of cv. Yellow were used to prevent plant losses that resulted from latent field infections by *P. carotovorum* (Blom and Brown 1999). More than 90% of the *P. carotovorum* inoculated rhizomes collapsed within 5 weeks after potting. Within the non-inoculated rhizomes, those treated with copper-based compounds showed significantly lower bacterial soft rot percentages than with a quaternary ammonium compound or potassium peroxymonosulfate + sodium chloride +

Table 1 Chemical compounds used to control postharvest pathogens of florists' crops

Pathogen	Host	Application	Chemical or active ingredient	Reference
A. alternata	Chrysanthemum	Preharvest	Hexaconazole, carbendazim 12% + mancozeb 63%, propineb, chlorothalonil, mancozeb, carbendazim 25% + iprodione 25%	Kumar et al. 2011
	Gerbera	Preharvest	Various chemicals	Nagrale et al. 2012
B. cinerea	Various crops	Preharvest/ postharvest	Sulfur, phthalimides, thiram, chlorothalonil, diethofencarb, prochloraz, and tebuconazole	Panagiotarou and Chrisaugi 1998
	Various crops	Preharvest/ postharvest	Folpet, captan, chlorothalonil, dichlofluanid	Ogawa et al. 1977
	Geraldton waxflower	Postharvest	Pyrimethanil, iprodione	Taylor et al. 1999
	Leucospermum	Postharvest	Benomyl, carbendazim + flusilazole, chlorothalonil, cyprodinil + fludioxonil, fenhexamid, iprodione, and pyrimethanil	Bezuidenhout et al. 2010
	Rose	Postharvest	Prochloraz, iprodione, dichlofluanid	Elad 1988
	Rose	Postharvest	Picro-cupric-ammonium formate	Hammer and Marois 1989
	Ruscus	Postharvest	Tebuconazole, polyoxin D, and polyoxin B	Elad et al. 1993
P. carotovorum	Calla lily	Preharvest	Quaternary ammonium compound, copper, potassium peroxymonosulfate + sodium chloride + other ingredients	Blom and Brown 1999

other ingredients during the first 6 weeks of forcing. During the remainder of the forcing period, there were no differences in weekly losses of rhizomes (Blom and Brown 1999).

3.2 Environmental Factors, Ethylene Inhibition, and Irradiation

3.2.1 Storage and Transport

Differences in *Botrytis* damage to cut roses of vars. Red Naomi, Aqua, and Avalanche were observed between wet and dry transport at low temperatures and high RHs (Harkema et al. 2013). *Botrytis* developed during dry or wet transport, but infection was significantly lower in dry transport conditions. These findings suggested that dry transport of roses had a significant positive effect on product quality by eliminating *Botrytis* development (Harkema et al. 2013).

Packaging of cut flowers during storage or transport may affect postharvest disease development. For example, package design (size and location of ventilation holes) had a significant effect on the proportion of flowers of rose cv. Sweet Promise with spotting caused by *B. cinerea* (van der Sman et al. 1996). Boxes with large ventilation holes that facilitated effective air ventilation around the packed flowers helped in RH reduction and avoidance of condensed water dispersion on the buds and, therefore, eliminated the chance of conidial germination and consequent infection. This resulted in a maximum of 42% infected flowers, whereas using commercial boxes resulted in 62% infected flowers (van der Sman et al. 1996).

3.2.2 1-Methylcyclopropane (1-MCP)

1-MCP is an ethylene inhibitor used to prevent the detrimental effects of ethylene to climacteric fruits, vegetables, and cut flowers (Serek et al. 1995). In recent research it was shown that 1-MCP may be used to control postharvest Botrytis blight of ornamental plants and cut flowers (Table 2). For example, 3.62 μL 1-MCP/L significantly reduced *B. cinerea* infection of *D. caryophyllus* cv. Idra di Muraglia and infection of *Cyclamen persicum* cv. Hallows Bianco Puro Compatto petals (Seglie et al. 2009, 2012). Furthermore, 1-MCP treatments in cyclamen at different rates ranging from 0.38 to 3.62 μL/L slowed *B. cinerea* infection by up to 28 days. Using the lowest concentration of 0.38 μL/L, 1-MCP slowed *B. cinerea* development on *R. hybrida* cv. Ritz by up to 3 days compared to the control flowers. It was concluded that 1-MCP can limit *B. cinerea* development, but its effect was depended on plant species and 1-MCP concentration (Seglie et al. 2009). This funding was

Table 2 Postharvest 1-MCP application on different ornamentals for management of *Botrytis cinerea*

Host	Treatment	1-MCP concentration	Reference
Carnation	Postharvest	0.38–3.62 μL/L	Seglie et al. 2009, 2012
Cyclamen	Postharvest	0.38–3.62 μL/L	Seglie et al. 2009, 2012
Rose	Postharvest	0.38–5.34 μL/L	Seglie et al. 2009; Favero et al. 2015

contradicted by a very recent study on *R. hybrida* cv. Avant Garde flowers which reported that 1-MCP did not protect cut roses artificially inoculated with *B. cinerea* (Favero et al. 2015).

3.2.3 Irradiation Treatments (Gamma, Ultraviolet-C (UV-C))

Gamma irradiation has been used as an alternative nonchemical treatment for pest and postharvest disease control in over 40 countries (Hallman 2011) (Table 3). Ionizing irradiation has been used for many years to sterilize plant and flower surfaces by killing the pests without damaging the plant tissues. Among its advantages is that treatment with gamma irradiation may improve the vase life of cut flowers (Cia et al. 2007). The effects of irradiation are dependent on fungal development, treatment dose, moisture condition, composition of treated products, and storage conditions. Although doses as low as 0.2 kGy were sufficient to protect against the most harmful insects, it was not possible to fully control postharvest fungal diseases of cut flowers (Blank and Corrigan 1995). Gamma irradiation at 4.0 kGy showed complete inhibition of spore germination and mycelial growth of *B. cinerea* in vitro (Chu et al. 2015). On the other hand, in vivo, antifungal activity of gamma irradiation against *B. cinerea* on cut rose flowers of vars. Shooting Star and Babe was dose dependent (Chu et al. 2015).

UV-C irradiation is another type of physical energy used to control postharvest spoilage. Exposure to low doses of UV-C can reduce storage rots in fruits, vegetables, and flowers (Darras et al. 2010a, 2012a; Wilson et al. 1997) (Table 3). This disease reduction was attributed to direct germicidal effects of UV-C on the pathogen and/or to defense response induction in the exposed host tissue (Darras et al. 2012a, 2015). Previous research has shown that exposure to low doses of UV-C (e.g., 0.5–2.5 kJ/m^2) may reduce *B. cinerea* postharvest infection on gerbera and freesia flowers by up to 70 and 75%, respectively (Darras et al. 2010a, 2012a, b). This reduction was attributed to direct fungicidal activity, although reductions of 55 and 24% were recorded as a result of an induced defense response. Additionally, UV-C irradiation at 2.5 kJ/m^2 reduced lesion size caused by *B. cinerea* on leaves of geranium (*P. hortorum*) plants by up to 50%, compared to the nonirradiated control plants (Darras et al. 2015). These disease reductions were the possible result of induction of defense which peaked 24 h post irradiation. Such defense responses were often associated with increases in polyphenol oxidase (PPO) and/or phenylalanine ammonia lyase (PAL) activities (Darras et al. 2012a).

Table 3 Irradiation treatments applied postharvest to various ornamentals for management of *Botrytis cinerea*

Host	Treatment	Doses	Reference
Freesia	UV-C – postharvest	0.5–10 kJ/m^2	Darras et al. 2010a
Geranium	UV-C – postharvest	0.5–10 kJ/m^2	Darras et al. 2015
Gerbera	UV-C – postharvest	0.5–10 kJ/m^2	Darras et al. 2012a, b
Rose	Gamma – postharvest	0.2–4 kGy	Chu et al. 2015

3.2.4 Organic and Inorganic Compounds

There is a significant amount of published research on alternative organic and/or inorganic compounds used as postharvest treatments to control diseases of ornamentals and cut flowers (Table 4). For example, sodium hypochlorite (NaOCl), a chlorine-containing compound, has been used to control bacterial and fungal contamination on fruits and vegetables, on processing equipment, and in flower vase solutions (Suslow 1997; van Doorn et al. 1990). Its strong oxidizing activity has a broad-spectrum antimicrobial impact. When NaOCl is dissolved in water, it ionizes to Na^+ and the hypochlorite ion in equilibrium with hypochlorous acid (HOCl), the active moiety (Dychdala 1983). HOCl damages microbe cell membranes, proteins, and nucleic acids by oxidative degradation upon contact (McDonnell and Russell 1999). A postharvest dip of 200 μL/L NaOCl for 10 s at 20 °C provided significant control against *B. cinerea* infecting rose flowers of cvs. Akito and Gold Strike (Macnish et al. 2010). In this experiment, Clorox® Ultra household bleach solution was more effective than the laboratory grade NaOCl. Additionally, when solution pH was reduced from 9.7 (unadjusted) to 7.0, efficacy of NaOCl was considerably improved. Treating cv. Gold Strike rose flowers in this pH-adjusted NaOCl solution resulted in drop in the level of infection on petals to lower levels to those recorded after postharvest dips with the conventional fungicides fludioxonil, copper, cyprodinil, and cyprodinil + fludioxonil. Applying NaOCl prior to a 3- or 10-day commercial shipment also provided the most consistent *B. cinerea* disease control for a wide range of rose cultivars compared to conventional fungicides (Macnish et al. 2010). Woltering et al. (2015) have shown that irrespective of the type of packaging and shipment conditions, roses that received a pre-shipment treatment with 100–150 mg/L NaOCl showed significantly lower *Botrytis* disease compared to the non-NaOCl-treated roses. The NaOCl treatment was generally more effective than comparable treatments with commercial fungicides (Woltering et al. 2015).

Calcium sulfate ($CaSO_4$) preharvest treatments were used to control postharvest *B. cinerea* infection on cut rose flowers (Capdeville et al. 2005). Application of 10–20 mM $CaSO_4$ 24 h prior to harvest resulted in disease severity reduction by up to 86%. In the non-inoculated assay, the maximum reduction was 86%, while in the inoculated assay, it was 76% (Capdeville et al. 2005).

GA_3 pulsing and spraying treatments at 20 mg/L for 24 h suppressed *B. cinerea* in detached rose petals of cvs. Mercedes and Sonata (Shaul et al. 1995). Since the

Table 4 Recent research on the effects of organic and inorganic compounds used to control *Botrytis cinerea* on ornamentals

Host	Treatment	Concentrations	Reference
Rose	NaOCl – postharvest	100–200 mg/L	Macnish et al. 2010; Woltering et al. 2015
Rose	$CaSO_4$ – postharvest	10–20 mM	Capdeville et al. 2005
Rose	GA3 – postharvest	20 mg/L	Shaul et al. 1995
Geraldton waxflower	S-carvone – postharvest	0.64 and 5.08 mM	Hu et al. 2009

studied concentrations of GA_3 were nontoxic to the fungus in vitro, the authors suggested that GA_3 probably activated natural defense responses. Although GA_3 was suggested by Shaul et al. (1995) as a potential alternative to conventional chemical fungicides, no further research on this topic is available.

S-carvone used in concentrations of 0.64 and 1.27 mM significantly reduced *B. cinerea* conidial germination in vitro on glass slides (Hu et al. 2009). Additionally, S-carvone used at concentrations of 2.54 and 5.08 mM inhibited *B. cinerea* spore germination and suppressed mycelial growth on agar. However, S-carvone treatments at 2.54 and 5.08 mM applied to cut waxflowers of cv. My Sweet Sixteen (*Chamelaucium floriferum* × *C. uncinatum* × *C. axillare*) sprigs generally gave no control of *B. cinerea* infection and on flower abscission (Hu et al. 2009).

3.3 Biological Agents/Microorganisms

Over the past 100 years, research has repeatedly demonstrated that phylogenetically diverse microorganisms can act as natural antagonists of various plant pathogens (McSpadden Gardener and Fravel 2002) (Table 5). The interactions between microorganisms and plant hosts can be complex. Interactions that lead to biocontrol can include antibiosis, competition, induction of host resistance, and predation (Cook and Baker 1983). When testing bacterial and fungal isolates from the environment for biocontrol activities, between 1 and 10% showed at least some capacity to inhibit the growth of pathogens in vitro. However, fewer isolates can suppress plant diseases under diverse growing conditions, and still fewer have broad-spectrum activity against multiple pathogenic taxa. Nonetheless, intensive screens have yielded numerous candidate organisms for commercial development. Some of the microbial taxa that have been successfully commercialized and are currently marketed as EPA-registered biopesticides in the United States include bacteria belonging to the genera *Agrobacterium*, *Bacillus*, *Pseudomonas*, and *Streptomyces* and fungi belonging to the genera *Ampelomyces*, *Candida*, *Coniothyrium*, and *Trichoderma* (McSpadden Gardener and Fravel 2002). Screening is generally a critical step in the development of biocontrol agents. The success of all subsequent stages depends on the ability of a screening procedure to identify an appropriate candidate. Primary screens for new biocontrol microbes are still undertaken, and it seems likely that continued prospecting will be required to diversify the potential applications of biocontrol as well as replace more widely used biocontrol products before resistance develops (Larkin and Fravel 1998).

Table 5 Biological agents used for the management of *Botrytis cinerea* infecting ornamentals

Pathogen	Host	Biological agents	Reference
B. cinerea	Cyclamen	*U. atrum*, *G. roseum*	Kohl et al. 1998
B. cinerea	Geranium	*R. glutinis* PM4, *B. subtilis*, *T. harzianum*, *G. catenulatum*	Buck 2004; Elmhirst et al. 2011
B. cinerea	Geraldton waxflower	*Pseudomonas* sp. 677	Beasley et al. 2001

In regard to ornamentals, *Pseudomonas* sp. 677 significantly reduced conidial germination and retarded germ tube elongation of *B. cinerea* (Beasley et al. 2001). None of the yeasts or fungal isolates tested were found effective in reducing conidial germination or retarding germ tube elongation in vitro, but several significantly inhibited *B. cinerea* growth in vivo. *Fusarium* sp., *Epicoccum* sp., and *Trichoderma* spp. were the most antagonistic in vivo. Of the isolates tested on waxflower, *Pseudomonas* sp. 677 was the most antagonistic against *B. cinerea* by delaying waxflower abscission by up to 3 days (Beasley et al. 2001).

The yeast *Rhodotorula glutinis* PM4 significantly reduced the development of lesions caused by *B. cinerea* on geranium, although efficacy varied between trials (Buck 2004). Treatment with *R. glutinis* in combination with azoxystrobin or trifloxystrobin at 7.5 μg a.i./mL significantly reduced lesion development, compared to treatment with the yeast or the fungicide alone (Buck 2004). Vinclozolin at 250 or 500 μg a.i./mL, in combination with *R. glutinis* PM4, significantly reduced the development of lesions caused by a *B. cinerea* isolate resistant to vinclozolin. Mancozeb did not increase biocontrol efficacy, while thiophanate methyl negatively affected it. Additionally, all combinations of *R. glutinis* PM4 with azoxystrobin, trifloxystrobin, or vinclozolin provided highly effective and consistent *B. cinerea* disease control not acquired with fungicide or yeast treatments alone (Buck 2004). The biological agents *B. subtilis*, *T. harzianum*, and *Gliocladium catenulatum* significantly reduced *B. cinerea* blight of geranium plants (Elmhirst et al. 2011). Reductions in lesion numbers of geraniums treated with *B. subtilis*, *T. harzianum*, or *G. catenulatum* were higher compared to the fungicide Captan 80 (Elmhirst et al. 2011).

Ulocladium atrum and G. roseum were used in preharvest trials in commercial greenhouses to control *B. cinerea* blight in cyclamen (Kohl et al. 1998). Both *U. atrum* and *G. roseum* were effective in reducing *B. cinerea* infection on the petioles. The study showed that both agents colonized all treated parts of the cyclamen providing adequate management of the disease via antagonism (Kohl et al. 1998).

3.4 Elicitors of Defense Responses

3.4.1 Jasmonic Acid (JA) and Methyl Jasmonate (MeJA)

The efficacy of MeJA has been tested postharvest on potted ornamental plants and on cut flowers (Table 6). Most tests were carried out in recent years and are still in progress. For example, pre- and postharvest treatments with MeJA on cut Geraldton waxflowers conferred variable protection against postharvest infections by *B. cinerea* (Dinh et al. 2007). JA and MeJA provided systemic protection to various rose cultivars (e.g., Mercedes, Europa, Lambada, Frisco, Sacha, and Eskimo) against *B. cinerea* (Meir et al. 1998). MeJA applied as a postharvest pulse significantly reduced *B. cinerea* lesion size on detached rose petals. In the same study, MeJA at 100–400 μM showed in vitro antifungal activity on *B. cinerea* spore germination and germ tube elongation. Similarly, a postharvest MeJA pulse, spray, or vapor treatment at 200 μM, 600 μM, or 1 μL/L, respectively, significantly reduced *B. cinerea* petal

Table 6 Recent research on elicitors of defense responses tested against postharvest pathogens of ornamentals

Pathogen	Host	Elicitor	Reference
Alternaria sp., *B. cinerea*	Geraldton waxflower	SA, MeJA, ASM	Beasley 2001; Zainuri et al. 2001; Eyre et al. 2006; Dinh et al. 2007
B. cinerea	Freesia	ASM, MeJA	Darras et al. 2005, 2006a, b, 2007
	Peonies	MeJA	Gast 2001
	Rose	ASM, MeJA	Meir et al. 1998

specking on cut *F. hybrida* inflorescences of cv. Cote d'Azur (Darras et al. 2007). Darras et al. (2005) demonstrated that MeJA vapor was more effective in reducing *B. cinerea* disease on freesia flowers at 20 °C than at 5 or 12 °C. At 20 °C, disease severity, lesion numbers, and lesion diameters were reduced by 58, 50, and 48%, respectively (Darras et al. 2005). Moreover, 1–100 μL/L MeJA postharvest vapor treatment reduced *B. cinerea* development on cut Geraldton waxflower cvs. Purple Pride and Mullering Brook sprigs (Eyre et al. 2006). Application of vapor MeJA to fresh cut peonies resulted in the lowest disease severity and in an improvement of vase life compared to the untreated controls (Gast 2001).

MeJA applied preharvest had variable responses against postharvest *B. cinerea* infection. MeJA was very effective in suppressing the development of postharvest *B. cinerea* disease for greenhouse-grown freesias when applied as a spray at 0.2 mM (Darras et al. 2006b). Dinh et al. (2007) reported that multiple sprays of ≤1000 μM MeJA to field-grown plants significantly reduced *B. cinerea* on Geraldton waxflower cv. My Sweet Sixteen cut sprigs that were non-inoculated or artificially inoculated with the pathogen. MeJA, applied as leaf spray at 10 mM, completely inhibited *P. carotovorum* development in calla lily (*Z. aethiopica*) leaves and afforded a long-lasting effect (Luzzatto et al. 2007). MeJA was more effective than acibenzolar-S-methyl (ASM) in long-term control suggesting that the defense response of calla lily against *P. carotovorum* involved the SA-signaling pathway in the short term, but the jasmonate-/ethylene-signaling pathway was required for durable protection (Luzzatto et al. 2007).

3.4.2 Acibenzolar-S-Methyl (ASM) and Salicylic Acid (SA)

ASM and SA have been applied preharvest on flowering crops to control postharvest decay caused by different pathogens. Preharvest SA sprays at 2000 μg/mL to Geraldton waxflower cv. CWA Pink plants reduced flower disease incidence caused by *Alternaria* sp. and *Epicoccum* sp. (Beasley 2001) (Table 6). However, SA treatment did not suppress postharvest *B. cinerea* infection on flowers of this cultivar. While SA may induce resistance, it can also be directly toxic to pathogens. Direct SA toxicity has been shown against *Cladosporium cladosporioides* and *B. cinerea* (Beasley 2001; Zainuri et al. 2001). Moreover, phytotoxicity was observed to treated Geraldton waxflower leaves (Beasley 2001). ASM has been

used both pre- and postharvest on freesia vars. Cote d'Azur and Dukaat to control postharvest *B. cinerea* infection (Darras et al. 2006b, 2007). Postharvest disease reductions on freesias treated with ASM ranged from 30 to 45% compared to the untreated controls (Darras et al. 2006b). Similar reductions (i.e., 26–44%) were recorded the second year of the glasshouse trials. However, no significant reductions were recorded when ASM was applied to var. Cote d'Azur freesia inflorescences postharvest, although significant reductions in fungal colony diameters and germ tube elongation were recorded in vitro against *B. cinerea* (Darras et al. 2007). It was suggested that a considerable time lag was needed for induction of defense responses by the host.

3.5 Antimicrobial Compounds Used in Vase Solutions

The use of antimicrobial and other compounds in the holding water at storage has been found to extend the vase life of various cut flowers (Damunupola and Joyce 2008; Hoogerwerf and van Doorn 1992; Knee 2000). A broad range of biocides has been used to prevent the proliferation of microorganisms in the vase solutions; however, their antimicrobial activity may be confounded by their other physico-chemical effects (Damunupola and Joyce 2008; Knee 2000). In this respect, the response of cut stems to biocides may vary between cut flower or foliage type, the specific microorganism involved, and other vase solution ingredients.

3.5.1 Hydroxyquinoline (HQ)

The effectiveness of HQ as a biocide in cut flower handling solutions has been known for decades (Damunupola and Joyce 2008; van Doorn 1997). Hydroxyquinoline sulfate (HQS) and citrate (HQC) are the most commonly used HQ compounds in flower handling (Halevy and Mayak 1981; Loubaud and van Doorn 2004; van Doorn 1997). Additionally, 8-HQS promotes stomatal closure (Burge et al. 1996), while both HQS and HQC promote flower longevity by acidifying the holding solution (Halevy and Mayak 1981). Generally, the quinoline esters are acidic (Weinberg 1957) and therefore may inhibit stem plugging by reducing solution pH. Since physiological plugging is mediated enzymatically (van Doorn 1997), the presence of 8-HQC may influence the activity of enzymes by altering the pH (Marousky 1969, 1971). Marousky (1972) considered that while 8-HQ compounds could help prevent microbial occlusion, their ability to reduce vascular blockage may be due to their ability to inactivate enzyme systems through pH adjustment.

3.5.2 Silver Nitrate ($AgNO_3$), Silver Nanoparticles (SNP), and Silver Thiosulfate (STS)

Silver is typically applied as the nitrate salt (i.e., $AgNO_3$) (Ketsa et al. 1995; van Doorn et al. 1991). It can act as antimicrobial agent (van Doorn et al. 1990), as an inhibitor of aquaporins in plants (Niemietz and Tyerman 2002), and/or as an ethylene-binding inhibitor during ethylene synthesis and action (Beyer 1976;

Serek et al. 2006; Veen 1979). Pretreatment with $AgNO_3$ as a pulse at 170 mg/L for 30 min was highly effective against bacterial growth in the basal stem segments of cv. Sonia roses (van Doorn et al. 1990). However, the effectiveness of $AgNO_3$ as a biocidal agent was highly variable as numbers of bacteria in the 5 cm basal were found to be $<10^2$ cfu/g FW in one experiment, but 1.9×10^6 cfu/g FW when repeated (van Doorn et al. 1990). $AgNO_3$ significantly increased vase life of *Viburnum tinus* inflorescences and reduced flower abscission, although it negatively affected flower opening and inflorescence fresh weight (Darras et al. 2010b). The effect of $AgNO_3$ in combination with other vase solution ingredients on the vase life and bud opening of cut *Dendrobium* flowers of cv. Pompadour was investigated by Ketsa et al. (1995). $AgNO_3$ plus glucose was not as effective in increasing bud opening and vase life as HQS plus glucose. However, HQS plus glucose was less effective in controlling microbial growth (i.e., 10^6–10^8 cfu/L and 10^4–10^5 cfu/L, respectively) (Ketsa et al. 1995). Research into the mechanism of the inhibitory action of Ag + on microorganisms revealed that the expression of cellular proteins and enzymes essential for ATP production was inactivated upon treatment, and DNA lost its replicative ability (Yamanaka et al. 2005).

Silver thiosulfate (STS) is used widely in the cut flower industry as dip, spray, or pulse treatment (Altman and Solomos 1995; Joyce 1993; Joyce and Beal 1999; Slater et al. 2001; van Doorn and Cruz 2000). The ability of STS to inhibit ethylene action was utilized to prolong vase life of various cut flowers (Cameron and Reid 1983; Joyce 1992; Mor et al. 1984; Premawardena et al. 2000; Yapa et al. 2000). Application of Ag + as STS to the flowers substantially reduced binding activity by substitution of Cu_2^+ (Beyer 1976). Cu_2^+ is involved in enzymatic reactions related to the biosynthesis and action of ethylene (Himelblau and Amasino 2000). In flowering stems that are sensitive to ethylene such as the *Heuchera sanguinea*, STS reduced flower abscission and prolonged vase life (Han 1998). However, STS may not be as effective in reducing the population of bacteria in vase solutions. For example, van Doorn et al. (1991) found that STS used at 656, 1312, or 2624 mg/L for 4 h was not effective in reducing the numbers of bacteria in petioles of *Adiantum raddianum* fronds, while $AgNO_3$ at 12.5 and 25.0 mg/L minimized them to zero. Furthermore, Hoogerwerf and van Doorn (1992) reported that STS did not control bacterial populations in solutions holding various cut flowers during transportation from growers to the retailers.

Pure colloidal silver nanoparticles (SNP) are potent and broad-spectrum antimicrobial agents. In the cut rose cv. Movie Star, pulsing 10 mg/L SNP plus 5% sucrose for 24 h and subsequently holding samples in 0.5 mg/L SNP plus 2% sucrose significantly delayed vascular blockage caused by bacterial contamination, and stomatal conductance was inhibited. This resulted in improvement of water balance in cut roses and significant increases in vase life by up to 11.8 days compared to the controls (Lü et al. 2010). Effects of SNP as a dip or spray pretreatment on the subsequent vase life and physiology of cut Lilium Oriental hybrid cv. Siberia and Lilium Asiatic hybrid cv. Dream Land were investigated by Kim et al. (2004). The vase life of Lilium "Siberia" florets was extended with a treatment comprised of 0.1% SNP plus natural chitosan. While ethylene production continuously decreased

and the climacteric-type respiration increased on the fifth day, these processes were not differentially affected by the various treatments. Furthermore, the antibacterial action of SNP and the mechanisms involved have not yet been fully elucidated, and interactions between SNP particles and bacterial species need to be studied (Pal et al. 2007). When cut gerbera flowers of cvs. Double Dutch and Red Explosion were held in a solution of 6 mg/L SNP, their vase life increased by up to 7.8 days compared to the controls (Oraee et al. 2011). At the same time, bacterial populations in the holding solutions decreased significantly.

3.5.3 Chlorine Compounds (NaOCl, DICA, ClO_2)

Many active chlorine (Cl) compounds with antimicrobial properties are commercially available. For example, the sodium salt of dichloroisocyanuric acid (Na-DICA) is a common slow-release (stabilized) chlorine compound used for sterilization of water in swimming pools (Jones et al. 1993; Joyce et al. 2000; Xie et al. 2007). Both DICA and household bleach or sodium hypochlorite (NaOCl) are extensively used in experimentation and flower handling (Akoumianaki–Ioannidou et al. 2010; Darras et al. 2010b; Faragher et al. 2002; He et al. 2006; Knee 2000; van Doorn et al. 1990). Chlorine activity involves the oxidation of cellular components in microorganisms, including essential enzymes in cell membranes and protoplasm (Bloomfield and Arthur 1989; Dychdala 1983). Five to 10 mg/L free available chlorine (FAC) units helped control bacteria in fresh, clean postharvest solutions (Xie et al. 2007). When chlorine compounds were added to vase water, a portion of the chlorine was consumed by water impurities (chlorine demand), which included inorganic reducing agents, like Fe_2^+, Mn_2^+, NO_2^-, and H_2S, as well as organic compounds, like amino acids. The chlorine atom ceases to maintain oxidizing capacity upon reduction to chloride (Cl−), and so its disinfectant property is lost or reduced (Dychdala 1983). Consequently, chlorine levels in postharvest vase solutions may decrease rapidly with time. High initial concentrations can be used to meet chlorine demand but may be phytotoxic (van Doorn et al. 1990). Joyce and Beal (1999) suggested that if symptoms of phytotoxicity are evident, then 0.5–1.0% (w/v) sucrose plus 25 mg Cl/L may be appropriate to reduce phytotoxic damage as well as to extend vase life. Chlorine dioxide (ClO_2) is another compound recently used in experimentation. The addition of 2 or 10 μL/L ClO_2 to clean deionized water extended the vase life of *Alstroemeria peruviana* cv. Senna, *Antirrhinum majus* cv. Potomac Pink, *D. caryophyllus* cv. Pasha, *G. jamesonii* cv. Monarch, *Gypsophila paniculata* cvs. Crystal and Perfecta, *L. asiaticum* cv. Vermeer, *Matthiola incana* cv. Ruby Red, and *R. hybrida* cv. Charlotte flowers by 0.9–13.4 days (7–77%) compared to the controls (i.e., 0 μL/L ClO_2) (Macnish et al. 2008). The beneficial effects of ClO_2 treatment were associated with the reduction of aerobic bacteria in vase water and on cut surfaces of flower stems.

DICA has been recommended as a vase solution biocide along with other ingredients, such as citric acid, sucrose, etc. (Akoumianaki–Ioannidou et al. 2010; Darras et al. 2010b; Jones 1991; Knee 2000), although concerns have been expressed over the use of combinations of chlorine compounds with citric acid (Faragher et al. 2002). There may be a decrease in the disinfecting efficiency of

chlorine with the decrease in pH (Dychdala 1983); moreover, chlorine compounds can corrode metal containers. The low chlorine concentrations used by researchers in experiments (e.g., 10 mg/L) (Ligawa et al. 1997) can be inappropriate for use by industry in handling solutions and consumers in vase solutions, where higher concentrations are required (e.g., 50 mg/L) (Jones 1991). In contrast, using DICA at 200 mg/L plus 2% sucrose was found to be effective in prolonging vase life of *Nerium oleander* inflorescences (Akoumianaki–Ioannidou et al. 2010). DICA alone at 100 mg/L did not increase the vase life of cut *Viburnum tinus* inflorescences (Darras et al. 2010b) but significantly increased vase life, flower opening rate, and flower fresh weight of cut *N. oleander* inflorescences when used at 200 mg/L plus 2% sucrose (Akoumianaki–Ioannidou et al. 2010). Knee (2000) found that DICA used alone at 200 mg/L was effective in extending vase life of cut alstroemeria, dianthus, and rose flowers by 2.6, 2.9, and 1.5 days, respectively, as compared to the controls.

3.5.4 Acidifiers

Acidic solutions provide an unfavorable environment for bacteria to develop and also help in rehydration of cut flowers. Water flow increased through rose stem segments with decreasing pH from 6 to 3 (Durkin 1979). In cut cv. Baccara roses, decreasing solution pH from 8 to 6 enhanced flower water relations, fresh weight maintenance, and vase life (Pompodakis et al. 2004). The positive effect of low pH was attributed to the reduction of microbial populations (Halevy and Mayak 1979; Pompodakis et al. 2004). Furthermore, low pH retarded stem blockage of roses in bacteria-free water (Marousky 1971). Bacterial growth was suppressed at low pH, although a population of yeasts and filamentous fungi developed over time (van Doorn 1997). Citric acid at 50 μg/L plus 2% sucrose had significant positive effects on vase life and flower opening scores of *N. oleander* inflorescences independent of storage temperatures tested (i.e., 2, 5 or 20 °C) (Akoumianaki–Ioannidou et al. 2010). Additionally, 50 μg/L citric acid plus 2% sucrose protected the flowers from low-temperature injuries (LTI) when observed at temperatures of 2 °C. It was suggested that the exogenous supply of sucrose might have prevented LTI in oleander-flowering stems, while compounds such as citric acid, DICA, or methanol may have facilitated the absorption and delivery of sucrose into cells and enhanced the ability of inflorescences to be stored at low temperatures (Akoumianaki–Ioannidou et al. 2010).

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Part II

Florists' Crops Diseases: Cut Flowers

Diseases of Anthurium

12

Anne M. Alvarez

Abstract

Anthuriums, characterized by a colorful spathe and spadix, are neotropical ornamentals belonging to the family Araceae. Nineteen *Anthurium* species with horticultural significance and value in breeding programs have been described, and among these, hybrids of two *Anthurium* species, *Anthurium andraeanum* Linden ex André and *A. antioquiense* Engler, are predominantly used in cut flower and/or potted plant production, respectively. Numerous hybrids of *A. andraeanum* Hort. and other species are grown throughout the tropics and are characterized by unique shapes and colors and a long shelf life. Bacterial pathogens have caused the most devastating diseases of anthurium and have limited the production of susceptible hybrids in Florida, the Caribbean, and the Pacific islands. Fungal and fungal-like diseases include anthracnose (black nose), foliage, and stem and root rots caused by *Phytophthora nicotianae*, *P. tropicalis*, and *Rhizoctonia solani*. Viral diseases have been described but do not cause appreciable economic damage. Anthurium decline caused by the burrowing nematode, *Radopholus similis*, currently poses a major challenge to anthurium production due to prohibition of available nematicides. Anthuriums can be successfully produced by constant vigilance and implementation of fully integrated disease management practices.

Keywords

Xanthomonas axonopodis pv. *dieffenbachiae* • *Ralstonia solanacearum* • *Acidovorax anthurii* • *Colletotrichum gloeosporioides* • *Pythium splendens* • *Phytophthora nicotianae* var. *parasitica* • *Rhizoctonia solani* • *Radopholus similis* • Integrated disease management

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© Springer International Publishing AG 2018

R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_13

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1 Introduction

Anthurium belongs to the family Araceae, which includes *Aglaonema*, *Dieffenbachia*, *Epipremnum*, *Philodendron*, *Syngonium*, *Spathiphyllum*, *Xanthosoma*, and other aroids. More than 1500 species have been described from Southern Brazil through Central America, Mexico, and the Caribbean islands (Kamemoto and Kuehnle 2006). Two *Anthurium* species, *A. andraeanum* [sect. Calomystrium] (Fig. 1) and *A. antioquiense* [sect. Porphyrochitonium] (Fig. 2), are most commonly used in the cut flower and/or potted plant industry, respectively (Kamemoto and Kuehnle 1996). The latter species is characterized by an upright spathe and spadix (tulip type), and its hybrids are usually smaller and used commercially as potted plants. In contrast, *A. andraeanum* cultivars are characterized by a wider spathe and are used as cut flowers desired for their long shelf life. These two species show differential susceptibility to the most devastating disease of aroids, bacterial blight caused by *Xanthomonas axonopodis* pv. *dieffenbachiae* (Vauterin et al. 1995) [= *Xanthomonas campestris* pv. *dieffenbachiae* (McCulloch & Pirone) Dye]. Other important bacterial diseases are bacterial wilt caused by *Ralstonia solanacearum* and leaf spot caused by *Acidovorax anthurii*. Fungal diseases, including anthracnose, foliar spots, and root rots are those common to other crops. Viral diseases have been described but do not pose major production problems. In contrast, root damage caused by the burrowing nematode, *Radopholus similis*, is a serious hindrance to production and export due to the lack of effective registered nematicides. Cultural guidelines to optimize nutrition, production, and shipping have been described (Chen et al. 2003). Specific management practices for diseases of anthurium are provided in the text, and additional information may be found in the introductory chapters on integrated disease management.



Fig. 1 Characteristic spathe and spadix of *Anthurium andraeanum* Linden ex. Andre, progenitor of modern anthurium hybrids (H. Kamemoto © 2017. All Rights Reserved.)



Fig. 2 Characteristic spathe and spadix of *Anthurium antioquiense* Engler. This species is currently used as a source of resistance to bacterial blight. Primary hybrids between *A. antioquiense* and *A. andraeanum* are backcrossed to *A. andraeanum* to achieve desirable cultural characteristics with improved blight resistance (H. Kamemoto © 2017. All Rights Reserved.)

2 Fungal and Fungal-Like Diseases

2.1 Anthracnose (Also Called Black Nose or Spadix Rot) [*Colletotrichum gloeosporioides* (Penz.) Sacc.]

Geographic occurrence and impact. Anthracnose occurs worldwide on a number of tropical and subtropical crops. When first reported, anthracnose was widespread on the Island of Hawaii but rare on Oahu. Plant damage was not severe, but black spots on the spadix limited marketability and caused more than 50% loss of flowers (Aragaki and Ishii 1960; Aragaki et al. 1968). When disease incidence is high, the spadix deteriorates rapidly resulting in the typical "black nose" disease.

Symptoms/signs. Small dark angular spots first appear on individual flowers on the spadix infected with *Colletotrichum gloeosporioides* (Fig. 3). Necrosis of tepal tissue forms a diamond pattern as the disease progresses. [Tepals are perianth segments or fused sepals surrounding the base of the anthurium flower.] In most cases, there is little indication that the spots will fuse. However, at a high level of infection, a spadix may be covered with more than 50 spots. Under warm, wet conditions, the entire spadix may be covered with mycelium (Fig. 4).

Biology and epidemiology. *Colletotrichum gloeosporioides* produces large numbers of asexual spores in open fruiting structures (acervuli) on anthurium flowers. Spores require moisture for germination, penetration, and infection, and during the rainy season, the disease may become severe. The fungus also can infect petioles and pedicles directly through the cuticle and/or through wounds (Aragaki 1968). Acervuli are produced on dead leaves and stems, and spores are carried by air currents, irrigation water, and splashing rain to leaves and spathes of other plants.

Management

- ***Cultural practices*** – Anthracnose incidence increases during warm moist months and should be monitored closely when conditions of high humidity occur. Culturing anthuriums in a covered greenhouse to keep rain off the flowers reduces infection. Fungicidal sprays should be applied when disease reaches the economic threshold.
- ***Sanitation*** – Infected flowers and plant debris should be removed and burned.
- ***Fungicides*** – Weekly or biweekly sprays of mancozeb or thiophanate methyl are commonly used at the recommended dosages. Visible chemical residues are unsightly for ornamentals, requiring the added step of washing flowers to restore their appearance. As a result, use of chemicals is generally discouraged (Norman and Ali 2012). Soluble, non-residue fungicides are more effective when anthuriums are cultured under a roof (Aragaki 1968). Other fungicides that can be used include chemicals such as propiconazole, bitertanol, and prochloraz (UWI 2004–2016).



Fig. 3 Anthracnose symptoms on spadix of *Anthurium andraeanum* var. "Kauai" caused by *Colletotrichum gloeosporioides*. Note small eruptions, which are the true flowers, and the tepals or perianth segments, which are fused sepals surrounding the base of the flower. Dark angular spots are infected tepal tissue (T. Amore © 2017. All Rights Reserved.)



Fig. 4 Anthracnose or "black nose" symptoms on an anthurium spadix caused by *Colletotrichum gloeosporioides* (J. Uchida © 2017. All Rights Reserved.)



Fig. 5 Anthracnose or "black nose" on spadix of *Anthurium andraeanum* var. "Kaumana" used as a susceptible check for screening for "black nose" resistance in breeding trials (A. Martinez © 2017. All Rights Reserved.)

- ***Resistance*** – In the breeding program in Hawaii, hybrid selections were screened for anthracnose resistance prior to cultivar release. The varieties Kaumana, Kansako No. 1, and Kozuhara are highly susceptible, and Kaumana is used as a standard to rate resistance in other cultivars (Fig. 5). Nitta Orange and Uniwai have intermediate resistance, whereas Abe Pink, Kansako No. 2, and Marian Seefurth are highly resistant (Aragaki 1968). Most of the varieties produced in Hawaii have now been indexed for anthracnose, and a reasonably large selection of resistant cultivars is available. The University of Florida also has a wide range of resistant cultivars (Norman and Ali 2012).
- ***Integrative strategies*** – Anthracnose can be effectively managed through the combination of cultural practices, fungicidal sprays, and deployment of resistant cultivars.

2.2 Stem and Root Rots (*Pythium splendens* Braun, *Phytophthora tropicalis*, and *Phytophthora nicotianae* var. *parasitica*)

Geographic occurrence and impact. *Phytophthora* and *Pythium* are fungus-like oomycetes which form motile spores (zoospores) and require free water for

multiplication and infection. These pathogens occur worldwide and cause considerable damage to susceptible crops.

Symptoms/signs. Early symptoms of root damage are yellowing and necrosis of leaves and spathes and necrosis at the base of the plant (Fig. 6). Leaves show characteristic water-soaked lesions (Fig. 7). Stems and leaves lose turgor, wilt, and deteriorate. The spadix also may be infected (Fig. 8), and the spathe will eventually decay (Fig. 9). Roots of affected plants have dark patches of decay. Sloughing off of root tissue is characteristic of several types of root diseases, and culture of the fungus is required for an accurate diagnosis.

Biology and epidemiology. *Phytophthora* and *Pythium* are oomycetes which form motile spores (zoospores) and require free water for multiplication and infection. Thus, as with most soilborne diseases, water-saturated soils are conducive to disease development.

Management

- ***Cultural practices*** – Use disease-free stock plants and light, well-drained, synthetic soil mixes.
- ***Sanitation*** – Plants with brown roots or other disease symptoms should be discarded. Benches and propagation equipment should be disinfested. Cutting shears, knives, and tools should be dipped in an appropriate disinfectant between plants (refer to ▶ Chap. 9, "Sanitation for Management of Florists' Crops Diseases").
- ***Fungicides*** – Fungicide drenches are commonly used to control *Pythium* and *Phytophthora*. Fungicides such as mefenoxam, aluminum tris/fosetyl-al, dimethomorph, fluopicolide, and phosphorous acid have been effective.



Fig. 6 Necrosis and decay of anthurium leaves caused by *Phytophthora nicotianae* (J. Uchida © 2017. All Rights Reserved.)



Fig. 7 Leaves of Pigtail anthurium (*A. scherzerianum*) showing decay due to *Phytophthora nicotianae* (J. Uchida © 2017. All Rights Reserved.)

- ***Soil disinfestation*** – Steam-sterilization of potting media is a common management method for soilborne diseases, and potting media should be treated with a fungicide drench. If containers are reused, they should be scrubbed and sterilized.
- ***Integrative strategy*** – A combination of all above control measures is needed for successful production.

2.3 Rhizoctonia Root Rot and Vascular Wilt (*Rhizoctonia solani*)

Geographic occurrence and impact. *Rhizoctonia* is a soilborne fungus that attacks the roots of numerous crops worldwide. The fungus often is associated with damping-off diseases that kill plantlets pre- or postemergence. Rhizoctonia root rots and damping-off diseases are a long-standing and well-known problem for nursery growers and can cause up to 100% loss of seedlings.

Symptoms/signs. *Rhizoctonia solani* attacks roots and lower portions of anthurium stems causing discoloration and rot. Leaves also are affected under particularly wet conditions when severe infections cause blight and decay. The presence of



Fig. 8 Dark angular flecks on spadix tissue caused by *Phytophthora tropicalis* (J. Uchida © 2017. All Rights Reserved.)

characteristic mycelial strands in decayed roots is a diagnostic feature that distinguishes *Rhizoctonia* infections from those caused by *Pythium* and *Phytophthora*.

Biology and epidemiology. *Rhizoctonia* produces mycelial mats containing resting structures (sclerotia) that are resistant to desiccation and low temperatures, permitting the fungus to survive for many years in soil in the absence of a susceptible host. These small, irregular brownish structures resemble soil particles and stick to surfaces of pots and other greenhouse materials without detection (Norman and Ali 2012). Plants are more susceptible to infection when soils are wet and roots are not well aerated.

Management

- ***Cultural practices*** – Provide good drainage and avoid waterlogged soils. Cultivate plants on raised benches.
- ***Sanitation*** – Disinfect pots, benches, and tools with appropriate materials, and use pathogen-free soils or pasteurize soils before use. Assure that propagation materials are pathogen-free and have no brown spots or areas of discoloration on



Fig. 9 Distorted spathe of Pigtail anthurium (*A. scherzerianum*) infected by *Phytophthora nicotianae* (J. Uchida © 2017. All Rights Reserved.)

roots. Potting media and additives such as wood chips and sphagnum moss should not be stored in contact with native soil.

- ***Fungicides*** – Several fungicides are active against *Rhizoctonia*, including thiophanate methyl, fludioxonil, and flutolanil (Norman and Ali 2012).
- ***Soil disinfestation*** – Soils and media mixes should be steam-sterilized prior to use. Chemical drenches (above) may be applied after plants have been established.

3 Bacterial Diseases

3.1 Anthurium Blight (*Xanthomonas axonopodis* pv. *dieffenbachiae* Vauterin et al. 1995) [= *Xanthomonas campestris* pv. *dieffenbachiae* (McCulloch and Pirone) Dye]

Geographic occurrence and impact. The disease was first reported in Brazil (Robbs 1960). In 1972 Hayward reported foliar symptoms on *A. andraeanum* at a few sites on the island of Kauai caused by a xanthomonad he then identified as *Xanthomonas*

dieffenbachiae (McCulloch & Pirone) Dowson (Hayward 1972). Anthurium blight, which includes both foliar and systemic phases of the disease, reached epidemic proportions and killed thousands of plants on the island of Hawaii between 1981 and 1985 (Nishijima and Fujiyama 1985; Nishijima et al. 1988). The epidemic reduced the number of anthurium growers from over 200 to fewer than 10 within 4 years (Shehata et al. 1989; Figs. 10, 11, and 12). Anthurium blight also was reported in California (Cooksey 1985), Venezuela (Guevara and Debrot 1985), Guadeloupe and Martinique (Hostachy et al. 1985), French West Indies (Prior et al. 1985), French Antilles (Rott and Prior 1987), Florida (Chase 1988), Jamaica (Young 1990a b), Tahiti (Mu 1990), the Philippines (Natural 1990), India and the Netherlands (Sathyanarayana et al. 1998), and Reunion Island (Soustrade et al. 2000). Bacterial blight continues to be a destructive disease in most anthurium production areas worldwide.

Symptoms/signs. The first symptoms of a foliar infection are small water-soaked spots at leaf margins which later become chlorotic (Nishijima 1985). Initial infection may occur through stomates and/or hydathodes (openings at leaf margins which lead directly to vascular tissues) (Figs. 13 and 14). Bacteria which enter stomates are generally restricted to mesophyll cells in the leaf, whereas infections occurring through hydathodes lead to rapid colonization of vascular tissue and progression through veins to petioles and the base of the plant, where bacteria can enter uninfected petioles and move upward into newly developing leaves (Alvarez et al. 2006). Systemically infected plants initially show yellowing and wilt followed by



Fig. 10 Anthurium culture under saran with 70% shade in Hawaii (A. Alvarez © 2017. All Rights Reserved.)



Fig. 11 Yellowing and necrosis on leaves of systemically infected plants, *Anthurium andraeanum* var. "Marian Seefurth" (R. Lipp © 2017. All Rights Reserved.)



Fig. 12 Field of *Anthurium andraeanum* var. "Paradise Pink" showing ~30% loss of plants due to bacterial blight (A. Alvarez © 2017. All Rights Reserved.)



Fig. 13 Water-soaked lesions surrounded by a yellow halo on the upper side of *A. andraeanum* leaf. At the top left, marginal water-soaking and chlorosis are evidence of invasion through hydathodes. The distinctly circular lesions distributed irregularly over the leaf surface are evidence of stomatal invasion (A. Alvarez © 2017. All Rights Reserved.)

rapid decline and death of the entire plant (Figs. 15, 16, and 17). Symptoms usually do not appear immediately after infection, giving rise to latent infections which are very difficult for the grower to assess. Colonization of leaf tissue by a bioluminescent strain of *X. axonopodis* pv. *dieffenbachiae* is detected in the dark by covering leaves with sensitive X-ray film, which turns black on exposure revealing the presence of the light-emitting pathogen (Fukui et al. 1996). Stages of disease progress 2–5 weeks after inoculation on plants maintained in a greenhouse at 30–35° C/86–95° F as shown in (Fig. 18). Symptom expression at different stages of disease development varies depending on temperature and other stress factors (Alvarez et al. 2006; Norman and Ali 2012).

Biology and epidemiology. The host range of *Xanthomonas axonopodis* pv. *dieffenbachiae* is generally restricted to aroids (members of the family Araceae). Bacterial strains associated with anthurium blight showed considerable diversity when characterized by bacteriological, serological, and pathogenicity tests (Alvarez et al. 1988; Lipp et al. 1992). Strains initially isolated from anthurium had a broader host range and were generally more virulent than strains isolated from other aroids, such as *Aglaonema*, *Colocasia*, *Epipremnum*, *Syngonium*, and *Xanthosoma* (Lipp et al. 1992). The pathogen also forms various subgroups which are genetically



Fig. 14 Circular water-soaked lesions on the underside of the leaf rarely show chlorosis. Elongated water-soaked lesions at the leaf margins are evidence of invasion through hydathodes. Chlorotic areas border the water-soaked tissues, which later become necrotic (A. Alvarez © 2017. All Rights Reserved.)

distinct (Louws and Alvarez 2000; Khoodoo et al. 2004). The bacteria are spread through contaminated cutting tools, hands, and clothing and through aerosols when sprinkler or rainwater impacts infected leaves. The most common means of spread is through latently infected propagative materials in which the presence of the pathogen is detected by specific immunodiagnostic assays (Alvarez et al. 1991). The impact is difficult to assess because plants appear healthy but nevertheless can result in widespread contamination of healthy stocks on planting benches and later kill the crop (Norman and Alvarez 1996). *Xanthomonas axonopodis* pv. *dieffenbachiae* can survive as latent infections in tissue-cultured microplants, and triple indexing is needed to ensure that pathogens are eliminated (Norman and Alvarez 1994). Nevertheless, pathogen-free microplants which are subsequently infected on the greenhouse bench may become systemically colonized without manifesting foliar symptoms (Ayin et al. 2016).

Management

- ***Cultural practices*** – Use of pathogen-free propagative materials is an important step for blight control. Tissue-cultured pathogen-free microplants are available from various commercial and private sources. Nevertheless, to reduce production



Fig. 15 Leaf margin of *A. andraeanum* showing water-soaking, necrosis, and chlorosis which progresses inward toward the midrib (A. Alvarez © 2017. All Rights Reserved.)

costs, growers continue to make cuttings from symptomless planting stocks, which may harbor latent infections that may not appear up for to 10 months after planting, so the latter practice, though less expensive, is risky (Norman and Alvarez 1996). Higher temperatures reduce the incubation period and increase leaf colonization by the pathogen (Fukui 1999a). Lowering shade-house temperatures through aeration or locating anthurium production at high elevations with corresponding cooler lower temperatures (18–24° C/64–75° F) reduces disease outbreaks.

- ***Sanitation*** – Destruction of infected plants and disinfestation of pruning and harvesting tools are essential to cut flower production. Tools are soaked in disinfectants for 30–60 s before touching the next plant, so fieldworkers may carry two to three tools or commercially available dispensers which permit sufficient bactericidal action between cuts. Commercially available disinfectants are generally broad-spectrum biocides.
- ***Fungicides/bactericides*** – Antibiotic sprays (streptomycin sulfate at 200 ppm combined with 200 ppm tetracycline) were initially used to retard the spread of bacterial blight, but the cost and potential development of antibiotic resistance in the bacterial population discouraged the use of antibiotics (Nishijima 1988). By increasing the dose and frequency of sprays, treated plants were protected and



Fig. 16 Anthurium leaf showing advanced necrosis and desiccation of tissues at leaf margins (A. Alvarez © 2017. All Rights Reserved.)

produced flowers in a field where no other plants survived (Fig. 19). In a field study research conducted in Hawaii, plants were sprayed six times over 3 weeks with high doses of streptomycin sulfate and tetracycline; then treatments were discontinued. Leaves became white and bleached following the applications, but foliage later recovered, the natural green color was restored, and plants survived and produced flowers. No resistant bacterial strains were detected 2 months following the last application (Alvarez et al. 1989). Nevertheless, field applications of antibiotics were abandoned and replaced by integrated pest management strategies that included a long list of improved production strategies including use of pathogen-free tissue-cultured planting stocks and strict surveillance when plants were propagated from cuttings (Alvarez et al. 1994). Judicious use of antibiotics is nevertheless warranted and can be effective for decontaminating planting stocks and protecting young plants on propagation benches (Vannini 2010). Products containing copper, mancozeb, and *Bacillus subtilis* are effective against *Xanthomonas* (Norman and Ali 2012). Anthurium leaves are sensitive to the high concentrations of copper used to reduce spread of other bacterial diseases caused by *Xanthomonas* so copper compounds must be used with attention to potential damage. Treatments with fosetyl aluminum 7 days prior to inoculation



Fig. 17 Late stages of foliar infection resemble symptoms of severe water stress (A. Alvarez © 2017. All Rights Reserved.)

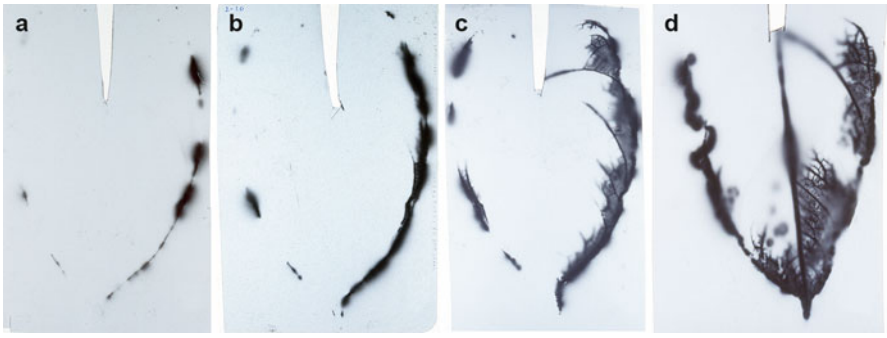


Fig. 18 Autophotographs of the same *Anthurium andraeanum* leaf taken successively 2, 3, 4, and 5 weeks after inoculation with a bioluminescent strain of the pathogen, *Xanthomonas axonopodis* pv. *dieffenbachiae*. (**a**) Two weeks after inoculation, dense areas of bacterial colonization are visible along the leaf margins; (**b**) 3 weeks after inoculation, the right leaf margin has been completely colonized, and bacteria are moving into the veins; (**c**) 4 weeks after inoculation, bacteria have colonized major veins. Bacteria in the topmost vein have reached the petiole, marked by the slit in the X-ray film; (**d**) 5 weeks after inoculation, the midrib has been colonized by bacteria coming from separate veins. The dichotomous pattern of veinlets is revealed when the X-ray film is exposed by bioluminescing bacteria (R. Fukui © 2017. All Rights Reserved.)



Fig. 19 High concentrations of antibiotics were used in early attempts to retard the progress of bacterial blight. Plants in treated rows (marked with signs) were the only surviving plants in a field of infected anthuriums (A. Alvarez © 2017. All Rights Reserved.)

with *X. axonopodis* pv. *dieffenbachiae* significantly reduced infection, possibly due to a concurrent drop in cellular pH from ~ 6.4 to ~5.4 (Alvarez et al. 1991).

- ***Biological control*** – Mixtures of antagonistic leaf-inhabiting bacteria have been successfully used to significantly reduce onset and spread of bacterial blight in the greenhouse and field (Fukui 1999b, c; Toves et al. 2008; Vowell 2009). A combination of four different species of bacterial antagonists sprayed to runoff at weekly intervals reduced field spread of disease to the same degree as application of chemical bactericides (Toves 2008; Vowell 2009). Specific combinations of the beneficial bacteria enhanced microplant growth (Fig. 20) and protected leaves from infection under the moist conditions on propagation benches (Toves 2008; Vowell 2009). Mixtures of beneficial bacteria can be efficiently prepared in the laboratory and applied to microplants when they are deflasked from axenic culture. Microplants are soaked for 30 min in a mixture of four bacterial species and then planted into community pots (Vowell 2009). Although these bacterial strains have yet to be formulated for large-scale commercial use, they have been consistently effective in greenhouse trials (Toves 2008; Vowell 2009).
- ***Soil disinfestation*** – Undecomposed plant materials remaining in infested fields are a major inoculum source for the subsequent crop although xanthomonads generally do not survive for long periods (< 6 weeks) in moist soil. Crop rotation, removal of infected plant debris, and frequent irrigation to accelerate



Fig. 20 Biological control can be achieved by immersing microplants in a mixture of beneficial bacteria immediately after deflasking. Growth enhancement of microplants (20 plants per pot) observed 6 weeks following the initial treatment is an added benefit. The top two community pots contain treated microplants; plants in the two lower pots were nontreated (A. Alvarez © 2017. All Rights Reserved.)

decomposition of anthurium residues are more effective than fumigation, although the latter practice is used for heavily infested fields (Vannini 2010).

- ***Resistance*** – Bacterial blight resistance in commercial anthuriums has been developed by hybridizing highly resistant *A. antioquiense* and its progeny with the more susceptible varieties of *A. andraeanum*. The classical strategy for developing resistance in the highly desired *A. andraeanum* cultivars was hybridization between the two species and subsequent backcrossing to *A. andraeanum* while selecting for desired qualities such as large spathe size, yield, novelty, color, and long shelf life. *Anthurium amnicola*, a new species discovered in 1972 by Robert L. Dressler in Coclé del Norte, Panama, has also been used as a source of blight resistance. The small lavender spathe is upright when young and bends downward as the flowers on the spadix mature (Fig. 21). This species has been used as a parent for variety development for potted plant production. Three hybrids, ARCS, ARCS Hawaii, and Lavender Lady, which are one-eighth *A. andraeanum* Hort. and one-half *A. amnicola* are resistant to the systemic phase of bacterial blight (Fukui et al. 1998; Amore, *personal communication*). Crosses between *A. andraeanum* and the miniature (dwarf) species, *A. antioquiense*, have resulted in relatively resistant compact hybrid *Anthurium* varieties that are widely used for potted anthurium production in Florida. In Hawaii, where the larger spathes of *A. andraeanum* are preferred, *A. andraeanum*-type hybrids are crossed with *A. antioquiense* and then backcrossed to *A. andraeanum* to produce hybrids with



Fig. 21 *Anthurium amnicola*, a new species discovered in 1972 by Robert L. Dressler in Coclé del Norte, Panama (T. Amore © 2017. All Rights Reserved.)

greater blight resistance yet having larger and broader spathes. Bacterial blight remains a constant challenge to production, but with integrated practices, the disease can be controlled. Various methods have been developed for rapid screening of hybrids in order to select cultivars with greater resistance (Fukui et al. 1998; Elibox and Umaharan 2008a, b, Elibox and Umaharan 2010; Ayin et al. 2016). Genetic engineering approaches have also been used to produce transgenic plants with blight resistance (Kuehnle et al. 2004a, b), but to date, no transgenic varieties are commercially available.

- ***Integrated management strategies*** – Anthurium blight has been successfully managed using a combination of approaches that start with pathogen-free planting stocks, disease-free fields, and rigorous sanitation (Alvarez et al. 2015). Tissue-cultured plantlets confirmed to be pathogen-free by triple indexing should be used as planting stocks. Leaf surfaces should be kept as dry as possible to avoid bacterial spread through aerosols and wounds. When overhead irrigation is used on propagation benches, biocontrol agents or biocides can be applied to closely growing microplants to reduce pathogen spread via irrigation water and aerosols. Frequent inspection followed by roguing of symptomatic plants from propagation benches and shade houses is essential. Decontamination of tools and worker's clothing is a standard practice, and highly susceptible plants are often cultivated and harvested separately in order to prevent spread to other sites. Highly susceptible cultivars are grown at higher elevations in the tropics where cooler temperatures (18–24° C/64–75° F) retard disease expression as well as plant growth. In warmer climates, growers often focus on the dwarf tulip-type hybrids that have greater blight resistance. A complete list of these and other disease management strategies for anthurium and other aroids with a clear rationale for each has been provided by Vannini (2010). Integrated disease management practices that have led to a productive industry in Florida have been described by Norman and Ali (2012).

3.2 Bacterial Wilt [*Ralstonia solanacearum* Smith (Yabuguchi 1995)]

Geographic occurrence and impact. *Ralstonia solanacearum* is a generally warm-weather pathogen present worldwide, predominantly in tropical and subtropical environments, but cold-tolerant strains also are prevalent in Europe. Anthuriums in Florida are affected by a group of strains formerly designated as race 1 and later more fully characterized as Race 1 Biovar 1 Phylotype II (Norman et al. 2009). A new strain of *Ralstonia solanacearum* (Phylotype II – sequevar 4NPB) was identified in 2002 during the dry season on a farm in Trinidad where local pink, white, and red cultivars were affected (University of the West Indies 2004–2016). *Ralstonia solanacearum* (race 1) was detected in the Netherlands on imported anthuriums in August 2015. Bacterial wilt of anthurium has not been observed in Hawaii or other islands of the Pacific although *R. solanacearum* is destructive on other crops throughout the Pacific and Asia.

Symptoms/signs. The first signs of bacterial wilt resemble water stress. Lower leaves of plants are yellow and petioles are flaccid. The bacteria colonize the vascular system rapidly causing stems and leaf veins to show a bronze color. A cross section of the stem or petiole will reveal vascular browning accompanied by abundant bacterial ooze, which is cream to brown in color. Plants do not recover after additional irrigation. As the disease progresses, leaves and stems become necrotic and plants die (Norman and Ali 2012; Figs. 22 and 23).

Biology and epidemiology. *Ralstonia solanacearum* spreads throughout anthurium farms via contaminated soil, water, tools, shoes, or worker contact with infected plants (Norman and Ali 2012). The bacteria survive for long periods in contaminated soil and are easily spread via infected cuttings. Detection of low



Fig. 22 Bacterial wilt of anthurium caused by *Ralstonia solanacearum* (D. Norman © 2017. All Rights Reserved.)

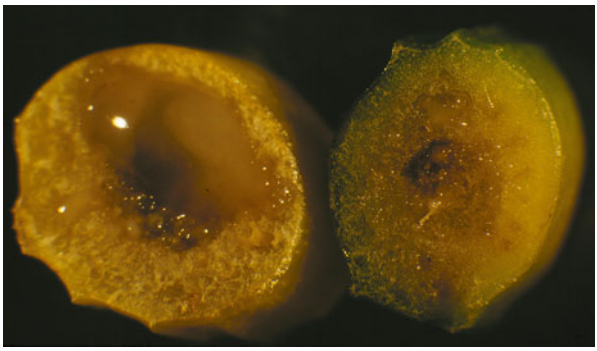


Fig. 23 Vascular discoloration and bacterial ooze of an anthurium infected with *Ralstonia solanacearum* (D. Norman © 2017. All Rights Reserved.)

populations in soil and potting medium has been improved by the development of specific immunodiagnostic and molecular assays (Paret et al. 2010b; Kubota et al. 2011a, b, c). Plants may be symptomless during cool weather, giving time for bacteria to colonize additional plants as latent infections (Norman and Ali 2012). Wilt symptoms generally appear rapidly during warmer weather (above 28° C/82° F).

Management

- ***Cultural practices*** – Use of pathogen-free propagation material followed by a strict sanitation program is needed to stop disease spread and eventually eradicate it from a production facility.
- ***Sanitation and plant quarantine*** – Sterilization of knives and clippers with a disinfectant containing a quaternary ammonium compound or diluted solution of bleach to prohibit spread is recommended (Norman and Ali 2012). Contaminated plant material and the supporting soil should be discarded. Soil should be removed completely from contaminated pots and trays followed by soaking in a bactericidal disinfectant. In the Netherlands, all imported anthurium planting stocks are screened for pathogens. Following the early detection of *Ralstonia solanacearum* on anthuriums in the Netherlands, eradication measures were undertaken immediately and affected plants were destroyed. All plants in two neighboring rows on either side of the infected plant were also destroyed. Specific hygiene measures for staff, equipment, and storage containers were imposed on companies that had infected plants. In addition, a specific surveillance program to confirm the absence of *Ralstonia* in anthurium plants was planned. (NPPO 2015).
- ***Fungicides/bactericides*** – Fungicides that contain phosphorous acid protect geranium plants from new infections, but once plants are systemically colonized by the pathogen, these chemicals are no longer effective (Norman et al. 2006).
- ***Soil disinfestation*** – Bacterial wilt of other crops (tomato, pepper, ginger) has been reduced through application of oil extracts of thyme, lemongrass, or palmarosa, all of which have biocidal activity against the common pathogen, *Ralstonia solanacearum* (Paret et al. 2010a; Pradhanang et al. 2003). As this

pathogen is not specific to anthurium, the same control measures should be effective for potted anthuriums. Botanicals are most effective in potting soils where soil applications can be closely monitored.

3.3 Bacterial Leaf Spot [*Acidovorax anthurii* (Referred to Earlier as Pseudomonas Blight)]

Geographic occurrence and impact. Bacterial leaf spot was first observed during the 1980s in Guadeloupe and Martinique (French West Indies) and Trinidad (Prior et al. 1985; Prior et al. 1986; Prior and Rott 1987) and confirmed in the 1990s in Trinidad and Tobago (Dilbar 1992; Gardan et al. 2000). The disease has not been observed in Hawaii or the Pacific islands. Bacterial leaf spot can cause extensive damage particularly during early stages of crop development, resulting in decreased cut flower production. Similar to bacterial blight, the labor and materials needed to control bacterial leaf spot are expensive, and this disease is responsible for decreased flower production in the Caribbean.

Symptoms/signs. Angular water-soaked leaf spots first appear on the undersides of leaves followed by necrosis of leaf tissue. Leaf lesions are surrounded by a chlorotic halo, typical of many leaf spot diseases caused by bacterial pathogens (Figs. 24 and 25). Lesions may expand to cover broad patches of leaf tissue which turns brown and becomes distorted. When the spathes are affected, the halos are often violet rather than yellow (Fig. 26). During periods of high humidity, bacterial ooze may emerge from margins of affected tissues. The disease also can become systemic when bacteria colonize vascular tissues. Water-soaked tissues occur at the junction between the petiole and veins resulting in leaf decay and abscission of flowers. Water-soaking also occurs at leaf margins, veins, and midribs. Multiple suckers may



Fig. 24 Bacterial leaf spot of anthurium caused by *Acidovorax anthurii* showing leaf spot and chlorotic halo (P. Rott © 2017. All Rights Reserved.)



Fig. 25 Anthurium leaf showing necrotic lesions caused by *Acidovorax anthurii* (P. Rott © 2017. All Rights Reserved.)



Fig. 26 Necrosis of the anthurium spathe caused by *Acidovorax anthurii* (P. Rott © 2017. All Rights Reserved.)

arise from the base of a dying plant, but they do not always show symptoms and may harbor latent infections (UWI 2004–2016).

Biology and epidemiology. *Acidovorax anthurii* is similar to bacterial pathogens in the genus *Pseudomonas* which are generally transmitted by watersplash, aerosols, and irrigation water. The bacteria survive in contaminated soils and are carried to healthy plants on tools and workers' hands, shoes, and clothing if plants are handled when wet.

Management

- ***Cultural practices*** – Propagative materials should be thoroughly inspected to ensure that they are pathogen-free and do not harbor latent infections. Avoid handling plants when fields are wet.
- ***Sanitation*** – Infected leaves and flowers should be removed and burned. When disease has become systemic, entire plants should be removed with the surrounding soil. Tools and clothes can be disinfected with 70% alcohol. Sodium

hypochlorite (10%) can be used for boxes and benches, but because it is corrosive, metal tools and equipment are better disinfested with alcohol or quaternary ammonium compounds or other disinfectants.

- ***Fungicides/bactericides*** – Leaves can be sprayed with copper fungicides to reduce infection, but some anthurium cultivars are sensitive to copper compounds and plants should be inspected for signs of phytotoxicity.
- ***Resistance*** – The search for resistant cultivars is underway at the St. Augustine campus of the University of the West Indies (Holder 2006). A two-phase screening method has been applied in an effort to determine sources of resistance. Several varieties have been identified that will be used in the future breeding program (UWI 2004–2016).

4 Viral Diseases

Viral diseases have been reported on anthurium, but following the advent of tissue culture to produce virus-free plantlets, they rarely have caused major problems for commercial anthurium production. Dasheen mosaic virus (DsMV) is an aphid-borne potyvirus virus that occurs worldwide and has been reported on 13 genera in the family Araceae (Zettler et al. 1970). Anthurium seedlings were susceptible following inoculation, but naturally infected anthuriums were not found in Florida (Hartman and Zettler 1972). DsMV is prevalent on taro, an edible aroid and important tropical food crop in the Pacific (Hu et al. 1995), but this virus is rarely found on anthurium. Although DsMV was reported on *Anthurium andraeanum* in Hawaii (Raabe et al. 1981), the virus did not cause major losses for the anthurium industry. Cucumber mosaic virus (CMV) caused severe mosaic and leaf deformation in commercial anthurium production in Brazil (Miura et al. 2013). Tomato spotted wilt virus (TSWV) vectored by a number of thrips species is occasionally observed on anthurium in Hawaii (Uchida et al. 1999) (Figs. 27 and 28).

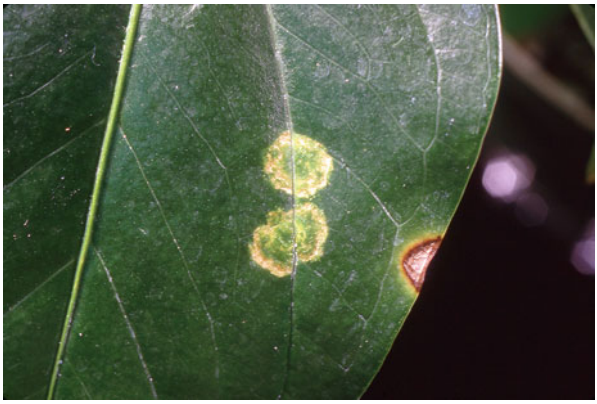


Fig. 27 Concentric rings of chlorosis and necrosis on *Anthurium andraeanum* leaf caused by tomato spotted wilt virus (*TSWV*) (J. Uchida © 2017. All Rights Reserved.)



Fig. 28 Symptoms of TWSV at later stage showing broad patches of necrotic and chlorotic tissue (J. Uchida © 2017. All Rights Reserved.)

5 Nematode Diseases

5.1 Anthurium Decline [Burrowing Nematode, *Radopholus similis* (Cobb) Thorne]

Geographic occurrence and impact. Burrowing nematode is a migratory endoparasite which occurs throughout the tropics and subtropics and feeds on roots of many plants including anthurium. *Radopholus similis* was first reported on anthurium in Hawaii (Sher 1954) where it caused a severe decline and continues to be a major problem during wet seasons. Root feeding by the nematode increases the incidence of secondary root rots and results in poor plant growth, decreased cut flower production, and decreased flower size and quality (Aragaki et al. 1984; Sipes and Lichty 2002). Burrowing nematode also is a problem in the West Indies and in anthurium glasshouse production in Europe, North America, and Japan (UWI 2004–2016).

Symptoms/signs. Small leaves and flowers, premature leaf yellowing, and poor plant vigor are characteristic of decline. Plants are stunted and lower leaves are

yellow and dry, showing signs typical of nutrient deficiency (Fig. 29). Stems, petioles, and peduncles are abnormally short, and the declining plants develop few suckers. Roots will have light orange lesions which later turn dark brown (Fig. 30). Basal stems and roots show severe rot about 6 months after initial infection (UWI



Fig. 29 Plant damage caused by burrowing nematode, *Radopholus similis* (T. Amore © 2017. All Rights Reserved.)



Fig. 30 Anthurium decline, caused by *Radopholus similis*, is characterized by stunting and necrosis of cataphylls (bract-like coverings of the stem). Burrowing nematodes feed on the roots causing elongated lesions (B. Sipes © 2017. All Rights Reserved.)

2004–2016). Dark lesions occurring on the stem just above the soil surface is a clear distinction between burrowing nematode damage and root rot caused by *Phytophthora* or *Rhizoctonia*. The nematodes will be visible with a dissecting scope.

Biology and epidemiology. Burrowing nematodes do not spread more than a few cm a year on their own, but they are moved relatively long distances by irrigation water, rain, or subsurface water movement. Nematodes can also be moved in infected planting material. Once nematodes penetrate the roots, they can spread to stem tissues and leaf petioles. As stem cuttings may be contaminated with *R. similis*, they are not considered reliable nematode-free propagation materials (Wang et al. 1999).

Management

- ***Cultural practices*** – Use of nematode-free planting materials is essential to disease management. Growing media should be steam-sterilized prior to use. The possibility of spread on footwear, tools, and equipment can be reduced through worker-awareness practices and by disinfecting items with appropriate biocides.
- ***Sanitation*** – Infected plant materials should be destroyed. Tissue-cultured anthurium plantlets can be used to ensure that planting materials are nematode-free (Higaki et al. 1995).
- ***Nematicides*** – Use of chemical nematicides is strictly regulated, and few effective nematicides are available for anthurium. In Hawaii, drenches with fenamiphos were effective in reducing root damage and enhancing plant growth (Fig. 31). Declining plants treated with fenamiphos produced 50% more flowers after 1 year, and the commercial value of the flowers from treated plants was approximately 60% greater. Although they are effective, chemical nematicides have restricted use, and availability will vary depending on location. In the Caribbean, growers may apply the nematicide aldicarb with a knapsack sprayer at manufacturer's specifications (5–10 g/m^2) (UWI 2004–2016). Applications are limited because aldicarb is highly toxic, and growers are advised to limit entry into treated fields during 3 months following treatment and then usually just to harvest mature cut flowers. Other chemicals such as oxamyl (3–5 L/ha) may be applied and repeated every 3 weeks.
- ***Control with natural products*** – Restrictions on use of chemical nematicides and subsequent lack of availability have stimulated research into greater exploration of natural products. Plant extracts from neem (*Azadirachta indica*) and leucaena (*Leucaena leucocephala*) are toxic to *R. similis* when applied to soils at 500 g/l and have been used in Trinidad and Tobago. Similarly, natural products (chitin, hydrolyzed protein, fatty acid-based products, and ground sesame plants) have been investigated for efficacy in burrowing nematode management on anthurium in Hawaii (Sipes and Delate 1996). None of the compounds tested reduced burrowing nematode damage as effectively as the synthetic nematicide, fenamiphos. However, ground sesame plant at 258 kg/ha reduced root and stem damage as compared to the untreated control (Fig. 32).
- ***Soil and root disinfestation*** – Nematodes in soil and plant debris were eliminated after a 3–6-month bare-soil, weed-free fallow period to achieve thorough



Fig. 31 Improved root and plant growth of anthurium plants treated with the nematicide fenamiphos (*left*) compared with the nontreated plants (*right*) (M. Aragaki © 2017. All Rights Reserved.)



Fig. 32 Reduced root damage and increased plant size (plant on *right*) following treatment with ground sesame plant used to reduce damage due to burrowing nematode. The nontreated plant is on the *left* (B. Sipes © 2017. All Rights Reserved.)

decomposition of plant parts (Hara et al. 2004). Hot water drenches can be used to disinfest roots of potted anthuriums. Temperature sensitivity of specific anthurium cultivars must be considered before applying hot water treatment to control burrowing nematode. Most cultivars are heat tolerant but "Marian Seefurth" may show some reduction in growth after 3 months. (Tsang et al. 2004). Roots and media should be drenched continuously in hot water (50° C/122° F) for 5–20 min depending on the severity of the infection and the heat tolerance of the anthurium cultivar. After 2 months, a second drench can be applied to further reduce the nematode populations. It is not necessary to treat bare roots by removing the soil (Tsang et al. 2004). Hot air at 50° C/122° F, 60% RH for 15 min may also disinfest stem pieces (Hara et al. 2004).

- ***Soilless media*** – Growing anthurium in soilless media in pots on raised benches offers another level of protection from nematode infection. When plants are already infected with burrowing nematodes, they suffer less damage if planted in soilless media, such as cinder rock, with less compost or peat. This practice is commonly used by anthurium growers in Hawaii (Wang et al. 1997a).
- ***Resistance*** – A screening method has been developed to assist in identification of resistant cultivars (Wang et al. 1997b). Among the most popular commercial *A. andraeanum* cultivars used for the cut flower industry in Hawaii, none was resistant to burrowing nematode. Cultivar "Nitta" was classified as the most susceptible (showing the highest nematode reproduction rate). Cultivars "Blushing Bride" and "Midori" were identified as nematode tolerant (suffering less damage from nematode infection). Cultivar "Ozaki" was highly intolerant (suffering most damage from nematode infection). Two closely related anthurium species, *A. pittieri* and *A. ravenii*, were more tolerant to burrowing nematode than "Midori" (Wang et al. 1998). In future breeding programs aimed to increasing tolerance against burrowing nematode, breeders should consider crossing *A. andraeanum* with other *Anthurium* species.

5.2 Other Plant-Parasitic Nematodes

Meloidogyne spp. (causal agents of root knot) are occasionally found on anthurium roots, but to date, root knot has not caused significant yield losses in anthurium production.

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Diseases of Carnation

13

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Abstract

Carnation (*Dianthus caryophyllus* L.) is one of the most popular and traditional cut flowers worldwide. This species has been used extensively by breeders for centuries, and as a result many cultivated hybrids exist. Several diseases affect quality. Among fungal diseases caused by soilborne pathogens, Fusarium wilt is the most devastating carnation disease worldwide. None of the management practices currently available completely control Fusarium wilt of carnation;

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© Springer International Publishing AG 2018

R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_14

only the integration of different control measures allows management of the disease. The most important bacterial diseases affecting carnation are caused by *Burkholderia* species. This cut flower is affected by eight viruses that reduce the quantity and quality of production. The vegetative propagation of the crop favors the dispersal of these pathogens as well as the fungi and bacteria that colonize xylem tissues. So, meristem-tip culture of healthy mother plants is recommended. Several plant pathogenic nematode species of different trophic habits can infect carnation. Nevertheless, only root-knot and cyst nematodes, of the genera *Meloidogyne* and *Heterodera*, respectively, are of significance because of their economic impact. The integration of different control practices is the best strategy for the management of most diseases.

Keywords

Dianthus caryophyllus • Carnation • *Fusarium oxysporum* f. sp. *dianthi* • *Burkholderia andropogonis* • *Burkholderia caryophylli* • *Carnation etched ring virus* • *Carnation ringspot virus* • *Meloidogyne* • *Heterodera*

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1 Introduction

Carnation (*Dianthus caryophyllus* L.) is one of the most popular and traditional cut flowers worldwide. It is an herbaceous plant belonging to the Caryophyllaceae family, native to the Mediterranean Region. *D. caryophyllus* has been used extensively by breeders for centuries, and as a result, many cultivated hybrids exist, each with a name that usually describes its features. Commercial production includes different cultivars of standard and mini (or spray) types of carnation with a large variety of colors and combinations.

Due to its excellent keeping quality, wide range of forms, ability to resist long distance transportation, and ability to rehydrate after continuous shipping, carnation is preferred by many growers to rose and chrysanthemum in several flower-exporting countries. Modern cut flower varieties of carnation have been selected for flower size, petal number, stem length, and disease resistance. In the nineteenth century, commercial growing was extensive in France and included both field and glasshouse production. After germplasm was transferred to the USA, carnation breeding and growing for the cut flower market became very popular in that country. Increased market demand in Europe and the USA in the early part of the twentieth century provided the impetus to ensure that carnation remained popular in the cut flower industry. Major producing countries are Colombia and Spain.

2 Fungal and Fungus-Like Diseases

2.1 Anther Smut [*Microbotryum dianthorum* (Liro) H. Scholz and I. Scholz (=*Microbotryum violaceum* (Pers.) G. Deml and Oberw.) = *Ustilago violacea* (Pers.) Roussel)]

Geographic occurrence and impact. This disease is distributed in Australia, Canada, England, Germany, Iran, Italy, New Zealand, Spain, Sweden, Switzerland, and the USA (English and Kinthan 1974; French 1989; Fischer 1953; Lindeberg 1959; Scholz and Scholz 1988; Vánky and Abbasi 2013; Vánky and McKenzie 2002; Vánky and Shivas 2008; Zundel 1953). This disease has not been common during recent decades.

Symptoms/signs. According to Horst (2013), the infected plants grow slowly and produce many weak axillary shoots. The stem internodes are shortened. Flower buds are short and squatty, while calyxes tend to split. The smut colonizes and alters the structures of female sex organs (the ovary becomes rudimentary and sterile) so both female and male flowers produce anthers filled with fungal teliospores appearing as a black sooty dust (sign) replacing pollen grains (Baker 1947). Sori also form on carnation petals (Antonovics and Alexander 1992; English and Kinthan 1974; Horst 2013; Smith et al. 2009).

Biology and epidemiology. The fungus enters through flowers (ovaries) or injured surfaces and grows systemically, so cuttings from diseased "mother plants" could be infected. Spores are spread on cuttings. Insect pollinators serve as vectors of the disease, but modern cultivars rarely produce anthers so the possibility of insect transmission is limited. *M. dianthorum* is an obligate pathogen. It is not transmitted by seeds nor persists in the environment, and the disease is restricted to perennial hosts that allow for transmission between living plants (Baker 1947).

Management

- ***Cultural practices*** – Removal of infected flowers or plant tissue should be done carefully to avoid spreading the spores.

2.2 Alternaria Blight (*Alternaria dianthi* Stevens and Hall)

Geographic occurrence and impact. This disease is distributed worldwide. It has been reported in Argentina, Australia, Brazil, Canada, China, Colombia, France, Germany, Greece, Italy, Japan, Korea, Mexico, New Zealand, South Africa, Tanzania, Thailand, Uruguay, the USA, and Zimbabwe (Alvarez 1976; Arbeláez 1988; Cho and Shin 2004; Cook and Dubé 1989; Farr and Rossman 2016; Fujioka 1952; Ginns 1986; Gorter 1977; Holevas et al. 2000; Koch de Brotos and Boasso 1955; Marchionatto 1944; Martínez Fernández 2008; Pennycook 1989; Riley 1960; Scaramuzzi 1953; Viennot-Bourgin 1946; Whiteside 1966; Zhuang 2005a).

Symptoms/signs. The initial symptoms on carnation leaves appear in the form of leaf spots which are circular or slightly oval, running parallel with the longitudinal axis of the leaf. The spots are purple to reddish brown in the center and soon develop a broad yellowish-green border. As the spots expand, the center becomes light brown or gray and adjacent spots tend to coalesce (Fig. 1). Healthy tissues between the spots often turn yellow or yellowish brown. Sometimes the infection takes place at



Fig. 1 Alternaria blight on leaves (S. Nakkeeran et al. Tamil Nadu Agricultural University, Coimbatore, Tamil Nadu, India © 2017. All Rights Reserved.)

the stem joints; in this case the attack usually involves both the leaves and the intermediate stems, resulting in blighting of the more distant parts of the plant and eventually killing the whole plants (Yu et al. 1989). The pathogen can also cause root and collar rot in rooted cuttings and plants that show dark-brown to black coloration (Rodríguez Rodríguez 1980; English and Kinthan 1974; Wolcan et al. 1999).

Biology and epidemiology. The dissemination of spores occurs by wind, water, tools, and animals. The pathogen needs free water to germinate and infect the leaves and stem. The fungus can survive in susceptible weeds or perennial crops (David 1991; Mamgain et al. 2013; Trujillo et al. 1989). Rot of the cuttings can be due to long periods of storage in cool chambers (English and Kinthan 1974).

Management

- ***Cultural practices*** – Avoid dense plantings and excessive irrigation, monitor plantings to detect early infections, and remove plant debris (Qazi et al. 2006). Also provide good air circulation by increasing plant spacing.
- ***Fungicides*** – Vaseem and Ghani (2005) tested fungicides for their efficacy against the spore germination in vivo and against the disease in the field. They found that systemic fungicides, in general, and ergosterol biosynthesis inhibitors (ESBIs) in particular were significantly superior to nonsystemic fungicides both in vitro and in the field. Among the systemic fungicides, penconazole followed by hexaconazole proved superior to the others. Field experimentation on fungicidal efficacy also yielded the superiority of systemic fungicides over nonsystemic ones.
- ***Integrative strategies*** – In integration of fungicide sprays with cultural practices, a combination treatment comprised of mancozeb with removal of diseased leaves and pinching proved to be significantly effective for the management of blight of carnation (Trujillo et al. 1989).
- ***Resistance*** – Disease tolerance of varying degree has been observed under natural condition in the available carnation germplasm. The cv. Tempo showed tolerance to the disease compared to other varieties (Vaseem and Ghani 2005).

2.3 Alternaria Flower Blight; Alternaria Petal Spot; Alternaria Leaf Spot (*Alternaria dianthicola* Neergaard)

Geographic occurrence and impact. The disease has occurred in Argentina, Australia, Austria, Canada, China, Denmark, Korea, Libya, Malawi, Malaysia, New Zealand, South Africa, and the USA (Hawaii) (Alippi et al. 1995; Cunnington 2003; Cho and Shin 2004; Duan et al. 2015; Farr and Rossman 2016; Ginns 1986; Gorter 1977; Johnston 1960; Miller 1993; Neergaard 1945; Pennycook 1989; Schmidt 1952; Trujillo et al. 1989).

Neergaard (1945) noted that the economic impact of *A. dianthicola* is significant and differs from Alternaria leaf spot. The disease weakens plants and attacks the petals before they have unfurled. Sharma (1994) reported that this disease is the major constraint in commercial carnation cultivation in India. This disease was very



Fig. 2 *Alternaria dianthicola* affecting sepals, petals, stem, and leaves of carnation (G.A. Lori © 2017. All Rights Reserved.)

destructive and caused significant economic losses to the flower producers being the most serious blossom disease of carnation in China (Duan et al. 2015).

Symptoms/signs. On immature flower buds and flowers, the initial symptoms are characterized by water-soaked areas. Light- to dark-brown lesions with purple margins occur on sepals and petals (Fig. 2) which then wither in a matter of days and are covered with dark conidiophores and conidia (Duan et al. 2015).

On fully grown plants, oval, pale-yellow, or brown spots with dark margins appear on the edges and tips of leaves and in due course expand to cover the leaf. Symptoms also occur on stems, beginning at the node and girdling the stem upward, producing pale-yellow–brown dark-bordered spots (David 1988). The spots may then become confluent, and under moist conditions, dense olive-black patches appear where the conidia are produced. Also, the fungus can cause the rot of basal tissues of the cuttings in the planting beds.

Biology and epidemiology. The lesions are covered with dark-brown powdery spores that are disseminated by wind and water. Because flower parts must be wet for at least 8 h before infections can occur, extended wet periods with light night rains favor outbreaks of this disease (Trujillo et al. 1989).

Management

- ***Cultural practices*** – Sharma (1994) recommends keeping the humidity low by providing good air circulation. To prevent disease outbreaks, any plant debris should be collected and destoyed.

2.4 Botrytis Blight; Gray Mold; Bud Rot; Blossom Blight (*Botrytis cinerea* Pers.: Fr.)

Geographic occurrence and impact. *Botrytis cinerea* is a ubiquitous worldwide fungal plant pathogen that causes enormous damage during both cropping and



Fig. 3 Botrytis bud blight symptoms in the apex (*left*) and characteristic sign on the dried bud (S. Nakkeeran et al. Tamil Nadu Agricultural University, Coimbatore, Tamil Nadu, India © 2017. All Rights Reserved.)

postharvest cycles of carnation. Even though flowers may be damaged before harvest, the disease is generally most damaging when the attacks take place after cutting and during packaging, shipping, or storing (Garcés De Granada 1992; Gibson et al. 2014).

Symptoms/signs. Carnation plants can be attacked in the field or during transport or storage. Flowers may carry the fungus asymptomatically during cutting and packaging, and symptoms develop later when the conditions are favorable (Garcés De Granada 1992). Early in the infection process, symptoms of Botrytis blight appear as water-soaked spots on the edge of petals and sepals. As the disease progresses, the flower is covered by a fluffy, grayish-brown mass of mycelium, conidiophores, and conidia (signs) (Fig. 3 right), and eventually entire flowers are affected (Garcés De Granada 1992; Whealy 1992). Infected buds fail to open (Fig. 3 left) and may sometimes abort. Under conditions of high relative humidity, the pathogen may produce sporogenous sclerotial bodies (Gibson et al. 2014).

Biology and epidemiology. Mycelium, conidia, and/or sclerotia of *B. cinerea* can survive as a pathogen or saprophytically on a large number of plant species including nursery plants, ornamentals, field crops, and vegetables. Sclerotia are considered the main structure of survival of the pathogen (Elad et al. 2007). Conidia are the main structure of dispersal and can be transported by air currents, insects, or water splash and deposited on the plant surface. High environmental humidity causes moisture to collect in the flowers and provides a favorable environment for the development of *B. cinerea*. Once spores germinate, the fungus enters the host plant through wounds or natural openings and becomes established in the flower petals (Gibson et al. 2014). Disease development is related to an increase in ethylene production from the infected tissues (Elad and Eversen 1995). Ethylene induces susceptibility to *B. cinerea* blight by enhancing the senescence of carnation flowers (Elad 1988).

Management

- ***Cultural practices*** – Environment control in the greenhouse is essential to prevent infections before harvest. Keeping the relative humidity (RH) below 85% provides excellent control of Botrytis blight, while proper plant spacing allows for better air circulation and reduces RH within the canopy (Gibson et al. 2014). To avoid the formation of free water on the flower surface, overhead watering is discouraged. Surface irrigation in the morning is recommended so that the foliage can dry as rapidly as possible (Whealy 1992; Gibson et al. 2014). Venting, use of fans to provide air movement above the canopy, and heating when carnations begin to show color help inhibit flower blight development (Whealy 1992; Gibson et al. 2014). Good aeration reduces surface moisture and thereby retards or inhibits *Botrytis* spore germination. After harvest, only blemish-free, nonsenescent flowers should be put in storage immediately under temperatures as close to freezing as possible. The best method to control Botrytis petal blight during postharvest is to prevent condensation. Temperature should be carefully controlled during transportation as the passage from cool storage to poorly refrigerated transports encourages the formation of free moisture on the flower surface (Garcés De Granada 1992).
- ***Sanitation*** – Sanitation practices in the greenhouse are crucial to achieve effective disease control both before and during the cropping cycle. Senescing tissues (including flowers and leaves) and infected plant material that provide the inoculum for new infections should be removed from the greenhouse as should wounded plants that provide a favorable environment for the infection process. The removed material should not be allowed to remain in trash cans within the greenhouses (Gibson et al. 2014).
- ***Fungicides*** – A variety of fungicides are available to control gray mold in the greenhouse. These include compounds containing azoxystrobin, boscalid + pyraclostrobin, chlorothalonil, copper, cyprodinil + fludioxonil, fludioxonil, fluoxastrobin, imazalil, iprodione, mancozeb, mancozeb + copper, polyoxin-D, and trifloxystrobin. Some of these fungicides are combinations of two different modes of action formulated by the manufacturer, and others can be mixed and applied simultaneously (e.g., chlorothalonil + thiophanate-methyl, iprodione + thiophanate-methyl) (Gibson et al. 2014; Gould 2012). Spraying of carnation flowers with fungicides before harvest can help reduce the infection during storage and transport (Whealy 1992).

  Some *Botrytis* populations have developed resistance to certain chemicals (including compounds containing cyprodinil, iprodione, or thiophanate-methyl) (Gibson et al. 2014; Gould 2012). As a result, the use of single or combined chemicals for extended periods of time should be avoided to reduce the possibility of developing fungicide resistance in the fungus.
- ***Biological control*** – Biological control agents such as *Bacillus subtilis*, *Streptomyces lydicus*, *S. griseoviridis*, and *Trichoderma harzianum* provide effective control of gray mold in the greenhouse (Gibson et al. 2014).
- ***Chemical control in cut flowers*** – Premature senescence caused by exposure to ethylene can be mitigated during postharvest by using inhibitors (Scariot et al. 2014). The gaseous ethylene antagonist 1-methylcyclopropene (1-MCP)

has been found to be effective in reducing damage caused by Botrytis blight in cut flowers of carnation (Seglie et al. 2012). The inclusion of 1-MCP in β-cyclodextrin-based nanosponge structures can overcome the practical difficulties in the application of this gaseous compound and has been shown to be effective in prolonging cut flower vase life and in reducing the development of gray mold (Scariot et al. 2014; Seglie et al. 2012).

2.5 Downy Mildew [*Peronospora dianthi* de Bary; *Peronospora dianthicola* Barthelet (Nom. inval., Art. 39.1 (Melbourne) Index Fungorum)]

Geographic occurrence and impact. The disease was reported from China, Colombia, the Czech Republic, France, Greece, Israel, Italy, Japan, and the USA (Ambrico et al. 2003; Arbeláez 1979; Ben Ze'ev et al. 2006; Duan et al. 2010; Farr and Rossman 2016; Gardner and Yarwood 1950; Holevas et al. 2000; Kayamori et al. 2012; Safránková 2012).

Incidence of 20% of diseased plants was recorded in Yunnan, China, from 2008 to 2010 (Duan et al. 2010).

Symptoms/signs. The disease first appears in the young leaves that show a pale-green color and turn yellow. Gray masses of sporangia (also named "conidia") develop firstly on the underside of the leaves and then on the upper surface (sign) (Fig. 4). Heavily infected plants are stunted and eventually show a bushy appearance due to the development of axillary buds.

Biology and epidemiology. *Peronospora dianthi* is a fungus-like microorganism belonging to the Kingdom Stramenopiles. It is an obligate parasite that only can live on a living host. It produces sporangia that when mature are transported by



Fig. 4 Symptoms/signs of downy mildew of carnation caused by *Peronospora dianthicola*: yellowing of new leaves (*left*), conidiophores, and conidia on both upper (*center*) and lower leaf surfaces (*right*) (Kayamori et al. 2012)

means of airflow, rain, irrigation, or field workers to young leaves. Less than 1 week after infection, abundant new sporangia arise from the underside of the leaves forming gray visible masses (sign) and then can develop on the upper leaf surface. Oospores (resting spores) are produced abundantly in leaves during autumn. The leaves eventually fall and become plant debris, and they carry the pathogen over the winter to spring when germinating oospores initiate the disease.

Management

- ***Cultural practices*** – Use proper spacing between beds and plants in beds, and remove weeds to maintain good air circulation and reduce leaf wetness.
- ***Fungicides*** – Disease can be controlled with copper oxychloride, mancozeb, zineb, metalaxyl, and fosetyl-Al (Arbeláez-Torres 1987). Also fludioxonil, imazalil, kresoxim-methyl, and propiconazole have been recommended (Gould 2012).

2.6 Fairy-Ring Leaf Spot [*Cladosporium echinulatum* (Berk.) G.A. De Vries,Teleomorph: *Mycosphaerella dianthi* (Burt.) Jorst]

According to Bensch et al. (2012), this species belongs to the *Cladosporium herbarum complex.*

Geographic occurrence and impact. The disease has been reported in Argentina, Australia, Canada, Chile, Colombia, the Czech Republic, Denmark, France, Finland, Germany, Italy, Kenya, Korea, New Zealand, Poland, Portugal, Romania, South Africa, the U K, the USA (Hawaii), and Venezuela (Alfieri et al. 1984; Arbeláez-Torres 1987; Bensch et al. 2012; Cedeño and Carrero 1997; Cho and Shin 2004; Cook and Dubé 1989; Crous et al. 2000; David 1997; French 1989; Ginns 1986; Gorter 1977; Marchionatto 1944; Mulenko et al. 2008; Sandoval et al. 2009; Trujillo and Nagata 1993).

This pathogen causes a loss in quality and quantity of harvested stems. In Colombia and Chile, *C. echinulatum* is one of the most important pathogens affecting the carnation foliage (Arbelaez Torres 1987; Sandoval et al. 2009).

Symptoms/signs. The pathogen affects the aerial organs of the plant, especially leaves and flowers (Fig. 5). The infection on leaves begins as purple specks which later enlarge and have tan to gray centers enclosed by a purple border. On the petals, light-brown lesions are produced which limit cut flower marketability (Cedeño and Carrero 1997).

Biology and epidemiology. According to Agrios (2005) and Latorre (2004), conidia are spread by wind, cultural practices, workers, diseased plants, or infected harvest residues. It is increased by high humidity during wet weather or wet leaves in greenhouses.



Fig. 5 Fairy-ring leaf spot symptoms on leaves (*left*) (S. Nakkeeran et al. Tamil Nadu Agricultural University, Coimbatore, Tamil Nadu, India © 2017. All Rights Reserved.) flower (*right*) (SM Wolcan)

Management

- ***Cultural practices*** – Keeping condensation in greenhouses under control, using ventilation and heating carefully, and only irrigating in the morning will help reduce this disease. Removal of senescing and dead plant tissue that provide a ready substrate for *Cladosporium* sporulation is important for the management of the disease (Arbeláez-Torres 1987).
- ***Fungicides*** – Mancozeb is recommended as a contact and preventive fungicide and inhibits spore germination; copper sulfate pentahydrate is a systemic product with therapeutic and preventative activities (Sandoval et al. 2009). This fungicide destroys the fungal cell wall, inhibiting spore germination. Ammonium bicarbonate, sodium bicarbonate, and potassium carbonate have been shown to be effective in the control of foliar pathogens, both in vitro and in vivo in carnations and other ornamental species. In Colombia, fungicides as propineb, captafol, zineb, tricarbamix, chlorothalonil, and triforine have been successfully tested to control the disease (Arbeláez-Torres 1987).
- ***Biological control*** – With regard to biocontrol, *Trichoderma virens* strain Sherwood has been able to control the damage caused by the pathogen, presenting a healing effect on the active wounds of *C. echinulatum* on carnation leaves (Sandoval et al. 2009).

2.7 Fusarium Stem Rot; Fusarium Stub Dieback; Fusarium Basal Rot; Fusarium Branch Rot; Fusarium Cutting Rot; Fusarium Pink [*Fusarium graminearum* Schwabe; *Fusarium culmorum* (W.G. Smith) Saccardo; *Fusarium avenaceum* (Fries) Saccardo; *Fusarium verticillioides* (Saccardo) Nirenberg; *Fusarium proliferatum* (Matsushima) Nirenberg]

Geographic occurrence and impact. The etiology of Fusarium stem rot/stub dieback/basal rot/branch rot/cutting rot is not clearly defined. Stub dieback is generally

associated with *Fusarium graminearum* (formerly *F. roseum* "Graminearum") (Nelson et al. 1975) and several other *Fusarium* species: *F. avenaceum*, *F. culmorum*, *F. verticillioides*, and *F. proliferatum* (Elad et al. 2014; Guba 1945; Robinson 1961; White 1938; Wickens 1935; Wolcan et al. 1995).

The three species, *F. avenaceum*, *F. graminearum*, and *F. culmorum*, were grouped together under the name *F. roseum* (Lk) emend. Snyd. and Hans. Snyder and Hansen's 1945 revised genus resulted in much of the literature after this time regarding Fusarium stem rot as referring to *F. roseum*.

The disease has been reported in Argentina, Australia, Denmark, England, France, Israel, New Zealand, Sweden, and the USA (Elad et al. 2014; Kalc Wright et al. 1997; Moreau 1953; Nelson et al. 1975; Nilson 1962; Robinson 1961; White 1929; Wolcan et al. 1995, 2007).

Fusarium culmorum is very common and widespread throughout New Zealand. It causes considerable damage as a basal rot of young plants and as a dieback of shoots (Robinson 1961). In some countries, the introduction of new cultivars bred for resistance to *F. oxysporum* f. sp. *dianthi* raises the potential problem of increasing the incidence of basal rot caused by *F. avenaceum* and *Fusarium* spp. In addition, these species can be carried on symptomless cuttings, which can result in disease introduction into clean glasshouses and spread onto adjacent, susceptible cultivars (Richardson 1987).

Symptoms/signs. The disease is divided into four phases (basal rot, basal stem rot, branch rot, and stub dieback) that affect different stages of production. Major losses occur due to basal cutting rot during propagation and basal stem rot of rooted cuttings shortly after planting (Fig. 6). Basal stem rot and branch rots are observed before flowering (Fig. 6). After flower harvest, damage occurs principally as a consequence of the dieback of stem stubs as the fungus grows down the stub into the main stem, reducing the number of flower shoots and plant productivity. Growth of the fungus rarely progresses down the stem for more than a few internodes and appears to be stopped when a junction with a larger branch of the main stem is reached. The fungus can grow into the main stem and girdle it, causing the death of the plant. Kalc Wright et al. (1997) found that on some occasions more than one



Fig. 6 *Fusarium* stem rot. *Left*: rooted cuttings. *Right*: necrotic plant before flowering with one healthy branch (S.M. Wolcan © 2017. All Rights Reserved.)

species was recovered from different parts of the same plant exhibiting different symptoms. In one instance, *F. acuminatum* Ell. and Everh. was isolated from healthy basal tissue, while 8 cm upward the same plant, *F. avenaceum*, was found invading through a wound in the stem. In two other cases, mixed infections of *F. avenaceum* and *F. oxysporum* were detected in plants with root and basal rots, while *F. avenaceum* was isolated from healthy stem tissue 3 cm above the soil level. Orange masses of sporodochia (conidia) of *F. graminearum* and black perithecia of *Gibberella zeae* on the rotten stem and branches are the main signs of the disease.

Biology and epidemiology. Environmental conditions suitable for plant production may also be favorable to pathogens. Cutting and flower collection produce exposed tissue surfaces, and intensive handling may result in wounds, all of which provide avenues for invasion by pathogens. General lack of adequate hygiene also favors disease spread (Kalc Wright et al. 1997). Under conditions of high relative humidity, the mycelium grows profusely on infected stubs. Although stub dieback occurs at all stages of plant growth, the most severe losses are sustained on 2-year-old plants during flower harvest. This may be related to the physiological age of the stem tissue or simply to the fact that the foliage canopy is so thick that it aids in keeping the relative humidity high (Nelson et al. 1975). Epidemics of stem rot and basal rot can occur during the summer months when it is not possible to control greenhouse environmental conditions which favor disease development. During the summer, plants may receive fertilizer rich in nitrogen that can induce lush, soft vegetative growth that Dorworth and Tammen (1969) found could favor the development of Fusarium stem rot. *Fusarium* spp. can spread aerially by means of microconidia and macroconidia, and most produce resistive chlamydospores which enable long-term survival. Survival of *F. graminearum* in association with plant debris, as occurs in many other hosts, is very likely.

Management

- ***Sanitation*** – Control consists of proper sanitation and cultural practices. Avoid over watering and excessively fertilizing, and provide good drainage and ventilation. Management practices consist of the removal of the inoculum source such as residues that contain *F. graminearum* sporodochia and *G. zeae* perithecia to avoid the rapid spread of the pathogen in the greenhouses (Kalc Wright et al. 1997; Nelson et al. 1975). Avoid injury to plants during cultural labors.

2.8 Fusarium Wilt (*Fusarium oxysporum* Schlectend.: Fr. f. sp. *dianthi* Prill. and Delacr.) Snyder and Hansen

Geographic occurrence and impact. Fusarium wilt of carnation has been reported worldwide (Fig. 7). Eleven races of *Fusarium oxysporum* f. sp. *dianthi* (*Fod*) have been distinguished. Race 1 is present in Colombia, France, Italy, and Spain (Baayen et al. 1997; Poli et al. 2013) and is closely related to race 8, which is present in Italy,



Fig. 7 Fusarium wilt of carnation caused *Fusarium oxysporum* f. sp. *dianthi* (G.A. Lori © 2017. All Rights Reserved.)

France, and Spain (Baayen et al. 1997). It has been proposed that race 8 might have arisen from race 1 by adaptation to resistant cultivars, as these two races differ in one or a few avirulence genes involved in host-pathogen recognition that are present in race 1 and absent in race 8 (Migheli et al. 1998). Race 2 is present worldwide (Aloi and Baayen 1993); race 3 has been reclassified as *F. redolens* Wollenw. f. sp. *dianthi* Gerlach (Baayen et al. 1997); race 4 is present in Colombia, Italy, Israel, Spain, and the USA (Baayen et al. 1997; Ben-Yephet et al. 1992; Cevallos et al. 1990; Garibaldi 1983); race 9 is present in Australia (Kalc Wright et al. 1996); and races 10 and 11 are present in the Netherlands (Brayford 1996). Races 5, 6, and 7 have been reported in France, the Netherlands, and the UK (Garibaldi 1983). However, only a few representatives of these pathotypes are currently available, and RAPD and RFLP profiles, vegetative compatibility groupings, and esterase groupings of representatives of these races were indistinguishable from those belonging to race 2 when tested (Manicom et al. 1990; Migheli et al. 1998). This is the most destructive disease of carnation.

Symptoms/signs. The pathogen infects the vascular system of affected plants, interfering in water and nutrient absorption. Externally, the infected plants appear chlorotic and show signs of wilting: lower leaves become yellow and dry, the stem often shrivels and turns grayish, and xylem tissues turn brown (Fig. 8; Baayen and De Maat 1987; Hood and Stewart 1957; Sharma and Sharma 2008). Symptoms commonly appear on one side of the plant, causing it to curl. Eventually, the symptoms quickly spread to the other side of the plant, resulting in its death (Brayford 1996).

Biology and epidemiology. *Fod* is present in the soil and infects young or mature plants at any time during the growing season via unwounded or wounded roots, crosses the root cortex, and enters the vascular tissues (Ben-Yephet and Shtienberg 1997; Brayford 1996). The colonization of the stem proceeds upward causing a brownish discoloration of the xylem tissues. Upon death of the plant, *Fod* produces pale-pink masses of macro- and microconidia on the surface of the colonized stem



Fig. 8 Carnation wilt symptoms, dark vascular system of infected plants (G.A. Lori © 2017. All Rights Reserved.)

and root tissues (Brayford 1996). *Fod* also produces resistive chlamydospores which enable long-term survival. *Fod* persists in the soil for long periods and can survive in and around greenhouses on benches, shoes, and even in the wood used for bench supports. The spores of the pathogen can remain viable for long periods and be spread by soil, wind, water, infected cuttings, and contaminated tools, equipment, and clothing (Rattink 1977). Fungus gnats are also effective vectors of *F. oxysporum* in greenhouses.

Management

- ***Cultural practices*** – Cultivation of carnations in raised benches allows for a better eradication of inoculum as the soil disinfestation is enhanced (Garibaldi and Gullino 1987). To avoid contamination of soils free of *Fod* propagules or recontamination of disinfested soils, the use of infected propagation material should be avoided. When possible, use cuttings produced from meristem tissue cultures of *Fusarium*-free mother plants (Garibaldi and Gullino 1987). Amendment of the soil with compost or manure with high levels of organic nitrogen reduces the incidence of Fusarium wilt. Amendments generate ammonia and/or nitrous acid, which are lethal to many pathogens, including *Fod* (Melero-Vara et al. 2011). These compounds are accumulated in the soil at concentrations that depend on its pH, organic matter content, buffering capacity, and nitrification rate (Lazarovits 2001). Melero-Vara et al. (2011) found that repeated amendment with poultry manure was effective in reducing Fusarium wilt incidence in carnation to



Fig. 9 Carnation greenhouse, soil disinfestation by solarization (G.A. Lori © 2017. All Rights Reserved.)

values similar to those obtained by the use of methyl bromide (MeBr). Suppression of fungus gnats will also prevent the spread of Fusarium wilt of carnation

- ***Soil disinfestation*** – Soil disinfestation is one of the most effective means of controlling Fusarium wilt of carnation. Soil disinfestation can be achieved by either physical or chemical treatments. Physical treatments include the application of soil solarization (Fig. 9). Elena and Tjamos (1997) found that soil solarization in greenhouses for periods of around 50 days was very effective in destroying *Fod* propagules down to a depth of 30 cm and significantly reduced the final wilt incidence. These effects were obtained even with differences of only 3–6 °C in soil temperature between solarized and control plots. However, the control of *Fod* by solarization is not easily achieved (Tjamos et al. 1999), and even in the cases where soil treatment with solar heat proves effective against Fusarium wilt, it is not practical as the treatment should be carried out during the time when carnations are grown (Garibaldi and Gullino 1987). Some of the limitations of soil solarization could be avoided by combining this practice with the use of fumigants at reduced dosages, which could shorten the duration of the heating period, or with biocontrol agents, thus making the method more attractive for growers (Gamliel and Katan 2009). Soil reductive sterilization (SRS) by induction of anaerobic conditions in the soil is an alternative to solarization in areas where temperatures are too low for the latter to be effective. By incorporating wheat bran into the soil, flooding, and covering it with polyethylene film, Yossen et al. (2008) achieved SRS and reduced disease incidence of Fusarium wilt in carnation by 84% and 98% in the first and second years of treatment, respectively. Another physical treatment for soil disinfestation is steaming, which can prove to be useful especially in greenhouses where growth media are used on raised benches (Tjamos et al. 1999). The control of *Fod* by steam disinfestation frequently results in incomplete eradication of the fungus (Garibaldi and Gullino 1987). Soil fumigation is the main chemical treatment for soil disinfestation in a number of countries. MeBr is the most powerful soil fumigant with a very broad spectrum of activity, but its use has been banned in a number of countries under the Montreal Protocol (UNEP 2006). Other fumigants such as dazomet (3,5, dimethyl-tetrahydro-l,3,5,(2H) thiodiazino-thione), methyl-isothiocyanate, and metam sodium

(sodium methyldithiocarbamate) strongly reduced *Fod* inoculum (Garibaldi and Gullino 1987). Nevertheless, these fumigants are not free of inconveniences. The biological vacuum that results from fumigation of soils affects its suppressiveness and explains the occurrence of subsequent increased attacks of *Fod* (Garibaldi and Gullino 1987).

- ***Fungicides*** – Garibaldi and Gullino (1987) found benomyl (benzimidazole) to be suitable for reducing infections of Fusarium wilt of carnation already present, in a temporary manner, at dosages of 10–20 g/m^2, while captafol (phthalimide) resulted in a prolonged reduction of inoculum at the same dosages. The authors reported that the appearance of resistant strains of *Fod* in France resulted in failures of soil treatments with benomyl. Among strobirulin fungicides, azoxystrobin (1–2 g/m^2) gave good results against *Fod*, while trifloxystrobin and kresoxim-methyl were only partially effective at 2 g/m^2. Better results were obtained for all strobirulins when they were applied by soil drenching (10 l/m^2 of water) compared to soil mixing (Gullino et al. 2002).
- ***Biological control*** – Even in the presence of a virulent pathogen and susceptible plant host, Fusarium wilt does not occur or is less severe in some soils that are referred to as "suppressive" (Mazzola 2002). It has been proposed that competition between nonpathogenic and pathogenic fusaria is the basic mechanism for suppression of Fusarium wilt diseases in these soils (Garibaldi and Gullino 1987; Mazzola 2002). Empirical methods, such as organic amendments, cultural practices (including crop rotation, tillage, and fertilization), and the introduction of antagonists, might influence ecological processes that impact microbial communities and have been proposed as a way of inducing soils' suppressiveness (Hornby 1983; Mazzola 2002). Drenching with *Trichoderma harzianum* and other biocontrol agents, such as chitinolytic bacteria *Arthrobacter* sp. and *Serratia liquefaciens*, has resulted in significant reductions in incidence of Fusarium wilt of carnation under field conditions (Garibaldi and Gullino 1987).
- ***Resistance*** – The development of cultivars resistant to Fusarium wilt of carnation is hindered by the presence of different races of *Fod* (Garibaldi and Gullino 1987). Resistance mechanisms seem to be variable between races: Demmink et al. (1989) suggested that resistance to race 1 in *Fod* is monogenic and dominant, whereas resistance of carnation cultivars to race 2 is usually partial and polygenic (Sparnaaij and Demmink 1977). As a result, the level of resistance achieved by breeding programs is usually operative against only one or a few *Fod* races (Garibaldi and Gullino 1987).

Furthermore, it has been proposed that the composition and aggressiveness of *Fod* populations might be constantly changing. The Spanish population of the pathogen, composed of isolates of races 1 and 2, shifted over time, as was revealed by molecular markers and pathogenicity tests, allegedly as a consequence of the continuous change in the commercial carnation cultivars grown by farmers (Gómez-Lama Cabanás et al. 2012). Similarly, Prados-Ligero et al. (2007) found that the resistance of several carnation cultivars against Italian *Fod* isolates of races 1 and 2 was variable and that different isolates of the same race induce different reactions

in a same cultivar. None of the management practices currently available completely control Fusarium wilt of carnation. As a result, only the integration of different control measures permits management of the disease (Garibaldi and Gullino 1987). For example, combining solarization with chemicals at reduced dosages (Tjamos et al. 1999), biocontrol agents (Gamliel and Katan 2009), incorporation of organic amendments into the soil before mulching (Tjamos et al. 1999), or other measures could temper the climate dependence of solarization or shorten the required duration of solar exposure, thus making the method more effective and acceptable to growers.

2.9 Powdery Mildew (*Erysiphe buhrii* U. Braun; Anamorph: *Oidium dianthi* Jacz.)

Geographic occurrence and impact. The disease was reported in different countries of the Northern hemisphere: Bulgaria, Finland, Guatemala, Israel, Japan, Poland, Romania, Switzerland, Turkey, the UK, the Ukraine, and the USA (Aleksandrova 1976; Amano 1986; Braun 1995; Farr and Rossman 2016; Mulenko et al. 2008; Saenz et al. 1995; Takahashi and Takamatsu 2003; Voytyuk et al. 2009). Although the fungus is cited in the whole world as affecting other plants of the Caryophyllaceae family including *Gypsophila* (▶ Chap. 19, "Diseases of Gypsophila") and several weeds (Braun 1995), powdery mildew has not been reported on carnation in the Southern hemisphere.

Symptoms/signs. White mycelium bearing conidiophores and conidia is the sign of the disease. It can grow densely and is observed on both surfaces of the leaves and calyces and at the junction of stem nodes and leaf bases. Below the mycelia, the plant tissues show chlorosis (symptom).

Biology and epidemiology. *Erysiphe buhrii* is an obligate parasite that can live only in living plant tissue. A full life cycle includes both sexual and asexual reproduction. The mycelium produces conidia (asexual stage) that are released when mature and can be transported by wind over long distances infecting near or distant plants. Conidia germinate only if the plant surface is water-free. The growth of the fungus is outside of the tissues (ectophytic growth) and only invades epidermal cells. It takes less than a week from infection to the production of new conidia, and then the cycle is restarted. Thus, many cycles may develop during the plant's production season, and the complete plantation can be affected in a short time. In several countries, the fungus' life cycle is completed with the formation of chasmothecia (sexual stage) in the absence of the host or when environmental conditions are unfavorable for the pathogen growth. Chasmothecia contains ascospores that can also serve as primary inoculum.

Management

- ***Cultural practices*** – After the growing season, remove crop residues. Remove weeds inside and outside the greenhouse to increase air circulation among the plants and avoid possible presence of alternative hosts. Avoid transferring inocula from infected plants to healthy ones by means of the hands, clothes, and tools.

- ***Fungicides*** – If infections of powdery mildew are common in the production area, preventive fungicides must be used. It is important to apply curative products when the first signs are detected because powdery mildew is extremely difficult to control once established. The fungus can develop resistance to repeatedly used fungicides; therefore, it is necessary to rotate fungicides having different modes of action. Azoxystrobin, copper fungicides, fludioxonil, imazalil, kresoxim-methyl, and propiconazole are recommended (Gould 2012).

2.10 Phytophthora Root Rot, Phytophthora Foot Rot, Phytophthora Wilt, Phytophthora Collar Rot, Phytophthora Blight [Mainly Caused by *Phytophthora nicotianae* Breda de Haan = *Phytophthora parasitica* Dastur (Previously Named as *P. nicotianae* var *parasitica* and *P. parasitica* var *nicotianae*) and Also by *Phytophthora cactorum* (L. & c.) Schroeter, *Phytophthora capsici* Leonian, *Phytophthora cryptogea* Pethybridge and Lafferty, *Phytophthora megasperma* Drechsler, *Phytophthora palmivora* Butler (Butler), *Phytophthora porri* Foister, and *Phytophthora* sp.]

Geographic occurrence and impact. The diseases are caused by different *Phytophthora* species around the world, mostly *P. nicotianae* = *P. parasitica* that has been recorded in Australia, Bulgaria, France, Greece, Japan, Korea, the Netherlands, Poland, South Africa, Taiwan, and the USA (Ann et al. 1990; Cho and Shin 2004; Farr and Rossman 2016; Garibaldi and Rapetti 1977; Gullino et al. 2002; Jin et al. 1998; Orlikowski et al. 1991; Rattink 1979; Sarejanni 1952; Uematsu et al. 1990). The other *Phytophthora* species that were recorded infecting carnation in different countries are *P. cactorum* in South Africa (Crous et al. 2000; Wijers 1937); *P. capsici* in Bulgaria, Korea, and Taiwan (Ann et al. 1990; Ilieva and Ivanova 1993; Jin et al. 1998); *P. citricola* in Italy (Garibaldi and Rapetti 1977); *P. cryptogea* in Taiwan (Ann et al. 1990); *P. palmivora* in Italy (Andreucci 1959), and *P. porri* in Greece (Kouyeas 1977). In Colombia, *Phytophthora* spp. occasionally affect carnation crops (Arbeláez-Torres 1987). During continuous rain in the typhoon seasons in Taiwan, *Phytophthora* spreads rapidly and destroyed 20% of the carnation crop (Ann et al. 1990).

Symptoms/signs. The symptoms occur in all growing stages of plants. *P. nicotianae* can invade the callus tissue of recently planted carnations causing a complete rot of the collar and first internodes and killing the plant (Ricci and Bonnet 1977). Symptoms of Phytophthora stem rot at the soil line may be mistaken for Rhizoctonia stem rot. The distinguishing symptom of infection by *Phytophthora* spp. is a rapid and complete wilting of the plants within 24–48 h. Leaves show a light greenish-gray discoloration; the tissues of the stem base have a brown, water-soaked appearance; and the roots rot.

Biology and epidemiology. The typical *P. nicotianae* life cycle includes both asexual and sexual phases. The asexual stage includes hyphae, sporangia that contain zoospores, and chlamydospores. The zoospores have two flagella, which enable them to swim in soil water to reach the roots of other plants that they infect. Chlamydospores are thick walled (resting spores), usually produced at the tips or in the middle of hyphae. *P. nicotianae* is predominantly heterothallic, requiring A1 and A2 mating types for the production of oospores. These oospores have a very thick wall and can survive in soil and plant debris for long periods. They germinate and produce sporangia over a wide range of temperatures (10 –35 °C), with the optimum between 25 °C and 30 °C. Wet seasons, overwatering, and poor drainage of soils are other conditions that favor the production of zoospores and the plant infection (Meng et al. 2014). These characteristics are similar for the other *Phytophthora* spp. recorded on carnation.

Management

- ***Cultural practices*** – Avoid high relative humidity and overwatering. Use well-drained soils. Control weeds inside and outside the greenhouse to improve air circulation among carnation plants, and eliminate possible alternative hosts of *Phytophthora* spp.
- ***Solarization*** **–** *S*oil mulching with clear double-layer polyethylene significantly reduced the percentage of carnation plants infected by *P. nicotianae* var. *parasitica* (Garibaldi and Tamietti 1989).
- ***Fungicides*** – The use of two strobilurin fungicides, azoxystrobin and kresoxim-methyl (1 g/m^2) applied as a soil drench or incorporation at transplant in potted plants, gave good results, similar to those obtained by using metalaxyl at the same rate (Gilardi et al. 2000). Gullino et al. (2002) evaluated the effectiveness of three strobilurin fungicides against the three important soil pathogens *Fusarium oxysporum* f. sp. *dianthi*, *P. nicotianae* var *parasitica*, and *Rhizoctonia solani* in the greenhouse. Azoxystrobin at 1–2 g/m^2 consistently gave good results, and trifloxystrobin and kresoxim-methyl appeared only partially effective when applied at 2 g/m^2. Fungicides applied by soil drenching at a rate of 10 l/m^2 of water gave better results compared to soil incorporation. Also etridiazole, mefenoxan, propamocarb hydrochoride, and pyraclostrobin are recommended (Gould 2012).

2.11 Pythium Root Rot [*Pythium aphanidermatum* (Edson) Fitzpatrick; *Pythium irregulare* Buisman; *Pythium ultimum* Trow.; *Pythium vexans* de Bary (Currently Named *Phytopythium vexans* (de Bary) Abad, de Cock, Bala, Robideau, Lodhi and Lévesque)]

Geographic occurrence and impact. *Pythium aphanidermatum* was recorded in Canada and China (Bolton 1984; Kimishima et al. 1991), *P. irregulare* in Argentina



Fig. 10 Young plants affected by *Pythium* sp. after transplanting in soil. Healthy plant below (S. Nakkeeran et al. Tamil Nadu Agricultural University, Coimbatore, Tamil Nadu, India © 2017. All Rights Reserved.)

and Japan (Frezzi 1956; Kimishima et al. 1991), *P. ultimum* in Argentina and Germany (Frezzi 1956; Gerlach 1986), and *P. vexans* in the USA (Raabe and Hurlimann 1972). *Pythium* species can cause damage ranging from slight injury to death of the carnation plantings and plants growing in soil or soilless media.

Symptoms/signs. Different *Pythium* species produce rootlet rot and brownish-black, water-soaked lesions at or near the base of the cuttings growing in rooting benches. Infection spreads upward in the stems and downward into the roots, discoloring all tissues and rapidly spreading to neighbor plants. With high humidity, white mycelia (sign) can be observed on infected tissues. When infected cuttings have no visible symptoms, they carry the inoculum to the production site where symptoms are expressed. *Pythium*-infected plants in soil beds exhibit root rot, discoloration of stems and basal leaves (Fig. 10), and reduction in plant stem vigor and height (plants remain stunted) and of number of flowers. Also, the pathogen can be found in soil, its natural habitat through survival structures, and it can infect healthy transplanted cuttings.

Biology and epidemiology. *Pythium* species are soilborne fungus-like microorganisms that belong to the same family as *Phytophthora*: Peronosporaceae, Kingdom Stramenopiles. Species of *Pythium* are common in most soils where they can live saprophytically or parasitically. When conditions are favorable for the fungus but less for the host, *Pythium* species can become very pathogenic. Young or soft tissue is preferentially attacked. As occurs with *Phytophthora*, in environments with excessive moisture or free water, the mycelia produce zoosporangia from which develop zoospores with two flagella. These spores are able to swim in soil water and

reach roots of other plants that are infected by means of germ tubes that penetrate the epidermis directly. Infection also depends on inoculum density, temperature, pH, and light intensity. Optimum temperature is dependent on the species of *Pythium* involved. Most species that affect carnation are polyphagous and when there are no carnation plants can survive infecting other plants (weeds or other crops) or remain in soil or plant debris as oospores (resting spores).

Management

- ***Cultural practices*** –Avoid high relative humidity and overwatering in rooting beds and in the production greenhouses. Use well-drained, well-leveled soil (avoid planting low areas where water accumulates). Control weeds inside and outside of greenhouses to keep air circulation among carnation plants, and avoid possible alternative hosts of *Pythium* spp.
- ***Biological control*** – Some species of *Pythium* act as mycoparasites. *P. oligandrum*, *P. acanthicum*, and *P. periplocum* are well known as parasites of *P. irregulare*, *P. ultimum*, and *P. vexans* (Van der Plaats-Niterink 1981).
- ***Fungicides*** – Etridiazole, mefenoxan, propamocarb hydrochoride, and pyraclostrobin are recommended (Gould 2012).

2.12 Rhizoctonia Cutting, Stem and Collar Rot [*Rhizoctonia solani* Kühn (Teleomorph: *Thanatephorus cucumeris* (A B Frank) Donk.)]

Geographic occurrence and impact. The disease has been reported in Argentina, Bulgaria, Canada, China, Colombia, Egypt, India, Italy, Israel, Korea, South Africa, and Turkey (Arbeláez-Torres 1987; Arici and Kazaz 2013; Bolton 1984; Chandel and Pathania 2003; Cho and Shin 2004; Eisa et al. 2000; Elad et al. 2014; Gullino et al. 2002; Lo et al. 1990; Mirkova and Maneva 2007; Wijers 1937; Wolcan et al. 1999). Farr and Rossman (2016) included reports from Australia, Brazil, Thailand, the USA, and Zimbabwe. In India, *R. solani* causes an emerging devastating disease in carnation rated next in importance to Fusarium wilt with a seedling mortality of 37% under conducive climatic conditions (Chandel and Pathania 2003). In Colombia and South Africa, Rhizoctonia rot is considered a minor disease, affecting only a few cultivars (Arbeláez-Torres 1987; Wijers 1937).

Symptoms/signs. Cuttings under propagation are especially susceptible to this disease. The symptoms occur mainly in rooting trays of cuttings and in rooted cuttings planted on the ground (especially if they are placed too deeply). In this case, the pathogen could have colonized the soil or could enter the soil through the infected rooted cuttings. Infected cuttings suddenly die. The most characteristic symptom is the grayish-green color and the flaccid aspect of the foliage of affected plants. Leaves become yellow, and when pulling the plants, roots remain in the soil, and the stem base shows dark discoloration and soft rot (Fig. 11). The symptom of stem rot at the soil line may be mistaken for Phytophthora stem rot.



Fig. 11 Rhizoctonia basal rot. Disintegrated roots and black basal stem rot (S. Nakkeeran et al. Tamil Nadu Agricultural University, Coimbatore, Tamil Nadu, India © 2017. All Rights Reserved.)

Biology and epidemiology. *Rhizoctonia* produces only mycelia and sclerotia. Both structures survive in soil and plant debris. Favored by warm, moist soil and poor drainage, the sclerotia germinate and produce mycelia that enter the plant through the wounded base of the stem. The prevalence of a warm humid climate favors the occurrence of stem rot. *R solani* populations are divided into anastomosis groups; among them AG-4, AG-2-2, AG-1, AG-6, and AG-7 are pathogenic on carnation causing different levels of disease on different cultivars (Lo et al. 1990; Trujillo et al. 1988).

Frequently, more than one soil pathogen is isolated from the same diseased plant (Arici and Kazaz 2013; Gullino et al. 2002; Wolcan et al. 1999). Mirkova and Maneva (2007) tested the pathogenicity of *R. solani*, *Phytophthora nicotianae*, *Fusarium oxysporum* f. sp. *dianthi*, and *Pythium* spp. and reported different synergisms among of the fungi on the different cultivars of carnation assessed.

Management

- ***Cultural practices*** – It is important to keep low humidity levels during critical periods, such as after pruning or during harvesting time. The risk of infection may be reduced by cultivation on sandy soils, with good drainage, using an optimal ventilation system, keeping the greenhouse free of weeds, practicing shallow planting of cuttings, and keeping a low or medium fertility level. Crop debris should be removed.

- ***Biological control*** – Elad et al. (1981) showed that *Trichoderma harzianum* gave best disease control when applied and established in the rooting mixture before transplanting in the field. The control agent was introduced into the rooting carnation plants as Speedlings in peat supplemented with *T. harzianum* preparation (15% by volume). When transferred to soil infested with *R. solani*, the treated plants had the lowest infection rate (13% diseased plants). This method was superior to broadcast application. In India, the reduction of *R. solani* infection occurred when *T. hamatum* was incorporated in the soil a week ahead of transplantation of cuttings and 45 d after establishment of cuttings (Chandel and Pathania 2003).
- ***Fungicides*** – Gullino et al. (2002) evaluated the effectiveness of three strobilurin fungicides against the three important soil pathogens *Fusarium oxysporum* f. sp. *dianthi*, *Phytophthora nicotianae* var *parasitica*, and *Rhizoctonia solani* in the greenhouse. They reported that the application of azoxystrobin at 1–2 g/m^2 showed consistently good results and that trifloxystrobin and kresoxim-methyl were partially effective when applied at 2 g/m^2. The application carried out by soil drenching with 10 l/m^2 of water produced better results compared to the soil mixing application. Also, captan, fludioxonil, iprodione, PCNB, and pyraclostrobin are recommended (Gould 2012).
- ***Integrated strategies: botanicals, biofumigant with crop residues, and biological control*** – Results obtained by Chandel and Sharma (2014) in several tests in vitro and in vivo suggested that the use of *Trichoderma harzianum* and *T. viride* along with commercial formulations of neem and cruciferous residues is a good alternative to conventional chemicals in managing stem rot of carnation. The effect of residues of cauliflower and cabbage (2 g/kg of soil) effectively reduced the disease and improved the evaluated parameters of the plants.

2.13 Rust [*Uromyces dianthi* (Pers.) Niessl = *Uromyces caryophyllinus* (Schrank) J. Schröt.]

Geographic occurrence and impact. This disease has been reported in Argentina, Australia, Brazil, Canada, Chile, China, Denmark, Greece, Israel, Kenya, Korea, Mexico, New Zealand, Poland, Portugal, South Africa, Uruguay, the USA, Venezuela, and Zimbabwe (Cho and Shin 2004; de Sousa Dias and Lucas 1980; Elad et al. 2014; Farr and Rossman 2016: Ginns 1986; Gorter 1977; Holevas et al. 2000; Lindquist 1982; Mujica and Oehrens 1967; Nattrass 1961; Pennycook 1989; Shivas 1989; Whiteside 1966; Zhuang 2005b). The disease causes a loss of aesthetic quality as well as a reduction in plant vigor (Ferrin and Rhode 1991).

Symptoms/signs. The first signs on leaves, stems, or flower buds are small, slightly raised blisters that eventually rupture, forming pustules filled with powdery reddish-brown urediospores. The pustules are surrounded by a yellow margin (symptom) (Fig. 12), and when infections are severe, entire leaves turn yellow and die. Stems may be girdled when several pustules develop around the shoot. The blackish-brown teliospores also form on stems, partially covered by epidermis. Flower production and quality is decreased. Plants may be attacked at any stage of development (Nelson 1960).



Fig. 12 Carnation rust (*left*). Detail of pustules of urediospores on leaves (*center*) and erumpent teliospores on stem (*right*) (S. Nakkeeran et al. Tamil Nadu Agricultural University, Coimbatore, Tamil Nadu, India © 2017. All Rights Reserved.)

Biology and epidemiology. Urediospores of this fungus are disseminated by wind, splashing water, or infected cuttings (Nelson 1960). Maximum urediospore germination of *U. dianthi* occurs at 10 °C (60.5%) and is minimal at 30 °C (27%). Disease is most severe at 15 °C, is minimal at 25 °C, does not occur at 30 °C (Polek 1993), and is favored by cool nights alternating with warm humid days that induces dew at night and the formation of a film of water on the leaf surface. Rust spores require free water for 9–12 h on the plant surfaces to germinate and infect. So this disease is most severe in open air culture and in plastic film greenhouses, where dew formation is common (Aloj and Garibaldi 1977). The pathogen enters the leaves through the stomates and grows between the host cells upon which it feeds (Ferrin and Rhode 1991). It is a heteroecious rust (pathogen with several growing stages on hosts in different families); the aecial stage is formed on *Euphorbia gerardiana* and *E. nicaeensis* (Lindquist 1982).

Management

- ***Cultural practices and sanitary methods*** – Rust spores require surface moisture to germinate and infect host plants. Reducing humidity and leaf wetness (e.g., avoiding overhead irrigation, spacing plants to lower humidity and taking advantage of prevailing winds, and using fans in greenhouses) will reduce the number of spores that successfully penetrate plant tissues.
- In nurseries or greenhouses, carefully remove rust-infected leaves and other debris, and discard or isolate infected plants. Place diseased plant material in plastic bags to remove them from the greenhouse; destroy the refuse by burning, burying, or rapid composting. At the end of the production cycle, clean up debris, and thoroughly clean and surface disinfect greenhouse benches and propagation areas using a commercially available product.
- ***Removal the alternate host*** – For heteroecious rusts, removal of the alternate host may help to disrupt the disease cycle. In most cases, it may be impossible to

completely eradicate alternate host plants from the vicinity of economically important hosts, but this tactic may remove a good deal of inoculum.

- ***Biological control*** – *Verticillium lecanii* (Zimm.) Viègas was found infecting uredia of *U. dianthi*, in a nursery. In the laboratory, *U. dianthi* was inoculated on to carnation leaf pieces, causing typical rust symptoms. In the presence of *V. lecanii*, either rust infection was prevented or the formation of urediospores was arrested, depending on the time of application of *V. lecanii* conidia. In a glasshouse, *V. lecanii* applied at the appropriate time reduced rust in carnation plants inoculated with *U. dianthi* (Spencer 1980).
- ***Fungicides*** – Dithiocarbamate sprays applied at weekly intervals prevented the buildup of the disease and gave better control than fortnightly sprays of a systemic material, oxycarboxin, or tetrachloroisophthalonitrile (Hill 1974). On the other hand, Hilal and Kamel (1990) found that rust was best controlled by bitertanol, while oxycarboxin and pyracarbolid were also effective. Under laboratory conditions, zineb, oxycarboxin, and dichlofluanid were the most efficient inhibitors of urediospore germination (Cifuentes and Arbeláez 1984). In the greenhouse trials, oxycarboxin appeared to be the best fungicide followed by triforine, bitertanol, dichlofluanid, zineb, and pyracarbolid. The fungicides oxycarboxin and triforine applied to the soil were significantly better than a foliar spray. The fungicides myclobutanil, propiconazole, and tebuconazole were tested both in the field and in a controlled environment for efficacy in controlling carnation rust. Myclobutanil and propiconazole provided exceptional control, whereas tebuconazole did not provide adequate control. Neem oil, a naturally produced fungicide, was included in the field trials only and provided moderate control (Polek 1993). Also fludioxonil, mancozeb, and sulfur are recommended (Gould 2012).

2.14 Sclerotium Soil Line Rot; Sclerotium Stem Rot, Southern Blight [*Sclerotium rolfsii* Sacc. (Teleomorph: *Athelia rolfsii* (Curzi) C.C. Tu and Kimbr.)]

Geographic occurrence and impact. This disease has been reported in Chile, Italy, Israel, South Africa, and Uganda (Andreucci and Andreucci 1955; Elad et al. 2014; Crous et al. 2000; Mosella and Verdugo 1984; Small 1920). Farr and Rossman (2016) included reports from Argentina, Australia, Brazil, Kenya, Mexico, New Zealand, Thailand, Uruguay, and Zimbabwe.

Symptoms/signs. *S. rolfsii* infects carnation stems at the soil line and may also infect plant roots. The infected basal stem becomes dark brown, and the xylem vessels of diseased plants are completely disorganized with a soft consistency. These symptoms are extended a few centimeters above and below ground level, completely affecting the root system. Lower leaves turn yellow and wilt first. The fungus produces characteristic cottony white and fibrous mycelia on infected tissues, plant debris, and the soil surface around the plant (sign). After few days, under hot humid conditions, abundant small (mustard seed size), round sclerotia creamy white at first and brown when mature

(signs) are produced. The disease in the infested plots is confined to a well-defined area showing patches of chlorotic and wilted plants that finally die.

Biology and epidemiology. Under favorable conditions, the pathogen rapidly produces infective mycelia and sclerotia. The disease is favored by a warm climate, moist soil, and poor drainage. High temperatures and humidity seem to be favorable to germination of the sclerotia and development of mycelia. The fungus has a wide host range and persists in soil, in plant debris, and in many weeds or cultivated plants. In winter, sclerotia remain in soil and plant disease debris, and during warm periods, they can germinate and infect new plants.

Management

- ***Cultural practices*** – Avoid dense plantations and excessive irrigation; eliminate weeds and crop residues; remove infected plants and soil surrounding the roots.
- ***Fungicides*** – The recommended products are azoxystrobin, fludioxonil, and PCNB (Gould 2012).

2.15 Sclerotinia Stem Rot; White Mold; Sclerotinia Flower Rot (*Sclerotinia sclerotiorum* (Lib.) de Bary)

Geographic occurrence and impact. The disease has been reported in Argentina, Colombia**,** Israel, India, Japan, and Korea (Anonymous 2012; Arbeláez-Torres 1987; Cho and Shin 2004; Elad et al. 2014; Vinod Kumar et al. 2015; Wolcan et al. 1999) but is occasionally present in Colombia (Arbeláez-Torres 1987) and in India was reported as highly destructive causing about 38.5% of stem rot incidence in five different locations (Vinod Kumar et al. 2015).

Symptoms/signs. Symptoms are paleness of the whole plant (Fig. 13 left) as a consequence of rot at the base of the stem. If infection occurs through ascospores that reach the highest parts of the plant via air currents, symptoms are bleaching of portions of the stem and leaves emerging from the infected nodes and also of the buds (Anonymous 2012). A sign of the disease is a cottony, white, dense mat of mycelial growth on the surface of the host and on the adjacent soil surface. The mycelium develops dense white bodies which become black, irregular, and hard when mature. These bodies are the sclerotia, which can be produced outside or inside the stem (Fig. 13 right).

Biology and epidemiology. The development of white mold is dependent on soil inoculum, high soil moisture, poor soil drainage, high plant density, and low temperature. Disease initiation is favored by cool and damp soil conditions. Sclerotia resist adverse environmental conditions. When these are conducive for the disease, the sclerotia germinate producing mycelia or apothecia (sexual fruiting body) that contain ascospores. These spores are airborne and spread the disease. Both mycelia and ascospores can initiate the infection.



Fig. 13 *Sclerotinia sclerotiorum*. Infected plant (*left*) and sclerotia formed inside the stem (*right*) (S. Nakkeeran et al. Tamil Nadu Agricultural University, Coimbatore, Tamil Nadu, India © 2017. All Rights Reserved.)

Management

- ***Cultural practices*** – Avoid dense plantations and excessive irrigation; eliminate weeds and crop residues; remove infected plants and soil surrounding the roots.
- ***Fungicides*** – The recommended products are chlorothalonil, copper hydroxide, mancozeb, and PCNB (Gould 2012).

2.16 Septoria Leaf Spot; Carnation Leaf Spot; Common Yellow Leaf Spot [(*Septoria dianthi* Desm = *Septoria spergulae* Westend.) Current Name *Caryophylloseptoria spergulae* (Westendorp) Verkley, Quaedvlieg and Crous (Verkley et al. 2013)]

Geographic occurrence and impact. The disease has been reported in Argentina, Australia, Brazil, Chile, Guatemala, India, Italy, Kenya, the Malay Peninsula, Mexico, New Zealand, Nicaragua, Portugal, South Africa, Uruguay, the USA (Hawaii), and Venezuela (Cook and Dubé 1989; de Sousa Dias and Lucas 1974; Farr and Rossman 2016; French 1989; Gorter 1977; Marchionatto 1950; Mendes da Silva et al. 1998; Mujica and Oehrens 1967; Nattrass 1961; Pennycook 1989; Raabe et al. 1981; Thompson and Johnston 1953).

Symptoms/signs. Light-brown spots with purple margins appear on leaves and stems. Small black specks are present at the center of the spots. These are the

spore-producing structures of the fungus (sign). Individual lesions may enlarge and coalesce with adjacent lesions to cause death of the leaf (Forsberg 1963). The symptoms are similar to leaf spot caused by *Alternaria dianthi* but tend to be found in the lower leaves (O'Neill 2008).

Biology and epidemiology. This disease is usually introduced with infected cuttings. Dissemination of the spores is by windblown rain and splashing water. High relative humidity favors the development of the disease and production of spores (Forsberg 1963).

Management

- ***Cultural practices*** – Control can be achieved by avoiding high humidity or wetting of foliage.
- ***Fungicides*** – The fungicides carbendazim and chlorothalonil should give control (Alford 2008). Also fludioxonil, kresoxim-methyl, and mancozeb are recommended (Gould 2012).

2.17 Verticillium Wilt [*Phialophora cinerescens* (Wollenweber) Van Beyma (syn. *Verticillium cinerescens* Wollenw.)]

Geographic occurrence and impact. This disease has been reported in Canada, China, Colombia, Denmark, England, France, Greece, New Zealand, Russia, Scotland, the Netherlands, and the USA (Arbelaez Torres 1987; Farr and Rossman 2016; Hellmers 1958; Sparnaaij and Demmink 1976; Voronenko et al. 1978). Damage caused by this fungus was especially severe until 1970, but a change in the method of cultivation of carnations and the introduction of more resistant cultivars lessened the damage for several years.

Symptoms/signs. Symptomatology is comparable to Fusarium wilt, and it is also caused by systemic vascular occlusion (Sharma and Sharma 2008). Initially symptoms include chlorosis of lower leaves which may have reddish pigmentation. Then the whole plant is affected. Plants wilt progressively, starting at the base and extending upward, and their leaves fade. This disease causes a brown discoloration of the vascular system of the stem and darkening of the plant base (Voronenko et al. 1978; Baker 1980).

Biology and epidemiology. The disease is transmitted through infected cuttings, soil, and contaminated tools (Arbeláez-Torres 1993). *P. cinerescens* is soilborne and survives saprophytically for several years and by means of resting structures in the absence of its host (Goicoechea 2009). When it comes into contact with the carnation plant, hyphae penetrate the root tips or through wounds and then colonize the vascular tissue. The pathogen invades the vessels and causes changes in the structure of living cells, simultaneously blocking the diseased vessels with polysaccharides and phenolic compounds produced by the host (Sharma and Sharma 2008). The optimum temperature for mycelial growth is between 10°C and 15°C and 18–23°C for sporulation and spore germination (Holley and Baker 1991).

Verticillium species are currently most often diagnosed by morphological examination using light microscopy or through DNA-based identification by sequencing of the ITS region followed by a blast search at GenBank (Inderbitzin and Subbarao 2014).

Management. The measures used to control this disease are basically the same as used in the control of *Fod*. However, there has been greater efficiency in controlling this disease (Guzmán et al. 1986).

- ***Cultural practices*** – Crop rotation has been used to reduce propagules that remain in the soil and may infect susceptible plants. However, the efficacy of this management strategy may be reduced because some propagules of *Verticillium* sp. can persist for long periods on plant debris and by infection of other crops and weed species (Goicoechea 2009). For that reason, removal of plant debris and elimination of weeds within and outside greenhouses should be applied.
  Soil amendment with organic animal or plant material has been used to reduce various plant diseases, including *Phialophora*-induced wilts (Cook and Baker 1983). "Culture indexing" to obtain pathogen-free propagative material is a very effective strategy (Baker 1980).
- ***Solarization*** – Soil solarization using clear plastic mulch is a common preplant practice in current agricultural systems in conducive climates. Alternative chemical methods for control, such as biofumigation, can increase the effectiveness of solarization under appropriate conditions.
- ***Resistance*** – A positive correlation between resistance to *P. cinerescens* and race 2 of *Fod* has been reported, suggesting that resistance may involve common mechanisms. Resistance to *P. cinerescens* is governed by at least two different gene pairs (Sparnaaij and Demmink 1976, 1977).
- ***Fungicides*** – Fumigation with methyl bromide prior to planting in order to reduce soilborne pathogens including *P. cinerescens* is very effective. Nevertheless, this compound has been categorized as a Class 1 ozone depleter and decreases the diversity of the microflora leading to extensive crop losses if pathogens are reintroduced to treated soil. This situation has led to a search for alternatives (Goicoechea 2009). The correct application of fungicides is complicated due to the wide range of alternative host plants of *P. cinerescens*. However, systemic fungicides were found to be useful in controlling the vascular wilt pathogens of carnation (Baker 1980); the 2-substituted benzimidazoles are effective in controlling Verticillium wilt. The fungicide must be repeatedly applied to the roots via soil drenches to suppress the pathogen.

Additional fungal diseases. The following fungal diseases of carnation have also been reported:

– **Anthracnose** [*Colletotrichum dianthi* (Westend.) B. Sutton] in Chile (Mujica and Vergara 1945)
– **Bud rot** [*Fusarium poae* (Peck) Woll.]; *Fusarium sporotrichioides* Sherb. in Germany, New South Wales, and the USA (Cooper 1940; White 1929)

- **Cercospora leaf spot** [*Cercospora dianthi* Muller and Chupp; currently *Cercospora apii* s. lat.] in Brazil, Cuba, and El Salvador (Farr and Rossman 2016)
- **Petal blight** (*Stemphylium floridanum* Hannon and G.F. Weber) in the USA (Alfieri et al. 1984; Hobbs 1963)
- **Phoma leaf spot** [*Phoma caryophylli* (Cooke); *Phoma dianthi* Sacc. and Malbr] in South America and the USA (Farr and Rossman 2016)
- **Phyllosticta leaf spot** (*Phyllosticta dianthi* Westend. 1857) in Brazil, South Africa, and Zimbabwe (Crous et al. 2000; Farr and Rossman 2016)

3 Bacterial and Phytoplasma Diseases

3.1 Bacterial Leaf Spot of Carnation [*Burkholderia andropogonis* (Smith 1911)]

Geographic occurrence and impact. *Burkholderia andropogonis* is widely distributed in Africa, Asia, Europe, Australasia, Oceania, and North and South America (Bradbury 1986). However, some strains of *B. andropogonis*, which appear to be specialized to particular hosts, are of limited distribution (Fahy and Persley 1983).

Symptoms/signs. *Burkholderia andropogonis* primarily affects parenchymatous tissue causing leaf spots, streaks, and stripes on a variety of hosts. Symptoms depend on the host which are mainly leguminous, gramineous, and ornamentals (Fahy and Persley 1983). On carnation, diseased leaves and stems show elliptical, pale-brown lesions, 5–10 mm in length with outer margins of water-soaked tissue (Fig. 14). Lesions on older leaves and stems have purple concentric zonations. The individual lesions often coalesce, and the lower leaves and stems become straw colored and withered. Young leaves and flower buds have water-soaked lesions on their tips (Shanks and Hale 1984).

Biology and epidemiology. *Burkholderia andropogonis* (formerly *Pseudomonas andropogonis*, *P. woodsii*, and *Phytomonas andropogonis*) has been reported infecting parenchymatous tissue causing leaf spots, streaks, and stripes on a variety of hosts (Fahy and Persley 1983). In ornamentals, it has been reported in *Bougainvillea* sp., *Strelitzia* sp., *Tradescantia* sp., and *Tulipa gesneriana* (Bradbury 1986) and also as an important pathogen of carnation (Smith 1911). Diagnosis is based on disease symptoms and isolation of the pathogen in semi-selective media. A PCR assay has been developed to detect the pathogen (Bagsic et al. 1995).

Management

- ***Chemicals*** – Using copper-based compounds is not feasible because they interfere with stomatal closing at night. Application of fosetyl-al at <7-days intervals provides adequate disease control (Trujillo and Nagata 1994).
- ***Resistance*** – Sixty-six carnation cultivars were screened for resistance to bacterial leaf spot in the USA and grouped into partially resistant, susceptible, and highly



Fig. 14 Bacterial leaf spot symptoms on leaves and flowers (J.D. Janse, Plant Protection Service, Wageningen, The Netherlands © 2017. All Rights Reserved, Retrieved from: www.atlasplantpathogenicbacteria.it)

susceptible. The partially resistant cultivars were Blaze, Danilo, Univ. Com. Sim 1, Cal Red, Improved New Pin Sim, Crowley Pink, Dusty, and Cal Improved White, with Cal Red and Cal Improved White the most resistant (Trujillo and Nagata 1994).

3.2 Bacterial Slow Wilt of Carnation, Also Named Bacterial Stunt of Carnation [*Dickeya dianthicola* (Formerly *Erwinia chrysanthemi* pv. *dianthicola*, *Pectobacterium carotovorum* pv. *dianthicola*, *E. carotovora* f. sp. *dianthicola*, *E. partheni* var. *dianthicola*, pv. *dianthi*)]

Geographic occurrence and impact. *Dickeya dianthicola* has been reported in Belgium, Denmark, France, Germany, Greece, Italy, Japan, the Netherlands, New Zealand, Norway, Poland, Romania, Sweden, Switzerland, the UK, and the USA (Bradbury 1986).

Symptoms/signs. Slow wilt symptoms include stunting and wilting. The disease is termed slow wilt as symptom development is often compared with that caused by *Burkholderia caryophylli*, the causal agent of bacterial wilt. *D. dianthicola* attacks the vascular tissue of the stem, often blocking the stem and causing cavities within it.



Fig. 15 Symptoms of slow wilt: Wilting (*left*) – vessels within the stem showing a brownish-yellow discoloration (*right*) (T. Saito, Minamiizu Experiment Farm, Shizuoka, Japan © 2017. All Rights Reserved, Retrieved from: www.atlasplantpathogenicbacteria.it)

With severe infections, vessels within the stem show a brownish-yellow discoloration (Fig. 15 left), excessive slime formation, and rotting at the base, together with a general wilting of the plant (Fig. 15 right). While minor cracks may appear at the stem surface, little bacterial slime appears to leak from the plant, thereby reducing the chances of bacterial transfer to neighboring plants. However, in severe infections, roots may fail to form, and in older plants, roots can completely rot, and this may lead to spread of the bacteria to neighboring plants via the soil. The disease tends to spread more readily in older plants, resulting in large gaps of 1–10 m in propagation beds (Hellmers 1958). In less severe cases, roots and shoots may continue to form, but young shoots become short and thick and leaves narrow (hence the alternative name for the disease, bacterial stunt). Roots of younger infected plants remain largely healthy, but brown discoloration can be found in the xylem vessels when the stem is cut.

Biology and epidemiology. Other hosts in which *D. dianthicola* has been shown to cause disease include potato (*Solanum tuberosum*), tomato (*Lycopersicon esculentum*), chicory (*Cichorium intybus*), begonia, *Chrysanthemum*, *Dahlia* spp. (including *Dahlia pinnata* and *D. variabilis*), artichoke (*Cynara scolymus*), *Kalanchoe* spp., *Hyacinthus* sp., and sedum (EFSA 2013). *D. dianthicola* is a vascular bacterium. Disease development is optimal at 25–27 °C, but the bacterium can grow at as low as 10–15 °C (Hellmers 1958). Secondary infection in transplanting beds may lead to sudden wilting and rotting at the stem base, presumably due to infection of roots through soilborne inoculum. In severe infections, plants often rot and die before cuttings are taken. However, in less severe cases (symptomless plants or those showing some stunting), bacteria may reach the upper parts of the plant and be transferred via cuttings. The bacterium can be identified by isolation in

semi-selective culture agar media followed by serological and biochemical analyses, bioassays, and immunological techniques (Czajkowski et al. 2015). In addition, PCR primer sets were developed to differentiate species of *Dickeya* (Pritchard et al. 2013).

Management

- ***Sanitation*** – Being a vascular bacterium, *D. dianthicola* has a potential latent infection stage, and as such, it is very efficiently transmitted by all vegetative multiplication techniques, emphasizing the importance of pathogen-free plants in the production system. There is no effective curative treatment that can be applied to *D. dianthicola*-infected plants in a production context; thus, it is best controlled through high levels of hygiene.
- ***Cultural practices*** – Grow plants in raised beds pasteurized by steam between crops. Use culture-indexed cuttings free of the pathogen. Destroy infected plants.

3.3 Burkholderia Wilt, also named Bacterial Wilt and Stem Cracking of Carnation [*Burkholderia caryophylli*]

Geographic occurrence and impact. The disease has been reported in Argentina, Austria, Belgium, Brazil, Colombia, China, Hungary, India, Italy, Japan, Norway, Poland, Sweden, Taiwan, Uruguay, Yugoslavia (Serbia and Montenegro), and the USA (Bradbury 1986; CMI 1976). In the past, it was found in Denmark, France, Germany, Ireland, the Netherlands, and the UK (EPPO/CABI 2015), but the disease did not establish there.

Symptoms/signs. Infected carnation plants develop grayish-green leaves before they turn yellow and die. Vascular discoloration and root rotting can also be observed (Saddler 1994). Under cool conditions and low soil temperatures, the carnation stems crack longitudinally between the nodes on the lower part of the stem. The outer layer (epidermis) separates easily from stem, which is sticky to the touch. Stem bases may show internodal cracking, developing into cankers (Fig. 16). Cutting diseased stems often reveals a brownish-yellow ooze (sign) (OEPP/EPPO 2006). Wilting symptoms are found in crops grown at high temperatures (>30 °C), whereas stem-cracking symptoms are more common at lower temperatures (<20 °C) (OEPP/EPPO 2006). Once wilting occurs, roots of infected plants are more or less rotten, the plants are easily pulled out of the soil, and, when cut, roots show discontinuous brown spots. This distinguishes Burkholderia wilt from that caused by *Phialophora cinerescens* which leaves the roots apparently symptomless (EPPO/CABI 2015). Plants may survive for 1–2 months, but invasion of secondary fungi, such as *Fusarium* spp., accelerates death. Severely infected cuttings wilt and die before roots form.

Biology and epidemiology. *Burkholderia caryophylli* (formerly *Pseudomonas caryophylli* and *Phytomonas caryophylli)* also causes rot in statice (*Limonium sinuatum)* and onion (Ballard et al. 1970; Jones and Engelhard 1984) and wilt of Russell prairie gentian (*Eustoma grandiflorum*) (Furuya et al. 2000) and baby's



Fig. 16 Symptoms of bacterial wilt and stem cracking (M. Scortichini, C.R. E.A.-Centro di Ricerca per la Frutticoltura, Roma, Italy © 2017. All Rights Reserved, Retrieved from: www.atlasplantpathogenicbacteria.it)

breath (*Gypsophila paniculata*). The bacterium enters plants through wounds and subsequently colonizes the vascular system of the stem and roots. The main source of infection is by the movement of infected cuttings taken from mother plants with latent infections. Infection can also occur from contaminated tools and water (Saddler 1994). Bacteria can pass from one cutting to another in the water of the propagating bed or, if the cuttings are held in water, before planting out. Bacterial slime is exposed when stems crack, and this inoculum may be transferred from one plant to another. Temperatures over 20 °C accelerate bacterial growth and, therefore, symptom expression, while at low temperatures, infected plants may show no symptoms (OEPP/EPPO 1978). The bacterium can be reliably detected by immunofluorescence staining (IFAS) and direct isolation on sorbitol neutral red agar (SNR) even in material with latent infection (Muratore et al. 1986). In addition, a real-time fluorescent PCR assay for specific detection of the pathogen has been developed (Shao et al. 2011).

Management

- ***Cultural and sanitary practices*** – Management of the disease is limited. Rapidly remove infected plants. Use disease-free cuttings. Avoid using cutting dips and avoid splashing water. Break rather than cut cuttings from stock plants. Disinfect tools. As the pathogen is soilborne, it is suggested to disinfect soil by steam or using fumigants such as chloropicrin, metam sodium, or metam potassium. Avoid overhead watering.

- ***Resistance*** – Two hundred and seventy seven carnation cultivars were screened for resistance to bacterial wilt in Japan. Most cultivars (74.7%) were highly susceptible, whereas three cultivars, Wiko, Nocto, and Sandrosa, possessed adequate resistance (Onozaki et al. 1999). Furthermore, Onozaki and coworkers (2002) developed a carnation breeding line called Carnation Nou No. 1 derived from an interspecific cross between carnation cultivar, Super Gold, and the highly resistant wild species, *Dianthus capitatus.* It has been shown that Carnation Nou No. 1 has the ability to produce resistant progeny and has a perpetual flowering habit.

3.4 Crown Gall (*Agrobacterium tumefaciens*, *Pseudomonas tumefaciens*, *Bacillus tumefaciens*, and *Phytomonas tumefaciens*)

Geographic occurrence and impact. Crown gall is one of the most widely distributed bacterial diseases and has been reported from all five continents (Hayward and Waterston 1965; CMI 1976).

Symptoms/signs. Crown gall tumors are observed most often at the base of stems and below ground on major roots. Once initiated, tumors continue to grow into large, friable masses that dissociate into small pieces as the tumor ages and dries (Kado 2010).

Biology and epidemiology. *Agrobacterium tumefaciens* is a soilborne bacterium that infects dicotyledonous plants from over 90 different plant families. Crown gall tumors are the culmination of *A. tumefaciens* plasmid-mediated transformation of cells surrounding the infection site. Infection sites where bacteria proliferate are established mainly through injuries caused by mechanical trauma, grafting, freezing temperatures, and root-invading insects. High moisture levels favor infections leading to transformation which take place in less than 3 h. Transformed cells proliferate into visible abnormal tumors. As the tumor grows, *A. tumefaciens* is shed into the surrounding soil, particularly when rain or sprinkler irrigation occurs (Kado 2010). *A. tumefaciens* naturally resides on the rhizoplane of woody and herbaceous weeds. Its presence in soils originates from galls that were broken or sloughed off from infected plants during cultivation practices or disseminated as infected plant material. Irrigation aids in further dissemination of *A. tumefaciens*. The pathogen is also spread by infected and infested planting material originating as nursery stock from uncertified sources. Diagnosis is based on isolation in selective and semi-selective media (Schaad et al. 2001) and bioassays and specific detection by PCR (Haas et al. 1995).

Management

- ***Cultural practices*** – A crop rotation program employing cereal crops followed by green manuring helps reduce the population size of *A. tumefaciens*. Fields that have grown cereal crops for a long period are favored as crown gall-free sites.

Fields previously used for growing fruit and nut crops can remain infested with *A. tumefaciens*. Certain weeds such as morning glory (*Ipomoea leptophylla*) can serve as natural hosts of *A. tumefaciens* and therefore perpetuate the survival of this pathogen in field soils (Kado 2002).

- ***Biological control*** – Nursery plants and transplants can be protected from crown gall by treating the seed, seedlings, or cuttings with commercial biological control agents such as *Agrobacterium radiobacter* strain K84 (New and Kerr 1972) or the genetically modified strain K1026 (Jones et al. 1988).

3.5 Fasciation [*Rhodococcus fascians* Formerly *Corynebacterium fascians*, *Bacterium fascians*, *Pseudobacterium fasciens*, and *Phytomonas fascians*], Also Called Leafy Gall

Geographic occurrence and impact. The disease is most serious on bulb, floral, and greenhouse crops. *R. fascians* is widely distributed and has been reported in Australia, Belgium, Canada, former Czechoslovakia, Denmark, Egypt, France, Germany, Hungary, Iran, Mexico, the Netherlands, New Zealand, Sweden, Russia, the UK, and the USA (Bradbury 1986; Putnam and Miller 2007).

Symptoms/signs. Fasciation is characterized by the excessive and abnormal proliferation of leaves, flowers, and shoots, including gall formation on leaves and at wounds on the stems on the plant host as a result of hyperplastic growth of the affected tissues (Kado 2010). Wounding of the host is not essential for infection; hence symptoms can be induced when the bacteria are present on the surface of healthy tissue. Nevertheless, when wounding does occur, symptoms are more severe and develop more rapidly. The continuous presence of the bacteria is required for symptom persistence, because they provide a continuous stimulus leading to progressive growth of the leafy gall. Symptoms vary depending on the host and the environment under which the host is grown. Multiple dwarfed flowers, leaves, and stems originating from a single meristematic region or a wound site are typical of fasciation in carnation.

Biology and epidemiology. In addition to carnation, *Rhodococcus fascians* has been isolated from a broad range of floral crops, including chrysanthemum (*Chrysanthemum morifolium*), jade plant (*Crassula* sp.), geranium (*Pelargonium* x *hortorum*), gypsophila, wallflower (*Cheiranthus cheiri*), dahlia (*Dahlia variabilis)*, primrose (*Primula* sp.), gladiolus (*Gladiolus* sp.), coral bells (*Heuchera* sp.), mullein (*Verbascum* sp.), petunia (*Petunia* sp.), stock (*Matthiola* sp.), stork's bill (*Pelargonium* sp.), snapdragon (*Antirrhinum majus)*, lily (*Lilium regale*), phlox (*Phlox* sp.), azalea (*Rhododendron* sp.), and polyanthus (*Primula* sp.). Virulent isolates of *R. fascians* pose a large conjugative plasmid that bears at least three loci required for pathogenicity (Putnam and Miller 2007). *R. fascians* grows epiphytically on aerial plant surfaces, where it is protected by a slime layer. The surface bacteria produce signals that initiate symptoms development including the novo meristem generation, or activation of existing meristems, resulting in a production

of a leafy gall. The pathogen is disseminated by splashing water in the form of rain or sprinkler irrigation. Moisture abundance is important for dissemination. Regions of low humidity with semiarid conditions are not conductive to the disease. The disease is seed-borne and the pathogen survives in soil and propagation material. The pathogen colonizes intercellular spaces through wounds (Cornelis et al. 2001). Growers who do not recognized that their plants are diseased risk propagating the bacterium along with their cuttings. The situation is complicated by the ability of the bacterium to persist on plant surfaces for many months prior to the production of symptoms (Putnam and Miller 2007). Diagnosis is made difficult by the similarly of symptoms caused by *R. fascians* and those produced by application of growth hormones which are commonly used in production of herbaceous ornamentals. Diagnosis must be confirmed by isolation of *R. fascians* and inoculation to a susceptible host to confirm pathogenicity. Both LAMP (loop-mediated isothermal amplification) and PCR methods have been recently developed for specific and rapid detection of *R. fascians* (Serdani et al. 2013).

Management

- ***Sanitation*** – Control bacterial fasciation primarily through good sanitation and use of pathogen-free plants.
- ***Cultural practices*** – Cultural control and plant hygiene are the main control methods. Systematic disinfection of greenhouses, materials, and protective clothing is essential. Disinfect cutting tools between crops. Use quaternary ammonium-based disinfectants on hard surfaces such as greenhouse pots, flats, benches, floors, and other structures, and use sterilized potting mixes. Avoid injuring the base of plants, especially when plants are wet. Keep the base of plants dry. Remove and dispose of infected plants, or prune and dispose of distorted tissue and do not propagate from those plants.
- ***Chemicals*** – Application of antibiotics that kill *R. fascians* results in remediation of the disease and normal outgrowth of shoots (Kado 2010). Control can also be attempted using copper containing chemicals**.**
- ***Integrative strategies*** – The role of insects in natural disease transmission is unknown, but under artificial conditions, aphids are capable of transmitting the disease (Putnam and Miller 2007), so it is recommended to reduce insect populations through chemical or biological means.

3.6 Phytoplasma Diseases

Aster Yellows [“*Candidatus* Phytoplasma asteris”]

Geographic occurrence and impact. Aster yellow disease of carnation has been reported in Canada (Northover 2007) and the USA (USDA 1960). The aster yellows phytoplasma has a very broad and extensive host range covering at least 60 families (Kado 2010) indicating a lack of specificity.

Symptoms/signs. Symptoms begin with a general yellowing of the foliage followed by growth distortions, such as the loss of apical dominance that results in shoot and floral proliferation into a witches' broom appearance. The yellowing leads to a general decline of the plant, culminating in death. Pigment expression is also altered by infection with the aster yellows phytoplasma. Phyllody and alteration of tissue pigments (purple, yellow, red, and bronze) have been observed in a number of hosts (Kado 2010).

Biology and epidemiology. *Candidatus* phytoplasma species are members of the bacterial class Mollicutes which are Gram-negative pleomorphic prokaryotes without cell walls. Currently, they are not cultivable in vitro and are considered obligate parasites that reside in the plant phloem. Phytoplasmas are able to replicate in the plant host and in the insects that vector them. In the USA and Canada, *C.* phytoplasma is vectored by the aster leafhopper or six-spotted leaf hopper (*Macrosteles quadrilineatus* = *M. fascifrons*).

Management. Management of phytoplasma diseases is based on monitoring and reduction of insect vector populations through chemical, physical, or biological means. Once a plant is infected, there is no curative treatment. Crop rotation is ineffective for aster yellows management due to the wide host range of phytoplasmas. Reflective mulches can be used as an alternative management strategy to control insect vector populations in situations where chemicals create a hazard to either the grower or field workers. The use of fine mesh netting to exclude vectors is also recommended.

4 Viral Diseases

In general, transmission of viruses through the process of vegetative propagation is very common in florists' crops like carnation. Loemmel et al. (1983) reported percentages of viral infection near 78% in carnation crops. These infections reduce the quantity and quality of the production. Management consists in the elimination of the viruses by meristem-tip culture and by a combination of heat treatment and meristem-tip culture (AAP 2015; CABI 2015a). Additional information on integrated disease management of insect-vectored viruses may be found in the introductory (▶ Chap. 4, "Insect Management for Disease Control in Florists' Crops").

4.1 Carnation Etched Ring Virus (CERV, Genus: *Caulimovirus*)

Geographic occurrence and impact. Distributed worldwide (Lawson and Hearon 1977); reported from the UK (Hollings and Stone 1961).

Symptoms/signs. Only members of the Caryophyllaceae are infected. The main symptoms are necrotic flecks, rings, and line patterns on the leaves of carnation.

These necrotic leaf patterns may enlarge to form blotches without conspicuous chlorosis. More severe symptoms including necrotic leaf spots, chlorosis, rings, and stem streaks and flecks (Fig. 17; Hakkaart 1968; Hollings and Stone 1961) are associated with mixed infections of CERV and *Carnation mottle virus*. In addition, expression of CERV symptoms are intensified in mixed infections with unidentified viruses (Hollings et al. 1968).

Biology and epidemiology. CERV is transmitted by the aphid *Myzus persicae* to *Silene armeria* and *Saponaria vaccaria* (Smith and Lawson, unpublished data). It is not reported to be transmitted by seeds or dodder.

Management. Management is based on the elimination of the virus by meristem-tip culture and/or a combination of heat treatment and meristem-tip culture (Paludan 1970). Control of the vectors with aphicides is recommended.

4.2 Carnation Italian Ringspot Virus (CIRV, Genus: *Tombusvirus*)

Geographic occurrence and impact. CIRV is reported in Germany, Italy, the UK, and the USA (Buttner et al. 1987; Hollings et al. 1970; Martelli et al. 1971).

Symptoms/signs. Slight stunting and transient chlorotic spots and rings in leaves (Martelli et al. 1971).

Biology and epidemiology. CIRV is transmitted by mechanical inoculation and grafting (Martelli et al. 1971).

Management. CIRV can be eliminated by thermotherapy (Hollings et al. 1970). Use virus-free propagative material and appropriate sanitary practices during propagation activities.



Fig. 17 Carnation etched ring virus (E. Dal Bó, CIDEFI-UNLP-CIC, Facultad de Ciencias Agrarias y Forestales, UNLP, Argentina © 2017. All Rights Reserved.)

4.3 Carnation Latent Virus (CLV, Genus: *Carlavirus*)

Geographic occurrence and impact. *Carnation latent virus* (CLV) has been reported in many countries: Europe [France, Germany, Lithuania, Spain, the Ukraine, and the UK (England and Wales)], Asia [India (Himachal Pradesh), Japan (Honshu), and the Korean Republic], North America [Canada (British Columbia) and the USA (Colorado and New York)], South America [Argentina and Venezuela], and Oceania [Australia (Victoria)] (CABI 2007; Kassanis 1955; González et al. 1990; Lastra et al. 1980). This extensive distribution is probably due to inadvertent international exchange of infected plantlets and germplasm previous to the identification of this virus.

Symptoms/signs. CLV causes few or no symptoms in carnation. There are no symptoms reported in another ornamentals.

Biology and epidemiology. CLV is transmitted by plant sap and/or vegetative multiplication. It is transmissible in a non-circulative (nonpersistent) manner by the aphid *Myzus persicae* to carnation, Sweet William, and sugar beet (Kassanis 1955). Seed transmission is not reported.

Management. The virus is controlled by the production, propagation, and distribution of virus-tested stock plants, within the framework of certification schemes. Meristem-tip culture was shown to be effective in obtaining virus-free plants (González et al. 1990). It is possible to eliminate CLV from sap containing the virus by thermotherapy for 10 min at 60 °C (Kassanis 1955; Mangal et al. 2002). Control of *M. persicae* is important.

4.4 Carnation Mottle Virus (CarMV, Genus: *Carmovirus*)

Geographic occurrence and impact. CarMV is widespread wherever carnations are grown. Its natural host range seems to be restricted to species of Caryophyllaceae, but the virus can infect over 30 species within 15 dicotyledonous families. It was reported in Brazil, China, Chile, Finland, Indonesia, Iran, Russia, and Taiwan (Alexandre et al. 2015; Bremer 1978; Chen et al. 2003; Gallardo and Alvarez 1979; Safari et al. 2009; Diningsih et al. 2015; Zhang 1992; Zhola and Ushkaura 1983). This extensive distribution is probably due to inadvertent international exchange of infected plantlets and germplasm previous to the identification of this virus.

Symptoms/signs. CarMV produces mild mottle and loss of vigor in carnation (Fig. 18). In many cases, plants are symptomless, and the disease is only detected when infected plants are compared with healthy plants. In other cases, plants show mild flower distortion or color-break symptoms, but no obvious leaf symptoms. Finally, many plants can also show chlorosis/mottle and malformation of leaves,



Fig. 18 *Carnation mottle virus* infecting carnation plant (E. Diningsih et al. Indonesia Ornamental Crop Research Institute (*IOCRI*) & Bogor Agricultural University, Indonesia © 2017. All Rights Reserved.)

chlorotic streaks, and stunting. Symptoms vary according to carnation cultivars. Although the infection leads to mild symptoms, it weakens the plant to infection by other pathogens.

Biology and epidemiology. Transmission of CarMV occurs by plant sap or by grafting. There is no consensus among authors in terms of transmission. According to Kassanis (1955), the virus is transmissible in a non-circulative (nonpersistent) manner by the aphid *Myzus persicae* to carnation, Sweet William, and sugar beet. On the other hand, Hollings and Stone (1964) reported no transmission by *Myzus persicae* and *Macrosiphum euphorbiae*. Unconfirmed circulative (persistent) transmission by *Aphis gossypii* was reported from India (Singh and Singh 1989). No transmission by seeds was observed.

Management. CarMV is considered to be invasive because it is highly stable in vitro and highly infectious. CarMV is readily transmitted mechanically, by contact between infected and healthy plants but, more usually, with virus-contaminated hands, tools, and equipment during horticultural practices (Hollings and Stone 1964). The virus also occurs in fresh river and sea waters (Koenig and Lesemann 1985; Kontzog et al. 1988), and susceptible plants can also be infected if grown in moist soil or perlite containing the virus (Goethals et al. 1973). The virus is best controlled by the production, propagation, and distribution of virus-tested stock plants, within the framework of certification schemes. Virus-tested plants have been produced in many countries by meristem-tip culture (Stone 1968; Kowalska 1974; Rybalko and Kharuta 1978; Pena Iglesias et al. 1979; Devergne et al. 1982) and by meristem-tip culture combined with heat therapy (38 °C for 3–5 weeks) (Goethals and van Hoof 1971; Kowalska 1974). Resistance to CarMV has been obtained experimentally by transferring the virus coat protein gene to plants, but such genetically modified carnations have not yet been used commercially (Bae and Yu 2002).

4.5 Carnation Necrotic Fleck Virus (CNFV, Genus: *Closterovirus*)

Geographic occurrence and impact. CNFV is distributed worldwide: in Europe [Belgium, Croatia, the Czech Republic, Finland, France, Germany, Italy (mainland Italy), Latvia, Lithuania, the Netherlands, Slovakia, Spain (mainland Spain), and the UK], Asia [India (Himachal Pradesh), Israel, Japan (Honshu), the Korean Republic, Sri Lanka], North America [the USA (California)], South America (Colombia, Venezuela), and Oceania (Australia, New Zealand) (CABI 2009; Inouye and Mitsuhata 1973).

Symptoms/signs. Severely affected seedlings are stunted and killed, but most chronically infected plants are symptomless. CNFV produces grayish-white necrotic spots and flecks sometimes followed by reddish-purple discoloration of leaves. On *D. barbatus*, veinal chlorosis and necrosis appear on fully expanded young leaves 2–3 weeks after inoculation by aphids, often appearing as yellow net symptoms. Affected leaves eventually show reddish discoloration and tip necrosis. Leaves produced subsequently show symptoms only at the leaf tips, and leaves developing still later are almost symptomless. Symptoms are greatly increased by mixed infection with other viruses, particularly with CarMV (Inouye and Mitsuhata 1973; Smookler and Loebenstein 1974; Mayhew 1979).

Biology and epidemiology. CNFV affects a few species of Caryophyllaceae: *D. caryophyllus* and *D. barbatus* (Inouye and Mitsuhata 1973). It is transmitted by *Myzus persicae* in a semi-persistent manner (Inouye and Mitsuhata 1973). Transmission by seed or by dodder was not tested.

Management. The management of CNFV consists of heat treatment combined with meristem-tip culture, followed by careful scouting for early detection and elimination of infected plantlets (Loebenstein 2006).

4.6 Carnation Ringspot Virus (CRV, Genus: *Dianthovirus*)

Geographic occurrence and impact. CRV is found wherever carnations are grown and is spread by vegetative propagation in temperate regions (Bremer and Lahdenpera 1981; Loemmel et al. 1983). It has been isolated from apple, pear, and sour cherry in Germany (Kleinhempel et al. 1980; Richter et al. 1978).

Symptoms/signs. Infection in carnations and *Dianthus barbatus* results in leaf mottle, ringspots, stunting, and leaf and flower distortion (Sweet William), sometimes with leaf tip necrosis (Kowalska 1974). The flowers are distorted and not marketable. CRV has been associated with a stony pit disease of pears (Richter et al. 1978) and decline diseases of sour cherry, apple, and grapevines (Kleinhempel et al. 1980). The virus also occurs in the weed *Stellaria media* (Fritzsche et al. 1979; Kleinhempel et al. 1980; Kegler et al. 1983). The disease symptoms are enhanced when carnations are co-infected with CarMV (Kemp

1964; Hollings and Stone 1965). CRV infections do not kill the host plants, but necrosis and other symptoms can become more severe at sustained temperatures between 15 °C and 20 °C.

Biology and epidemiology. CRV is not transmitted by insects or soil-inhabiting fungi (Hiruki 1987). Transmission by adult nematodes like *Longidorus elongatus*, *L. macrosoma*, and Xiphinema diversicaudatum has been reported (Fritzsche and Schmelzer 1967), but the evidence supporting these claims is considered to be inadequate (Brown and Trudgill 1984). Spread in orchard soils without vector involvement is suspected (Tremaine and Dodds 1985).

Management. Selection, roguing, indexing, and meristem-tip culture are all effectively employed to ensure that stock plants are free of the virus (Rybalko and Kharuta 1978; Stone 1968). The virus can be readily eliminated from several hosts by heat treatment and also by meristem-tip culture (Rybalko and Kharuta 1978). The nearly worldwide establishment of a virus-free certified carnation propagation program has ensured that epidemics of this virus have not reoccurred (Ebbels 1979).

4.7 Carnation Vein Mottle Virus (CVMoV, Genus: *Potyvirus*)

Geographic occurrence and impact. CVMoV occurs wherever carnations are extensively grown; it is uncommon in greenhouse carnations in the UK (Kassanis 1955) and countries of N.W. Europe where diagnostic tests have been done, though commoner in S. Europe, and widespread in *Dianthus barbatus* in gardens in the UK (Hollings and Stone 1971). The virus also occurs in China, the USA, and Venezuela (Brierley and Smith 1957; Lastra et al. 1980; Li et al. 2014).

Symptoms/signs. The symptoms are similar to CarMV. In *Dianthus caryophyllus*, it causes diffuse chlorotic spotting and mottling, with spots and flecks of darker green on some of the veins of young leaves. Older leaves are usually symptomless. Symptoms may be very slight in some cultivars of the Sim group in winter, but more conspicuous in others and in seedling clones (Hollings and Stone 1971; Kassanis 1955). Flower yield may be decreased, and color break and distortion may occur, especially in summer (Brierley 1964; Hakkaart 1964). Symptoms are intensified when the plants are co-infected with CarMV.

Biology and epidemiology. Adults and larval stages of *Myzus persicae* transmit the virus in a non-circulative (nonpersistent) manner (Kassanis 1955; Hollings and Stone 1971). There is no evidence for virus multiplication within the vector *M. persicae* f. *dianthi* (= *M. polaris*) (Brierley and Smith 1957). No seed transmission was detected in *Dianthus barbatus* or *Chenopodium quinoa* (Hollings and Stone, unpublished). Moreover, *Cuscuta campestris* did not transmit the virus from *Chenopodium quinoa* to *C. quinoa* (Hollings and Stone, unpublished).

Management. Carnation plants can be freed from the virus with some difficulty by heat treatment (Brierley 1964), but readily by meristem-tip culture (Stone 1968).

4.8 Carnation Yellow Fleck Virus (CYFV, Genus: *Closterovirus*)

Geographic occurrence and impact. CYFV has been reported in Australia, Israel, Japan, and New Zealand (Inouye and Mitsuhata 1973; Sutton and Taylor 1971; Smookler and Loebenstein 1974).

Symptoms. CYFV causes yellow mottling, streaking, flecking, and necrotic discoloration of carnation leaves (Smookler and Loebenstein 1974).

Biology and epidemiology. The virus is transmitted by the aphid *Myzus persicae* (Smookler and Loebenstein 1974) in a semi-persistent manner. Transmission through seed or by dodder was not tested.

Management. The use of virus-free propagative material and aphid management is essential.

4.9 Additional Viral Diseases of Carnation

- *Cucumber mosaic virus* (CMV, genus: *Cucumovirus*) (Italy, Mexico, Yugoslavia Štefanac and Wrischer 1983; De La Torre-Almaráz et al. 2016; Lovisolo et al. 1968)
- *Impatiens necrotic spot virus* (INSV, genus: *Tospovirus*) (Iran, the USA (University of Massachusetts 2016; Shahraeen et al. 2002))
- *Tomato spotted wilt virus* (TSWV, genus *Tospovirus*) (Greece, Iran, Chatzivassiliou et al. 2000; Ghotbi et al. 2005)

5 Nematode Diseases

Carnation hosts a wide range of plant-parasitic nematode species. Some of them, such as *Meloidogyne incognita*, *M. arenaria*, and *M. javanica*, infect carnation among a long list of many other susceptible plant species. On the contrary, other species, such as *Heterodera trifolii*, *H. daverti*, and *Paratylenchus dianthi*, include carnation within a narrow list of susceptible species. Every trophic group of plant-parasitic nematode is represented in carnation-associated nematofauna.

5.1 Sedentary Endoparasitic Nematodes

Geographic occurrence and impact. Root-knot nematodes have been reported parasitizing carnation in almost every place in which the crop is cultivated. This

includes Australia, Bulgaria, Colombia, Costa Rica, Cyprus, Egypt, Greece, India, Iraq, Israel, Italy, Kenya, Korea, Portugal, New Zealand, Spain, and the USA (Arbeláez 1999; Cho et al. 1996; Katalan-Gateva and Milkova 1982; Khanna and Khan 1990; Kimenju et al. 2014; Knight et al. 1997; Koliopanos 1979; Lamberti et al. 1987; Levin 2005; López and Salazar 1988; Melero-Vara et al. 2012; Minz 1958; Mostafa Fatma et al. 2014; Nobre Maleita et al. 2012; Philis 1979; Singh and Majeed 1991; Stirling et al. 1992). On the other hand, cyst nematodes have been reported as carnation pests only in Australia, France, Italy, and the USA (Cuany and Dalmasso 1975; Del Sorbo et al. 2003; Stirling et al. 1992; Wang and Riggs 1999).

The economic importance of sedentary nematodes has not been completely assessed, but losses between 10% and 27% have been reported (Cho et al. 1996; Philis 1979; Ravichandra 2014).

Symptoms/signs. Aboveground symptoms result from the impairment in normal root physiology and include stunting, lack of vigor, moderate wilting, and nutrient deficiency (Trujillo et al. 1989). Roots infected by root-knot nematodes are usually characterized by the development of galls. Severe infections lead to reduction in flower number and size.

It has been demonstrated that infection by root-knot nematodes enhances synergistically the damage provoked by other pathogens, such as *Fusarium oxysporum* f. sp. *dianthi* (Schindler et al. 1961) and *Burkholderia caryophylli* (Stewart and Schindler 1956).

Biology and epidemiology. Five species of *Meloidogyne* (Tylenchida: Heteroderidae), including *M. javanica*, *M. arenaria*, *M. incognita*, *M. hapla*, and *M. hispanica*, have been reported parasitizing carnation (Di Vito 1979; Horst 2013; Knight et al. 1997; Nobre Maleita et al. 2012). *Heterodera trifolii* is the most important cyst nematode associated with carnation, even though two other nematodes of the genus, *H. daverti* and *H. schachtii*, can be found (Cuany and Dalmasso 1975; Del Sorbo et al. 2003; Wang and Riggs 1999). Both genera display a very specialized and evolved parasitism strategy. Root-knot and cyst nematodes present six developmental stages: eggs, four juvenile stages, and adults. This last phase is characterized by sexual dimorphism, with females displaying spherical or lemonlike shapes (Siddiqi 2000). Roots are infected by juveniles of the second stage (J2) that migrate inside the roots until reaching vascular cells. Successive molts take place within the root. After the last molt, the females adopt a sedentary habit and induce morphological changes in the host to establish permanent feeding sites (giant cells, in the case of root nematodes, and syncytia, in the case of cyst nematodes). *Meloidogyne* females lay a large number of eggs included in a gelatinous matrix secreted through the anus (egg masses) (Jepson 1987). On the other hand, *Heterodera* females retain the eggs inside their body as the external cuticle turns thicker and harder becoming a protective shield (cyst) (Baldwin and Mundo Ocampo 1991). Both cysts and egg masses represent the source of primary inoculum from which the next generation will start successive infections.

5.2 Migratory Endoparasitic Nematodes

Three migratory endoparasitic nematodes of the genus *Pratylenchus* have been mentioned infecting carnation. *P. pratensis* has been found in India (Khanna and Jeevan 1999), *P. coffeae* in Italy (Ambrogioni and Rapetti 1992), and *P. penetrans* in Bulgaria (Katalan-Gateva and Milkova 1982) and New Zealand (Knight et al. 1997). These nematodes are commonly known as "root-lesion nematodes." Some morphological adaptations (cephalic framework heavily sclerotized, robust stylet) allow these nematodes to penetrate the cell wall (Castillo and Vovlas 2007). They migrate intracellularly until reaching the endodermis, which constitutes a barrier for their advance (Thomason et al. 1976). The physical damage leads to the appearance of necrotic zones and detachment of cortical tissues (Zunke 1990). No particular research concerning either pathogenicity or impact has been carried out in carnation up to the present time.

5.3 Semi-Endoparasitic Nematodes

Carnation has been cited as a natural host for *Rotylenchulus macrodoratus*, a kidney-shaped semi-endoparasitic nematode that occurs only in the Mediterranean area (Inserra and Vovlas 1980). On the other hand, experimental observations carried out in Egypt showed that carnation is moderately susceptible to the "reniform nematode" *Rotylenchulus reniformis* (Mostafa Fatma et al. 2014).

5.4 Root Ectoparasitic Nematodes

Several species of ectoparasitic nematodes attack carnation roots in many crop locations worldwide. These species find their habitat in the rhizosphere and feed on epidermal or subepidermal cells making use of their long stylets. Their damage potential becomes apparent when reaching high population levels. Special importance within this trophic group is reserved for the "pin nematodes" of the genus *Paratylenchus*. *P. dianthi* is a key pest of carnation in South Italy (Pennacchio et al. 1985), while *P. curvitatus*, *P. hamatus*, and *P. projectus* are mentioned in other countries such as India and New Zealand (Ravichandra 2014; Khanna and Jyot 2002; Knight et al. 1997). Other ectoparasites such as *Helicotylenchus varicaudatus*, *Criconemella curvata*, *Mesocriconema* spp., and *Hoplolaimus* spp. infect carnation and may occasionally become of economic relevance (Anonymous 2006; Noel and Lownsbery 1984; Khanna and Jyot 2002).

5.5 Aerial Endoparasitic Nematodes

Here are included nematode species whose infecting forms inhabit the soil, though infection takes place after migration upward until reaching tissues located at the

ground level (*Ditylenchus* spp.) or above (*Aphelenchoides* spp.). Carnation is mentioned as host of *Ditylenchus dipsaci* and *Aphelenchoides fragariae* (CABI 2015b; Kohl 2011; Ravichandra 2014), though the economic impact of both is very limited.

Management. As occur with most crops, the main management strategy for pathogenic nematodes in carnation consists in reducing the initial inoculum by means of soil disinfestation. Chemical control resorts to nematicides classified as fumigants (i.e., gaseous or highly volatile nonspecific biocides used preplant) and nonfumigant nematicides (NFNs). Fumigants, such as 1,3-dichloropropene, dazomet, and non-fumigants, such as fenamiphos and carbofuran, have been tried for preplant control of nematodes in carnation (Melero-Vara et al. 2012; Nagesh and Parvatha Reddy 2005). The use of methyl bromide is discouraged, not only because of restrictions and environmental and job safety concerns, but also because of extreme sensitivity of carnation to bromine residues. More environmentally friendly approaches of soil disinfestation, such as solarization, soil amendments, and biological control, have been also assayed (Melero-Vara et al. 2012; Nagesh and Parvatha Reddy 2005). No cultivar with gene-to-gene resistance to root-knot nematodes is currently available. Nevertheless, the susceptibility of the most cultivars varies to a great extent, and some of them perform satisfactorily in heavily infested soils (Cho et al. 1996)

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Diseases of Celosia

14

Ann B. Gould

Abstract

Celosia (*Celosia* L.), generally known as cockscomb or woolflower, is a genus of ornamental and edible tender annuals in the Amaranthaceae. Ornamental cultivars are subject to few diseases, although fungal and bacterial leaf spots, Botrytis blight, Rhizoctonia root and stem rot, damping-off, and root-knot nematode may occur where humidity is high or where good growing practices, sanitation, and other cultural disease management practices that reduce inoculum and spread of propagules are not followed.

Keywords

Alternaria spp. • *Macrophomina phaseolina* • *Rhizoctonia solani* • *Meloidogyne* spp.

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R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_31

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1 Introduction

Celosia L., also known generally as celosia or as woolflower (plumed flowers), cockscomb (flowers crested by fasciation), or spiked (wheat) cockscomb, is a genus of ornamental and edible herbaceous tender annuals in the Amaranthaceae (pigweed family). Of tropical origin, the genus is thought to originate in Africa or Asia. Cultivated species include *C. argentea* L. (silver cockscomb) (preferred scientific name), *C. cristata* L. (crested cockscomb), *C. nitida* Vahl (West Indian cockscomb), *C. palmeri* S. Watson (Palmer's cockscomb), *C. trigyna* L. (woolflower), and *C. virgata* Jacq. (albahaca) (ITIS 2016). In the older literature and in trade, cockscombs may be listed as variants of *C. argentea* (e.g., *C. argentea* var. *argentea*, *C. argentea* var. *cristata*, *C. argentea* var. *plumosa*, *C. argentea* 'Spicata', or *C. plumosa*). The leaves and brightly colored flowers are edible: *C. argentea* is cultivated as a leafy green in tropical regions of Africa, South America, and Southeast Asia (National Research Council 2006). As an ornamental, celosia adds colorful interest in gardens (beds, borders), containers, as cut flowers or in dried floral arrangements, or indoors with sufficient light. Celosia is also an herbaceous annual weed in many cropping systems (e.g., groundnut, pearl millet, cotton, maize) in India (Nalini et al. 2015; Ravindra et al. 2008).

Celosia is propagated from seed, and in warm weather the annual grows well in full-sun and in moist but well-drained soils, flowering until mid- to late-summer in temperate regions (Nau 1991; Gilman and Howe 1999). The annual is subject to few

diseases; leaf spots, Botrytis bight, damping-off, and root, stem, and crown rots (non-water molds or oomycetes) may occur under conditions of high moisture; other reported issues include powdery mildew, root-knot nematode, and phytoplasma and virus diseases (Blake and Williamson 2015; Farr et al. 1989; Horst 2008; Horst 2013; Pundt 1993; Raabe et al. 2009; Wehlburg et al. 1975; Wilson 1932; Tanne et al. 2000). Cold damage and high soluble salts in growing media may also affect celosia production (Chase and Daughtrey 2012).

Most of the disease information in this chapter pertains to *Celosia* sp. grown as an ornamental. In some regions of the world, however, both edible and ornamental crops are cultivated; the distinction between these is made where appropriate.

2 Fungal and Fungus-Like Diseases

2.1 Alternaria Leaf Spot and Blight (= Nimbya Leaf Spot) [*Alternaria* sp.; *A. alternata* (Fr.) Keissl.; *A. celosiicola* Jun. Nishikawa & C. Nakashima (Basionym: *Nimbya celosiae* E.G. Simmons & Holcomb (≡ *A. celosiae* (E.G. Simmons & Holcomb) Lawrence, Park & Pryor)), *A. gomphrenae* Togashi]

Geographic occurrence and impact. Species of *Alternaria* have a wide host range, causing leaf spots and blights on numerous ornamental crops. One of several leaf spot diseases of celosia, Alternaria leaf spot (as one or more of the species listed above) in production (plug trays and finishing) and landscape has been reported in the USA (on *Celosia argentea* and C. *argentea* var. *cristata*, Farr et al. 1989, United States Agricultural Research Service 1960; on *Celosia* sp., Ivey and Iverson 2005), Japan (on *C. argentea* var. *plumosa* and *C. argentea* var. *cristata*, Nishikawa and Nakashima 2013), South America (on *C. argentea*, Cúndom and Cabrera 2002), Thailand (on *Celosia* sp., Seemadua et al. 2011), other parts of South and Southeast Asia and the Caribbean (on *C. plumosa*, Yuchida 1998), and in Africa on *C. argentea* grown as a vegetable (Yarger 2007). Holcombe (1978) reported that a species of *Alternaria* (not listed above) from alligatorweed, *A. alternantherae*, in Louisiana was pathogenic to ornamental *C. argentea* inoculated in the greenhouse, causing severe infection and moderate to severe defoliation. Outdoors, the disease is more common during wet seasons (Pirone 1978).

Symptoms/signs. Leaf spotting appears on older leaves as small, reddish purple spots that turn tan to gray (concentric rings may be evident) with a red to purple border as they enlarge (up to 8 mm diameter) (Seemadua et al. 2011) (Fig. 1). Spots may also appear on bracts, petioles, or stems, and blighting of leaves, shoots, and flowers may occur; plants are most heavily diseased during cool, wet weather or during stress (Agrios 2005; Yuchida 1998). Although Alternaria leaf spot may occur during plug production, it is most evident at finishing or in landscape plantings (Chase and Daughtrey 2012).



Fig. 1 Alternaria leaf spot and blight (R.J. McGovern © 2017. All Rights Reserved.)

Biology and epidemiology. Abundant *Alternaria* spores (conidia) are produced in lesions during moist weather. Conidia are pale to brown, multicellular with longitudinal and transverse septa, and borne in chains on short conidiophores. The fungus survives adverse conditions as mycelium or spores associated with infected plant debris or seeds and is spread by moving air, splashing water (rain or irrigation), mechanically during handling, or movement of contaminated seeds or plant material (Agrios 2005; Gleason et al. 2009).

Management. Management of leaf spot diseases benefits from strategies that reduce inoculum: discard severely diseased plants, use pathogen-free seed, minimize moisture and humidity in the canopy (e.g., careful watering, avoiding overhead irrigation, proper spacing and ventilation), and consider a protectant fungicide or biological product if conditions conducive to disease development are expected. Sanitation practices include cleaning production areas or gardens of diseased plants and debris to reduce any surviving inoculum (Chase and Daughtrey 2012; Yuchida 1998). In addition, leaf spot diseases may be more severe on plants weakened by environmental factors or poor growing conditions (Douglas 2012).

2.2 Botrytis Blight, Botrytis Stem Rot (*Botrytis cinerea* Pers.:Fr.)

Geographic occurrence and impact. Also known as "gray mold," Botrytis blight is a very common and widely distributed disease of over 170 families of cultivated plants, including flowering crops (Elad et al. 2016a). *Botrytis cinerea*, a nonhost specific pathogen, readily colonizes nonliving organic matter as a saprophyte and, under the right conditions, can also attack leaf, floral, stem, bud, fruit, root, bulb, and crown tissues as a necrotroph. In celosia, the pathogen causes a stem canker and blight on the inflorescence and leaves when humidity is high, causing direct crop loss and significantly reducing yield or suitability as cut flowers or in dried arrangements.



Fig. 2 Botrytis leaf and stem blight (North Carolina State University © 2017. All Rights Reserved.)

Symptoms/signs. On hosts affected by gray mold, leaves and petals may appear water soaked at first, progressing to irregular, sunken spots or larger lesions that fade to tan to dark (Chase et al. 1995) (Fig. 2). Flowers will fail to open if infected during the bud stage. Under conditions of high humidity, the fungus produces a characteristic gray, fuzzy growth (mycelium and spores) on infected tissue (Agrios 2005). This aerial mycelium may also be evident on affected celosia stems, which turn gray to black and may break at the point of infection.

Biology and epidemiology. Epidemics of gray mold are common in enclosed structures, such as greenhouses, and outdoors where moisture is high. Abundant conidia (asexual spores) and aerial mycelium of *B. cinerea* are associated with diseased tissues when humidity is high (>90%) and a thin film of water is present for 4 h or more (Carisse 2016). Spores are hyaline, ovoid, and borne in bunches (botryose) on branched conidiophores. These spores readily disperse by air currents throughout the greenhouse or outdoors, penetrating host tissues, especially floral organs, most often through wounds. Spores may also lodge on stems and other plant parts, readily penetrating wounds to cause stem cankers or cutting rots during propagation.

On many hosts, the fungus produces sclerotia: black, elliptical (up to 5 mm/0.2 in. diameter) survival structures that are associated with dead, colonized tissues. Sclerotia, which consist of a compact mass of hyphae with a melanized rind, are resistant to adverse conditions and serve as an inoculum source for new infections. The fungus also survives the noncropping period as mycelium in plant debris, and can remain asymptomatically as an endophyte in plant tissues (Elad et al. 2016b).

Management

- ***Cultural practices*** – Botrytis diseases benefit from strategies that reduce moisture (tissue wetness and humidity) and wounding. Since moisture is required for spore germination and penetration, careful watering, avoiding overhead irrigation or watering early in the day, and proper spacing and ventilation in production areas are required (Elad 2016). In shops, keep flowers and cooler floors dry

(Wright 2011). Avoid unnecessary mechanical injury during production, harvest, and transport to reduce the number of infection courts for the fungus. In celosia marketed as cut flowers, plants are harvested 3–4 week after the flower matures. To avoid issues with gray mold after harvest, these hardened stems must be dry and kept at a constant temperature at storage and during transport to prevent condensation (Zuck 2015). Stems that are wounded by poor handling at harvest or by inadequate packing prior to transport may become colonized by the fungus when moisture becomes available. Process flowers quickly and carefully, ensuring good air flow to keep blooms dry. Control insects during production, which create wounds in plants.

Sanitation practices reduce inoculum; these include cleaning production areas or outdoor beds of diseased plants and debris where the fungus may have survived in absence of its host (Chase and Daughtrey 2012; Elad 2016). In addition, senescent and dead plant tissues are readily colonized and may serve as a "base" from which the fungus spreads to healthy tissue.

Proper management of Botrytis blight includes monitoring. Purchase clean stock and ask suppliers about Botrytis management practices and shipping conditions. Look for symptoms (including fluffy gray mold on infected plant parts) when weather conditions favor the pathogen (wet, overcast, and very humid weather). If symptoms are present but sporulation is not evident, place affected tissue in a moist chamber for 1–2 days to encourage the development of aerial mycelium.

Avoid plant stress. High salts, compacted planting media, uneven watering, and fertilization that does not meet the needs of the plant reduce the ability of plants to resist disease.

- ***Chemical management*** of Botrytis diseases on ornamental hosts, including celosia, includes application of preventive fungicides when environmental conditions are conducive to disease development. Chemical applications combined with cultural management strategies to reduce inoculum may be necessary to control Botrytis blight in many cropping situations, especially for high value plants (Fillinger and Walker 2016). Due to the genetic plasticity of the *Botrytis* fungus (Carisse 2016), fungicide resistance has developed to several commonly used classes of fungicides worldwide (Beever et al. 1989; Bollen and Scholten 1971; Katan 1982; Leroch et al. 2011; Leroux et al. 2002; Lamondia and Douglas 1997; Yourman and Jeffers 1999; Zhao et al. 2010). A treatment of fungicide mode of action, resistance development, and fitness costs in *Botrytis* sp. may be found in reviews compiled by Fillinger and Walker 2016, Kretschmer et al. 2009, and Leroux 2007. As some populations of *Botrytis* may have partial resistance to antifungal compounds, strategies to reduce fungicide resistance development include reducing inoculum through cultural practices, alternating or tank-mixing compounds with differing modes of action, and development of new chemistries to maintain a diversity of mode of actions (Beever and Brien 1983; Fillinger and Walker 2016; Rosslenbroich and Stuebler 2000).

- ***Biological control*** – In addition to synthetic fungicides, the use, efficacy, and durability of biological control agents and biopesticides for gray mold management have been assessed (Nicot et al. 2016) in an effort to identify materials that can be applied to Integrated Crop Management (ICM) or organic cropping methods. Many of these products inhibit the activity of *B. cinerea* in the greenhouse or laboratory, but these vary in efficacy and mode of action and few are commercialized. Those biologics with activity against *Botrytis* in ornamental plants include formulations containing *Reynoutria sachalinensis*, *Bacillus subtilis* QST 713, *Gliocladium catenulatum* J1446, *Streptomyces griseoviridis* K61, and *S. lydicus* WYCD108. Other materials include neem, soybean oils, ammonium chloride, hydrogen dioxide, and potassium bicarbonates. Although these products have general labels for ornamental plants, none include celosia-specific phytotoxicity or application information.

2.3 Cercospora Leaf Spot (*Cercospora celosiae* Syd.)

Geographic occurrence and impact. *Cercospora* spp. cause a leaf spot or blight on a wide variety of host plants (Agrios 2005). *C. celosiae* has been reported on celosia in the USA (on *Celosia cristata*, Ellett 1989; on *C. argentea* and *C. argentea* var. *cristata*, Farr et al. 1989), Asia (on *C. cristata*, *C. plumosa*, Braun and Sivapalan 1999; on *C. argentea* var. *cristata*, Groenewald et al. 2013, Nguanhom et al. 2015; on *C. argentea*, To-Anun et al. 2011), South America (on *Celosia* sp., Standen 1952), Africa (on *C. argentea* grown as a vegetable [Legos spinach], Yarger 2007), Malaysia (on *C. cristata*, Turner 1967), and the Indian subcontinent (on *C. argentea*, Kew Royal Botanic Gardens 2015). As with other leaf spot diseases of celosia, the disease is more common during wet seasons (Pirone 1978).

Symptoms/signs. Leaf spots on celosia appear as irregularly shaped, pale to brown lesions (up to 7 mm/0.3 in. diameter) surrounded by a red to dark brown border (Groenewald et al. 2013); infected tissues sometimes drop away from the leaf, leaving holes (known as shot-holes).

Biology and epidemiology. *C. celosiae* spores (conidia) are hyaline, straight to curved, multiseptate, and are borne on pale to olive-brown groupings of conidiophores within small, brown compact masses of hyphae (fruiting structures) called stromata (Groenewald et al. 2013). Conidia are easily detached and spread by moving air. Moisture or high humidity and warmer temperatures favor disease development, and the fungus survives adverse conditions associated with seed or within stromata in old leaf lesions (Agrios 2005).

Management. Management of leaf spot diseases benefits from strategies that reduce inoculum as described for Alternaria leaf spot in Sect. 2.1. In addition, use of disease-free seed and potting material is helpful (Agrios 2005).

2.4 Charcoal Rot, Collar Rot (*Macrophomina phaseolina* (Tassi) Goid.)

Geographic occurrence and impact. *Macrophomina phaseolina* is a widely distributed soilborne pathogen that affects the roots and lower stems of more than 500 host plants, notably soybean, maize, cotton, and sunflower, causing a root rot, charcoal rot, collar rot, and damping-off and seedling blight worldwide (Ijaz et al. 2013; Sinclair 1982; Smith and Carvil 1997). In a pot culture experiment, Singh et al. (1990) reported that a *M. phaseolina* isolate from chickpea was pathogenic to a *Celosia argentea*, a new host. The fungus was also reported in Pakistan in an outdoor planting of *C. argentea* in 2011 (Rehman et al. 2015). In the USA, charcoal rot (as *Sclerotium batatitcola*) of celosia (*C. argentea* var. *cristata*) has been reported in Texas (US Agricultural Research Service 1960).

Symptoms/signs. In general, disease symptoms caused by *M. phaseolina* include a rot of the roots and lower stem. Infected seedlings exhibit sunken cankers at the soil line. Aboveground, leaf yellowing and premature leaf drop and plant death are common. Rehman et al. (2015) reported symptoms of charcoal/collar rot on *C. argentea* as chlorosis and wilt of the lower leaves during warm dry weather. In addition, the roots and stem collar were shrunken and shredded. In *C. argentea* grown as a vegetable, infection by *M. phaseolina* results in dark spots on the leaves (Yarger 2007).

Biology and epidemiology. *M. phaseolina* produces numerous small, black, opaque microsclerotia (50–150 μm diameter) just under the epidermis that serve as survival structures in soil and crop residues and as a source of primary inoculum (Gupta et al. 2012; Sinclair 1982).

Typically, development of charcoal rot is favored by hot, dry weather. Rehman et al. (2015) noted that charcoal/collar rot of celosia in Pakistan was more severe during warmer, dry conditions (30°C/82°F and <25% soil moisture). Microsclerotia may survive associated with host debris or seed or for extended periods, perhaps years, in dry soil; at warm temperatures, these structures germinate on root or seed surfaces and penetrate the epidermis directly or through natural openings by means of appressoria (Gupta et al. 2012). Infected seeds may fail to germinate, and infected seedlings may damp-off. *M. phaseoli* is reported to be a poor competitor in soil, and survival in wet soils is significantly reduced (Sinclair 1982).

Management. Strategies that improve plant health help to reduce disease incidence. In addition, high plant density and injury due to insects predispose plants to disease (Ijaz et al. 2013). To manage charcoal rot in cropping systems, irrigate to avoid drought, especially during exceptionally warm periods, and fertilize at optimal levels. The pathogen has a wide host range; weed control and long-term crop rotations to less susceptible species may reduce inoculum (Gupta et al. 2012; Sinclair 1982).

In general, fungicides do not provide acceptable or consistent control for charcoal rot in cropping systems, although the fungicide thiophanate-methyl and extracts of neem (*Azadirachta indica*) and *Eucalyptus camaldulensis* reduced mycelial growth of *M. phaseolina* isolated from *C. argentea* in vitro (Rehman 2015). In other cropping systems, thermotherapy, oil seed treatments, and various plant extracts and fungicides have been screened for efficacy in vitro with varying success (Ijaz et al. 2013).

Some reduced disease development in arid regions has been observed in soils amended by organic composts, animal manure, mustard cake, and weed residues (Gupta et al. 2012). Lodha et al. (2014) found that populations of actinomycetes antagonistic to *M. phaseolina* (soil suppressiveness) increased in soils amended with a combination of mustard and weed residue compost. In general, management of charcoal rot might be best served through a combination of cultural practices that includes crop rotation, maintenance of plant health, irrigation, low sowing density, and organic matter inputs (Gupta et al. 2012).

2.5 Fusarium Canker, Blight, Leaf Spot (*Fusarium lateritium* f. sp. *celosiae* (Abe) Matuo) [≡ *Fusarium lateritium* Nees) (tel. *Gibberella baccata* (Wallr.) Sacc])

Geographic occurrence and impact. *Fusarium lateritium* is a soilborne fungus that causes a canker or blight on a broad range of woody plants worldwide (Booth 1971; Lawrence et al. 1985). A disease of ornamental celosia (*Celosia cristata*) caused by a new *Fusarium* species, described in this report as *Fusarium celosiae* sp. nov., was first recorded in Japan in 1928 (Abe 1928). Symptoms observed included damping-off of seedlings and leaf blight and stem canker of mature plants. The pathogen (recorded as *Fusarium* sp., *F. celosiae*, *F. lateritium*, or *F. lateritium* f. sp. *celosiae*) has since been reported to cause a stem or collar rot of celosia (*Celosia* sp., *C. argentea*, or *C. cristata*) in Australia (Summerell et al. 2011); stem rot, wilt, or canker in the southern USA (Blake and Williamson 2015; Chase et al. 1995; Farr et al. 1989; Ivey and Iverson 2005; Jacobi 2000), leaf spot or blight in Nigeria (Afanide et al. 1976a,b), and damping-off (Dodge and Rickett 1948).

Symptoms/signs. Symptoms of *Fusarium* disease in celosia are varied; the disease affects seedlings (as a seedling blight or damping-off) and more mature plants (as a leaf spot, leaf blight, root, stem, or collar rot, wilt, or stem canker). Seedlings may damp-off or exhibit brown-black, water-soaked spots (Abe 1928; Dodge and Rickett 1948). Mature plants may develop rusty brown spots or blotches with concentric, red-brown rings on leaves. On stems, rusty spots form on leaf stalks and stems, which dry out, canker, and crack (Chase et al. 1995). Flowers may also be affected. Afanide et al. (1976a) reported that although young seedlings in the field were susceptible, disease incidence was greater on more mature plants.

Biology and epidemiology. *F. lateritium* is probably a species complex that is not well-characterized (Summerell et al. 2011). Macroconidia of the pathogen are cylindrical or curved (canoe-shaped) with 3–7 septa, and the basal cell has a characteristic beak or hook. In culture, spores are produced abundantly in orange sporodochia. Chlamydospores are formed singly or in chains (Booth 1971; Nelson et al. 1983). Perithecia of the teleomorph, *Gibberella baccata*, form in culture and occasionally on diseased plant material (Lawrence et al. 1985). In the original description of this pathogen on celosia, Abe (1928) reports the fungus grows best at 28–32°C/82–90°F.

Fusarium spp. survive as resistant hyphae, chlamydospores, or immature perithecia associated with plant debris in soil (if soilborne) or (if a leaf pathogen) aboveground plants parts. These fungi are dispersed passively through splashing water, movement of infected plant material, of debris or infested soil (if soilborne), or may be airborne (if pathogenic to aboveground plant parts) (Burgess 1981). Booth (1971) reported that *F. lateritium*, unlike other *Fusarium* species, is rarely recovered from seed; the association of this fungus with seed of celosia is not known.

Management

- ***Cultural practices*** – Environmental stress (e.g., moisture stress, freeze damage) can predispose hosts to diseases caused by *Fusarium* sp. (Sinclair and Lyon 2005). Fusarium diseases are difficult to control (Chase 2013a); diseases caused by these fungi benefit from general control measures in place for management of soilborne diseases in production systems, such as using clean stock and soilless mix uncontaminated by *Fusarium* inoculum, inspecting new stock and monitoring for symptoms of disease, rotating with nonsusceptible crops, managing soil moisture and drainage, proper fertilizing, sanitation to reduce inoculum, and using resistant plant material where available.
- ***Fungicides and biocontrols*** – Fungicides labeled for *Fusarium* sp. on celosia are preventive materials applied as a soil drench, foliar spray, or pre-potting mix and include certain phenylpyrroles, thiophanates, dithiocarbamates, QoI fungicides, or combination products. A biologic with activity against *Fusarium* in celosia, *Bacillus subtilis* var. *amyloliquefaciens* strain FZB24, is applied as a soil or transplant drench. Other products with activity against *Fusarium* in ornamental plants include formulations of *B. subtilis*, *Streptomyces lydicus*, *Trichoderma* sp., hydrogen dioxide, and potassium bicarbonate.

2.6 Phytophthora Root and Stem Rot, Damping-off (*Phytophthora cryptogea* Pethybr. & Laff.; *P. drechsleri* Tucker; *P. nicotianae* Breda de Haan (syns. *Phytophthora nicotianae* var. *parasitica*, *P. parasitica*)

Geographic occurrence and impact. *Phytophthora* spp. cause diseases with high economic impact on a wide variety of dicots, including vegetable, field, and fruit crops, forest trees, and ornamental plants, including a wide variety of bedding plants,

worldwide. These organisms may be host specific or have a broad host range. The pathogen may attack both belowground (e.g., roots, tubers, corms) and aboveground (stems and twigs, buds, foliage, fruit) plant parts (Agrios 2005).

Phytophthora disease on celosia has been reported in the USA and Canada (as *Phytophthora* sp. on *Celosia* sp., Blake and Williamson 2015, Ivey and Iverson 2005, Western Committee on Crop Pests 2014; as *P. nicotianae* var. *parasitica* on *C. argentea* var. *cristata*, Farr et al. 1989; as *P. cryptogea* on *C. argentea* var. *cristata* [cockscomb], Middleton et al. 1944), Britain (as *P. cryptogea* [foot rot] on *Celosia* sp., Moore 1959), Argentina (as *P. drechsleri* and *P. parasitica* on *C. plumosa*, Frezzi 1950), and Nigeria (as *P. cryptogea* on *C. argentea* grown as a vegetable [Legos spinach], Yarger 2007).

Symptoms/signs. Symptoms of Phytophthora diseases usually include chlorosis, blight or dieback, wilt, decline or rapid collapse, and death (Banko and Stefani 2000). Those reported on *Celosia* sp. include damping-off, root and stem rot, and collar rot (Blake and Williamson 2015; Farr et al. 1989; Ivey and Iverson 2005; Western Committee on Crop Pests 2014; Yarger 2007).

Biology and epidemiology. As oomycetes (water molds), most *Phytophthora* species disperse short distances as motile zoospores through moving water, or long distances through movement of diseased plant material or soil. The pathogen survives adverse environmental conditions as chlamydospores, oospores (where formed), and mycelium within infected plant tissues, which serve as a source of disease inoculum. Environmental conditions that favor disease development include overwatering or poor drainage (Banko and Stefani 2000).

Management. Banko and Stefani (2000) and Hagan et al. (1996) included *Celosia* cultivars (*C. argentea* Apricot Brandy, Castle Pink, New Look, or Prestige Scarlet) in studies of bedding plant susceptibility to *P. nicotianae*. In both reports, these cultivars had excellent survival rates in infested beds and might be useful as alternatives to more susceptible species such as *Antirrhinum*, *Catharanthus*, and *Viola* (pansy) or intensive chemical treatment programs (Hagan 2004). Refer to ▶ Chap. 7, "Fungicides and Biocontrols for Management of Florists' Crops Diseases" for additional strategies.

2.7 Powdery Mildew (*Erysiphe celosiae* Tanda; *Leveillula taurica* (Lév.) G. Arnaud)

Geographic occurrence and impact. Normally a ubiquitous and important disease of ornamental crops, official reports of powdery mildew of celosia (*Celosia argentea*) are few, although trade publications occasionally mention the occurrence of this disease.

First reported on *Celosia* sp. in Italy (not reliably known as *Erysiphe communis*), Tanda (2000) described a powdery mildew disease on feather cockscomb



Fig. 3 Powdery mildew on Celosia (Armstrong Growers © 2017. All Rights Reserved.)

(*C. argentea* var. *argentea*) leaves and stems planted in a garden in Tokyo. The fungus was described as a new species: *Erysiphe celosiae* Tanda. Reports of another powdery mildew fungus, *Leveillula taurica* on other species of *Celosia* occurred in parts of Africa, the Persian Gulf, South Asia, and parts of Europe (Amano 1986). An *Oidium* sp. was recorded on *Celosia* sp. from Russia (Amano 1986), and *Erysiphe betae* (a pathogen of beet) was reported on *Celosia* sp. collected in Germany (Braun 1998). While reexamining the taxonomic state of these fungi, Ale-Agha et al. (2008) concluded that the record of *E. communis* on *C. argentea* from Italy likely belonged to *E. celosiae* and that further work is needed to confirm that *E. celosiae* and *E. betae* are indeed separate species.

Symptoms/signs. In general, powdery mildews appear as a white to tan superficial mycelium on the surface of affected leaves and other aerial plant parts during warm, humid weather (Fig. 3). Asexual spores (or conidia) are produced on this powdery growth and are distributed by air currents to other susceptible plants. Young plants and tissues are often more susceptible to this disease. Although the fungus does not directly kill the cells it invades, infection does result in reduced photosynthesis and increased water loss. As a result, the growth rate and aesthetic value of infected plants may be reduced. Leaves may be stunted, curled, or twisted; in highly susceptible plants, new growth, flowers, and buds can be destroyed (Agrios 2005; Chase et al. 1995).

Tanda (2000) describes symptoms and signs of powdery mildew on *C. argentea* as irregular, white patches on leaves and stems (but not flowers), often covering the entire leaf surface. Affected leaves are reddish purple, but further impact of the disease on host development is not mentioned.

Biology and epidemiology. The conidia (asexual spores) of *E. celosiae* are produced singly (pseudoidium type) on conidiophores with one to two septa. Cleistothecia are globose and dark brown with simple or sometimes branched

appendages, each containing 4–8 asci. Ascospores (sexual spores, 2–4 per ascus) are ovoid and light yellow (Tanda 2000). Conidia are easily dispersed by air currents and do not require free moisture to sporulate. Cleistothecia serve as survival structures during noncropping periods and a source of primary inoculum to new crops. Powdery mildew fungi are host specific, reaching epidemic proportions when only one plant type is grown.

Management. To manage powdery mildew in ornamental plantings, improve air movement around plants through proper spacing and weed control, and increase the amount of sunlight that reaches foliage. Rake old leaves and prune shoots infected the previous growing season to reduce inoculum. Overhead watering increases humidity and encourages disease development. Practices that promote succulent growth, including nitrogen fertilizing, should be avoided on susceptible hosts (Chase et al. 1995).

2.8 Pythium Blight, Damping-off (*Pythium irregulare* Buisman; *P. ultimum* Trow)

Geographic occurrence and impact. *Pythium* spp. are soilborne oomycetes (water molds) that cause seed and seedling damping-off as well as root and soft rots in a wide variety of hosts worldwide (Agrios 2005), including bedding plants and other floriculture crops (Múnera and Hausbeck 2016; Stephens and Powell 1982). Pythium disease on celosia has been reported in the USA, Canada, and Great Britain as *Pythium* sp. on *Celosia* sp. (Blake and Williamson 2015; Chase et al. 1995; Ivey and Iverson 2005; Raabe et al. 2009; Western Committee on Crop Pests 2014) or *C. argentea* var. *cristata* (Farr et al. 1989; Moore 1959) and in Nigeria as *P. aphanidermatum* on *C. argentea* grown as a vegetable (Legos spinach) (Yarger 2007).

Symptoms/signs. In addition to causing root disease, blight, and soft rot of fleshy plant parts that touch the soil, *Pythium* sp. are significant damping-off pathogens (where seeds fail to germinate or developing seedlings die in seeding beds or shortly after transplant). Symptoms of Pythium disease on celosia include root rot and damping-off (Blake and Williamson 2015; Chase and Daughtrey 2012; Ivey and Iverson 2005; Raabe et al. 2009; Stephens and Powell 1982; Vea and Palmer 2015); symptoms above ground include yellowing and wilt of leaves and flowers, and plant stunting (Chase et al. 1995). When root-knot nematodes are present the disease can be devastating (Fig. 4).

Biology and epidemiology. As oomycetes (water molds), most *Pythium* species produce sporangia and motile zoospores, which disperse short distances in moving water. The organism survives as chlamydospores and oospores, which can be dispersed long distances in diseased plant material or associated soil. Root rot and damping-off of *Pythium* occurs when soils contaminated with the pathogen are saturated and poorly aerated (Agrios 2005).



Fig. 4 Severe wilt and plant death caused by combined infection by *Pythium* sp. and root-knot nematode (Erin Rosskopf. USDA.)

Celosia is susceptible to several species of *Pythium*, notably *P. ultimum* and *P. irregulare* (Chase 2012). In a greenhouse study, Stephens and Powell (1982) evaluated the pathogenicity of five *Pythium* species to nine commonly grown bedding plants, including *C. argentea* Red Fox. In general, *P. ultimum* was the species of primary concern; damping-off of celosia due to *P. aphanidermatum*, *P. spinosum*, *P debaryanum*, *and P. irregulare* was also significant.

Management

- ***Cultural practices*** – Management of Pythium diseases in ornamental plant production benefits from strategies to reduce inoculum and optimize crop production: use pathogen-free potting materials, mix, and tools, monitor crops for disease, and optimize crop production through proper use of water and fertilizer (Chase 2013b; Chase and Daughtrey 2012).
- ***Fungicides*** – In general, chemical management of Pythium damping-off in celosia is difficult; several products with some efficacy in greenhouse trials include azoxystrobin, cyazofamid, etridiazole, fenamidone, mefenoxam, alone or in combination (Chase 2010d, 2012, 2013; Chase and Daughtrey 2012; Vea and Palmer 2015), but products do not tend to provide satisfactory control under severe disease pressure (Chase 2010b). Efficacy depends on *Pythium* species and isolate (Chase 2010c); Chase (2012) warns that accurate diagnosis is needed to select the most effective products for disease control. In addition, populations of

Pythium resistant to metalaxyl are present in the bedding plant industry, which contributes to failure of efficacy in some growing situations (Chase 2012; Múnera and Hausbeck 2016). Information on biocontrols for *Pythium* management may be found in ▶ Chap. 7, "Fungicides and Biocontrols for Management of Florists' Crops Diseases."

2.9 Rhizoctonia Root and Crown Rot, Blight, Damping-off [*Rhizoctonia solani* Kühn (tel. *Thanatephorus cucumeris* (A.B. Frank) Donk)]

Geographic occurrence and impact. The genus *Rhizoctonia* encompasses a group of ubiquitous soilborne fungi that exist as saprophytes or as opportunistic (facultative) plant pathogens on a wide range of host plants, including ornamentals.

Rhizoctonia disease on celosia has been reported in the USA and Canada as *Pellicularia filamentosa* (syn. *R. solani*) on *Celosia* sp. (cockscomb) (Pirone 1978) or as *Rhizoctonia* sp. or *R. solani* on *C. argentea* var. *cristata* (cockscomb) (Farr et al. 1989; United States Agricultural Research Service 1960), *C. argentea* (Farr et al. 1989), or *Celosia* sp. (cockscomb, woolflower) (Beale and Bachi 2005; Blake and Williamson 2015; Evans-Ruhl et al. 1980; Ivey and Iverson 2005; Raabe et al. 2009; Western Committee on Crop Pests 2014). The pathogen is also regarded as a significant pathogen on *C. argentea* grown as a vegetable (Legos spinach) in parts of Africa (Yarger 2007).

Symptoms/signs. *R. solani* primarily attacks the stem at the soil line in most crops, particularly young seedlings, causing a sunken canker that girdles the stem and kills distal tissues (Pundt 1993) (Fig. 5). It is a significant pre- and postemergence damping-off pathogen; attacks roots, the crown, and propagative plant parts; attacks leaves where brown, irregularly shaped spots develop; and can also cause an aerial (or web) blight when humidity is high (Chase 2011). Aboveground symptoms of *R. solani* are similar to those caused by other root and vascular diseases: small, pale yellow or green leaves, wilt, stunt, poor yield, and plant death. In celosia, damping-off is of prime concern (Peltier 1916; United States Agricultural Research Service



Fig. 5 Rhizoctonia stem blight (*left*), and leaf spot and blight (*right*) (R.J. McGovern © 2017. All Rights Reserved.)

1960; Yarger 2007), although root, crown, and stem rots are also reported (Evans-Ruhl et al. 1980; Farr et al. 1989; Raabe et al. 2009).

Biology and epidemiology. The thallus, or vegetative body, of *R. solani* is a sterile mycelium (lacking spores); the fungus survives as mycelium or as small sclerotia in soil, debris, propagative plant parts (such as tubers), or in infected perennial plants. The fungus is easily dispersed with infested soil particles that cling to propagation materials and tools, with moving water, or infected plant material. Diseases caused by *Rhizoctonia* are common during wet, warm weather or inside heated greenhouses with overhead irrigation (Chase 2011). Aerial (or web) blight may also occur when humidity is high. Plants that are weakened from adverse environmental conditions are more susceptible to disease (Agrios, 2005). In a pathogenicity study characterizing fungal isolates associated with damping-off, all *Rhizoctonia* isolates recovered from bedding plants, including *C. argentea* Red Fox, were from anastomosis group 4 (Ag-4) (Stephens et al. 1982).

Management

- ***Cultural practices*** – Management of Rhizoctonia diseases in ornamental production benefits from a combination of cultural practices with the use of fungicides where appropriate. Soilborne propagules are easily dispersed with soil particles or infested plant material and debris; prevention is key. To keep inoculum levels low, clean growing areas, remove debris, avoid placing plants directly on the ground, and use pathogen-free potting materials, mix, and tools. Other management tactics include: inspect incoming stock and monitor existing plants for symptoms of disease, reduce humidity and leaf wetness in growing areas, and optimize plant health through proper use of water and fertilizer (Beytes 2014). Incorporation of composted hardwood bark to potting mix has been shown to reduce Rhizoctonia damping-off of *C. argentea* Red Fox compared to standard sterile mixes (Nelson and Hoitink 1982; Stephens et al. 1981), due perhaps to forced competition with other microorganisms or reduced movement of the pathogen in the compost medium.
- ***Fungicides*** – Several fungicide products with some efficacy in celosia greenhouse trials include fludioxonil, polyoxin-D, some strobilurins, and thiophanate-methyl (Chase 2009, 2010a; Chase and Daughtrey 2012). Accurate diagnosis of the pathogen is vital; fungicides that control the soilborne oomycetes (such as *Pythium* and *Phytophthora*) do not control *Rhizoctonia*, and other factors, such as strain of the pathogen and host cultivar, may be in play.

2.10 Verticillium Wilt (*Verticillium albo-atrum* Reinke & Berthold; *Verticillium dahliae* Kleb.)

Geographic occurrence and impact. The soil-inhabiting fungus *Verticillium* affects more than 300 plant species in temperate regions worldwide, including trees, shrubs, vines, other woody and herbaceous ornamentals, and field and vegetable crops (Agrios 2005; Klosterman et al. 2009). Two species that are best

characterized and most associated with crop loss are *Verticillium dahliae* and *V. albo-atrum*. *Celosia* sp. has been reported to be susceptible to Verticillium wilt in several disease compilations in the USA (Douglas 2008; McCain et al. 1981), but original sources are lacking.

Symptoms/signs. Symptoms of Verticillium wilt are indicative of water stress in the plant: lower leaf chlorosis and droop are evident and v-shaped blotches or scorch may appear at leaf tips. Symptoms progress to the upper canopy, often appearing on one side of the plant. Discoloration (brown to black streaking) of the vascular tissue in roots, tubers, and stems may be evident. Symptoms will vary depending on the host and environmental conditions, which may be more severe during dry periods. The symptoms associated with Verticillium are similar to Fusarium wilt, so an accurate diagnosis is needed to differentiate the two diseases (Douglass 2008).

Biology and epidemiology. The *Verticillium* fungus survives in perennial host plants or in soil as microsclerotia (*V. dahliae*) or thick-walled mycelium (*V. albo-atrum*) or as mycelium in plant debris or in seed and other propagative plant parts, which serve as sources of inoculum for disease development. The fungus penetrates plant roots and grows into the xylem, where it produces conidia that spread acropetally through the plant, producing a mycelium that eventually colonizes neighboring vascular and cortical tissues (Klosterman et al. 2009). Conidia are round or egg-shaped and produced at the end of phialides arranged in whorls (verticillate) on simple or dichotomously branched conidiophores (Gilman 1945). Symptoms occur as the xylem function is impaired from fungal infection, toxic compounds, or an ineffective response of the host to restrict movement of the fungus. Wilhelm (1955) reported that microsclerotia of *V. albo-atrum* can persist 8 years in the field or 15 years in culture.

Management. Verticillium wilt in herbaceous crops is not subject to satisfactory chemical control and is difficult to manage once plants are affected by the disease. In an effort to delay disease development until harvest, water and mulch plants with minimal symptoms to avoid additional moisture stress and fertilize as needed based on a soil test (Douglas 2008). Little information on the susceptibility of celosia cultivars to Verticillium wilt is available, so general practices in place to prevent management of soil diseases in production systems are needed. Preventive measures that impact the source and level of inoculum, such as those practiced in other cropping systems, include: inspect incoming stock and monitor existing stock for disease, avoid planting susceptible species in soils known to be infested, plant crops in commercial beds in long rotations to reduce populations of microsclerotia below economic thresholds, control alternative weed hosts, reduce host plant stress, and avoid movement of contaminated soil and debris through proper sanitation practices (Kamal 1985). Other strategies implemented in other cropping systems with varying success include solarization or fumigation of soils (both of limited use), organic soil amendment (Gamliel et al. 2000), green manures (Larkin et al. 2011), and biological control (Pegg and Brady 2002).

Additional Fungal Diseases The following diseases of celosia (ornamental or vegetable varieties) have been reported:

- **Leaf spot disease** of *C. cristata* (cockscomb) caused by ***Colletotrichum capsici* (Syd.) Butler & Bisby** in India (first report) (Rao 1969)
- **Phyllosticta leaf spot (*Phyllosticta celosiae* Thüm.)** of *C. cristata* in India (Rao 1963), of *C. argentea* in New Jersey, USA (Farr et al. 1989), and of *C. argentea* var *cristata* in Quebec, Canada (Shoemaker and Creelman 1957)
- **Sclerotinia rot (*Sclerotinia sclerotiorum* (Lib.) de Bary)** on *Celosia* sp. (Saharan and Mehta 2008), reported in Canada (Western Committee on Crop Pests 2014)
- **Southern blight (*Sclerotium rolfsii* Sacc)** on *Celosia* sp. in Kentucky (Beale and Bachi 2006)
- **Thielaviopsis root rot (*Thielaviopsis* sp.)** on *Celosia* sp. in Louisiana (Ivey and Iverson 2005)
- **White rust (*Albugo bliti*)** on *C. argentea* grown as a vegetable (Legos spinach) in Nigeria (Yarger 2007)
- **Wet (or stem) rot (*Choanephora cucurbitarum*)** on *C. argentea* grown as a vegetable (Legos spinach) in Nigeria (Yarger 2007)

3 Bacterial and Phytoplasma Diseases

3.1 Bacterial Leaf Spot (*Dickeya chrysanthemi* (syn. *Erwinia chrysanthemi* Burkholder et al.); *Pseudomonas syringae* pv. *tabaci* (Wolf & Foster) Young, Dye & Wilkie; *Xanthomonas* sp. Dowson)

Geographic occurrence and impact. Bacterial leaf spot diseases are common throughout the ornamentals industries worldwide. Causal agents are reported in Asia as *Erwinia chrysanthemi* (syn. *Dickeya chrysanthemi*) in *C. cristata* (Yufan 1990), South America as *Pseudomonas syringae* pv. *tabaci* in *C. argentea* (Brioso 2005), and in the USA as *Xanthomonas* sp. in *Celosia* sp. (Chase and Daughtrey 2012).

Symptoms/signs. In celosia, leaf spot is reported as circular spots with light brown margins (Yufan 1990).

Biology and epidemiology. Bacterial pathogens such as *Erwinia* and *Pseudomonas* survive in plant debris, on or in infected plants or seed, or may survive as reservoirs in other plants (Schuster and Coyne 1974). In greenhouses, these organisms are easily disseminated with water splash from overhead irrigation, and they can also be spread by animals and insects or as infected plants/plant parts or infested packing material, machinery, and tools. As bacteria penetrate

plants through wounds or natural openings, plants injured during cultivation or rough handling are more susceptible to disease.

Management. Management of bacterial diseases in ornamentals includes use of pathogen-free seed or culture-indexed stock, and monitoring incoming and existing stock for disease. Strict sanitation practices are also needed: destroy infected plants, clean and disinfest all tools, potting materials, and benches, use pathogen-free mixes, and wash hands during handling. Use practices that keep foliage surfaces dry and reduce splashing and humidity. Once disease occurs, chemical control is not satisfactory (Moorman 2016).

3.2 Seedling Blight (*Pseudomonas syringae* van Hall)

Geographic occurrence and impact. Seedling blight was reported on *C. cristata* var. *pyramidalis* bedding plants in Australia (Wellings and Stovold 1977).

Symptoms/signs. Seedlings develop chlorotic spots on leaves; leaf and shoot blight and plant death occurs.

Biology and epidemiology. Symptoms develop during cool weather accompanied by driving rain; symptoms are more severe at cooler (14–18°C/57–64°F) than warmer (25°C/77°F) weather.

Management. Refer to management strategies discussed in Sect. 3.1.

3.3 Phytoplasma Diseases ("*Candidatus* Phytoplasma spp.")

Geographic occurrence and impact. Phytoplasma diseases are reported in more than 700 plant species, including ornamentals, fruit and vegetable crops, timber, and shade trees worldwide, where economic losses may be substantial (Lee et al. 2000). A disease of celosia (*Celosia plumosa*, *C. cristata*) caused by a phytoplasma was first reported in by Tanne et al. (2000) in Israel, where severe loss (100%) of early seedings occurred. Subsequently, phytoplasma disease was identified in the Baltic States, Middle East, Oceana, and South America: Lithuania (*C. argentea*, Samuitienė and Navalinskienė 2006); Iran (*C. argentea*, Babaie et al. 2007; *C. cristata*, Aldaghi and Bertaccini 2015); Solomon Islands (Guadalcanal) (*C. argentea*, Davis and Tsatsia 2009); and Brazil (*C. argentea*, *C. spicata*, Eckstein et al. 2012).

Symptoms/signs. Symptoms of phytoplasma disease in the records above are typical of that for other hosts (Lee et al. 2000) of these organisms: yellows, phyllody, malformed leaves and flowers, stunt, and witches' brooms (Fig. 6). In addition, noted was lack of flowering in *C. cristata* in Israel (Tanne et al. 2000) and dieback in *C. argentea* in Iran (Babaie et al. 2007).



Fig. 6 Witches' broom symptoms caused by phytoplasmas (Edna Tanne © 2017. All Rights Reserved.)

Biology and epidemiology. Phytoplasmas are phloem-limited, biotrophic prokaryotes that lack a cell wall; they vary in size and shape (pleomorphic, < 1 μm in diameter), have a small genome, and require a phloem-feeding vector (such as leafhoppers) for transmission, in which the bacteria circulate and multiply (Agrios 2005). Phytoplasmas may also be spread through grafting, vegetative propagation, or via dodder (Lee et al. 2000); Eckstein et al. (2012) established transmission of phytoplasma via dodder (*Cuscuta subinclusa*) from an infected *C. argentea* plant to other celosia and periwinkle plants, where the development of leaf chlorosis, stunt, and shoot proliferation were noted. Phytoplasmas move within plants through sieve-tube elements from source to sink (Christensen et al. 2004). Phytoplasmas overwinter in the phloem of infected perennial plants or within vectors (Bertaccini and Duduk 2009).

Techniques used to characterize phytoplasma diseases include observation of symptoms, electron microscopy, and serological and molecular methods (Lee et al. 2000). Until recently, these organisms could not be cultured (Contaldo et al. 2012), making study difficult. Authors reporting phytoplasmas in *Celosia* sp., characterized strains by PCR-RFLP analysis of the conserved regions of 16S rDNA sequences. Most strains from celosia were speciated as "*Candidatus* Phytoplasma asteris" (16SrI aster yellows group) (Tanne et al. 2000; Samutiene and Navalinskiene 2006;

Aldaghi and Bertaccini 2015); others as "*Ca.* Phytoplasma aurantifolia" (16SrII) (Davis and Tsatsia 2009), "*Ca*. Phytoplasma trifolii" (16SrVI clover proliferation group) (Babaie et al. 2007), and the X-disease group (16SrIII-J) (Tanne et al. 2000; Eckstein et al. 2012).

Management. Management of phytoplasma diseases includes preventive techniques such as use of disease free or resistant stock, monitoring, removal of infected plant material and weeds, and vector control. It is not known whether control measures for phytoplasma disease incidence in celosia have been implemented or attempted in countries where the disease has been detected.

4 Viral Diseases

Celosia sp. are subject to several economically important plant virus, but not much is published on the resistance or susceptibility of cultivars to the diseases they cause. Celosia is recorded most often as susceptible to cucumber mosaic, tomato spotted wilt, tobacco mosaic, and beet curly top diseases. In Africa, celosia mosaic, a virus disease of Legos spinach (*C. argentea* L., cultivated as a vegetable), was described in 1998 (Owolabi et al. 1998). Celosia also serves as a diagnostic host for other viruses, and there are a number of reports where sap transmission occurs experimentally. These more often described virus diseases of celosia are included here; a list of minor virus disease records or where celosia is included in transmission experiments are found in Table 1.

4.1 Cucumber Mosaic (*Cucumber mosaic virus*)

Geographic occurrence and impact. *Cucumber mosaic virus* (CMV) (*Cucumovirus*: *Bromoviridae*) is distributed in subtropic, tropic, and temperate regions worldwide and affects more than 1200 species in 100 families of agricultural, horticultural (including ornamental) crops. The virus also occurs in perennial and annual weeds that may serve as a source of inoculum for crop plants (Zitter and Murphy 2009). The virus is easily spread and causes crop loss in hosts such as bean and pea, beet, cucurbits (cucumber, melon, pumpkin, squash, watermelon), pepper, tomato, potato, and spinach. Ornamentals affected by this virus disease include, among others, columbine, dahlia, delphinium, geranium, gladiolus, ixora, marigold, narcissus, petunia, phlox, and viola.

Celosia has been reported as host to CMV in the USA (on *Celosia* sp., *C. argentea* var. *cristata*, *C. argentea* var. *plumosa*, Dragich et al. 2014; Price 1940; Provvidenti 1975), India (*C. argentea*, as a weed in banana production, Mali and Rajegore 1980), and Africa (*C. argentea* grown as a vegetable, Aliyu et al. 2014). The report by Provvidenti (1975) records the first natural infection of celosia, where 70% of the *C. argentea* var. *plumosa* Fairy Fountains in a New York

Table 1 Minor viruses and those experimentally transmissible to *Celosia* sp.

Virus	Occurrence	Natural transmission	Natural hosts	Celosia hosts	Symptoms in celosia	Mechanism of experimental transmission	References
Alphaflexiviridae							
Cactus virus X (CVX) (*Potexvirus*)	Europe, USA	Mechanical, grafting	Cultivated *Cactaceae*	*C. argentea*	Systemic chlorotic spots	Sap inoculation	Koenig et al. 2004; Liao et al. 2003
Foxtail mosaic virus (FoMV) (*Potexvirus*)	USA	Mechanical	Foxtail, foxtail millet, other gramineae, and dicots	*C. plumosa*	Chlorotic local lesion	Sap inoculation	Paulsen and Niblett 1977
Betaflexiviridae							
Poplar mosaic virus (PopMV) (*Carlavirus*)	Poplar growing range	Infected cuttings	*Populus* sp.	*C. argentea*	Necrotic spots or rings with chlorotic halo	Sap inoculation	Schmelzer 1966
Closteroviridae							
Beet yellows virus (BYV) (*Closterovirus*)	Worldwide in beet growing regions	Aphids (e.g., *Myzus persicae*, green peach aphid)	*Beta vulgaris* (beetroot, sugarbeet); chrysanthemum, spinach	*Celosia* sp., *C. argentea*, *C. cristata* (cockscomb)	Moderately severe symptoms (not described)	*M. persicae*	Bennett 1960; Ruppel 1970; Russell, 1965
Luteoviridae							
Beet western yellows virus (BWYV) (*Polerovirus*)	North America, Europe, Asia	Aphids (e.g., *M. persicae*)	*Beta vulgaris*, lettuce, radish, spinach	*C. argentea* var. *cristata*	Not described	*M. persicae*	Duffus 1972; Smith 1972

Potyviridae							
Tobacco etch virus (TEV) (*Potyvirus*)	North, South America	Aphids (e.g., *M. persicae*)	Solanaceous crops (e.g., pepper, tomato, tobacco), perennial weeds	*C. argentea*	Local lesions include chlorotic spots	Sap inoculation	Holmes 1946; Laird and Dickson 1963; Purcifull and Hiebert 1982
Secoviridae							
Arabis mosaic virus (ArMV) (*Nepovirus*)	Europe	*Longidorus* sp., *Xiphinema* sp. nematodes	Celery, cucumber, lettuce, rhubarb strawberry, among others	*C. argentea*	Local reaction: chlorotic, necrotic spots; systemic leaf mosaic and distortion	Sap inoculation	Samuitienė et al. 2008
Tombusviridae							
Carnation Italian ringspot virus (CIRV) (*Tombusvirus*)	Europe, USA	Unknown	*Dianthus* sp.	*C. argentea*	Necrotic local lesions	Sap inoculation	Büttner et al. 1987; Hollings et al. 1970
Carnation mottle virus (CarMV) (*Carmovirus*)	Worldwide	Mechanical	*Dianthus* sp., begonia	*C. argentea*	Faint chlorotic local lesions; systemic infection symptomless	Sap inoculation	Brunt and Martelli 2008; Hollings and Stone 1964
Lisianthus necrosis virus (LNV) (*Necrovirus*)	Japan, Taiwan	*Olpidium*, mechanical	Lisianthus, carnation, calla lily	*C. argentea*	Local lesion	Sap inoculation	Chen et al. 2000; Chen et al. 2006

(*continued*)

Table 1 (continued)

Virus	Occurrence	Natural transmission	Natural hosts	Celosia hosts	Symptoms in celosia	Mechanism of experimental transmission	References
Virgaviridae							
Ribgrass mosaic virus (RMV) (*Tobamovirus*)	Worldwide	Mechanical, grafting	Ribgrass, broadleaf plantain, foxglove, Chinese cabbage, other solanaceous and cruciferous plants	*C. argentea*	Local reaction: necrotic spot; systemic malformed leaves	Sap inoculation	Kim et al. 2010
Tobacco mosaic virus (TMV) (*Tobamovirus*)	Worldwide	Mechanical	Wide host range, including tobacco, tomato, other solanaceous plants	*C. argentea*	Local reaction: chlorotic spots; systemic chlorotic spot and mottle	Sap inoculation	Holmes 1946
Tobacco rattle virus (TRV) (*Tobravirus*)	Brazil, Europe, Japan, USA	*Trichodorus* sp. and other nematodes	Wide host range, including many herbaceous ornamental plants, bean, pepper, potato, spinach	*C. argentea*	Local reaction: brown lesions; systemic leaf distortion, vein banding	Sap inoculation	Samuitienė et al. 2009

greenhouse was affected by the virus. Although CMV occurs in Africa (Nono-Womdim 2003) where *C. argentea* is widely grown as a vegetable, reports of CMV on this host on this continent are lacking.

Symptoms/signs. Symptoms of cucumber mosaic are systemic in most hosts, varying in severity depending on the species and growth stage of the crop affected (Zitter and Murphy 2009). Symptoms of typical virus symptoms may appear in infected hosts, affecting leaves (mosaic, mottle, ringspot, rolling, distortion, or yellowing), fruit and floral parts (malformation, rugosity, ringspots, or discoloration), or the entire plant (stunt, yellowing, development of axillary shoots, or death). Recorded symptoms in naturally infected plumed celosia (*C. argentea* var. *plumosa*) included ring spot, mottle, necrosis, and tissue distortion (Provvidenti 1975; Mali and Rajegore 1980) and in sap-inoculated *C. argentea* included local leaf lesions (chlorotic spots, oak leaf pattern, vein banding) (Hollings 1959) or leaf mosaic and mottle, reduced stem girth, and stunt (Aliyu et al. 2014).

Biology and epidemiology. Transmission of CMV throughout its host range is by one of 80 species of aphid (Hemiptera: *Aphidoidea*), through seed, or dodder (Bennett 1940; Zitter and Murphy 2009). The virus does not persist in the aphid vector; the insect acquires the virus in less than 60 sec and remains infective for a few minutes to hours, depending on the aphid species. The efficiency of transmission depends on the host, aphid species, virus strain, and environmental conditions. Seed transmission is common in some hosts (chickweed, for example). Specific transmission (vector, seed) data for CMV in celosia is lacking, although Provvidenti (1975) reported that the green peach aphid *Myzus persicae* was effective in transmission experiments in a New York greenhouse epidemic and that transmission of this virus through seed was not established. Crop hosts (including volunteer plants) and weeds (such as chickweed, common milkweed, and yellow toadflax) serve as sources of inoculum for many hosts of CMV (Zitter and Murphy 2009).

Management. Management of CMV includes monitoring, use of virus-free stock, weed control to remove sources of inoculum (particularly key weed species close to the crop), and control or exclusion of the aphid vector. Resistant cultivars are used for management of CMV in cucumber in spinach (Zitter and Murphy 2009); success is more variable in other crops. In a greenhouse study, Provvidenti (1975) inoculated four cultivars of cockscomb (*C. argentea* var. *cristata*) (Jewel Box, Kardinal, Fireglow, and Toreador) and five cultivars of plumed celosia (*C. argentea* var. *plumosa*) (Fairy Fountains, Forest Fire, Golden Triumph, Pampas Plume, and Red Fox) with CMV recovered from infected Fairy Fountains and chickweed. Although all plants were symptomatic (including ringspot, mottle, necrosis, and deformed tissues), disease severity varied in intensity depending on cultivar and viral isolate. No further data on disease development among the individual cultivars was presented; the report suggests, however, that all inoculated celosia were susceptible to this viral disease. Information on aphid virus vector control may be found in ► Chap. 4, "Insect Management for Disease Control in Florists' Crops."

4.2 Beet Curly Top (*Beet curly top virus*)

Geographic occurrence and impact. *Beet curly top virus* (BCTV) (*Curtovirus*: *Geminiviridae*) has a very wide host range (300 species from 44 families), primarily sugar and table beet (*Beta vulgaris*), other Amaranthaceae (chard, spinach, chenopods), solanaceous crops (pepper, potato, tomato), cucurbits (cucumber, melon, pumpkin, squash), and many others, including weeds (Harveson 2015). This virus disease likely originated in the Mediterranean Basin (Bennett and Tanrisever 1957) and occurs in Canada, Mexico, Central and South America, and parts of the USA (western, north-central, mid-western, and southern states) (EPPO/CABI 1997a).

Host range of BCTV in celosia includes *C. argentea* var. *cristata* (Smith 1972), *C. cristata* and *C. plumosa*, recorded by Dreistadt (2001) in California, and *C. argentea* (feather cockscomb) artificially inoculated by leafhoppers reared on infected beet (Freitag and Severin 1936). Although BCTV is one of the earliest recorded virus diseases of celosia, the economic impact and distribution of this disease is not known.

Symptoms/signs. General symptoms on many crops include yellows, leaf curl and distortion, vein clearing, and decline; in some economic crops, the virus disease is lethal. Symptoms in celosia include distorted foliage and blossoms, stunt and development of many axillary shoots, and plant death. Blossoms may appear blotched, mottled, or streaked with discolored, dwarfed heads (Dreistadt 2001; Freitag and Severin 1936).

Biology and epidemiology. Transmission of BCTV is characterized in studies of beet. The virus is vectored by two species of leafhoppers in the genus *Ciculifer* (Hemiptera: Cicadellidae): *C. tenellus* (USA, Mediterranean Basin), and *C. opacipennis* (Mediterranean Basin) (EPPO/CABI 1997a). BCTV is circulative but not propagative; a four-hour latent period is required between vector acquisition and transmission. The virus is not transmitted to eggs, and leafhopper adults remain infective for a considerable period (3 months or more) (Harveson 2015; Stahl and Carsner 1923). There are no reports that BCTV is seed or tuber transmitted in beet (EPPO/CABI 1997a). The disease appears where populations of the vector and weed hosts that serve as inoculum reservoirs occur. Movement of infected plant host material results in long distance disease spread.

Management. Curly top in beet is best managed by using resistant varieties where identified that are adapted to local growing conditions. Management in celosia includes monitoring and use of virus-free stock, weed control to remove sources of inoculum, and control or exclusion of vector (leafhopper) populations (Dreistadt 2001).

4.3 Tomato Spotted Wilt, Impatiens Necrotic Spot (*Tospovirus*)

Geographic occurrence and impact. Tospoviruses in the Bunyaviridae family, such as *Tomato spotted wilt virus* (TSWV) and *Impatiens necrotic spot virus* (INSV),

affect over 1000 monocots and dicots worldwide and cause economic loss in many vegetable, field crop, cucurbit, solanaceous (such as potato, tomato, eggplant), herb, and herbaceous ornamental crops (such as aster, begonia, chrysanthemum, and impatiens), as well as many weeds (Cho et al. 1986; German et al. 1992; Kazinczi et al. 2007; Sherwood et al. 2003). TSWV and INSV are associated with outbreaks in ornamental crops throughout the USA and Europe; while TSWV is more readily associated with vegetable and field crops, INSV is more common in the greenhouse (Daughtrey et al. 1997). Reports of tospovirus in celosia include TSWV in the Mediterranean (*Celosia* sp., EPPO/CABI 1997b), USA (*C. cristata*, Hausbeck et al. 1992), Greece (*C. cristata*, Chatzivassiliou et al. 2000), and Israel (*Celosia* sp., Antignus et al. 1997) and INSV in the USA (*Celosia* sp., Hudelson et al. 2015, McDonough et al. 1999).

Symptoms/signs. General symptoms of tospoviruses vary with host species and age of infection, strain of virus, and environmental factors (German et al. 1992; Mumford et al. 1996). Symptoms include chlorosis and necrosis in local lesion hosts and compromised leaf, stem, and fruit tissues (bronzing, chlorosis, line patterns, necrosis, ring spot, silvering, stunt, and wilt) in systemic hosts. Infection is more severe or may be lethal in younger plants (Moyer et al. 1999; Sherwood et al. 2003). Symptoms in celosia are typical of that seen in other crops, including leaf mottle, mosaic, and ring spot, necrosis, and stunt (Antignus et al. 1997; Hausbeck et al. 1992).

Biology and epidemiology. Tospoviruses are naturally vectored in a circulative (persistent) and propagative manner by several thrips (Thysanoptera: *Thripidae*) in the *Frankliniella*, *Scirtothrips*, and *Thrips* genera. Of these, an important vector of tospovirus in ornamental crops is the polyphagous western flower thrips (*Frankliniella occidentalis*) (Kazinczi et al. 2007). Tospovirus outbreaks in countries such as Israel (Antignus et al. 1997), Greece (Chatzivassiliou et al. 2000), and Egypt (El-Wahab et al. 2011) have been associated with population increases of this insect, most likely enhanced by long-distance movement of infected plant material (Kazinczi et al. 2007; Kirk and Terry 2003). Only the first and early second larval stages are able to acquire tospoviruses, and only immature thrips that acquire these viruses or adults derived from such immatures are vectors. Adult thrips remain viruliferous for life; but tospoviruses are not transovarial. Seed transmission is not well documented. As with other viruses of ornamental plants, tospovirus diseases can appear, especially in greenhouse crops, where populations of thrips and nearby reservoirs of inoculum, such as weeds, overlapping crops, and unsold diseased plants occur (Daughtrey et al. 1997).

Management. Viruses are not subject to direct chemical control. As such, management of tospovirus in ornamental crops includes strategies that reduce inoculum and disease spread: use stock free of disease (especially in nurseries), monitor existing stock for symptoms and thrips populations, exclude vectors from greenhouses, rogue diseased stock, and remove weeds and other plants that may harbor virus and are

feeding host for thrips vectors. Many ornamental plants, including bedding plants, are susceptible to tospoviruses; cultivars of celosia resistant to these virus diseases have not been identified. In countries where tospovirus incidence is rare, the introduction of western flower thrips and other tospovirus vectors to locales where susceptible crops are grown should be prevented (EPPO/CABI 1997b). Information on thrips virus vector control may be found in ▶ Chaps. 4, "Insect Management for Disease Control in Florists' Crops," and ▶ 20, "Diseases of Lisianthus," Sect. 4.2, Bunyaviridae.

4.4 Celosia Mosaic (*Celosia mosaic virus*)

Geographic occurrence and impact. A previously undescribed virus isolated from vegetable-grown *C. argentea* from farms near Lagos, Nigeria, was characterized and its host range and transmission determined (Owolabi et al. 1998). Incidence of this disease in farms in the region varied from 0.6% to 27.5% (Taiwo and Owolabi 2004). The host range of the proposed new virus, *Celosia mosaic virus* (CIMV) (*Potyviridae*) was limited to a few species of other amaranths and solanaceous crops. Since the first report, the virus has not been recorded outside Nigeria.

Symptoms/signs. Symptoms of CIMV in *C. argentea* include leaf curl and mosaic (Owolabi et al. 1998). In transmission experiments, symptoms on 3- to 4-week seedlings (reduced leaf size and number, plant height, and shoot fresh and dry weight) were more severe than that of older plants (5–7 week) (Owolabi and Taiwo 2001).

Biology and epidemiology. Owolabi et al. (1998) reported that CIMV is transmitted in a nonpersistent manner by the aphid species *Aphis spiraecola* and *Toxoptera citricida* (Hemiptera: *Aphidoidea*). The authors found that the virus was not transmitted through seed. In general, virus incidence is higher during wet seasons (Taiwo and Owolabi 2004).

Management. In addition to management strategies for plant viruses that reduce insect transmission, Taiwo and Owolabi (2004) suggest that management of CIMV in celosia might also benefit from removing sources of inoculum, such as volunteer crops.

5 Nematode Diseases

5.1 Root-Knot (*Meloidogyne* spp.)

Geographic occurrence and impact. Root-knot nematodes (*Meloidogyne* sp.) (Tylenchida: Heteroderidae) are soil-dwelling, obligate parasites that feed on plant roots. These pathogens have a wide host range and most are distributed in tropical to

subtropical regions and greenhouses worldwide (Mitkowski and Abawi 2003). *Celosia* spp. are reported moderately to highly susceptible to three species of root-knot: *M. incognita* (Kofoid & White) Chitwood (root-knot or southern root-knot, races 1 and 3), *M. javanica* (Treub) Chitwood (sugarcane eelworm), and *M. arenaria* (Neal) Chitwood (peanut root-knot) (Goodey et al. 1965; McSorley and Fredrick 1994).

The disease is reported in warmer regions of the USA as *Meloidogyne* sp. on *Celosia* sp. (Kaiser et al. 1983), *C. argentea* (Hagan 2005; United States Agricultural Research Service 1960), and *C. plumosa* Fresh Look Mix *(M. incognita* race 3, Hagan and Arkidge 2009). In other parts of the world, root-knot on *C. argentea* was recorded in Brazil (*M. javanica*, Filho et al. 1971), Nigeria (*M. incognita* on *C. argentea* grown as a vegetable, Daramola et al. 2015), and Pakistan (*M. incognita*, Anwar et al. 2009; *M. incognita* race 1 and *M. javanica*, Maqbool et al. 1986).

Symptoms/signs. Feeding sites for root-knot nematodes are collections of cells (giant-cells) that enlarge to funnel carbohydrate and plant nutrients to the animal. Growth regulators from the nematode cause neighboring cells to increase in size, forming the large multinucleate "knots" or galls that comprise the symptom for root-knot disease. These knots can be inconsequential to quite large, disrupting root function, limiting the capacity to transport water and nutrients, and reducing the market value of root crops (Agrios 2005). As such, aboveground symptoms include wilt, stunt, chlorosis, and reduced yield. Younger plants with minimal root development may die (Mitkowski and Abawi 2003). Root-knot can frequently occur with other soilborne diseases such as Pythium root rot (Fig. 4.)

Symptoms of root-knot on celosia include leaf chlorosis, necrosis, droop, poor development and premature drop, progressive wilt, stunt, reduced stem diameter, and reduced yields (Anwar et al. 2009; Caveness and Wilson 1977; Tobih et al. 2011). Gall formation is heavy in susceptible plants; affected roots may rot and, as reported for other crops (Carter 1975; Jacobsen et al. 1979; Mai and Abawi 1987), be associated with other soilborne pathogens such as *Fusarium* and *Rhizoctonia*. Anwar et al. (2009) isolated *Fusarium* sp., *Pythium* sp., and *Rhizoctonia* sp. from galled and rooted roots of *C. argentea* (cockscomb) in a landscape planting.

Biology and epidemiology. Like other nematodes, the root-knot nematode undergoes molts from egg, through four larval stages (J1, J2, J3, J4), to the adult male or female. Root-knot nematodes are sedentary endoparasites: the first molt occurs in the egg; the J2 larvae, or infective larval stage, emerges from the egg and penetrates plant roots near the root tip. The larva establishes a feeding site (giant-cell) near developing vascular cells, becomes sedentary, and molts three more times to the adult. The mature, sac-shaped (globose) female may deposit 500 or more eggs within a protective gelatinous matrix inside or outside the root (Mitkowski and Abawi 2003). The life cycle varies but may be as brief as 2 week depending on host species, food availability, and temperature.

Root-knot nematodes survive up to 1 year as eggs associated with plant debris or in soil and are easily dispersed with contaminated plant material or soil particles.

Since these animals are biotrophs, distribution in the field is uneven and the highest populations of eggs and larvae are associated with plant roots. Damage to crops depends on factors such as nematode species and race, preplant nematode population density, host species and cultivar, and environmental conditions, especially temperature or drying (Barker and Olthof 1976).

Management

- ***Cultural practices*** – Use of resistant plant material where identified is of prime importance for root-knot control (Hagan 2005). In general, however, *Celosia* sp. are considered moderately to highly susceptible to root-knot nematodes (Daramola et al. 2015). Celosia cultivars reported as susceptible to one or more root-knot species (*M. incognita* race 1 or 3, *M. arenaria*, and *M. javanica*) in field or greenhouse studies include *C. argentea* Century Mix (McSorley and Frederick 1994); *C. argentea* Bombay and Kurume (Kokalis-Burelle and Rosskopf 2013); *C. plumosa* Apricot Brandy, Castle Scarlet, Fireglow, and Kimono Cream (Walker et al. 1994); and *C. plumosa* Fresh Look Mix, Fresh Look Gold, Fresh Look Yellow, and Bombay Orange (Hagan and Arkidge 2009). Other less susceptible bedding plants may be acceptable alternatives, such as ageratum, alyssum, amaranth, cosmos, globe amaranth, lantana, marigold, melampodium, salvia, scabiosa, sunflower, tickweed, vinca, and zinnia (Hagan and Arkidge 2009; McSorley and Frederick 1994), but caution is needed: susceptibility or resistance to root-knot can be cultivar or nematode dependent, and testing of different cultivars may be limited. Other management options for root-knot nematodes include crop rotation, cover crops or organic amendments, use of nematode suppressive plants (such as French marigold), heat treatments (such as steam and soil solarization), and sanitation practices to reduce spread (Perry and Ploeg 2010). Some of these strategies have potential in Nigeria where *C. argentea* is grown as a vegetable; Tobih et al. (2011) reported that *Chromolaena odorata*, a perennial herb in the sunflower family, applied as an organic mulch to nursery plots of *C. argentea* was, compared to controls, moderately effective, and neem compost reduced root-knot nematode populations and increased yields in field trials (Olabiyi et al. 2013). Caveness and Wilson (1977) suggest that vegetable celosia crops should be grown in a rotation scheme to keep nematode populations below economic thresholds. Further strategies to manage nematode diseases may be found in ▶ Chap. 8, "Soil/Media Disinfestation for Management of Florists' Crops Diseases."

Additional Nematode Pathogens Other species of pathogenic nematodes associated with the roots or rhizosphere soil of *Celosia* spp. include: *Mesocriconema mutabile*, *Paratrichodorus minor*, *Paratylenchus* spp., and *Xiphenema americanum* (Elmore et al. 2007) as well as unidentified species of *Belonolaimus*, *Criconema*, *Helicotylenchus*, *Hoplolaimus*, *Pratylenchus*, *Rotylenchus*, *Scutellonema*, *Tylenchus*, and *Tylenchorhynchus* (Anwar et al. 2009; Daramola et al. 2015).

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Diseases of China Aster

15

Harukuni Horita and Robert J. McGovern

Abstract

China aster, *Callistephus chinensis* (L.) Nees., family Asteraceae, is a popular ornamental flower worldwide. Diverse cultivars have been bred for garden, potted, and cut flowers. Numerous diseases have been reported in the cultivation of China aster. Frequently occurring and highly damaging diseases are caused by *Botrytis cinerea*, *Fusarium oxysporum* f. sp. *callistephi*, and an ever-increasing number of viruses, including tospoviruses. Recent studies on management strategies for China aster diseases include chemical, cultural, and physical control, sanitation, host resistance, and their integration.

Keywords

Botrytis cinerea • *Fusarium oxysporum* f. sp. *callistephi* • *Tospovirus* spp.

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R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_15

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1 Introduction

China aster, *Callistephus chinensis* (L.) Nees., family Asteraceae, is a popular flower worldwide. China aster seeds were first introduced from China into France in 1731, and cultivation of the flower spread across Europe (Tsurushima 1983). Diverse cultivars for garden flowers were bred in France, Germany, and the UK. China aster was introduced into Japan from China in the 1700s, and many cut flower cultivars such as the Matsumoto series have been bred. In the 1800s, China aster was introduced into the USA, and a vast array of different cultivars was developed for cut flower production. Fusarium wilt has caused the greatest damage to the China aster cultivation, and a number of control measures have been developed for this challenging disease. Outbreaks of other China aster diseases that limit its production have also been reported such as those caused by viruses and phytoplasma. This chapter covers diseases that occur in China aster and describes their epidemiology and management. Also refer to the chapters in the introductory part "Components of Integrated Disease Management" for general strategies applicable to florists' crops.

2 Fungal and Fungus-Like Diseases

2.1 Botrytis Blight, Gray Mold (*Botrytis cinerea* Pers.)

Geographic occurrence and impact. Botrytis blight or gray mold occurs in many floral plants and other commercial crops worldwide and causes extensive damage both during production and after harvest (Horita 1995; Williamson et al. 2007).

Symptoms/signs. Initially brown lesions are observed in the margins of young leaves. The lesions rapidly enlarge to blight entire leaves. Blighted tissues expand to the petioles and stems. Lesions may girdle stems, which exhibit an internal brown discoloration, and stems die above the infection site. When stems are infected at the soil line, the whole plant ultimately wilts and dies. The pathogen also causes petal



Fig. 1 Botrytis gray mold symptoms/signs: leaf blight with profuse sporulation (*left*), flower blight with gray mycelium (*right*) (H. Horita © 2017. All Rights Reserved. (*left*), R. J. McGovern © 2017. All Rights Reserved. (*right*))

blight under cool, moist conditions. Growth of *B. cinerea* on leaves, stems, and flowers is indicated by the presence of a profuse gray mold which consists of masses of fungal hyphae and spores (Fig. 1).

Biology and epidemiology. *Botrytis cinerea* is an airborne pathogen which infects over 200 hosts including all floral crops. The fungus can survive as a pathogen or saprophyte in infected plant debris. *Botrytis cinerea* also produces small, black stress-resistant sclerotia. Sclerotia and mycelia in infected tissues enable long-term survival during unfavorable periods. When disease-conducive conditions return, quiescent mycelia become active, and sclerotia germinate and produce mycelia and conidiophores. The conidia of *Botrytis* are dispersed by wind and rainfall and cause new infections. *Botrytis cinerea* is favored by cool, moist conditions. A severe outbreak of gray mold in China aster was observed during the rainy season in cultivated fields in Japan (Nishikado and Inoue 1956). Seed infection by *Botrytis* sp. of China aster was reported in Poland (Rymar 1984).

Management

- ***Cultural practices and sanitation*** – Remove dead and dying leaves and flowers from plants and the soil surface. In greenhouse cultivation, avoid overhead irrigation, or if this is not possible, water early in the day to reduce the duration of leaf wetness. Increasing plant spacing will increase ventilation and also reduce leaf wetness. Bertus (1967) reported control of *B. cinerea* and *Alternaria tenuis* on China aster seed by treatment with aerated steam at 53 °C/127 °F for 30 min.
- ***Fungicides*** – Rymar (1984) found that carbendazim, benomyl, and thiophanate methyl were effective in treating aster seed contaminated with *Botrytis* sp.. However, widespread resistance to thiophanate methyl and iprodione has occurred in *B. cinerea* in floral crops (Daughtrey et al. 2000). Thiophanate methyl resistance frequency in China aster isolates of *B. cinerea* in Japan was reported to be 31% (Irie et al. 1989). The fungicide azoxystrobin and natural product D-limonene inhibited the development *of B. cinerea* in China aster in Poland

(Nawrocki 2013). It is important to rotate fungicides with different modes of action in order to prevent the development of resistance. Information on fungicide mode of action and resistance management strategies can be found at the Fungicides Resistance Action Committee (FRAC) website: http://www.frac.info/.

2.2 Fusarium Root Rot, Wilt (*Fusarium oxysporum* Schlechtend.: Fr. f. sp. *callistephi* (Beach) W.C. Snyder & H.N. Hans; *Fusarium* sp.)

Geographic occurrence and impact. Fusarium wilt of China aster has occurred in Australia, the Czech Republic, Germany, Japan, New Zealand, Poland, and the USA (Cunningham 1931; Hanning and Orlicz-Luthardt 2002; Kodama and Horita 1995; Nečas and Kobza 2008; Rataj-Guranowska et al. 2007; Simmonds 1956; Snyder and Hansen 1940). Disease incidences of Fusarium wilt up to 71.2% were reported in the USA (Connecticut and Florida) (Elmer and McGovern 2013).

Symptoms/signs. Initial symptoms are observed in the seedling stage as damping-off. The stems of tiny seedlings rot at the soil line, the cotyledons wither, and the plants fall over. At the transplant stage, infected plants are stunted, have brown lower leaves, wilt during the heat of the day, and die. Brown lesions occur in the stem bases, and black streaks extend to the upper stems at harvest. Vascular discoloration is visible when infected stems are cut lengthwise. White mycelia and masses of salmon-colored spores are produced on stem bases of dead and declining plants (Fig. 2).

Biology and epidemiology. *Fusarium oxysporum* f. sp. *callistephi* produces three types of asexual spores, microconidia and macroconidia, which enable aerial dissemination, and thick-walled, stress-resistant chlamydospores which enable the



Fig. 2 Fusarium wilt symptoms: at the transplant stage (*left*), at harvest (*center*), root rot and vascular discoloration (*right*) (H. Horita © 2017. All Rights Reserved. (*left*), R.J. McGovern © 2017. All Rights Reserved. (*center*, *right*))

survival of the pathogen for many years even in the absence of aster plants. All of the *F. oxysporum* f. sp. *callistephi* isolates are highly virulent to China aster; however, some isolates also exhibit a lower virulence to *Dianthus caryophyllus*, *Narcissus* sp., and *Tulipa gesneriana* (Rataj-Guranowska et al. 2007). *Fusarium oxysporum* f. sp. *dianthus* showed a low level of virulence in China aster but may aggravate Fusarium wilt caused by *F. oxysporum* f. sp. *callistephi* (Orlicz-Luthardt et al. 2000). Four races of *F. oxysporum* f. sp. *callistephi* were identified under experimental conditions based on differential pathogenicity to China aster cultivars (Armstrong and Armstrong 1971). Sources of the pathogen are plant debris in the soil and seed infection (Neergaard 1977). Elmer and McGovern (2013) attributed serious outbreaks of Fusarium wilt in the USA to a high incidence (12%) of the pathogen on seed. The pathogen is also moved on infested tools, equipment, and shoes. Warm temperatures (~27 °C/80 °F) favor the disease, as well as acidic soils and soils that have been disinfested by steam or fumigation and subsequently re-infested by *F. oxysporum* f. sp. *callistephi*, e.g., through infected transplants (Baker 1953), presumably because of the elimination of microorganisms which are antagonistic to the pathogen.

Management

- ***Sanitation and cultural practices*** – Sodium hypochlorite was effective in eliminating the fungus from seeds (Elmer and McGovern 2013). Rymar (1984) found that carbendazim, benomyl, and thiophanate methyl were effective in treating aster seed contaminated with *Fusarium* sp.. The reuse of propagative containers such as transplant trays should be avoided without thorough chemical or steam disinfestation. The use of pathogen-free transplants is essential. Avoid fertilizing with ammoniacal sources of nitrogen. Lime the soil to 6.5–7.0.
- ***Soil disinfestation*** – Elmer and McGovern (2013) demonstrated that soil disinfestation with methyl bromide + chloropicrin, 1,3-dichloropropene + chloropicrin, or metam sodium maintained Fusarium wilt at a low level in the USA (Florida). The incidence of Fusarium wilt was reduced by 73.4% by soil solarization conducted for 3–4 weeks in Taiwan using clear 0.025 mm-thick polyethylene mulch (Huang and Sun 1991).
- ***Resistance*** – Nečas and Kobza (2008) in the Czech Republic classified China aster cultivars according to their reactions to artificial inoculation with *F. oxysporum* f. sp. *callistephi* as resistant, moderately resistant, and sensitive. The "Matsumoto", "Einf. Madeleine", and "Americká kráska" series were found to be resistant in these trials. The "Bouquet Powderpuff Mix", "Benary's Princess Formula Mix", "Finest Mixed", and "Matsumoto Formula Mix" series were rated as resistant in a greenhouse trial, and the "Bouquet Powderpuff Mix", "Astoria Mix", "Aster Sandy Mix", and "Aster Composition" (Stokes Seeds) series were moderately resistant to the fungus in field trials in Connecticut (Elmer and McGovern 2013). Interestingly, the densities of *Trichoderma viride*, *T. polysporum*, and *Penicillium jensenii*, fungal antagonists of *F. oxysporum* f. sp. *callistephi*, were found to be higher in the root system of a resistant aster cultivar than in a susceptible one in a field experiment in Poland (Manka and Kacprzak 1998).

- ***Fungicides and biocontrols*** – The biocontrol *Pythium oligandrum* and the fungicide azoxystrobin were effective in reducing infection of China aster plants by *F. oxysporum* in Poland (Nawrocki 2013).

2.3 Itersonilia Petal Blight (*Itersonilia perplexans* derx)

Geographic occurrence and impact. To date, Itersonilia petal blight of China aster has only been reported in the USA (Florida, Hawaii) (McGovern and Seijo 1999; J. C. Trolinger personal communication). In Florida, 10–50% postharvest losses occurred (McGovern and Seijo 1999).

Symptoms/signs. Initially the fungus causes pinpoint, bleached lesions on petals. The lesions enlarge and result in bleached streaks on ray flowers. Eventually the entire blossom turns brown (Fig. 3).

Biology and epidemiology. Growth, spore production, and infection by *I. perplexans* are favored by plentiful rainfall, high RH (>70%), and cool temperatures (10–15 °C/ 50–59 °F) (Brown et al. 1964; Horita et al 2005; Horst 1997). Often symptoms of the disease are not apparent until after postharvest processing. Two groups of *I. perplexans* have been reported which are specific to Asteraceae or Apiaceae. In host range tests, China aster isolates were pathogenic to Asteraceae, including such florists' crops as chrysanthemum, gerbera and sunflower, but not to Apiaceae (McGovern et al. 2006). *Itersonilia perplexans* was isolated from *Emilia fosbergii*, Asteraceae, a weed species growing adjacent to a China aster production facility that has had a long history of Itersonilia petal blight outbreaks (Fig. 3). Since *Emilia* isolates produced severe petal blight on China aster, this weed represents a remarkable reservoir of this cool-season fungus that enables it to survive in a subtropical climate.

Management

- ***Cultural practices*** – The use of effective sanitation methods, such as removal of non-harvested flowers and weed hosts, especially Asteraceae, is critical in controlling Itersonilia petal blight.

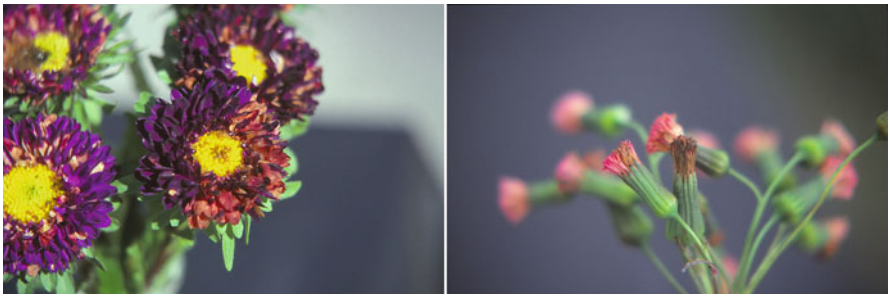


Fig. 3 Itersonilia petal blight symptoms: China aster (*left*), weed host – *Emilia fosbergii* (*right*) (H. Horita © 2017. All Rights Reserved.)

- ***Fungicides*** – McGovern et al. (2006) found that myclobutanil, propiconazole, and potassium bicarbonate were generally more effective than azoxystrobin in lowering the severity levels of petal blight on detached flowers in controlled environment experiments.

2.4 Rhizoctonia Stem and Root Rot, Blight [*Rhizoctonia solani* Kühn, (Teleomorph: *Thanatephorus cucumeris* (A.B. Frank) Donk)]

Geographic occurrence and impact. This disease has occurred in China aster in Finland (Linasalmi 1952), Japan (Ito et al. 2006), and the USA (Ullstrup 1936).

Symptoms/signs. Initially, infected plants have light brown lesions on stems at the soil line. The lesions enlarge to girdle the stems and spread to the root system. As stem girdling and root rot progresses, infected plants become stunted, wilt, and die. Occasionally, blight symptoms appear on the lower leaves in contact with the soil. Eventually dark brown hyphae are readily visible on the surface of stems at the soil line. Small, black survival structures, sclerotia, may form in mycelia of the fungus.

Biology and epidemiology. *Rhizoctonia solani* is a major pathogen with worldwide distribution which infects most cultivated crops and weeds. China aster isolates of *R. solani* belong to hyphal anastomosis group AG-2-2 (Ito et al. 2006). They are very active at high temperatures (35 °C/95 °F) (Ullstrup 1936). The fungus survives as hyphae and sclerotia in plant debris or soil, and seed infection has also been detected in China aster (Crosier 1968).

Management

- ***Cultural practices*** – Rapidly remove infected plants. Excessive irrigation and, where possible, high temperatures should be avoided.
- ***Soil disinfestation*** – Soil disinfestation strategies include fumigation, steam, or soil solarization and preventative fungicide application.
- ***Fungicides and biocontrol***s – Prior to planting or transplanting, incorporate PCNB or *Trichoderma* spp. into the top 2.5 cm of soil; spray the bases of seedlings with thiophanate methyl, iprodione, or *Trichoderma* spp. (Koike et al. 2016). A talc-based formulation of *Pseudomonas fluorescens* combined with thiophanate methyl applied to soil was found to be effective in increasing germination of China aster seed and in decreasing root rot incidence in India (Babu et al. 2013).

2.5 Additional Fungal and Fungus-Like Diseases

The following fungal diseases of China aster have also been reported:

– **Alternaria leaf spot**, **flower blight** [*Alternaria alternata* (Fr.) Keissl. *Alternaria* sp.] (the USA, Baker and Davis 1950a)

- **Anthracnose** [*Colletotrichum gloeosporioides* (Penz.) Penz. and Sacc., (teleomorph: *Glomerella cingulata* (Stoneman) Spauld. & H. Schrenk; *Colletotrichum* sp.] [the USA (Florida), Alfieri et al. 1994]
- **Ascochyta leaf spot** [*Ascochyta asteis* (Bres.) Gloyer; *Ascochyta* sp.] (Germany, Gloyer 1924)
- **Camarosporium leaf spot** (*Camarosporium eriogonii* Ellis & Everh.) [the USA (Florida), Alfieri et al. 1994]
- **Downy mildew** (*Basidiophora entospora* Rose & Cornu) [the USA (Florida), Alfieri et al. 1994]
- **Phialophora wilt** [*Phialophora asteris* (Dowson) Burge & Isaac] (UK, Burge and Isacc 1974)
- **Phomopsis stem canker** [*Phomopsis callistephi* Tehon & E. Y. Daniels] (the USA, Tehon and Daniels 1925)
- **Phymatotrichopsis root rot** [*Phymatotrichopsis omnivora* (Duggar) Hennebert (syn. *Phymatotrichum omnivorum*)] (the USA, Farr et al. 1989)
- **Phytophthora root and foot rot** [*Phytophthora cryptogea* Pethybr. & Laff., *Phytophthora* sp.] (Ireland etc. Erwin and Ribeiro 1996)
- **Powdery mildew** [*Oidium asteris-punicei* Peck (teleomorph: *Golovinomyces cichoracearum* (DC.) V.P. Heluta) (as "*cichoraceorum*") (syn: *Erysiphe cichoracearum*)]; *Erysiphe polygoni*] (Japan, Sato et al. 1992)
- **Pythium root and stem rot**, **wilt** (*Pythium splendens* H. Braun, *P. ultimum*, *Pythium* sp.) (Japan, Tasugi and Siino 1940)
- **Rust** [(*Coleosporium asterum* (Dietel) Syd. & P. Syd.] [the USA (California), (Raabe and Pyeatt 1990)]
- **Sclerotinia rot** [*Sclerotinia sclerotiorum* (Lib.) de Bary] (Japan, Narita 1998)
- **Sclerotium blight** [*Sclerotium rolfsii* Sacc. (teleomorph: *Athelia rolfsii* (Curzi) Tu & Kimbrough] [the USA (Florida), Alfieri et al. 1994]
- **Septoria leaf spot** [*Septoria atropurpurea* Peck, *S. callistephi* Gloyer (1921) (teleomorph: *Mycosphaerella* sp.)] (Poland, Madej 1970)
- **Stemphylium leaf spot** [*Stemphylium botryosum* Wallr., (teleomorph: *Pleospora tarda* E. Simmons); *S. lycopersici* (Enjoji) W. Yamamoto (syn. *S. floridanum* Hannon & G.F. Weber)] (the USA, Baker and Davis 1950b)
- **Verticillium wilt** (*Verticillium albo-atrum* Reinke & Berthier) (the USA, Baker et al. 1940)

3 Bacterial and Phytoplasma Diseases

3.1 Aster Yellows ('*Candidatus* Phytoplasma asteris')

Geographic occurrence and impact. Aster yellows in China aster has been reported in Canada, Germany, Japan, Myanmar, and the USA (Illinois) (Babadoost 1988; Lee et al. 2004; Murayama et al. 1963; Win et al. 2011).

Symptoms/signs. The China aster phytoplasma produces a range of symptoms which include yellowing of leaves, stunting of whole plants, and yellow-green discoloration of flowers (Fig. 4). Flower virescence (greening of flowers) has also been observed in Myanmar (Win et al. 2011).

Biology and epidemiology. '*Candidatus* Phytoplasma asteris,' which comprises the aster yellows group, is associated with diseases of over 100 plant species. The China aster phytoplasma strains belong to the 16SrI group based on analysis of the 16S rDNA sequences (Lee et al. 2004). A different group of phytoplasma, 16SrII, was reported in China aster in Myanmar (Win et al 2011). Aster leafhoppers [*Macrosteles fascifrons* (Hemiptera: Cicadellidae)] acquire the phytoplasma and spread it to new plants (Babadoost 1988). Infection of aster yellows in temperate areas occurs by migration of leafhoppers that overwinter on infected host plants in warmer areas. However, high temperatures (approximately 31 °C/88 °F) inactivate the phytoplasma in leafhoppers and plants.

Management

- ***Cultural practices*** – Rapidly remove infected plants. Eradicate susceptible hosts (including weeds) around China aster production sites. Use reflective mulches in field-grown asters to disorient the vectors. In greenhouse cultivation, use physical barriers such as fine mesh nets or screens to exclude the leafhoppers.



Fig. 4 Aster yellows symptoms: infected plant (*left*), healthy plant (*right*) (Whitney Cranshaw © 2017. All Rights Reserved.)

3.2 Bacterial Wilt [*Dickeya* sp. (Formerly *Erwinia chrysanthemi*]

Geographic occurrence and impact. This disease has occurred in Japan (Komatsu et al. 2002).

Symptoms/signs. Early symptoms include water-soaked, black lesions in the stem at the soil line. The rot gradually expands, petioles exhibit a black rot, and the foliage yellows. The plants eventually wilt and die. Infected stems reveal a typical black discoloration of the vascular tissue when dissected (Fig. 5).

Biology and epidemiology. Komatsu et al. (2002) identified the causal pathogen as *Erwinia chrysanthemi*. Samson et al. (2005) proposed that *Pectobacterium chrysanthemi* (=*Erwinia chrysanthemi*) and *Brenneria paradisiaca* be reclassified as a novel genus, *Dickeya*, which includes six species. However, *Dickeya* from China aster has not yet been speciated. The bacterium is very active at warm temperatures and spreads to new plants by splashing water from infected plants and via infested tools.



Fig. 5 Bacterial wilt symptoms: stem rot at the soil line (Komatsu et al. 2002)

Management

- ***Cultural and sanitation practices*** – Rapidly remove infected plants. Disinfest tools.
- ***Resistance*** – Greenhouse trials conducted in Japan indicated that the China aster cvs. Stella and Top Rose were partially resistant to the disease (Komatsu et al. 2002).

3.3 Additional Bacterial Disease

The following bacterial disease of China aster has also been reported:

– **Bacterial black spot** [*Pseudomonas viridiflava* (Burkholder 1930) Dowson 1939] (Japan, Moriwaki et al. 2013)

4 Viral Diseases

4.1 Bromoviridae *Cucumber Mosaic Virus* (CMV, Genus: *Cucumovirus*)

Geographic occurrence and impact. This disease has occurred in Korea (Oh et al. 2008) and in the USA (Ferreira 1992).

Symptoms/signs. Symptoms on China aster are a severe leaf mosaic.

Biology and epidemiology. In experiments using dsRNA, RT-PCR, and restriction enzyme analysis, CMV isolated from China aster belongs to subgroup IA. CMV is spread by a number of aphids (Hemiptera: Aphididae), including the green peach aphid, *Myzus persicae*, in a noncirculative (nonpersistent) manner. The virus infects numerous vegetables, ornamentals, and weeds.

Management. Virus management is based on reduction of vector populations through chemical or biological measures. In greenhouse cultivation, use physical barriers, such as fine mesh nets or screens, to exclude the aphids, or reflective mulches to disorient them in field-produced asters. Weeds growing near China aster production sites should be removed in order to eliminate reservoirs of the virus and its vectors.

4.2 Bunyaviridae (Genus: *Tospovirus*)

Geographic occurrence and impact. Thus far five viruses in genus *Tospovirus*, Bunyaviridae, have been detected in China aster: *Chrysanthemum stem necrosis virus* (CSNV), *Groundnut ringspot virus* (GRSV), *Tomato chlorotic spot virus* (TCSV), and *Tomato spotted wilt virus* (TSWV) (Alexandre et al. 1999; Bakardjieva et al. 1998; Chatzivassiliou et al. 2000; Ishikawa et al. 2002; Kim et al. 2006;

Kitajima 2015; Latham and Jones 1997; Marchoux et al. 1991; Momonoi et al. 2011; Ramasso et al. 1994; Sether and DeAngelis 1992). Alexandre et al. (1999) found that one China aster plant in Brazil was coinfected with CSNV + GRSV. The geographic occurrence of these viruses in China aster is indicated in Table 1. CSNV symptoms were observed on about 6,000 China aster plants in a commercial facility in Japan (Toyama Prefecture) (Momonoi 2011). The incidence of TSWV was 10% in seedlings in a greenhouse in Japan (Ishikawa et al. 2002). In open-field surveys in Greece, the incidence of TSWV was 37.7% (Chatzivassiliou et al. 2000).

Symptoms/signs. The range of symptoms caused by tospoviruses in China aster is presented in Table 1.

Biology and epidemiology. The tospoviruses are vectored in a circulative and propagative manner (they enter and replicate within the insect's hemolymph) by a number of thrips species (Thysanoptera: Thripidae) (Table 1). Only first- and early second-instar larvae can acquire tospoviruses, and only immature thrips that acquire these viruses or adults derived from such immatures are vectors. Adult thrips remain viruliferous for life; but tospoviruses are not transovarial. Tospoviruses may be disseminated over long distances in vegetatively propagated plant material; but seed infection has not been reported. Therefore, thrips population dynamics and the proximity of infected reservoir plants are the determining factors in *Tospovirus* outbreaks. Momonoi et al. (2011) detected CSNV in chrysanthemum, tomato, China aster, and lisianthus in the same transplant facility and hypothesized that viruliferous *Frankliniella occidentalis* and *F. schultzei* were dispersed from chrysanthemum to China aster and other plants.

Management

- ***Cultural practices*** – Use virus-free transplants, fine mesh screening for greenhouse culture to exclude thrips, and reflective mulches in the field to disorient the vectors. Workers who handle plants should avoid wearing yellow- and blue-colored clothing which attracts thrips.
- ***Sanitation*** – Removal of weeds and other hosts of tospoviruses and their vectors from greenhouses and the surrounding area is essential. Momonoi (2011) reported that removal of host plants and weeds in and around a transplant facility alone was not effective for controlling CSNV. However, CSNV and thrips could be eradicated by these methods when practiced during the winter season. Similarly, Kakizaki (2005) reported that *F. occidentalis* was eradicated by the removal of a greenhouse plastic film during the winter (approximately 4 months) in Hokkaido, the northern part of Japan, presumably by cold temperatures.
- ***Scouting*** – Thrips populations should be routinely monitored in the greenhouse and field through the use of yellow or blue sticky cards and/or tapping foliage over white paper.
- ***Insecticides*** – Resistance management of the limited number of insecticides effective against the two major *Tospovirus* vectors, *F. occidentalis* and *Thrips tabaci*, is a major challenge (Martin et al. 2003; Reitz and Funderburk 2012; Robb

Table 1 Tospoviruses infecting China aster

Viruses	Occurrence in China aster	Transmission[a]	Other natural hosts	Symptoms in China aster	References
Chrysanthemum stem necrosis virus (CSNV)	Brazil, Japan	*Frankliniella occidentalis*, *F. schultzei*	Lisianthus (*Eustoma grandiflorum*), chrysanthemum (*Chrysanthemum morifolium*), tomato (*Solanum lycopersicum*)	Mosaic, necrotic spot, and lines on leaves (Fig. 6)	Alexandre et al. (1999) and Momonoi et al. (2011)
Groundnut ringspot virus (GRSV)	Brazil	*F. gemina*, *F. intonsa*, *F. occidentalis*, *F. schultzei*	Lisianthus, chrysanthemum, tomato	Mosaic, necrotic spots, and lines on leaves	Alexandre et al. (1999)
Tomato chlorotic spot virus (TCSV)	Brazil	*F. intonsa*, *F. occidentalis*, *F. schultzei*	15 species in six plant families	Not determined	Kitajima (2015) and Polston et al. (2013)
Tomato spotted wilt virus (TSWV)	Australia, Bulgaria, France, Italy, Greece, Japan, Korea, the USA	*F. bispinosa*, *F. cephalica*, *F. fusca*, *F. gemina*, *F. intonsa*, *F. occidentalis*, *F. schultzei*, *T. palmi*, *T. setosus*, *T. tabaci*	>900 species in >90 monocotyledonous and dicotyledonous plant families	Necrotic spots and ring spots and yellowing in leaves (sd) (Fig. 7)	Bakardjieva et al. (1998); Marchoux et al. (1991); Chatzivassiliou et al. (2000); Ishikawa et al. (2002); Kim et al. (2006); Latham and Jones (1997); Ramasso et al (1994) and Sether and DeAngelis (1992)

[a]Transmission data from Riley et al. (2011)



Fig. 6 CSNV symptoms: brown stem lesions (Momonoi et al. 2011)



Fig. 7 TSWV symptoms: yellowing and necrotic spots (S. Sugiyama © 2017. All Rights Reserved.)

et al. 1995). Therefore, insecticides cannot be viewed as a stand-alone treatment for either thrips or virus, even if resistance management regimes such as rotation and limited usage are rigorously maintained.

- ***Biological control*** – Biological control should be used preventively and not when thrips populations are high. Commonly used biocontrols for thrips management in greenhouses include predatory mites such as *Neoseiulus cucumeris* (formerly known as *Amblyseius cucumeris*) (Mesostigmata: Phytoseiidae); predatory bugs,

Orius spp. (Hemiptera: Anthocoridae); and entomopathogenic nematodes such as *Steinernema feltiae* (Rhabditida: Steinernematidae) and *Thripinema nicklewoodi* (Tylenchida: Allantonematidae).

4.3 Virgaviridae *Tobacco Rattle Virus* (TRV, Genus: *Tobravirus*)

Geographic occurrence and impact. This disease has occurred in Japan, where losses in China aster of up to 63% occurred (Komuro et al. 1970), and in the USA (Florida) (Anderson 1954).

Symptoms/signs. Symptoms of TRV vary based on the cultivar of China aster. The common symptom is concentric yellow ring spots on the foliage. Infected plants are slightly stunted. Occasionally, chlorotic chevron or vein-banding patterns on foliage or malformation of flowers occur.

Biology and epidemiology. TRV is transmitted by nematodes in the *Paratrichodorus* and *Trichodorus* species, known as stubby root nematodes (Brown et al. 1989). TRV on China aster is reported to be only transmitted by *Paratrichodorus minor* (syn. *Trichodorus minor*) (Komuro et al. 1970). However, TRV is not transmitted through the seeds of China aster (Komuro et al. 1970).

Management

- ***Cultural practices*** – Rapidly remove infected plants. Rotate crops with nonhost plants. Spinach is useful as a nematode trap plant for TRV (Komuro et al. 1970).
- ***Soil disinfestation*** – Reduce nematode populations by soil disinfestation using fumigation, steam, or soil solarization.

4.4 Additional Viral Diseases

The following viral diseases of China aster have also been reported:

– **Potyviridae**, *Bidens mottle virus* (BiMoV, genus: *Potyvirus*) (the USA, Logan et al. 1983)
– **Rhabdoviridae**, *Callistephus chinensis chlorosis virus* (CCCV, genus: *Nucleorhabdovirus*) (Brazil, Kitajima and Costa 1979)

5 Nematode Diseases

The following nematode diseases of China aster have been reported:

– **Foliar nematode** (*Aphelenchoides ritzemabosi*) (Germany, Burckhardt 1972)
– **Reniform nematode** (*Rotylenohulus reniformis* (the USA, Linford and Yap 1940)
– **Root knot nematode** (*Meloidogyne incognita*) (India, Nagesh et al. 1999)

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Diseases of Chrysanthemum

16

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Abstract

Chrysanthemums originated in China, but its major phenotypic selection occurred in Japan. The plant is sold as a cut flower, as a potted flowering plant, or as a garden plant. By manipulating day length, the plant can be produced year-round. Intensive production during the first half of the twentieth century resulted in devastating diseases, such as those caused by the fungi *Ascochyta*, *Septoria*, and *Verticillium*, which severely threatened the chrysanthemum industry, but are no longer problems because of the widespread use of fungicides and the clean stock programs employed by the key producers. However, several new major diseases of chrysanthemums have emerged that limit production and affect quality including bacterial infections, root rots, rusts, and viral and viroid infections.

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R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_16

Keywords

Alternaria spp. • *Botrytis cinerea* • *Fusarium oxysporum* f. sp. *chrysanthemi* • *Pythium* spp. • *Puccinia horiana* • INSV • TSWV • Viruses • Bacteria • Phytoplasmas • Viroids

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1 Introduction

Chrysanthemum, *Chrysanthemum* x *morifolium* Ramat. (pro sp.) (syn. *Dendranthema grandiflora*, *D. grandiflorum*), known as chrysanthemum, florist's chrysanthemum, or florist's daisy, is a flowering plant native to Asia (Anderson 1987; Anon 1996, 2010; Dole and Wilkins 2005; Gillow and Gortzig 1964). The many thousands of different cultivars developed over the centuries of this herbaceous perennial are grown according to use: as a cut flower, as a potted flowering plant, or garden plants. The commonly grown potted and cut chrysanthemums are grown year-round for indoor decorating (Mark Smith, personal communication). Garden chrysanthemums are sold in spring and fall, but especially in the fall for their vivid seasonal colors (Dole and Wilkins 2005). Chrysanthemum cultivars can also be classified by flower characteristics (Table 1). For chrysanthemums, a nearly infinite number of combinations of

Table 1 Chrysanthemum flower types and common ray florets

Flower types	**Description**
Single/daisy	Flat disks + 1–2 rows of ray florets
Anemone	Single with petaloid disk florets
Decorative/double/cushion	Majority ray florets, mostly lacking disk florets
Button	Tiny decorative
Common ray floret types	
Flat	
Incurved	
Quill	
Spoon	

flower sizes, color patterns, and ratios of disk florets to ray florets exist (Wendy Bergman, personal communication; Gillow and Gortzig 1964).

Chrysanthemum originated in China and the first cultivated species was likely in China over 2000 years ago (Gillow and Gortzig 1964). Japan is sometimes referred to as the flower's native land since that country was where the chrysanthemum was really developed and became foremost in use (Gillow and Gortzig 1964; Horst and Nelson 1997). Figures of the flower were used on the swords of the Japanese emperor in 1186 AD (Horst and Nelson 1997). Chrysanthemum received its current name after it reached the Western world when, in 1753, Karl Linnaeus combined the Greek words *chrysos*, meaning gold, and *anthemom*, a flower (Gillow and Gortzig 1964; Horst and Nelson 1997).

Initially, chrysanthemum was a seasonal crop, but when horticultural research determined that day length affected flowering (Garner and Allard 1920), subsequent studies applied this newfound knowledge to the chrysanthemum enabling it to become a year-round crop (Laurie 1930; Poesch and Laurie 1935; Post 1934, 1942). Specifically, the knowledge of a 14.5-h photoperiod being critical for flower bud initiation and a 13.5-h photoperiod being critical for flower development (Horst 1985) provided the foundation for the chrysanthemum industry to really develop so that chrysanthemum became one of the most important year-round flowering crops. Commercially, cuttings are produced by specialty propagators who maintain pathogen-indexed stock (Dole and Wilkins 2005). Globally, chrysanthemum is one of the most famous cut flowers with a high ornamental value, occupying an irreplaceable position in international flower commerce (Fan et al. 2015). In Europe, approximately 700 million cut chrysanthemums, 225 million santini chrysanthemums (small flowered cut mums) and 250 million pot mums are sold annually (Noel Vincent, personal communication). In Japan, chrysanthemum is cultivated primarily for ornamental use and occupies the highest level of production among cultivated flowering plants, with more than 6000 ha in production (Matsurra et al. 2007; Tsukiboshi et al. 2007). According to the latest industry statistics, the wholesale values of chrysanthemum crops for the USA in 2015 were $123.7 million for garden chrysanthemums, $16.7 million for 'florist' chrysanthemums (flowering potted plants), and $11.5 million for cut chrysanthemums resulting in a total value of $151.9 million (Anon 2016).

Two key publications (Horst 1985; Horst and Nelson 1997) on chrysanthemum diseases include many of the earlier descriptions of disease biology/epidemiology to which there have been very few more recent studies. Some of the diseases, such as *Ascochyta*, *Septoria*, and *Verticillium*, which severely threatened the chrysanthemum industry in the twentieth century, are no longer commonly encountered because of the widespread use of fungicides and the clean stock programs employed by the key producers. Noninfectious disorders are not included in this chapter and are discussed in detail by Horst and Nelson (1997).

This chapter presents a review of chrysanthemum diseases of primary importance and encountered most frequently during production. Chrysanthemum diseases discussed in this chapter may be encountered on cultivars grown for cut, pot and garden mum production. Specific management practices are presented when available. Refer to introductory chapters of this handbook for a broader discussion of disease management principles.

2 Fungal and Fungus-Like Diseases

2.1 Alternaria Leaf Spot and Blight (*Alternaria* spp.)

Geographic occurrence and impact. *Alternaria alternata* (Fr.: Fr.) Keissl. 1912 is cosmopolitan; it is reported on chrysanthemum in the USA (California and Florida), New Zealand, Puerto Rico, the Virgin Islands, the West Indies, Mexico, and India (Alfieri et al. 1994; Domínguez-Serrano et al. 2016; Farr and Rossman 2017; French 1989; Minter et al. 2001; Pennycook 1989; Shamala and Janardhana 2015). *Alternaria tenuissima* (Nees and T. Nees: Fr.) Wiltshire 1933 is also cosmopolitan causing leaf spots on multiple genera in multiple families; specific reports on chrysanthemum are from South Africa, Mexico, and China (Crous et al. 2000; Domínguez-Serrano et al. 2016; Gorter 1977).

Symptoms/signs. Leaf spot symptoms are often observed on the edges of the leaves and petals and are brown to blackish, rounded to oval, sometimes zonated with or without halos; sometimes leaves are entirely yellow (Domínguez-Serrano et al. 2016). Disease progression can result in necrotic leaves. Infected flower petals have 1–3 mm/0.04–0.12 in. reddish-brown lesions, which also can progress to entirely necrotic petals (Fig. 1) (Engelhard 1970). The symptoms on flowers are similar to those caused by *Botrytis*, *Helminthosporium*, or *Stemphylium.* Symptoms caused by *Alternaria* or *Stemphylium* have been described as ray speck (Horst and Nelson 1997; Tammen 1963). Sporulation after incubation at high humidity is necessary to distinguish the pathogen.

Biology and epidemiology. The disease occurs most frequently under production conditions that are stressful to the host. Disease spread is by infected plant material, splashing water, and air movement (Chase 2005). Spores require wetness for germination and infection of the host plant and disease development is enhanced by high relative humidity (RH).



Fig. 1 Alternaria petal spot showing dark necrotic lesions on sepals. (Penn State Department of Plant Pathology and Environmental Microbiology © 2017. All Rights Reserved.)

Management

- ***Cultural practices*** – ways of working that minimize leaf wetness duration will help control this disease (i.e., avoid overhead watering or water plants early in the day). Remove diseased plants and fallen leaf debris from the growing area. Avoid wide temperature fluctuations during shipping. Good air circulation should be provided. Avoid condensation development on leaf surfaces by providing heat and ventilation at sunset with greenhouse-produced crops. Use pathogen-free plants (Chase 2005).
- ***Fungicides*** – The following fungicides generally ranged from very good to excellent in controlling *Alternaria* on ornamentals: iprodione, trifloxystrobin, chlorothalonil, fluoxastrobin, myclobutanil, azoxystrobin, azoxystrobin + benzovindiflupyr, fludioxonil, pyraclostrobin + boscalid, cyprodinil + fludioxonil, copper sulfate pentahydrate, triadimefon, triflumizole, and triticonazole (Chase 2005, 2016; Singh and Milne 1974). Arun Kumar et al. (2011) found hexaconazole to be very effective in controlling *A. alternata* under field conditions followed in efficacy by chlorothalonil and mancozeb. According to Chase, if fungicides are effective on one species of *Alternaria*, they are often effective on other species. The use of thiophanate-methyl can actually make the disease more severe since this class of compounds does not control *Alternaria* (Chase 2005).
- ***Resistance*** – In India, the cultivars Chandini and Karnel were most susceptible to blight showing the highest disease incidence (100%) and severity (100%),while the most resistant cultivar examined was Rajawhite (Shamala and Janardhana 2015). Intergeneric hybrids were more resistant against both aphid infestation and Alternaria leaf spot inoculation than the *C. morifolium* "Nannongxiaoli" parent, but less than the *Artemisia vulgaris* "Variegata" parent (Zhu et al. 2014). Transgenic chrysanthemum plants that expressed a harpin gene demonstrated induced resistance to Alternaria leaf spot and earlier flowering than the wild type (Xu et al. 2011).
- ***Biological control*** – Compost teas consisting of various combinations of biofertilizers (vermicompost, paddy straw, neem powder, cow dung, and fish meal) and biocontrols (*Pseudomonas fluorescens* and *Trichoderma viride*) suppressed Alternaria leaf blight and, in some cases, were more effective than the fungicides iprodione + carbendazim (Deepthi and Reddy 2014).

2.2 Anthracnose (*Gloeosporium carthami* (Fukui) S. Hori and Hemmi, *G. chrysanthemi*, *Colletotrichum carthami*)

Geographic occurrence and impact. Many species of the genus are cosmopolitan, but the economic impact on chrysanthemum is sporadic and occurs under warm, moist conditions. The disease is also reported on safflower and pot marigold (Uematsu et al. 2012).

Symptoms/signs. Dark brown, sunken lesions on leaves sometimes extend over half the leaf and usually affect leaves only. As lesions progress, stems turn dark. Anthracnose lesions usually begin as small, tan leaf spots, which as they enlarge, become the site of dark acervuli producing rose to orange conidial masses.

Biology and epidemiology. As with most anthracnose diseases, conidia are released in the spring from acervuli that overwinter on last year's debris. Inocula can also be transmitted on seeds, transplants, cuttings, utensils, trays, and pots.

Management

- ***Cultural practices and sanitation*** – Avoid the use of overhead irrigation. Use disease-free stock plants for propagation. The fungus can be eliminated by steam-pasteurizing the rooting medium and by using disinfested cutting tools to minimize contamination and potential spread of the pathogen. Plant injury due to careless cultural practices should be avoided.
- ***Fungicides*** – Preventive applications of fungicides containing azoxystrobin + benzovindiflupyr, chlorothalonil, mancozeb, pyraclostrobin + boscalid, and thiophanate-methyl are effective.

2.3 Ascochyta Ray Blight [*Ascochyta chrysanthemi* (*Mycosphaerella ligulicola* Baker, Dimock, and Davis)]

Geographic occurrence and impact. The disease was first observed in North Carolina in 1904 and was later observed wherever chrysanthemums were grown including England, Japan, the USA (Florida), and Australia (Alfieri 1966; Stevens 1907). Once considered a key disease of chrysanthemum, which had to be controlled, this disease is no longer a major threat to commercial production. This is likely attributed to sanitation practices and regular fungicide use. It could be argued that this disease could again be prevalent if either of these practices was compromised.

Symptoms/signs. Symptoms can develop on all aboveground plant parts as well as on the roots (Baker et al. 1960). Leaf spots are brown to black and variable in size, from circular to wedge shaped, usually starting at the margins. Stem lesions are elongate, brown to black, slightly sunken areas about 2.5 cm/1 in. long often associated with the point of leaf attachment. Flower symptoms are a light brown necrosis at the base of the petals and extend into the receptacle (Fig. 2). Infected cuttings are first noted by the leaf symptoms which may extend quickly to the stems.

Biology and epidemiology. Perithecia and pycnidia serve as the overwintering structures, which give rise to abundant conidia when warm rains appear. Conidia and ascospores can germinate and infect within a few hours (Baker et al. 1949). The fungus can readily survive as mycelium on roots of infected plants (Chesters and Blakeman 1966).

Management

- ***Cultural practices and sanitation*** – The use of disease-free planting stock is very important for preventing outbreaks.
- ***Fungicides*** – Azoxystrobin + benzovindiflupyr, chlorothalonil, iprodione, iprodione+thiophanate methyl, mancozeb and myclobutanil are all effective in controlling Ascochyta. Chlorothalonil gave satisfactory control when applied to



Fig. 2 Ascochyta ray blight (R.K. Jones © 2017. All Rights Reserved.)



Fig. 3 Botrytis petal blight (R.K. Jones © 2017. All Rights Reserved.)

plants before flower (Singh and Milne 1974). A benomyl cutting dip was shown to be effective (Judd and Walton 1973).

2.4 Botrytis Blight (*Botrytis cinerea* Pers.:Fr 1794)

Geographic occurrence and impact. Worldwide in occurrence, but mostly a problem in greenhouse cut flower production. The disease can be very destructive when environmental conditions are conducive (Daughtrey et al. 1995).

Symptoms/signs. Light brown spots form on lower petals (Fig. 3). Browning spreads to other petals. Infected tissues become covered with dusty gray spores. Infected flower buds may fail to open. A grayish-brown mycelial growth may develop on the entire bud and leaves and extend into the stem (Fig. 4).

Biology and epidemiology. It is a highly water-dependent fungus and a certain leaf wetness duration is necessary for spore germination. The fungus is ubiquitous and



Fig. 4 Botrytis foliar blight with masses of mycelia and conidia (Jay Pscheidt © 2017. All Rights Reserved.)

survives as a pathogen or saprophyte on a wide range of plant hosts (Daughtrey et al. 1995). The fungus survives as mycelium in plant tissue (Yunis and Elad 1989) and as sclerotia (Ellis and Waller 1974). Conidia of *B. cinerea* are easily spread by air currents and water splash, making Botrytis blight one the most commonly encountered and difficult-to-control diseases of chrysanthemum. The fungus can infect all aboveground tissue but is most economically damaging to the floral structures. Conidia of the pathogen must remain wet for 5–8 h for infection by *B. cinerea* to take place (Jewett and Jarvis 2001). However, during germination before the buildup of inoculum, periods of dryness (desiccation) can kill the spores.

Management

- ***Cultural practices and sanitation*** – To avoid gray mold disease incidence, the RH set point is generally kept low. This costs high amounts of energy through dehumidification by heating and ventilating the greenhouses (Daughtrey et al. 1995). Greenhouse environment systems should be programed to avoid RH above 85–90%. Overhead irrigation should also be avoided. Postharvest handling to control *B. cinerea* and other problems involves avoidance of plant injury and storage at 4 °C/39.2 °F (Anon 2015). Growers should remove infected and blighted tissue from all plants to reduce inoculum.
- ***Fungicides and biocontrols*** – Chemical control of Botrytis blight has received much attention due to the propensity of the pathogen to develop resistance (Leroux 2007; Moorman and Lease 1992). Fungicide resistance has been found with products containing benomyl, fenhexamide, iprodione, and thiophanate-methyl due to overuse and the global movement of plant material in the horticultural trade. In order to avoid the development of resistant strains, a rotation of alternative classes of fungicides needs to be used. The fungicides commonly used include chlorothalonil, singly and in combinations, along with iprodione, fludioxonil, fenhexamid, strobilurins and triflumizole. More recently, combination products such as azoxystrobin + benzovindiflupyr, cyprodinil + fludioxonil, and pyroclostobin + boscalid that include two active ingredients with different modes of action on Botrytis are being employed in Botrytis management programs with

great success. There are several biorational and biological products that can be used to suppress *Botrytis* in production. Biorational products include bicarbonates and polyoxin D and biological products include *Bacillus subtilis*.

2.5 Chrysanthemum Brown Rust (*Puccinia chrysanthemi*, Roze; P. tanaceti D.C.)

Geographic occurrence and impact. Brown rust of chrysanthemum caused by *Puccinia chrysanthemi* is commonly found among chrysanthemums grown outdoors. However, the disease does occur in production greenhouses and fields. Generally, this fungus is only a serious problem in coastal areas, which have favorable temperature and moisture conditions.

Symptoms/signs. Brown rust produces pale yellow flecks on both surfaces of the leaf, which develop into dark chocolate brown powdery pustules typically found on the underside of the leaves. Rings of secondary pustules form around these pustules and the central pustule often turns brown to black. Brown rust can be easily distinguished from white rust by the pustule and spore color (Fig. 5) and the occurrence of uredospores. Brown rust produces dark red-brown teliospores (Kahn and Wheeler 1969; Peterson et al. 1978).

Biology and epidemiology. The rust pustules are composed of rings of the uredial stage, which produce urediospores. These spores serve as the primary inoculum for outbreaks. Infection occurs at 16–27 °C (60–81 °F).

Management

- ***Cultural*** – Purchase cuttings from a reputable commercial source. Keep foliage dry and maintain low humidity if possible. Remove old plant debris.

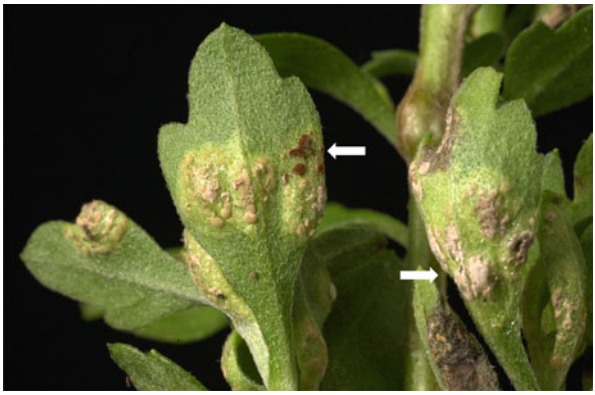


Fig. 5 Rust symptoms and signs: Brown rust (brown pustules, *left*) white rust (white pustules, *right*) (Tracey Olson © 2017. All Rights Reserved.)

- ***Fungicides*** – Spray every 7–10 d with a preventive fungicide (McCain and Gonot 1979). Preventive and eradicant fungicides are efficacious on both brown rust and white rust. See CWR management for details on these fungicides.

2.6 Chrysanthemum White Rust (*Puccinia horiana* Henn.)

Geographic occurrence and impact. Chrysanthemum white rust originated in Japan (Hiratsuka 1957) and is now established in the Far East, Europe, Africa, Asia, Central America, and South America. In 1895, it was found in Japan and was described in 1901 by Hennings (Hennings 1901). When the disease was discovered in England in 1963, it spread rapidly to other countries and became a disease of international importance. Several countries placed quarantines on this disease, which restricted the distribution of chrysanthemums. In the USA, the disease was first found in June of 1977 on a hobbyist planting in New Jersey (Peterson et al. 1978; O'Keefe 2014).

Symptoms/signs. Chrysanthemum white rust appears as yellow spots up to 4 mm, which occur first on the upper leaf surface (Fig. 6). Lesions have a dimpled look due to the center being slightly sunken and in time become necrotic. The pustules are waxy when touched. Pustules are common on young leaves and flower bracts, but they can be found on any green tissue. It was noticed that on certain cultivars, purple spots rather than yellow spots are produced (Tracey Olson, personal communication). Prominent pustules subsequently develop on the lower surfaces of the leaves corresponding with the spots. The pustules are pinkish buff initially and then become white with age and sporulation (Fig. 6) when basidiospores are produced. Teliospores are hyaline to yellow microscopically in contrast to those of *P. chrysanthemi*. *P. horiana* lacks the uredospore stage.

Biology and epidemiology. Chrysanthemum white rust infects 13 different species of chrysanthemum or closely related genera: *Leucanthemella* and *Nipponanthemum*. The fungus produces two types of spores: teliospores and basidiospores. Teliospores are produced in the pustules unless they are aggressively brushed off. They can survive for up to 8 wk if they remain in pustules on detached leaves at a 50% RH or less. They die sooner under moist conditions. When the RH is 96–100% for at least 3 h, teliospores produce basidiospores. In a greenhouse study, teliospores survived for a maximum of 28 d in dry soil and 7 d in moist soil. In a growth chamber simulating winter conditions in the North Eastern USA, teliospores survived a maximum of 35 d (Bonde et al. 2013). It appears that teliospores are not able to survive typical North Eastern winters since they would become nonviable before actually being frozen. The teliospores produce basidiospores, which are actually the "infectors" and can cause an epidemic if conditions are right. They spread from plant to plant by splashing water and human handling. The basidiospores must have a film of water in order to



Fig. 6 Early symptoms of chrysanthemum white rust – yellow spots on the upper leaf surface (*upper photo*); unopened rust pustules on the lower leaf surface (*middle photo*); sporulating pustules on the lower leaf surface (*lower photo*) (Tracey Olson © 2017. All Rights Reserved.)

cause infection. Host penetration can occur in 2 h in wet conditions at the optimum temperature of 17 °C/63 °F. Basidiospores can travel up to about 0.8 km/0.5 mi in wind currents during moist weather (Zandvoort 1968). They survive only 5 min when the RH is 80% or below and less than 60 min when the RH is 90% (Firman and Martin 1968).

Management

- ***Cultural practices*** – Chrysanthemum white rust has been excluded by the use of quarantine regulations, but increasing frequency of finds have resulted in pressures to change existing regulations. Measures that are effective in preventing chrysanthemum white rust are using a disease-free source and scouting the crop regularly. Do not comingle imported cut mums with a growing crop since flowers can harbor spores, and maintain low humidity and dry foliage especially in greenhouses.
- ***Fungicides*** – Schedule regular applications of preventive fungicides if in an area where the disease has previously been reported. Refer to ▶ Chap. 7, "Fungicides and Biocontrols for Management of Florists' Crops Diseases."

2.7 Fusarium Stem Rot (*Fusarium solani* Mart.)

Geographic occurrence and impact. Fusarium stem rot of chrysanthemum was first observed in Florida in the 1970s. It appears to be less severe in flowering plants but can be very destructive in rooted cuttings (Engelhard et al. 1976). It is not a systemic disease but exhibits a more localized canker lesion.

Symptoms/signs. On cuttings, red to brownish lesions develop on the stem which may develop into a black, wet, soft decay. On older plants producing cuttings, stem dieback occurs where cuttings have been harvested. Many times, the infections will progress unilaterally with one side of the plant unaffected. On older flowering plants, wilt may occur followed by leaf chlorosis (Engelhard et al. 1976).

Biology and epidemiology. *Fusarium solani* is a soilborne disease and likely gains entry into propagation beds through contaminated soil. The fungus is also easily transmitted on plant material, pots, and trays. The fungus overwinters as mycelium and as resistive chlamydospores. *Fusarium solani* also produces two other asexual spores, microconidia and macroconidia, which can be dispersed aerially and by water splash. These later spores initiate the stem dieback symptoms (Trolinger unpublished).

Management

- ***Cultural practices and sanitation*** – Sanitation should be the first line of defense. Growers should use clean cutting stock, disease-free soil, and disinfested cutting utensils. Regular scouting and removal of any diseased plants from the stock planting is effective in avoiding disease spread. The disease can also be minimized by avoiding deep planting and overfertilization. Maintaining a soil pH of 6.2 or above and avoiding ammonium nitrogen sources will suppress Fusarium stem rot.
- ***Fungicides*** – Although Tepper et al. (1983) found several fungicides restricted radial growth, none of them suppressed the disease on potted chrysanthemum plants. Stem dieback on chrysanthemum stock plants can be prevented by postharvest sprays of chlorothalonil and/or benomyl (Trolinger unpublished).

2.8 Fusarium Wilt [(*Fusarium oxysporum* f. sp. *chrysanthemi* Littrell, G.M. Armstr., and J.K. Armstr. 1970; *F. oxysporum f.* sp. *tracheiphilum* (E. F. Sm.) W. C. Snyder and H. N. Hans. 1940) (Race 1)]

Geographic occurrence and impact. Fusarium wilt (FW) first appeared in 1939 but was described as being caused by *F. oxysporum* f. sp. *chrysanthemi* in 1965 by Armstrong and Armstrong. The disease can cause significant losses that can carry over due to the persistent nature of the disease. The disease is most severe in warm climates (Locke et al. 1985).The disease is reported most severe on *Chrysanthemum morifolium*, but the pathogen can incite symptoms on Paris daisy (*Argyranthemum frutescens*), African daisy (*Osteospermum* sp.), and gerbera (*Gerbera jamesonii*) (Garibaldi et al. 2009; Minuto et al. 2008).

Symptoms/signs. Symptoms of FW can be quite variable depending on the cultivar and soil temperature. In some cultivars, the disease is somewhat unique in that symptoms begin on the youngest foliage first and then progress to the older leaves (Fig. 7), as opposed to the more typical FW expression where symptoms appear at the base of the plant and move upward. The pathogen may invade one section of the vascular system and produce a one-sided or sectored symptom in the plant. Wilting is more common at temperatures about 32 °C (Gardiner et al. 1987) and symptom onset can be delayed at cooler temperatures. For example, studies showed that under an equal amount of inoculum, disease development was fast at temperatures of 27–32 °C (80–90 °F), while cooler soil temperatures of 18–24 °C (64–75 °F) delayed the onset of symptoms by several weeks (Emberger and Nelson 1981; Horst and Nelson 1997). A characteristic feature of FW is the presence of vascular discoloration (Fig. 8). Although the fungus infects through young feeder roots, it is not generally associated with a root rot. However, many times root systems will be compromised and smaller.

Biology and epidemiology. Fusarium wilt in chrysanthemum can be caused by two formae speciales, *F. oxysporum* f. sp. *chrysanthemi* (*Foc*) and *F. oxysporum* f. sp. *tracheiphila* (*Fot*), and three races of the former have been identified (Armstrong and Armstrong 1965; Armstrong et al. 1970; Troisi et al. 2013). These pathogens can be differentiated based on differential chrysanthemum cv. reactions and through genetic analysis (Nelson et al. 1981; Troisi et al. 2013). Disease development is dependent upon or can vary with cultivar and environmental conditions. In general, conditions that favor plant growth also promote disease. The fungus persists in decaying host tissues as mycelium and as chlamydospores, which have the increased capability to endure harsh environmental conditions (Booth 1971; Nash et al. 1961; Nelson et al. 1981). The fungus can sporulate on infected tissue (Fig. 9), which allows spores (microconidia and macroconidia) to be distributed by air, wind-driven rain, and/or overhead irrigation. The fungus can also be spread by fungus gnats (*Bradysia* spp.) and



Fig. 7 Fusarium wilt: early symptoms, yellowing of leaves and thinning of canopy (*upper photo*); advanced symptoms, wilting and tissue necrosis (*lower photo*) (Nancy Rechcigl © 2017. All Rights Reserved.)



Fig. 8 Fusarium wilt: vascular discoloration in stem (R.J. McGovern © 2017. All Rights Reserved.)



Fig. 9 Fusarium wilt: sporulation (white sporodochia) of *F. oxysporum* f. sp. *chrysanthemi* on necrotic stems (Nancy Rechcigl © 2017. All Rights Reserved.)

shore flies (*Scatella* spp.). Additional information on fungus gnats and shore flies may be found in introductory chapter (▶ Chap. 4, "Insect Management for Disease Control in Florists' Crops").

Management

- ***Cultural practices*** – Purchase culture-indexed cuttings free of the pathogen. Plant in pasteurized soil or soilless mix free of the pathogen. Maintain soil pH between 6.5 and 7.0. Use nitrate rather than ammonium forms of fertilizer.
- ***Suppressive media*** – Considerable research has been conducted on the disease-suppressive effects of compost in the planting medium. Chef et al. (1983) observed disease suppression when the potting medium was amended with mature composted hardwood bark (CHB), but disease suppression was less significant when green CHB was used. Incorporation of composted sewage sludge into a *Pinus* bark-based substrate significantly reduced FW, but addition of biofertilizer, fish hydrolyzate, chitosan, and *Trichoderma* had no effect on the disease (Pinto et al. 2013).
- ***Fungicides/fumigants*** – Most FW diseases are not effectively suppressed by fungicides. However, research has been conducted that shows combining a high lime, all nitrate fertilization regime with a systemic fungicide gave complete control of FW on pot-grown chrysanthemum (Engelhard and Woltz 1973). In separate research, the fungicides benomyl and thiophanate-methyl effectively reduced FW, and the combination of benomyl + lime + nitrate provided good control but caused slight stunting and chlorosis in potted chrysanthemums (Strider 1985a). Fungicides containing fludioxonil and azoxystrobin applied as a soil treatment have demonstrated good protection against F. oxysporum in chrysanthemum and other crops (Rechcigl and Trolinger, unpublished). Other fungicides which can be rotated are iprodione, thiophanate-methyl + iprodione, triticonazole, boscalid + pyraclostrobin, azoxystrobin + benzovindiflupyr and fluoxastrobin.
  In the past, preplant fumigants such as methyl bromide + chloropicrin, metam sodium, etc. were routinely used to control soilborne pathogens such as *F. oxysporum* in many crops. However, increasing concern over the sustainability of such a strategy has led to the development and use of safer approaches to soil disinfestation such as steam, soil solarization, anaerobic soil disinfestation, and

alternative fumigants and fumigation practices (refer to ▶ Chap. 8, "Soil/Media Disinfestation for Management of Florists' Crops Diseases").

- ***Resistance*** – Strider (1985b) reported that of the chrysanthemum cvs. tested, Cirbronze, Puritan and Tuneup were the most resistant to *Foc*, while Cirbronze, Jamboree, Pinktive, Puritan, Trophy, Tuneup, and Yellow Delaware were highly resistant to *Fot.* However, others indicated that "Cirbronze" and "Yellow Delaware" were highly susceptible to *Foc* (Anon 2012; Engelhard and Woltz 1971). Engelhard and Woltz (1971) indicated that the cvs. Dillon Beauregard, Stingray, and Tinsel remained symptomless after inoculation with *Foc*. Knowledge of the specific races involved in FW outbreaks is necessary to effectively develop/deploy resistance/tolerance to the pathogens effectively. More recent studies of current cultivars are not available.
- ***Integrative strategies*** – *Trichoderma viride* (T-l) and a benomyl-resistant biotype (T-I-R9), alone or in combination with *Aspergillus ochraceus*, reduced disease by at least 50% (Locke et al. 1985). Application of Tl-R9 in combination with as few as two benomyl drenches provided control equal to a commercial integrated control program. Fusarium wilt in potted chrysanthemum was reduced by 81–92% by a number of *T. harzianum* isolates and by 50–70% by a number of botanical extracts (Singh and Kumar 2011). Zhao et al. (2016) suggested that the combination of a bioactive fertilizer with the soil fumigant dazomet could effectively control Fusarium wilt in field-produced chrysanthemums in China. Slow sand filtration, alone and in combination with the application of biocontrol agents (*Streptomyces griseoviridis*, *Trichoderma* spp.), and a nutrient solution pH higher than 6.0, produced significant reductions in *Foc* infection of gerbera plants grown in closed soilless systems (Minuto et al. (2008).

2.9 Itersonilia Petal Blight [*Itersonilia perplexans* Derx 1948 (Basidiomycetes, Incertae sedis)]

Geographic occurrence and impact. Reported in Asia (Japan), Australia, Europe (Austria, Greece, the Netherlands, Portugal), New Zealand, North America (Canada, USA: California, Florida) and South America, this fungus causes a flower blight in chrysanthemum and *Callistephus* (China aster), but also a seedling blight, root canker, leaf spot/necrosis, and dieback in other crops (Koike and Tjosvold 2001). The host range also includes *Anethum graveolens*, *Heracleum*, and other Apiaceae, *Emilia*, *Helianthus*, and other Asteraceae.

Symptoms/signs. Only the flowers are affected and the initial symptoms of pinpoint, necrotic, red to brown specks enlarge on older florets but do not spread on younger florets. Disease progresses on the older florets until the entire floret becomes straw to brown colored. At this stage, the symptoms are difficult to distinguish from Botrytis petal blight. Itersonilia petal blight symptoms on China aster are very similar to chrysanthemum.

Biology and epidemiology. Cool temperatures favor this fungus; 18 °C (65 °F) is the optimum temperature for growth. In 12 h, lesions develop on infected florets held

at 10–15 °C (50–59 °F) but develop at a slower rate at 1 and 21 °C (33 and 70 °F). Near 100% RH encourages pinpoint lesions to enlarge rapidly, whereas 70% RH results in no enlargement. A minimum period of 8–12 h of moisture is required for infection. Outer florets, which are older, are more susceptible to rapid fungal growth than younger florets. Floret tips are more susceptible than the bases. Any bruising of the flowers hastens infection. Two groups of *I. perplexans* have been reported, which are specific to Asteraceae or Apiaceae. Cross-pathogenicity has been reported in isolates from certain hosts. Isolates found pathogenic on chrysanthemum were equally virulent on China aster, gerbera, and sunflower (McGovern et al. 2006).

Management

- ***Cultural practices and sanitation*** – Sanitation is strongly encouraged, such as roguing infected plants and removing non-harvested flowers. Eliminating weed host, especially the Asteraceae, is critical in controlling Itersonilia petal blight.
- ***Fungicides*** – McGovern et al. (2006) found that myclobutanil, propiconazole, and potassium bicarbonate were generally more effective than azoxystrobin in lowering the severity level of petal blight on China aster in controlled environment experiments. These fungicides were likewise effective in reducing petal blight in chrysanthemum in Hawaii (unpublished, Trolinger).

2.10 Leaf Spots of Chrysanthemum *Bipolaris setariae* (Sawada) Shoemaker; *Cochliobolus setariae* (Ito and Kuribayashi in Ito) Drechs.ex Dastur (teleomorph); *Septoria chrysanthemi* Halst. in Seym. and Earle; S. *leucanthemi* Sacc. and Speg.; *Cylindrosporium chrysanthemi* Ellis and Dearn; *Cercospora chrysanthemi* Heald and F. A. Wolf; *Phyllosticta chrysanthemi* Ellis and Dearn

Geographic occurrence and impact. Leaf spots occur sporadically but can be damaging if not suppressed. Most outbreaks occur in humid areas where rainfall is abundant.

Symptoms/signs. Symptoms consist of spots on the leaves. These spots are first yellowish and then become dark brown and black, increasing from 0.05 to 2.5 cm (⅛ to 1 in.) or more in diameter. Leaves may wither prematurely. The lower leaves are infected first. Many similar fungi can cause leaf spot diseases on a wide range of flowers and ornamentals. Leaf spots sometimes have yellowish margins and increase in size until they coalesce. Numerous tiny, black fruiting bodies of the fungus can sometimes be found in the center of the lesion depending on the fungal pathogen. Affected leaves drop prematurely.

Biology and epidemiology. Specific information on the biology and epidemiology of each leaf spot pathogen on chrysanthemum is not available. However, conidial

germination of these fungi is favored by prolonged leaf wetness and high RH. Inoculum is spread by wind-driven rain and air. Most of these fungi produce overwintering structures like pycnidia that provide the inoculum for new infections when favorable environmental conditions return.

Management

- ***Cultural practices*** – Remove and destroy infected leaves and clean up and destroy dead plant debris between crop cycles to reduce inoculum. Provide good air circulation to minimize leaf wetness. Avoid overhead irrigation in the morning.
- ***Fungicides*** – If disease is severe enough to warrant chemical control, fungicides containing azoxystrobin + benzovindiflupyr, chlorothalonil, mancozeb, myclobutanil, propiconazole, or thiophanate-methyl may be used.

2.11 Phytophthora Root and Stem Rot [*Phytophthora cactorum* (Lebert and Cohn) J. Schröt. 1886; *Phytophthora chrysanthemi* Naher et al. 2011; *Phytophthora cryptogea* Pethybr. and Laff. 1919; *Phytophthora nicotianae* Breda de Haan 1986; *Phytophthora tentaculata* Kröber and Marwitz 1993]

Geographic occurrence and impact. *Phytophthora* does not impact chrysanthemum as extensively as *Pythium*, another oomycete, but when present, causes losses and sometimes death of plants. Species reported are *P. cactorum* in Japan and other countries; *P. chrysanthemi* initially only in Japan, but recently reported in the USA (Lin et al. 2017); *P. cryptogea* in the USA (California) and Scotland; *P. nicotianae* in the USA (Florida and Alabama); and *P. tentaculata* in Spain and Germany on *Chrysanthemum frutescens* and *C. leucanthemum*. *P. tentaculata* is reported in the USA, but not currently on florists' chrysanthemum; it is a concern for further introduction into the USA (Erwin and Ribeiro 1996; Frankel et al. 2015; Kagiwata 1972; Kröber and Marwitz 1963; Kroon et al. 2004, 2012; Martin et al. 2012; Naher et al. 2011).

Symptoms/signs. Symptoms include basal stem rot resulting in yellowing/wilting of the plant top. Root rot is commonly observed as well (Fig. 10). Other associated symptoms are poor plant size, chlorosis, and vein reddening (Lin et al. 2017). A laboratory diagnosis is necessary for best management since these symptoms are similar to those caused by other root rot pathogens. Leaf blight resulted on artificially inoculated chrysanthemum cultivars Capri and Vermillion with an isolate of *P. nicotianae* from hibiscus (Semer and Raju 1985). A blossom blight caused by *P. nicotianae* additionally was described on the cultivar Debonair growing in Alabama (Mullen et al. 2001). Various other cultivars (Grace, Hot Salsa, Jennifer, Raquel, Spotlight, and Yellow Triumph) developed blossom blight when artificially inoculated under high humidity conditions.



Fig. 10 Phytophthora root and stem rot (R. Wick © 2017. All Rights Reserved.)

Biology and epidemiology. *Phytophthora* belongs, like *Pythium*, to the oomycetes, a diverse group of fungus-like eukaryotes that was recently placed in the new kingdom Stramenopila including diatoms, brown algae, and golden-brown algae (Beakes and Sekimoto 2009). Oospores, chlamydospores, or mycelium of *Phytophthora* in infected roots or stems or in soil survive cold winters or hot, dry summers. Motile zoospores are ultimately produced and further disseminate the fungus in irrigation, surface water, and rain. Wet conditions and saturated soils favor disease. Ideal temperatures vary by species from cooler to warmer conditions (Agrios 2005; Shew and Lucas 1991).

Management

- ***Cultural practices*** – Cultural conditions contribute to disease problems. Plants that are stressed are more prone to infection so sound horticultural practices are key in preventing this disease. Prevent prolonged saturated media conditions and use media that provides good aeration to avoid compaction. Inspect roots on a regular basis and avoid high soluble salts or extensive drying of the root system. Sound sanitation practices should always be implemented.
- ***Fungicides*** – All of the oomycetous microorganisms have a short generation time and great reproductive capacity (Dick 1992) and, thus, management is necessary. Drench at transplanting or 7 d after sticking unrooted cuttings. Fungicide

rotations are important in order to avoid resistance issues. Resistance to mefenoxam has been reported. Fungicides found to be effective for suppression of *Phytophthora* are fenamidone; cyazofamid; aluminum tris; mono- and dibasic sodium, potassium, and ammonium phosphites; mono- and dipotassium salts of phosphorous acid; mandipropamid; oxythiopiprolin; dimethomorph; ametoctradin + dimethomorph; and fluopicolide. Free available chlorine at 2 ppm at the output of irrigation systems is required to achieve good control of *Phytophthora* spp. in irrigation water (Hong et al. 2003).

2.12 Plectosporium Blight/Leaf Spot/Cutting Rot [*Plectosporium tabacinum* (J.F.H. van Beyma) M.E. Palm, W. Gams, and Nirenberg (Palm et al. 1995)]

Geographic occurrence and impact. This pathogen is cosmopolitan on various plant parts in various plant families. The disease was described and characterized on chrysanthemum in Japan as a cutting rot in field plantings (Satou et al. 2010, 2013) and has been identified in diagnostic laboratories in the USA as a leaf spot/blight of outdoor plantings (field or pots) of the crop (M. Daughtrey, T. Schubert personal communication; J. Trolinger, unpublished data). A wide host range in which vegetable and ornamental plants can become infected interchangeably and the ability to survive generally as a saprophytic soilborne organism (Dillard et al. 2005; Satou et al. 2013) make this pathogen a concern to crop production.

Symptoms/signs. Black discoloration and decay of stems were determined to be caused by *P. tabacinum* after unrooted cuttings (cv. Jimba No. 2) were transplanted into a production field in Japan and failed to initiate roots (Satou et al. 2010) (Fig. 11). Whether the pathogen was imported with the cuttings or was already present in the field was not determined. A leaf-spotting disease initially on the undersides of leaves (no spotting on the upper leaf side) was noted in the USA in outdoor chrysanthemum plantings in 2007 and likewise, *P. tabacinum* was determined to be the causal agent of leaf spot on cucurbits (Bost and Mullins 1992; England et al. 2007; Zitter 1996; Mullen and Sikora 2003) and snap beans (Dillard et al. 2005). On chrysanthemum leaves, brown spots occur and spread on the lower leaf surface but often do not extend to upper surface until the entire leaf becomes blighted (Fig. 12). Under wet conditions, a slightly whitish to buff sporulation (conidia and mycelia) can sometimes be found in leaf lesions.

Biology and epidemiology. High moisture and RH favor the disease (Tomioka et al. 2011; Satou et al. 2013). In snap bean, a continuous 48-h leaf wetness duration at 23–27 °C (73–80 °F) was essential for rapid symptom development (Dillard et al. 2005). This organism is generally known as a soilborne pathogen, but Satou et al. (2013) reisolated the pathogen from chrysanthemum cuttings taken from mother plants produced by original transplanted cuttings inoculated with the fungus, indicating the fungus was transmitted by endosymbiosis, as are the pathogens *Fusarium*



Fig. 11 Plectosporium cutting rot (Mamoru Satou © 2017. All Rights Reserved.)



Fig. 12 Plectosporium leaf spot (Nancy Rechcigl © 2017. All Rights Reserved.)

spp. and *Verticillium* spp. This disease could potentially affect a chrysanthemum crop as a cutting-borne disease and/or as a soilborne disease. A pumpkin isolate was pathogenic to chrysanthemum (Satou et al. 2013), and a zucchini isolate was

pathogenic to snap bean (Dillard et al. 2005) indicating isolates have cross-infectivity. Distribution of *P. tabacinum* appears to be increasing in cucurbits in the USA (Quesada-Ocampo et al. 2015), and the leaf-spotting disease has been additionally noted on chrysanthemum. Additional studies are needed on this disease.

Management

- ***Cultural practices*** – The use of clean planting stock and clean potting soil mixes is important for managing any soilborne disease (Agrios 2005).
- ***Resistance*** – There are differences in susceptibility between chrysanthemum cultivars. Satou et al. (2013) found 'Jimba' and 'Jimba No. 2' more susceptible to cutting rot than 'Oki-no-Shiranami'. Likewise, in an outdoor setting where the leaf-spotting disease is found, not all cultivars are affected.
- ***Fungicides*** – Satou et al. (2013) showed that captan and thiophanate-methyl as a basal stem dip into powder or emulsion formulations are effective in controlling the disease. Azoxystrobin alternated weekly with chlorothalonil as sprays (as with pumpkin, Mullen and Sikora 2003) and/or mancozeb sprays have been effective on the chrysanthemum leaf-spotting disease. A recently marketed combination fungicide of azoxystrobin + benzovindiflupyr is registered in the USA for leaf spot diseases caused by *Plectosporium* spp.

2.13 Powdery Mildew [*Golovinomyces cichoracearum* (DC.) V.P. Heluta 1988 (Ascomycetes, Erysiphales)], *Erysiphe cichoracearum* DC. 1805

Geographic occurrence and impact. The pathogen is cosmopolitan and has been reported on *Chrysanthemum morifolium* in the USA (Alaska, California, Florida, Illinois, North Carolina, and South Dakota), Canada, Puerto Rico, the Virgin Islands, the West Indies, Japan, Korea, New Zealand, and the UK (Anon 2015). The disease is not usually damaging but can threaten crop quality and stress the plant.

Symptoms/signs. The disease appears as ash-gray powdery growth on leaves and occasionally stems (Fig. 13). As the lesions develop, the leaf may become puckered or distorted and severely infected leaves will shrivel and drop from the plant (Bradshaw 2015).

Biology and epidemiology. Braun and Cook (2012) have described *E. chrysanthemi*, *G. artemisiae*, and *G. cichoracearum* in detail. The fungus is an obligate parasite and, generally, overwinters as mycelial mats in plant tissue and/or as sexual fruiting bodies called chasmothecia. During warm and humid weather in the spring, the chasmothecium absorbs water and splits open, and the ascospores are dislodged to initiate the first round of infection. After a mycelial mat has formed, chains of asexual conidia are evident within a few days, completing the disease cycle. The disease is most serious during hot, humid weather. Unlike most fungal diseases, free water is not required for powdery mildew infection and actually inhibits germination; high humidity encourages disease development.



Fig. 13 Powdery mildew (Penn State Department of Plant Pathology and Environmental Microbiology © 2017. All Rights Reserved.)

Management

- ***Cultural practices*** – Powdery mildew can be avoided by routine scouting, proper plant spacing, good air circulation, low RH, and adequate light levels.
- ***Fungicides and biocontrols*** – Protectant fungicides should be used just before or when first symptoms appear. The active ingredients, copper, azoxystrobin, azoxystrobin + benzovindiflupyr, pyraclostrobin, triflumizole, myclobutanil, triadimefon, propiconazole, sulfur, potassium bicarbonate, or thiophanate-methyl, are effective when used according to label instructions. Bicarbonate-based products and horticultural oils can be used to supplement a routine program when powdery mildew is first observed. A few commercial preparations of the biocontrol *Bacillus subtilis* strain QST 713 are available and useful when rotated with other products.

2.14 Pythium Blight and Root Rot (*Pythium aphanidermatum* (Edson) Fitsp; *P. carolinianum* Matthews, *P. debaryanum* Hesse, *P. helicoides* Drechsler, *P. dissotocum* Drechsler; *P. oedochilum Drechsler*; *P. sylvaticum* Campbell et Hendrix; *P. ultimum* Trow var. *ultimum*, and Other *Pythium* spp.

Geographic occurrence and impact. *Pythium* is widely distributed, occurring in most agricultural soils, and attacks a wide range of plants. Several species cause disease in chrysanthemum (Liu et al. 2007; Moorman et al. 2002; Paternotte and de Kreij 1993; Tsukiboshi et al. 2007; Van der Plaats-Niterink 1981). In the USA, *P. aphanidermatum* is frequently isolated from diseased chrysanthemums in hot weather. Pythium diseases are one of the most frequently found chrysanthemum diseases.

Symptoms/signs. Pythium root and basal stem rot and stunting are the most common symptoms on chrysanthemum (Fig. 14). Foliar symptoms on cuttings in propagation or on young plants, sometimes referred to as aerial *Pythium*, also occur



Fig. 14 Pythium root rot and wilt (*left*) compared to healthy plant (*right*) (Jane Trolinger © 2017. All Rights Reserved.)



Fig. 15 Pythium aerial blight (Jane Trolinger © 2017. All Rights Reserved.)

(Fig. 15). Symptoms are very dependent on high RH and soil moisture. Water-soaked, sloughed roots and dark brown necrotic stems with a wet appearance can distinguish Pythium root rot from other root rot diseases. Overall, wilt results from the weakened root system and sometimes only the wilted plant is apparent. Likewise, foliar symptoms of *Pythium* are necrotic and usually have a wet appearance rather than dry. In extremely wet, humid environments, *Pythium* mycelium can appear on the leaves but disappears quickly with mechanical contact such as by human touch or cutting tools. Microscopic observation can be used to distinguish *Pythium* from other root rot diseases.

Biology and epidemiology. *Pythium* is a soilborne pathogen which, like *Phytophthora*, belongs to a diverse group of fungus-like eukaryotes that was recently placed in the new kingdom Stramenopila including diatoms, brown algae, and golden-brown algae (Beakes and Sekimoto 2009). It survives in soil in a dormant state largely as chlamydospores or oospores usually associated with plant debris; hyphae and zoospores are likewise infective units but generally survive for shorter periods. *Pythium* can survive in dust and soil particles in the greenhouse as well as in soiled transplant trays and pots. In the greenhouse, soiled hands, tools, hose nozzles, and contaminated plant material are the primary means of introducing *Pythium* into the growing medium. In both greenhouse and field situations, abundant soil moisture is conducive to disease development, and the splashing of water causes spread. High soil moisture with high temperatures (27–32 °C [80–90 °F]) enhances wilting and root rot, whereas with low temperatures (10–16 °C [50–60 °F]), symptoms similar to those of nutrient deficiencies develop (likely no stem lesions or distinct wilting, but some sloughed roots). Hasan (1988) observed a synergistic interaction when *Pratylenchus coffeae* and *Pythium aphanidermatum* and/or *Rhizoctonia solani* coinfected chrysanthemum.

Management

- ***Cultural practices*** – Cultural conditions contribute to disease problems. Plants that are stressed are more prone to infection so sound horticultural practices are key in preventing this disease. Prevent prolonged saturated media conditions and use media that provides good aeration to avoid compaction. Inspect roots on a regular basis and avoid high soluble salts or extensive drying of the root system. Sound sanitation practices should always be implemented.
- ***Fungicides*** – Drench at transplanting or 7 d after sticking unrooted cuttings. Fungicide rotations are important in order to avoid resistance in the pathogen. The same fungicides that are active against *Phytophthora* should be effective against *Pythium*, but need to be trialed since not all fungicides that control Phytophthora are effective on Pythium. Fungicides that can be used to control Pythium include: fenamidone, pyraclostrobin, etridiazole, cyazofamid, propamocarb, fosetyl-Al, and phosphorus acid. *Pythium* isolates resistant to mefenoxam have been reported.

2.15 Rhizoctonia Root, Stem and Cutting Rot, Web Blight [*Rhizoctonia solani* Kuhn (syn., *Thanetephoros cucumeris* A. B Frank) Donk]

Geographic occurrence and impact. This disease is cosmopolitan and infects more than 400 species of plants in 75 families including important flower and vegetable crops as well as weeds (Daughtrey et al. 1995; Willets and Wong 1980; Boland and Hall 1994). This disease occurs wherever chrysanthemums are grown. Given its wide distribution, *Rhizoctonia solani* is very common in propagation of chrysanthemums.



Fig. 16 Rhizoctonia root and stem rot in cuttings (R.K. Jones © 2017. All Rights Reserved.)

Symptoms/signs. Cutting rot can be one of the more common symptoms and may often serve as the main method of infection. Infected roots decay very rapidly and reddish-brown lesions may appear just below the soil line. Root rot is also one of the most common symptoms. Aboveground symptoms are typical of most root rots including stunting and chlorosis followed by wilt, defoliation, and death (Fig. 16). During warm, humid conditions, *Rhizoctonia* can develop into a web blight where the aerial mycelium grows out of the soil medium onto the leaves that are in contact and create a weblike growth on the leaves and stems.

Biology and epidemiology. Chrysanthemums are most susceptible during the propagation phase. Wojdyla et al. (1988) reported artificially inoculated shoots developed symptoms over a wide range of temperatures 15–35 °C (59–95 °F) but had an optimum temperature between 25 and 28 °C (77–82 °F). The fungus can grow saprophytically on the surface of soil and thrives in potting media that is moist and warm. In India, the lesion nematode, *Pratylenchus coffeae*, increased the severity of Rhizoctonia stem and root rot (Hasan 1988).

Management

- ***Cultural practices and sanitation*** – Sanitation practices should be followed to avoid introducing the pathogen. Choose disease-free stock. Clean up greenhouses after a disease outbreak. Rhizoctonia stem and root rot can be suppressed using the same sanitation protocols for other soilborne diseases.

- ***Fungicides and biocontrols*** – Many fungicides have shown efficacy against *Rhizoctonia* on chrysanthemum including the strobilurins, fludioxonil, iprodione, pentachloronitrobenzene, thiophanate-methyl, and triflumizole. Beckerman and Koeing (2011) obtained excellent suppression with fludioxonil and mefenoxam + thiophanate-methyl. A variety of antagonists/biological control organisms have been evaluated for their ability to affect Rhizoctonia cutting rot on chrysanthemums. Tschen (1991) found in a research trial that *Aspergillus*, *Gliocladium*, *Paecilomyces*, and *Trichoderma* showed activity against *Rhizoctonia* root rot.

2.16 Sclerotinia Stem Rot, Cottony Stem Rot [*Sclerotinia sclerotiorum* (Lib. de Bary 1884)]

Geographic occurrence and impact. This disease is cosmopolitan and infects numerous plant species including flower and vegetable crops as well as weeds (Daughtrey et al. 1995; Willets and Wong 1980; Boland and Hall 1994). On *Chrysanthemum morifolium*, this disease was reported in Argentina, Canada, Korea, New Zealand, and Scotland and in the USA: Arizona, California, Florida, Hawaii, Louisiana, Michigan, North Carolina, and Virginia. On other *Chrysanthemum* species, the disease was reported in Argentina, Australia, Brazil, China, Kenya, Korea, New Zealand, Taiwan, and Tanzania and in the USA: California, Montana, Texas, Virginia, and Washington (Farr and Rossman 2017; Wright and Palmucci 2003).

Symptoms/signs. The pathogen causes a stem rot or blight and/or head rot in chrysanthemum; cottony stem rot is another name for the disease referring to the clumps of cottony white fungal hyphae (sign of this pathogen under humid conditions) often growing from the blighted stem and/or leaves (Agrios 2005; Horst and Nelson 1997; Willets and Wong 1980) (Fig. 17). Soft watery spots soon becoming brown are the first symptoms on the leaves. A dark green wet rot is the first symptom when plants are first attacked in the basal stem area (Fig. 18). The stem is quickly girdled and killed; the plant then wilts and dies. Sclerotinia wilt is yet another



Fig. 17 Sclerotinia stem rot: note white cottony fungal mycelium (Jane Trolinger © 2017. All Rights Reserved.)



Fig. 18 Basal rot of stem caused by *Sclerotinia* (Jane Trolinger © 2017. All Rights Reserved.)



Fig. 19 Black sclerotia of *Sclerotinia sclerotium* (*arrow*) inside rotted stem (Jane Trolinger © 2017. All Rights Reserved.)

descriptive disease name of the general wilted appearance. Infrequently, flower rot occurs in an infection apparently not related to stem or leaf blight; initial symptoms are brownish discoloration on the tips of lower petals and a brown watery rot of the flower receptacle resulting in petal drop (Holcomb and Motsinger 1968; McClellan 1941; Plakidas 1939). In addition to the cottony white fungal hyphae, other signs of *Sclerotinia* are sclerotia, highly resistant resting structures. Sclerotia are hemispherical, black, and 2–10 mm/0.08–0.4 in. in diameter and develop on the stem or in the pith cavity (McRitchie 1982) (Fig. 19). The sclerotia are actually compacted hyphae so they are white at first; the outer rind blackens with age and the sclerotia look like black beans. These sclerotia provide a reliable means of field diagnosis. Cutting

them in half should always show a white to buff-white center with a black outer shell.

Biology and epidemiology. *Sclerotinia* can survive as a pathogen or as a saprophyte on a large number of crops. Sclerotia are the primary means of survival; ascospores and mycelium in infected tissues have limited survival compared to the sclerotia (Cook et al. 1975; Willets and Wong 1980). Sclerotia produced in or on infected tissues can survive for up to 3 y in moist soil, up to 7 y in dry soil, and up to 9 y in a refrigerator (Cook et al. 1975; Strider DL personal communication). Infection is initiated by germinating sclerotia or by hyphae or by ascospores. Ascospores are produced when sclerotia are exposed to cold temperatures producing apothecia (tiny, pinkish to buff saucer-shaped structures). Apothecia mature under moist conditions, forcibly eject ascospores into the air over a period of 2–3 wk to inoculate aboveground plant parts (Abawi and Grogan 1975; Agrios 2005). Host cells are killed in advance of the fungal hyphae once infection is initiated. Germination of sclerotia and mycelial growth are favored by aerobic conditions, low temperatures (15–20 °C/59–68 °F areideal), and high RH. Ascospores are triggered by moist weather to germinate, and for them to cause infection, the RH must be above 90%. Under dry conditions, they can remain dormant for several weeks (Sharma et al. 2015). Disease development in the field is favored by low temperatures (15–20 °C/59–68 °F). For sclerotia to form apothecia, the temperature must be below 20 °C, and for the apothecia to mature and eject ascospores, the humidity must be high (Abawi and Grogan 1979; Coley-Smith and Cooke 1971). Ascospores may be windborne (up to 25 m/82 ft) and a single sclerotium may produce 2.3×10^8 ascospores (Abawi and Grogan 1979; Schwartz and Steadman 1978; Wu et al. 2008).

Management

- ***Cultural practices and sanitation*** – In the field, remove and discard infected plants immediately when infection is observed. In the greenhouse, do not reuse pots or transplant trays where the disease has been found without disinfestation; clean the benches and floor with a product registered for greenhouse cleaning. Soil disinfestation – when possible, steam sterilization is preferable due to its reliability in killing the sclerotia. Exclusion of the pathogen and complete eradication, if introduced, are necessary in order to continue crop production. Cultural measures, such as crop rotation in the field, for disease management in chrysanthemum are not an option because of the lengthy survival of sclerotia and broad host range of the fungus. Establish good drainage in the field – sclerotia require free water to germinate (Coley-Smith and Cooke 1971). Space plants for free air flow. Start with planting material that is pathogen-free.
- ***Fungicides and biocontrols*** – Prior to planting, the fumigant chloropicrin has been used effectively in the field (Besemer and McCain 1969). In the greenhouse, drench with thiophanate-methyl or pentachloronitrobenzene when *Sclerotinia* is found.

Use a spray rotation on remaining healthy plants chosen from the following fungicides: chlorothalonil, chlorothalonil + thiophanate-methyl, pyraclostrobin, cyprodinil + fludioxonil (commercial formulation), and azoxystrobin + benzovindiflupyr (commercial formulation). *Paraconiothyrium minitans* (W.A. Campb.) Verkley 2004 = *Coniothyrium minitans* W.A. Campb. 1947 is a commercially available biological fungicide with a specific antagonistic action against the sclerotia of *Sclerotinia* spp. (Ojaghian 2009). It can be used primarily as a soil treatment product in the greenhouse or open field. It destroys sclerotia irrespective of the crop. Other fungal antagonists such as *Clonostachys rosea* (Link: Fries) Schroers, Samuels, Seifert, and W. Gams (syn. *Gliocladium roseum* (Link) Bain), and *Trichoderma* spp. are able to parasitize the sclerotia (Jones et al. 2003; Turner and Tribe 1976; Whipps and Budge 1990). Rabeendran et al. (2006) showed *T. hamatum* LU593 applied as transplant treatments, as solid substrate soil amendments, or as a spore drench gave consistent disease control.

2.17 Southern Blight (*Sclerotium rolfsii*)

Geographic occurrence and impact. Southern blight is a problem in warm and wet tropical and subtropical areas but can appear in northern climes when infested material is shipped north. *Sclerotium rolfsii* causes disease on a wide range of agricultural and horticultural crops. Current estimates place the number of host genera at over 270 in the USA alone. Along with chrysanthemum, southern blight affects several ornamentals in the genera *Narcissus*, *Iris*, *Lilium*, and *Zinnia* (Farr et al. 1989; Mullen 2001).

Symptoms/signs. Southern blight is primarily a disease of stems and crown tissue, but when environmental conditions are optimal, it may infect any part of a plant including roots, petioles, leaves, and flowers. Symptoms are first observed as dark brown lesions on the stem at or just beneath the soil level; the first visible symptoms are progressive yellowing and wilting of the leaves. Following this, the fungus produces abundant white, fluffy mycelium on infected tissues and the soil. A diagnostic feature of southern blight is the presence of uniformly round, mustard seed-size sclerotia that first appear as white and then turn brown and then sometimes black. These can be differentiated from those of *Sclerotinia* where sclerotia are usually black and irregular in shape.

Biology and epidemiology. The disease is more prevalent in warmer regions than colder ones. Sclerotia are the main overwintering structures and the fungus can also survive as mycelium in infected tissue. The inoculum, however, does not tolerate freezing temperatures. Sclerotia are disseminated by cultural practices (infested soil and contaminated tools), infected transplant seedlings, water (especially through irrigation), wind, and possibly on seeds. The fungus rarely produces basidiospores (the sexual stage of reproduction).

Management

- ***Cultural practices and sanitation*** – Sanitation is extremely important as most inocula originate on infested stock and/or soil. A proper cleanup after an infestation occurs must be practiced. Good sanitary practices include roguing, eliminating weed hosts, and avoiding crop injury during cultivation. Rotating out of infested fields must be practiced, but care must be exercised to pick a non-susceptible rotation crop. Deep plowing to bury the sclerotia can also reduce inoculum. A dense canopy increases disease incidence; thus, increasing plant spacing can help keep infection down. Soil solarization has been beneficial in select situations in southern regions.
- ***Fungicides*** – Fungicides that are known to have activity against southern blight are pentachloronitrobenzene, azoxystrobin, azoxystrobin + benzovindiflupyr, flutolanil, fluoxastrobin, fludioxonil, and triticonazole.

2.18 Verticillium Wilt (*Verticillium albo-atrum* Reinke and Berthier, *V. dahliae* Kleb.)

Geographic occurrence and impact. Verticillium wilt of chrysanthemum tends to be a temperate disease and not commonly reported from warmer regions. It was first observed in 1923 in New Jersey, USA. The disease was one of the most destructive soilborne diseases in the industry before the 1940s (Dimock 1940) but has been minimized due to culture-indexed cuttings and sanitation practices (Dimock 1956). Although its importance has declined, it can still cause problems in field plantings.

Symptoms/signs. The first sign of Verticillium wilt is a chlorosis and wilt of the margins of the lower leaves often followed by the leaves on the whole stem wilting as the lower leaves dry up and defoliate. Early on, the disease can be just one sided on the stems. In some cultivars, wilting does not occur. Symptoms can resemble Fusarium wilt. Severely affected plants rarely produce flowers.

Biology and epidemiology. The fungus produces abundant microsclerotia in the host tissue after it has died, which provides for long-term survival in soil. The pathogen can be spread on cutting tools, pots, and transplant trays. Most infections, however, result from infected stem cuttings (Horst and Nelson 1997). Alexander and Hall (1974) studied the infection process on chrysanthemum and reported that the fungus infects very young roots shortly after the plant cutting is potted. Colonization of vessels of stem and leaves is initially discontinuous and was likely a result of conidia, but stems and petioles soon become clogged which restricted water flow.

Management

- ***Cultural practices and sanitation*** – Sanitation has been the number one strategy for eliminating *Verticillium* from the industry. Using clean, culture-indexed,

cutting stock, and steam-disinfested soil or soilless potting mixes will essentially eliminate *Verticillium*.

- ***Resistance*** – There are a wide range of reactions among cultivars to *Verticillium* wilt, but the success of sanitation has lessened the importance of developing resistant cultivars.

2.19 Additional Fungal Diseases Reported

For an up-to-date list of fungi associated with chrysanthemum, access http://nt.ars-grin.gov/fungaldatabases/fungushost/FungusHost.cfm and enter "chrysanthemum" in the host name box (Farr and Rossman 2017).

3 Bacterial and Phytoplasma Diseases

3.1 Aster Yellows ["*Candidatus* Phytoplasma asteris" and Other *Phytoplasma* spp.]

Geographic occurrence and impact. "*Candidatus* Phytoplasma asteris" has been reported to occur on all arable continents and causes aster yellows (AY) in a large number of cultivated crops including florists' chrysanthemum (CABI 2016a). Recent reports of AY induced by a number of *Phytoplasma* spp. include China and India ["*Candidatus* Phytoplasma asteris" (Min et al. 2009; Raj et al. 2007a)], Okinawa ["*Candidatus* Phytoplasma aurantifolia" (Naito et al. 2007), and Serbia ("*Candidatus* Phytoplasma solani" Mitrović et al. 2013).The pathogen caused losses of 70–80% in one of the largest chrysanthemum gardens in Yangling, Shanxi Province, China (Min et al. 2009).

Symptoms/signs. Phytoplasmas can produce a range of symptoms which may include stunting of whole plants, yellowing of leaves, necrosis, yellow-green discoloration of flowers (virescence), the modification of flowers into shoots and leaves (phyllody), and the proliferation of shoots (witches' brooms) due to suppression of apical dominance (Fig. 20).

Biology and epidemiology. Phytoplasmas are pleomorphic prokaryotes that lack cell walls. "*Candidatus* Phytoplasma asteris," which comprises the aster yellows group, is associated with diseases of over 100 plant species. Aster yellows phytoplasma strains belong to the16SrI group based on analysis of the 16S rDNA sequences (Lee et al. 2004). Aster leafhoppers [*Macrosteles fascifrons* (Hemiptera, Cicadellidae)] acquire the phytoplasma and spread it to new plants (Babadoost 1988). Infection of aster yellows in temperate areas occurs by migration of leafhoppers that overwinter on infected host plants in warmer areas. However, high temperatures (approximately 31 °C/88 °F) inactivate the phytoplasma in leafhoppers and plants.



Fig. 20 Aster yellows symptoms in chrysanthemum flowers: virescence (*left*), phyllody (*right*) (Naito et al. 2007).

Management

- ***Cultural practices*** – Rapidly remove infected plants. Eradicate susceptible hosts (including weeds) around chrysanthemum production sites. Use reflective mulches in field-grown mums to disorient the vectors. In greenhouse cultivation, use physical barriers such as fine mesh nets or screens to exclude the leafhoppers.

3.2 Chrysanthemum Phloem Necrosis (Phytoplasma-Like Organism)

Geographic occurrence and impact. In the 1970s and 1980s, a number of important cut flower cvs. in the USA, especially the Marble group (Pink, Blue, White, etc.), which accounted for a major portion of the market (~70%), exhibited foliar deterioration and loss of vigor attributed to infection by a phytoplasma-like organism (McGovern et al. 1989). Other researchers attributed the symptoms to manganese toxicity (Dienelt and Lawson 1991). Chrysanthemum phloem necrosis was also reported to occur in a number of field-grown garden chrysanthemum cultivars in the USA (NY) (McGovern and Horst 1986).

Symptoms/signs. The earliest cytological symptoms of the disease are collapse and necrosis of phloem cells, hence the name of the disease, chrysanthemum phloem necrosis (CPN). Macroscopic symptoms include yellowing of leaves, necrotic flecks primarily adjacent to leaf veins, premature foliar deterioration and desiccation starting with the lowest leaves, and occasional flower distortion (Figs. 21 and 22).



Fig. 21 Progression of chrysanthemum phloem necrosis symptoms from *left* to *right* (R.J. McGovern © 2017. All Rights Reserved.)



Fig. 22 Chrysanthemum phloem necrosis symptoms in a garden chrysanthemum (R.J. McGovern © 2017. All Rights Reserved.)

Biology and epidemiology. The pathogen is transmissible via stock-scion grafts to chrysanthemum and by means of dodder (*Cuscuta epithymum*) to *Catharanthus roseus* (Horst et al. 1983; McGovern et al. 1989). Symptoms of CPN appear to be exacerbated by high temperature (32 °C/90 °F). Interestingly, symptoms of the disease were more severe when chrysanthemums were grown with a low level of a commercial fertilizer which furnished plants with 150 ppm $MnSO_4$ than with a moderate level of the same fertilizer that supplied 600 ppm of the nutrient (McGovern 1986).

Management

- ***Cultural practices*** – Fertilize plants adequately.
- ***Antibiotics*** – Symptoms of CPN were significantly reduced by treatment with oxytetracycline HCl and tetracycline HCl but not by Na penicillin G (McGovern 1986).
- ***Resistance*** – Differences in susceptibility to CPN were observed among garden chrysanthemum cvs.: severe symptoms were observed in Lavender Pink and Yellow Starlet and mild symptoms in Quaker White, Ivory, White Stardom, and Stargazer (McGovern and Horst 1986).

3.3 Bacterial Blight and Soft Rot [*Dickeya chrysanthemi* (syn. *Erwinia chrysanthemi*)]; [*Pectobacterium carotovorum* (syn. *Erwinia carotovora*)]

Geographic occurrence and impact. The disease has been reported in China, Hungary, Italy, Japan, the Netherlands, Poland, and the USA (Bazzi et al. 1987; Burkholder et al. 1953; Horita 1995; Janse and Ruissen 1988; Jiang et al. 2002; Schollenberger 2004; Vegh et al. 2014) and is likely to occur wherever florists' chrysanthemums are produced because of the global distribution of the pathogens (Bradbury 1977a, b). Losses to bacterial blight were estimated to be up to 25% in Florida, USA, and the disease was particularly damaging to cut flower production (McFadden 1958).

Symptoms/signs. Internally infected plants exhibit vascular discoloration and reddish-brown rot of the pith. External symptoms may include grayish discoloration and collapse of infected stems, foliar blight, wilting, and death. Droplets containing bacteria may be exuded from infected tissue (Fig. 23). Stock plants may be systemically infected and not show symptoms.

Biology and epidemiology. *Dickeya* and *Pectobacterium* soft rotting bacteria have broad host ranges including many fruit, ornamental, and vegetable crops (Bradbury 1977a, b).The disease is favored by warm (>27 °C/80 °F), wet conditions (Burkholder et al. 1953). The bacteria are spread in diseased plant material, infected plant debris, contaminated tools, water and soil, and handling. The bacteria are opportunistic pathogens and require an impaired host and favorable environmental conditions to cause disease. Wounding during periods of favorable moisture and temperature facilitates infection and disease development.

Management

- ***Cultural practices*** – Always buy from a reputable breeding company which sells certified stock. Remove and discard affected plants. Avoid over-misting during propagation. McGovern et al. (1985) found that high rates of a complete fertilizer (473, 105, and 413, N, P, and K, respectively) and $(NH_4)_2SO_4$ (400 ppm N) increased rot in chrysanthemum cuttings and should be avoided.



Fig. 23 Bacterial blight and soft rot symptoms caused by *D. chrysanthemi*: pith maceration producing hollow stem (*upper photo*, Jane Trolinger © 2017. All Rights Reserved.) and stem discoloration, and collapse (*lower photo*, R.J. McGovern © 2017. All Rights Reserved.)

- ***Sanitation*** – Sanitation is the key to preventing this disease. Remove and discard affected plants; workers should sanitize their hands following handling diseased plants and before resuming routine activities. Contaminated work areas and tools should be disinfested.
- ***Bactericides*** – McFadden (1958) reported reduction of cutting rot caused by *D. chrysanthemi* following dipping in a streptomycin solution.
- ***Resistance*** – No cultivar resistance to bacterial blight in chrysanthemum has been reported. Interestingly, cutting rot was consistently reduced following prior infection with a symptomless strain of the *Chrysanthemum chlorotic mottle viroid* (McGovern et al. 1988).

3.4 Bacterial Fasciation/Leafy Gall [*Rhodococcus fascians* (syn. *Corynebacterium fascians*)]

Geographic occurrence and impact. This pathogen is globally distributed and causes sporadic losses especially in flower, bulb, and greenhouse-grown crops (Bradbury 1967; CABI 2016b).

Symptoms/signs. In florists' chrysanthemum, the bacterium induces shortening and thickening of the stem near the base and the development of aborted and deformed leaves; the plants are stunted and produce an abnormal number of buds (Pirone 1978). Incidence of severe fasciation symptoms increases with progressively longer periods of infection (Oduro 1975).

Biology and epidemiology. *Rhodococcus fascians* has a broad host range encompassing over100 species of ornamentals, vegetables, and fruits (Goethals et al. 2001; Putnam and Miller 2007).The disease is most severe during warm wet conditions. The bacterium survives on and in infected plant material and contaminated soil (Faivre-Amiot 1967) and seed (Pataky 1991). Water is an important means for disseminating *R. fascians* (Putnam and Miller 2007).

Management

- ***Cultural practices*** – Use pathogen-free propagative material.
- ***Sanitation*** – Outbreaks of the disease frequently occur in production facilities that fail to maintain rigorous sanitation. These strategies include roguing infected plants, isolating newly acquired stock plants from plants in production and monitoring them for disease symptoms, routinely renewing stock plants, disinfesting work surfaces and plant containers, and sterilizing growing media (Putnam and Miller 2007). Soil may be sterilized by steam (82.2 °C/180 °F for 30 min or 71.1 °C/160 °F for 1 h (Pataky 1991)).

3.5 Bacterial Leaf Spot/Bud Blight (*Pseudomonas cichorii*)

Geographic occurrence and impact. The disease has been reported in Brazil, Canada, Japan, New Zealand, the Netherlands, and the USA (Broadbent and Matteoni 1990; Horita 1993; Neto et al. 1976; Janse 1987; McFadden 1959; Young et al. 1987).

Symptoms/signs. Water-soaked lesions that give rise to large, black, circular or angular, necrotic spots on leaves, buds, and stems are typical symptoms (Fig. 24). Bud blight will also occur during prolonged wet periods. The bacterium can also cause internal necrosis in chrysanthemum stems (Jones et al. 1983).

Biology and epidemiology. *P. cichorii* causes diseases in several ornamental and vegetable crops. Host specificity is not known to exist in *P. cichorii*. Optimal temperature for the bacterium is 20–28 °C (68–82 ° F). Periods of high humidity and wetness, which are promoted by overhead irrigation and high plant density, are necessary for infection and disease development as is generally true for other foliar bacterial diseases. Jones et al. (1985) found that bacterial leaf spot was exacerbated by high light intensity and fertilizer rate and prolonged misting. The bacterium is primarily spread by water splash and over long distances by infected plant material. Broadbent and Matteoni (1990) also reported transmission of *P. cichorii* by the leaf miner *Liriomyza trifolii* (Diptera, Agromyzidae).



Fig. 24 Initial symptoms of bacterial leaf spot (*upper photo*, Jeff Gerberick © 2017. All Rights Reserved.) and advanced symptoms (*lower photo*, Paul Bachi © 2017. All Rights Reserved.)

Management

- ***Cultural practices*** – Use pathogen-free cuttings. If possible, avoid overhead watering or water early in the day to promote rapid leaf drying. Increase plant spacing to increase air circulation.
- ***Sanitation*** – Rogue infected plants; ensure that workers wear disposable gloves or disinfest their hands before resuming routine activities.
- ***Bactericides*** – McFadden (1959) found that sprays of either tribasic copper or streptomycin sulfate controlled the disease. He recommended that sprays should be applied weekly to susceptible cvs. and stock plants, especially during rainy weather.

3.6 Crown Gall [*Rhizobium radiobacter* (syn. *Agrobacterium tumefaciens*)]

Geographic occurrence and impact. There have been reports of infection of florists' chrysanthemum by *R. radiobacter* from Japan, New Zealand, Poland, the UK, and the USA (Ogawa et al. 2000; New and Milne 1975; Schollenberger 2005;



Fig. 25 Symptoms of crown gall in flowering chrysanthemum (Liz Hunt © 2017. All Rights Reserved.)

BSPP 2014; Miller 1975). However, given the global distribution of the pathogen, crown gall may occur wherever the crop is grown. Another *Rhizobium* sp., *R. skierniewicense* sp. nov., has been reported to cause crown gall of chrysanthemum in Poland (Pulawska et al. 2012). At present, crown gall does not appear to be a major problem for chrysanthemum production but diligence in keeping a crop free of this pathogen needs to be consistent.

Symptoms/signs. The bacterium causes gall formation on the crown, roots, stems, or branches of a plant and stunting. Galls are firm with roughened, irregular surfaces (Fig. 25). Inoculated cuttings can develop galls in 6 to 10 d depending on the susceptibility of the cultivar (Trolinger, unpublished data).

Biology and epidemiology. *Rhizobium radiobacter* can survive in the soil and in association with plant debris and herbaceous perennials (Miller and Putnam 2005). Flowering potted plants are infrequently infected by *R. radiobacter* unless they are brought into contact with contaminated field soil or are propagated from infected stock plants. The bacterium enters plants through wounds that occur on the roots or at the crown just under the soil surface. It can also enter stems and leaves through wounds. *Rhizobium radiobacter* is spread by detached galls, contaminated soil, irrigation, and uncertified infected stock plants. Because *R. radiobacter* genetically transforms its host plant, it has become a widely used tool in the genetic modification of many crops including chrysanthemum.

Management

- ***Cultural practices*** – Always buy from a reputable breeding company which sells certified stock.
- ***Sanitation*** – Preventive measures include frequent hand-washing (antibacterial soap and nail brush), foot baths, and sterilizing tools and benches (quaternary ammonium compounds with a thorough water rinse). Other disinfectants include

blends of acids, surfactants and wetting agents, and hydrogen dioxide. When field soil is used in a potting medium, it should be pasteurized. Discard symptomatic plants and remove them completely from the premises. Do not reuse pots or trays from which infected material was removed without disinfestation.

- ***Biological control*** – *Agrobacterium radiobacter* strain K84 was developed as a biological control for the closely related *Rhizobium radiobacter* (Kerr and Htay 1974); *A. radiobacte*r produces the bacteriocin (an antibiotic-like chemical) agrocin 84 which inhibits sensitive strains of *R. radiobacter*. Miller and Miller (1976) reported control of crown gall on stems, roots, and leaves of chrysanthemum by drenching *R. radiobacter*-infested soil with *Agrobacterium radiobacter* strain K84. Soil drenches and plant sprays with oxytetracycline controlled the disease, while drenching infested soil with vancomycin HCl significantly reduced crown gall development. However, another research group found that the biocontrol was less effective; the incidence of crown gall in *A. radiobacter*-treated plants was 63.6% compared to 97.5% in the control (Faivre-Amiot et al. 1982). Moreover, Putnam and Miller (n.d.) found that only 3 of 23 *R. radiobacter* isolates screened were sensitive to the genetically modified biocontrol *A. radiobacter* strain K1026.
- ***Resistance*** – Some degree of cultivar resistance to *R. radiobacter* exists in florist's chrysanthemum. Miller and Crane (1975) found that 10% of 237 cvs. were resistant to the bacterium. Likewise, Schollenberger (2005) reported that only 3 of 29 cvs. were resistant to the pathogen: Epidote Jaune, Epidote Orange, and Epidote Rouge, all potted chrysanthemums.

3.7 Additional Bacterial Diseases Reported

- **Bacterial leaf spot** (***Pseudomonas syringae***) (Italy, Carta 1993)
- **Bacterial wilt [*Ralstonia solanacearum* (syn. *Pseudomonas solanacearum*)]** (USA, Kelman 1953)
- **Pith necrosis** (***Pseudomonas corrugata***) (Italy, Fiori 1992)
- **Pith necrosis** (***Pseudomonas viridiflava***) (Crete, Greece, Goumans and Chatzaki 1998)

4 Virus and Viroid Diseases

4.1 Pospiviroidae *Chrysanthemum stunt viroid* (CSVd), Genus *Pospiviroid*

Geographic occurrence and impact. The genus also contains a number of other important viroids including the *Potato spindle tuber viroid* (PSTVd) for which chrysanthemum has been shown to be an artificial host. During the 1940s, CSVd was a serious threat to chrysanthemum production in the USA, which was averted by the isolation of mother blocks and viroid indexing (Brierly and Smith 1951; Diener and Lawson 1973; Dimock 1947).The viroid has been distributed worldwide (Matsushita 2013). Recent outbreaks of chrysanthemum stunt have occurred in the

ROK where the disease incidence in 64 cvs. ranged from 9.7 to 66.8 (Chung et al. 2005). In India, 70% of the cvs. screened were infected with CSVd (Singh et al. 2010).

Symptoms/signs. Symptoms of CSVd in chrysanthemum depend on the cultivar and environment and may include reduction of plant growth including flower size, early flower development, delayed rooting, leaf spots and distortion, and flower bleaching (Horst et al. 1977) (Figs. 26, 27, and, 28). Some cvs., although infected,



Fig. 26 Viroid symptoms in chrysanthemum leaves (*left* to *right*): viroid-free, CSVd, PSTVd, and CChMVd (R.J. McGovern © 2017. All Rights Reserved.)



Fig. 27 Normal flower (*left*) reduced size, bleached flower caused by CSVd (*right*) (Horst 2008).



Fig. 28 Flower trial showing how *Chrysanthemum* stunt affects growth and timing of flowering (Jane Trolinger © 2017. All Rights Reserved.)

remain symptomless and may provide a reservoir for the viroid. Symptoms are exacerbated by high temperature (26–29 °C/79–84 °F) and light intensity (Brierley 1952).

Biology and epidemiology. Viroids are the smallest known autonomous infectious nucleic acids, consist solely of a small, non-encapsidated, circular RNA genome of about 250–400 nt which apparently does not encode any protein and replicate and move systemically in host plants (Di Serio et al. 2014). The genome of CSVd is 354–356 nt in size consisting of a rodlike structure with a central conserved region (Cho et al. 2013). Natural infection of CSVd has occurred in a number of cultivated and wild plants mainly in the Asteraceae (Matsushita 2013) (Table 2). CSVd is easily transmissible by mechanical inoculation as are viroids in general and can be efficiently spread by contact with contaminated pruning tools, farm implements, clothing, and hands. Graft transmission and foliar/root contact between neighboring plants are also effective means of spread (Hammond and Owens 2006). The first indexing program for this viroid was based on graft transmission (Raju and Olson 1985).

Reports of seed transmission of the viroid in chrysanthemum are contradictory, but it has been confirmed in tomato. Seed transmission of CSVd in chrysanthemum was not demonstrated by Brierley (1952) or Hollings and Stone (1973), but Monsion et al. (1973) presented evidence for seed transmission of this viroid. On tomato, CSVd was detected in pollen from infected plants, and the application of this pollen to flowers on healthy plants resulted in infected seedlings of tomato (Kryczynski et al. 1988; Mink 1993). Kryczynski et al. (1988) proposed the possibility that plant species differ in their ability of transmitting viroids through pollens and seeds.

Table 2 Characteristics of CSVd and CChMVd viroids (based on Cho et al. 2013)

Characteristics	CSVd	CChMVd
Disease	Chrysanthemum stunt	Chrysanthemum chlorotic mottle
Symptoms	Light green young leaves, chlorotic spots, stunting, small leaves and flowers, decreased rooting ability, and early flowering	Yellow-green mottling, chlorosis, dwarfed size, and delayed flowering
Family and genus	Pospiviroidae, *Pospiviroid*	Avsunviroidae, *Pelamoviroid*
Genome size	354–356 nt	398–401 nt
Replication method	Asymmetric rolling circle mechanism	Symmetric rolling circle mechanism with the hammerhead ribozymes
Replication localization	Nucleus	Chloroplast
Structure	Rodlike structure including central conserved region (CCR)	Branched conformation including hammerhead ribozymes
Transmission	Sap, grafting, and seed	Sap, grafting
Host	Chrysanthemum, *Petunia hybrida*, tomato, *Gynura aurantiaca*, *Ageratum*, dahlia, *Senecio*, *Vinca major*, *Argyranthemum frutescens*, and many plants belonging to the families Solanaceae and Asteraceae	Restricted to chrysanthemum

Circumstantial evidence of seed transmission in chrysanthemum has been observed though not absolutely proven (J. Trolinger, unpublished).

Management

- ***Cultural practices*** – It is critical to use only certified viroid-free propagative material produced through isolation of mother blocks and virus/viroid indexing. Because of the possibility of seed transmission in chrysanthemum, seedlings used in breeding programs should not be precluded from a pathogen-free indexing program. Newly acquired plant material should be isolated and monitored to ensure that it is disease-free before including in general production.
- ***Sanitation*** – Pruning tools should be disinfested before moving from plant to plant. Sodium hypochlorite and sodium hydroxide plus formaldehyde have been reported to be effective in disinfesting tools contaminated with viroids (Garnsey and Whidden 1971; Matsuura et al. 2010; Roisstacher et al. 1969; Singh et al. 1989).
- ***Resistance*** – The chrysanthemum cvs. Sei no Issei and Mari Kazaguruma and progeny from the self-pollination of Utage were found to be resistant to CSVd (Omori et al. 2009; Nabeshima et al. 2012). The cv. Okayamaheiwa also showed strong resistance against CSVd, and after crossing with two susceptible cultivars, Sei-elza and Anri, a number of the first generation progeny were strongly resistant

to the viroid (Matsushita et al. 2012). Ogawa et al. (2005) demonstrated that a single tolerance gene in transgenic chrysanthemum plants could attenuate both CSVd- and TSWV-incited diseases.

4.2 Avsunviroidae *Chrysanthemum chlorotic mottle viroid* (CChMVd), Genus *Pelamoviroid*

Geographic occurrence and impact. CChMVd is a potential global threat to florists' chrysanthemum production. Recent outbreaks of CChMVd have occurred in China, India, Japan, Korea, the Netherlands, and the USA (Cho et al. 2013; Dimock et al. 1971). A survey of the viroid in Akita Prefecture Japan found CChMVd at an incidence of 20%; in a number of cases, the infection was symptomless (Yamamoto and Sano 2005).

Symptoms/signs. The symptoms of CChMVd vary by chrysanthemum cv. and may include mild mottling, often followed by a general chlorosis of new leaves, dwarfing, and delayed flowering (Figs. 26 and 29).

Biology and epidemiology. The genome of CChMVd is 398–401 nt in size and has a branched conformation including hammerhead ribozymes (Cho et al. 2013). The only known natural host of this viroid is florists' chrysanthemum. Horst (1975) detected a symptomless strain of CChMVd that cross-protects against the symptom-producing viroid. A comparison of CSVd and CChMVd is presented in Table 2.

Management. Refer to Sect. 4.1, *Chrysanthemum stunt viroid.*

4.3 Betaflexiviridae

Geographic occurrence and impact. *Chrysanthemum virus B* (CVB), family Betaflexiviridae and genus *Carlavirus*, is distributed globally (CABI 2016c). In



Fig. 29 Chrysanthemum cultivar Velvet Ridge exhibiting symptoms of *Chrysanthemum chlorotic mottle viroid* (Syngenta Flowers © 2017. All Rights Reserved.)



Fig. 30 Chrysanthemum cultivar Blanche exhibits a curly top symptom when coinfected with CVB and CSVd (Syngenta Flowers © 2017. All Rights Reserved.)

2003, a survey of chrysanthemum production in Himachal Pradesh, India, detected CVB at an incidence of 40.62–94.66% (Verma et al. 2003).

Symptoms/signs. Single infections of CVB may produce a mild mosaic (in the early literature, CVB was also referred to as Chrysanthemum mild mosaic virus), vein clearing, or necrotic streaks but are often symptomless. However, synergistic symptoms may be produced in mixed infections of CVB with other chrysanthemum viruses and viroids (CABI 2016c) (Fig. 30).

Biology and epidemiology. CVB has a very narrow host range consisting of florists' chrysanthemum and *Gynura aurantiaca* (symptomless host) (Moran 1987). The virus is transmitted in a non-circulative (nonpersistent) manner by five species of aphids (Hemiptera, Aphididae): *Myzus persicae*, *Macrosiphum euphorbiae*, *Acyrthosiphon* (*Aulacorthum*) *solani*, *Coloradoa rufomaculata*, and *Macrosiphoniella* (*Pyrethromyzus*) *sanborni*; mechanically; and by grafting (Hollings 1957).

Management

- ***Cultural practices*** – As is the case with other viruses and viroids, the foundation of CVB control is the use of certified virus/viroid-free propagative material produced through isolation of mother blocks and virus/viroid indexing.
- ***Sanitation*** – Sound phytosanitary practices should be implemented such as the removal and destruction of infected plants and disinfestation of pruning tools before moving from plant to plant.
- ***Vector management*** – Monitoring and reduction of aphid populations should be practiced through chemical and/or biological measures, vector exclusion by physical barriers such as fine mesh screens, vector disorientation in field-grown crops through the use of reflective mulches, and elimination of and avoidance of growing near established, infected chrysanthemum crops. Also refer to ► Chap. 4, "Insect Management for Disease Control in Florists' Crops."

4.4 *Bromoviridae*

Geographic occurrence and impact. Major crop losses are caused worldwide by viruses in this family. *Tomato aspermy virus* (TAV) and *Cucumber mosaic virus* (CMV), genus *Cucumovirus*, are members of this family that can cause significant damage to florists' chrysanthemum wherever it is produced. Verma et al. (2007) detected TAV and CMV at incidences of 26.2% and 42.5%, respectively, when screening 80 chrysanthemum cvs. in India.

Symptoms/signs. Severe flower distortion, size reduction, and color breaking are typical symptoms of TAV (Hammond et al. 1986, 1987) (Fig. 31). Plant stunting, necrotic ring spots, mosaic, yellowing, and green vein-banding in leaves have also been reported (Raj et al. 2007b; Rishi 2009). However, some TAV-infected chrysanthemum cvs. do not exhibit leaf symptoms or loss of vigor (Hollings and Stone 1971). Symptoms produced by CMV depend on the virus strain and chrysanthemum cv. and may include yellow spotting, stunting, mosaic, and mild to severe chlorosis and necrosis (Verma et al. 2004; Srivastava et al. 1992).



Fig. 31 Symptoms of TAV including flower distortion and yellowing of leaves (Raj et al. 2007b)

Biology and epidemiology. The natural host range of TAV is narrow and includes florists' chrysanthemum, tomato, *Canna* spp., and *Lilium* spp. (Hammond et al. 1986, 1987). On the other hand, the natural host range of CMV encompasses a very large number of ornamental, vegetable, and weed species (Garcia-Arenal and Palukaitis 2008).TAV and CMV are transmitted in a noncirculative (nonpersistent) manner by 10 and more than 80 aphid spp., respectively, including *Myzus persicae* (Garcia-Arenal and Palukaitis 2008; Kennedy et al. 1962). TAV and CMV are mechanically and graft transmissible and by seed in some plants not including chrysanthemum.

Management. Refer to the Betaflexividae, Sect. 4.3 above, and ▶ Chap. 4, "Insect Management for Disease Control in Florists' Crops."

4.5 *Bunyaviridae*

Geographic occurrence and impact. Plant viruses in this family and the genus *Tospovirus* infect a vast range of cultivated plants plus weed species and pose a serious worldwide threat to both greenhouse and field flower crops and most other major horticultural crops. Four of these viruses have been detected in chrysanthemum: *Chrysanthemum stem necrosis virus* (CSNV), *Impatiens necrotic spot virus* (INSV), *Iris yellow spot virus* (IYSV), and *Tomato spotted wilt virus* (TSWV). The geographic distribution of these viruses in chrysanthemum is indicated in Table 3.

Symptoms of CSNV were observed in two chrysanthemum cvs., at an incidence of more than 70% (approximately 30,000 plants), representing approximately 1000 m^2 of greenhouses of one grower in Hiroshima Prefecture, western Japan (Matsuura et al. 2007). One greenhouse grower in Belgium sustained losses that ranged in three cvs. from 50 to nearly 100%; the most susceptible cvs. were killed. All infected cvs. were propagated from cuttings imported from Brazil (De Jonghe et al. 2013). INSV and TSWV, alone and in combination, were reported to decrease chrysanthemum production by up to 80% in Colombia (Vasquez and Angarita 1999).

Symptoms/signs. See Table 3 for symptoms caused by tospoviruses in chrysanthemum.

Biology and epidemiology. These unusual plant viruses, closely related to viruses that infect animals and insects, are transmitted in nature by thrips (Thysanoptera, Thripidae) in a circulative (persistent) and propagative manner. Only the first and early second larval stages are able to acquire tospoviruses, and only immature thrips that acquire these viruses or adults derived from such immatures are vectors. Adult thrips remain viruliferous for life, but tospoviruses are not transovarial. Tospoviruses are actually difficult to mechanically transmit. It is not impossible, but unlikely, that tospoviruses will spread by normal handling of plants. The risk of spreading tospoviruses is higher on cutting knives or pruning equipment because sap transmission is possible.

Table 3 Tospoviruses infecting florists' chrysanthemum

Viruses	Occurrence in chrysanthemum	Transmission[a]	Other natural hosts	Symptoms in chrysanthemum	References
Chrysanthemum stem necrosis virus(CSNV)	Belgium, Brazil, Japan, Slovenia, the UK	*Frankliniella occidentalis*, *F. schultzei*	China aster (*Callistephus chinensis*), *Senecio* x *cruentus*, lisianthus (*Eustoma grandiflorum*), tomato (*Solanum lycopersicum*)	Severe necrotic streaks on stems, chlorotic and necrotic spots and rings on leaves, and leaf distortion	Alexandre et al. (1999), De Jonghe et al. (2013), Duarte et al. (1995), Matsuura et al. (2007), Mumford et al. (2003), Nagata et al. (1998), and Vozelj et al. (2003)
Impatiens necrotic spot virus (INSV)	North America, Colombia, the Czech Republic, Iran, Japan, Mexico, Poland, the Netherlands, Serbia, Slovenia, the USA	*F. fusca*, *F. intonsa*, *F. occidentalis*	At least 300 species in 85 different plant families	Chlorotic mottle/spots and necrosis on leaves Fig. 32	Balukiewicz and Krzyciński (2005), Daughtrey et al. (1997), Ghotbi et al. (2003), Horst (2008), Kondo et al. (2011), Mertelik et al. (2002), Ochoa et al. (1996), Moran (1994), Pappu et al. (2009), Stanković et al. (2013), Vasquez and Angarita (1999), and Vozelj et al. (2003)
Iris yellow spot virus (IYSV)	Poland	*F. fusca*, *T. tabaci*	Amaryllis (*Hippeastrum hybridum*), iris (*Iris hollandica*), lisianthus, onion, and related species, Peruvian lily (*Alstroemeria* sp.), spiny sow thistle (*Sonchus asper*), and a number of taxonomically diverse weed spp.	Not described	Bag et al. (2014), Balukiewicz and Krzyciński (2005), Derks and Lemmers (1996), Doi et al. (2003), Kritzman et al. (2000), Nischwitz et al. (2012), and Okuda and Hanada (2001)
Tomato spotted wilt virus (TSWV)	North America, Australia, Iran, Mexico, Poland, ROK, Serbia, Slovenia, the USA	*F. bispinosa*, *F. cephalica*, *F. fusca. F. gemina*, *F. intonsa*, *F. occidentalis*, *F. schultzei*, *T. palmi*, *T. setosus*, *T. tabaci*	>900 species in >90 monocotyledonous and dicotyledonous plant families	The symptoms vary by chrysanthemum cv. and environment and may include chlorotic leaf spots, leaf and axillary shoot necrosis, black streaks in the stem, wilting, apical bud death, and the death of young plants	Balukiewicz and Krzyciński (2005), Chung et al. (2006), Daughtrey et al. (1997), Horst (2008), Moran (1994), Nour et al. (2013), Ochoa et al. (1996), Pappu et al. (2009), Stanković et al. (2013), and Vozelj et al. (2003)

[a]Riley et al. (2011) and Srinivasan et al. (2012)



Fig. 32 Yellow mottle in leaf caused by INSV (Kondo et al. 2011)

Management

- ***Cultural practices*** – Use only certified virus/viroid-free propagative material produced through isolation of mother blocks and virus/viroid indexing. Newly acquired plant material should be isolated and monitored to ensure that it is disease-free before including in general production.
- ***Sanitation*** – In a greenhouse or field situation, all symptomatic plants should be removed and destroyed or removed from the premises. Sound phytosanitary practices should be practiced with tools (clean tools) since sap transmission is possible.
- ***Vector control*** – All stages of thrips must be controlled in order to control tospoviruses. Thrips, especially western flower thrips (*Frankliniella occidentalis*, WFT), is considered one of the most serious pests of ornamental crops. In addition to vectoring the tospoviruses, WFT damage plants directly by feeding. Control of thrips pupae in the growing medium is best accomplished by drench applications of insecticides. Daily scouting should be accomplished in conjunction with spraying rigorously or using a well-monitored biological control program to control all stages of thrips. Allen and Matteoni (1991) demonstrated that petunia is a useful indicator plant for thrips carrying TSWV. Matsuura et al. (2006) reported that verbena plants attracted western flower thrips (thrips trap crop) reduced their colonization of chrysanthemum until flower bud initiation and markedly suppressed TSWV incidence on chrysanthemums until flowering.
- ***Resistance/tolerance*** – Ogawa et al. (2005) demonstrated that a single tolerance gene in transgenic chrysanthemum plants could attenuate both TSWV- and CSVd-incited diseases. Sherman et al. (1998) used TSWV nucleocapsid (N) gene constructs in *Agrobacterium*-mediated transformation of chrysanthemum cv. Polaris which produced highly TSWV-resistant lines. Also refer to ► Chap. 4, "Insect Management for Disease Control in Florists' Crops."

Table 4 Other viruses reported but less common in chrysanthemum

Family/virus	Occurrence in florists' chrysanthemum	Transmission	Other natural hosts	Symptoms in chrysanthemum	References
Betaflexaviridae, *Potato virus X* (PVX, genus, Potexvirus)	China	Mechanical	Potato (*Solanum tuberosum*), *Brassica campestris* ssp. *rapa*	Often found in mixed infections with other viruses	Brunt et al. (1996) and Wu et al. (2002)
Nepoviridae, *Tomato ring spot virus* (ToRSV), *Tobacco ring spot virus* (TRSV), and *Arabis mosaic virus* (ArMV) (genus, *Nepovirus*)	Iran	Mechanically and by nematodes (*Xiphinema* spp.)	Many ornamentals (carnation, gerbera, sunflower, zinnia, etc.) and other cultivated hosts and weeds	Necrotic or chlorotic local lesions following mosaic, ring spots, or mottle symptoms on infected host plants; TRSV symptoms disappear soon after infection	Ghotbi and Shahraeen (2009)
Potyviridae, *Potato virus Y* (PVY, genus, *Potyvirus*); *Zucchini yellow mosaic virus* (ZYMV, genus *Potyvirus*)	China	Many species of aphids	PVY infects many cultivated and wild herbaceous hosts especially in the Solanaceae. ZYMV mainly infects the Cucurbitaceae	Yellowing and mottling of leaves	Liu et al. (2014) and Niu et al. (2015)

Other viruses reported but less common in florists' chrysanthemum are listed in Table 4.

5 Nematode Diseases

5.1 Foliar Nematode [*Aphelenchoides fragariae* (Ritzema Bos) Christie and *A. ritzemabosi* (Schwartz) Steiner and Buhrer]

Geographic occurrence and impact. *Aphelenchoides fragariae* has an extensive host range including 250 species of plants in 50 families. The primary floricultural families affected are Asteraceae, Bignoniaceae, Gesneriaceae, Liliaceae,



Fig. 33 Symptoms of foliar nematodes (Horst 2008)

Primulaceae, and Ranunculaceae. The host range of *A. ritzemabosi* is not as extensive but does include chrysanthemum and other members of the Asteraceae.

Symptoms/signs. Leaves become discolored quickly after invasion. The initial reaction is usually an overdevelopment of yellow leaf pigment which soon becomes greenish-brown patches. The affected area enlarges to vein-delimited lesions and blackens. Large V-shaped necrotic lesions are characteristic of foliar nematode infection of chrysanthemum leaves (Fig. 33). The V-shape opens on the leaf margin and is surrounded by yellow. The lower leaves, usually infected first, wilt and abscise from bottom to top. Shoots turn brown and die. Bud symptoms are rosetting and growth deformation which result from nematodes feeding in the buds (Esser 1966).

Biology and epidemiology. *Aphelenchoides* spp. can enter the host tissue through the leaf stomata and wounds and likely by direct penetration. Nematodes migrate from the soil to young plants usually entering a leaf touching the soil; they move up the stem and enter other leaves. The nematodes then migrate in leaf tissue and become more numerous eventually killing the leaves. The infested leaves then drop and the cycle continues as the nematodes return to the soil and then travel back up the plant stem. Some host plants have a high degree of tolerance to the nematodes and can harbor large populations while showing few or no symptoms. This is significant in that symptomless carriers can easily introduce destructive numbers of *Aphelenchoides* into clean stock (Esser 1966).

Management. Nematode-free mother stock is essential in producing nematode-free cuttings. An indexing system for clean stock production should include measures that would exclude nematodes from the plant material. Sanitation practices resulting in both clean water and disinfested soil are essential for field grown chrysanthemums. Refer to general management strategies for clean water systems and disinfested soil either by physical or chemical means (▶ Chaps. 9, "Sanitation for Management of Florists' Crops Diseases," and ▶ 8, "Soil/Media Disinfestation for Management of Florists' Crops Diseases"). Commercial potting mixes without natural soil should be nematode-free.

5.2 Additional Nematode Diseases Reported in Chrysanthemum

Lesion nematode (***Pratylenchus penetrans***) (widely distributed, Ravichandra 2014).

Root knot nematode (***Meloidogyne* spp.**) (widely distributed, Ravichandra 2014).

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Diseases of *Delphinium*

17

Stephen N. Wegulo

Abstract

Delphinium (Larkspur) is a field- or greenhouse-grown cut flower of significant economic importance. Diseases are a major limiting factor in the profitable cultivation of delphinium. Notable among these diseases are root, crown, and stem rots; powdery mildew; black leaf spot; aster yellows; and several viral diseases. Yield loss can be reduced and profitability increased by concerted efforts to manage delphinium diseases. Management tactics include host resistance and cultural, chemical, biological, and physical control. Integration of as many of these tactics as practically possible will maximize the effectiveness of disease management. This chapter presents information on the occurrence, symptoms, biology and epidemiology, and management of individual diseases of delphinium. Details are provided for diseases on which research has been done and published. Some diseases have only been observed and research has not been done or published. Such diseases are only briefly mentioned.

Keywords

Blight • Leaf spot • Root and crown rot • Wilt • Mildew • Aster yellows • Viral diseases

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R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_17

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1 Introduction

The genus *Delphinium* consists of 300 or more species belonging to the family Ranunculaceae (Bassett and Bassett 2007). True delphiniums are herbaceous perennials. The common name, larkspur, is shared with a separate, closely related genus, *Consolida*, comprising annual species. Flower structure distinguishes the two genera. Flowers of species in the genus *Consolida* have a single petal and a single carpel (seedpod), whereas flowers of species in the genus *Delphinium* have two or four petals and three or more carpels (Bassett and Bassett 2007; Mabberley 1997). Delphiniums are native to the northern hemisphere, although a few species occur along the mountain ranges of tropical Africa. They produce a range of beautiful flower colors including blue, pink, purple, cream, and white which contribute significantly to the aesthetic value of homes, gardens, and the landscape and are grown commercially for cut flowers. In 2006, the wholesale value of larkspur (*Delphinium* spp. and *Consolida* spp.) as a cut flower in the USA for

operations with sales worth $100,000 or more was $ 8 million, with California accounting for 80% of the sales (Jerardo 2007).

Diseases can significantly reduce marketability of delphiniums as plants or cut flowers. The most common diseases include powdery mildew, bacterial leaf spot, and root and crown rots. Delphiniums can also be attacked by viruses and to a lesser extent by nematodes. The diseases found in a given location or geographical area will vary depending on environmental conditions. Losses due to diseases can be reduced by integrating available management tactics which include host resistance, cultural practices, physical control, chemical control, and biological control.

2 Fungal and Fungus-Like Diseases

2.1 Botrytis Blight (*Botrytis cinerea*)

Geographic occurrence and impact. Botrytis blight, also known as gray mold, has been reported on delphinium in the USA (Horst 2013; Pirone 1978; Pscheidt and Ocamb 2016) but is a ubiquitous disease of many plants worldwide.

Symptoms/signs. Symptoms include blighting of stems, petioles, leaves, and flowers (Fig. 1); seedling damping off; stem cankers; and rots. A basal rot is common on delphiniums grown in the greenhouse; affected plants wilt and fall over. They develop a soft rot and a gray mold develops on the basal tissue. The fungus sporulates profusely on all affected parts of the plant. Sclerotia (compact masses of mycelia) can form inside or on the surface of infected plants (Agrios 2005; Pirone 1978).

Biology and epidemiology. Botrytis blight is favored by high relative humidity and cool temperatures. The optimum temperature range for growth and sporulation of *B. cinerea* is 18–23 °C. However, the pathogen can remain active outside this temperature range and can cause considerable losses in products stored at temperatures as low as 0–10 °C. The greenhouse environment is especially favorable for



Fig. 1 Botrytis blight on delphinium flowers (L. Jones © 2017. All Rights Reserved.)

growth and sporulation of *B. cinerea*. Free water on the plant surface is required for infection to occur. During favorable conditions, *B. cinerea* sporulates profusely, producing a mass of gray mycelium and spores. Sclerotia are a means of long-term survival of the fungus. They germinate by producing mycelia which can infect plants directly or occasionally they can germinate carpogenically by forming apothecia and ascospores. The fungus also can survive as mycelium in soil and plant debris. Spores are disseminated by air currents and will germinate and infect healthy plants if they land on wounds or senescent plant parts. Insects such as moths, thrips, and fruit flies (Holz et al. 2004) also can spread spores from infected to healthy plants.

Management

- ***Cultural practices*** – Scouting for detection of diseased plants coupled with sanitation and good cultural practices can be effective in reducing damage and losses caused by *B. cinerea*. Removal and destruction of infected plants and infested plant debris can help to reduce the amount of inoculum. Good aeration keeps the plant surface dry, which prevents spores from germinating. In the greenhouse, humidity can be reduced by ventilation and heating.
- ***Resistance*** – There are no data on resistance of delphinium cultivars to Botrytis blight. It is likely that some cultivars have good resistance to the disease. Where such cultivars are identified, they should be planted and highly susceptible cultivars should be avoided.
- ***Fungicides*** – Application of chemical and biological fungicides was shown to be effective in controlling Botrytis blight on lisianthus (Wegulo and Vilchez 2006c) and is likely to be effective on delphinium as well (see *Botrytis blight* in ► Chaps. 20, "Diseases of Lisianthus," and ► 7, "Fungicides and Biocontrols for Management of Florists' Crops Diseases").

2.2 Diaporthe Blight (*Diaporthe arctii*)

Geographic occurrence and impact. Diaporthe blight occurs infrequently. It has been reported in the USA (Anonymous 1960; Boewe 1964) mainly on annual larkspur (Pirone 1978).

Symptoms/signs. After plants have reached the flowering stage, the lower leaves become dry and brown but remain attached to the stem. Brown lesions appear near the base of the stem which may be completely girdled, resulting in death of the entire foliage. Pycnidia form in the stems, uppermost leaves, petals, and seed capsules. During wet weather, a cottony mass of mycelium covers the crown and uppermost roots (Forsberg 1975; Mulford and Weiss 1939; Pirone 1978).

Biology and epidemiology. The anamorph of *Diapothe* is *Phomopsis*. Perithecia form and survive on dead plant debris. Ascospores are released from perithecia and cause infections on leaves, usually after flowering. The pathogen spreads to the stem

through the petioles. Lesions appearing on petals and seed capsules may result from direct infection by ascospores or spread from the stem. Infected seed can be a means of pathogen introduction in noninfested areas. However, the role of pathogen transmission from infected seed to the plant in disease development has not been elucidated. Wet weather promotes fungus growth especially on the crown and roots.

Management. Remove and destroy infected plants and infested plant debris. Ploughing to bury infested plant debris can reduce inoculum load. Plant pathogen-free seeds in noninfested sites (Forsberg 1975; Mulford and Weiss 1939; Pirone 1978).

2.3 Fusarium Stem Canker and Wilt (*Fusarium oxysporum* f. sp. *delphinii*)

Geographic occurrence and impact. Stem canker and wilt has been reported in the USA, UK, and Japan (Alfieri et al. 1984; Anonymous 1960; Kondo et al. 2013; Ryan 1966; Shaw 1973).

Symptoms/signs. Brown to black water-soaked lesions appear on stems and petioles resulting in cankered areas when the lesions coalesce (Fig. 2). Cankers may cover over 30 cm of the stem on large plants. Numerous salmon-colored sporodochia cover the cankered areas (Fig. 3). Discoloration of internal tissues is visible in cross-sections of stems and petioles (Fig. 4), and the pith cavity is filled with white mycelium. Affected plants wilt and have poorly developed root systems. Leaves may yellow and droop. Invasion of the vascular system occurs when the fungus reaches the crown and roots, eventually resulting in plant death (Forsberg 1975; Kondo et al. 2013; Laskaris 1949; Pirone 1978).

Biology and epidemiology. *F. oxysporum* f. sp. *delphinii* produces three forms of asexual spores: macroconidia, microconidia, and chlamydospores (Laskaris 1949). Macroconidia and microconidia are formed in sporodochia on stems and can be disseminated by windblown rain or splash dispersal. Chlamydospores are formed directly from vegetative hyphae or by conidial transformation and are the means by which long-term survival of the fungus in plant debris or soil is achieved. The sexual stage of *F. oxysporum* has not been reported. Infections were initiated when plants were sprayed with a spore suspension (Laskaris 1949) or by dipping roots in a spore suspension (Kondo et al. 2013), implying that in nature infections can start in the root zone when chlamydospores germinate or on stems when macroconidia and microconidia are dispersed by wind or water splash from rain or irrigation. In pathogenicity tests in a glasshouse, leaves of delphinium plants maintained at 20–30 °C began wilting 11 days after inoculation and entire plants began wilting 18 days after inoculation (Kondo et al. 2013), indicating that disease development is favored by warm temperatures.



Fig. 2 Symptoms of stem canker and wilt on delphinium plants naturally infected with *Fusarium oxysporum* f. sp. *delphinii* (Reprinted from Kondo et al. (2013), with permission from The Phytopathological Society of Japan and Springer Japan)

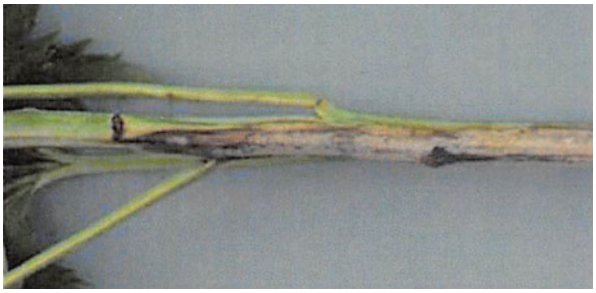


Fig. 3 Orange sporodochia (not visible) give a salmon color to cankered areas on a delphinium plant naturally infected with *Fusarium oxysporum* f. sp. *delphinii* (Reprinted from Kondo et al. (2013), with permission from The Phytopathological Society of Japan and Springer Japan)

Management. Inoculum in infested sites or fields can be reduced by removing and destroying infected plants or infested plant debris, soil disinfestation through fumigation or solarization, or by not planting delphinium in these locations for several years. Fungicides that have been effective against other formae speciales of



Fig. 4 Discoloration of vascular bundles in delphinium caused by *Fusarium oxysporum* f. sp. *delphinii* (Reprinted from Kondo et al. (2013), with permission from The Phytopathological Society of Japan and Springer Japan)

F. oxysporum on ornamental crops are likely to be effective against *F. oxysporum* f. sp. *delphinii*. Screening delphinium cultivars for resistance against stem canker and wilt is desirable. Cultivars found to be highly susceptible should be avoided. Although not confirmed, seed transmission of *F. oxysporum* f. sp. *delphinii* is likely. Disease free seed or transplants should be used.

Additional wilt pathogens. Delphinium wilt can also be caused by *Verticillium albo-atrum* (Horst 2013).

2.4 Leaf Spot and Crown Rot (Diplodina Disease) (*Ascochyta aquilegiae*; Formerly *Diplodina delphinii*)

Geographic occurrence and impact. This disease has been reported in the USA (Laskaris 1950). It occurs infrequently.

Symptoms/signs. Crown and root rot is the most common form of the disease, but it can also manifest itself as stem cankers, leaf spots, shoot or petiole soft rot, or flower and seed pod blight (Forsberg 1975; Laskaris 1950). Lesions on stems are brown to black and variable in size and shape. Pycnidia appear in concentric rings on the surfaces of lesions. Diseased petiole tissues appear brown and water soaked. The pathogen spreads rapidly down the petiole and causes a brown, water-soaked area to form around the base of the petiole. Pycnidia form in infected petiole tissue, especially at the point where the petiole and leaf blade are attached. During wet weather, the pathogen attacks young basal shoots, causing a watery brown to black soft rot. Affected basal shoots may wilt suddenly during the growing season. On leaves, tarry black lesions of variable size and shape form on the upper surface

whereas light brown lesions form on the lower surface. These symptoms are indistinguishable from those of black spot caused by *Pseudomonas syringae* pv. *delphinii*. Root and crown rot symptoms are similar to those caused by other pathogens. A dark brown to black discoloration of vascular tissues is characteristic of the disease, in contrast to the brown discoloration caused by *Rhizoctonia solani* or *Fusarium oxysporum* f. sp. *delphinii*.

Biology and epidemiology. The pathogen survives on host plant debris and in crowns of living plants. When mature pycnidia are wetted, numerous conidia ooze out in long, gelatinous coils and are spread by splashing water. Disease development is favored by wet weather, high humidity, and excessive fertilization.

Management

- ***Cultural practices*** – Plant delphiniums on raised beds. Remove and destroy diseased plants. Space plants so that there is adequate air circulation which helps to keep plant surfaces dry. Avoid overhead irrigation or practices that keep plant surfaces wet for extended periods of time. Fields with a history of the disease should not be replanted with delphinium for at least 2 years (Laskaris 1950; Pscheidt and Ocamb 2016; UC IPM 2009).
- ***Fungicides*** – There are no efficacy data on fungicides for *Diplodina* disease in delphinium. However, broad spectrum fungicides labeled for use on ornamental crops may be effective if applied preventively or before severe symptoms develop.

Additional leaf spot pathogens. Additional pathogens causing leaf spots on delphinium include *Cercospora delphinii*, *Ovularia delphinii, Phylosticta* spp., *P. delphinii, Ramularia delphinii*, and *Septoria delphinella* (Horst 2013). *Synchytrium aureum* causes leaf gall (Horst 2013).

2.5 Sclerotinia Wilt and Stem Rot (*Sclerotinia sclerotiorum*)

Geographic occurrence and impact. Sclerotinia wilt and stem rot of delphinium has been reported in the USA (Forsberg 1975; Horst 2013). The pathogen is widely distributed and causes problems on a large number of ornamental and vegetable plants.

Symptoms/signs. On delphinium, a general wilting of the newer leaflets and shoots occurs first followed by whole branches, especially the lower ones. Cankers occur on the lower stem and become extensive, causing plants to wilt and fall over. A dense white mycelium forms on the surface of the stem. Black sclerotia form on (Fig. 5) and inside the stem (Forsberg 1975).

Biology and epidemiology. *S. sclerotiorum* has a wide host range that includes at least 64 plant families (Purdy 1979). It survives as sclerotia on the soil surface or in



Fig. 5 Black sclerotia of *Sclerotinia sclerotiorum* on stem bases of delphinium (S. Wegulo © 2017. All Rights Reserved.)

the top soil for long periods of time. Sclerotia can germinate carpogenically following cold temperature conditioning to form apothecia which when mature release ascospores. The ascospores are dispersed by wind and infect the plant by germinating to form mycelia on senescent flowers or in wounds on the stem. The mycelium grows into the plant tissues which are killed as a result of enzymatic processes and the production of oxalic acid (Lumsden 1979; Purdy 1979). Sclerotia can also germinate eruptively to form a mycelium which directly infects plant parts it contacts. Disease development is favored by cool, wet conditions.

Management

- ***Cultural control*** involves removal of infected plants, spacing plants far apart to allow sufficient air circulation, and avoiding frequent or excessive watering.
- ***Fungicides*** labeled in the USA for *S. sclerotiorum* such as iprodione and thiophanate-methyl can be applied to control *Sclerotinia* wilt and stem rot in delphinium.
- ***Biological control*** – A commercial formulation of the biocontrol agent *Coniothyrium minitans* can be incorporated into the top 2.5–5 cm of soil (Pscheidt and Ocamb 2016).

Additional stem rot pathogens. Stem rot on delphinium can also be caused by *Phytophthora* spp. (Horst 2013).

2.6 Sclerotium Root and Crown Rot (*Sclerotium rolfsii, S. delphinii, Pellicularia rolfsii*)

Geographic occurrence and impact. Sclerotium root and crown rot has been reported in the USA (Forsberg 1975; Horst 2013).

Symptoms/signs. The lower leaves become discolored and the young shoots wilt, followed within a few days by death and drying of the entire plant (Fig. 6) (Forsberg 1975; Pirone 1978). The roots and crown become black or dark brown and rotted. Severely infected plants are easily pulled up exposing the diseased roots and crown (Fig. 7). In wet weather, small round sclerotia form on infected plant parts. The sclerotia are initially white to cream colored and change to dark red or chocolate brown when mature.

Biology and epidemiology. The causal fungi survive in the soil or plant debris as sclerotia which are dispersed by means of moving water, infected plants, or in infested plant debris. Under wet conditions, the sclerotia germinate eruptively by forming mycelia which infect plant parts they contact. Following infection, a mycelium rapidly grows within the plant, killing the tissues and causing yellowing of leaves and sudden wilting. Disease development is favored by warm temperatures and the presence of moisture (Agrios 2005).

Management

- ***Cultural practices*** – Remove and destroy diseased plants by burying or burning. Ensure good drainage. Plowing to bury sclerotia can reduce inoculum buildup over time.
- ***Soil disinfestation*** – Soil solarization for 2–3 months during the summer in conducive climates can kill sclerotia.
- ***Fungicides*** registered for root and crown rot can be applied to plants or as a soil drench to control the disease.

2.7 Pythium Crown and Root Rot (*Pythium* spp.)

Geographic occurrence and impact. Pythium crown and root rot has been reported on delphinium in the USA (Anonymous 1960).

Symptoms/signs. Leaves turn yellow then brown. Plants may be stunted and wilt in advanced stages of disease development. Brown, water-soaked lesions can be seen on the collar area of the stem, just below the soil line. The whole stem may be girdled by these lesions. Roots turn brown starting at the tips. The cortical layer consisting of rotted cells can easily be pulled from the white vascular tissue or can slough off if the rot is extensive. The root system is reduced and in severe infections plant death may occur (Bloch et al. 1976).

Biology and epidemiology. Pythium spp. produce a white mycelium that grows rapidly and gives rise to sporangia. A sporangium germinates directly by giving rise to a germ tube or by producing a short hypha bearing a balloon-like vesicle at its end. Within the vesicle, 100 or more zoospores are produced. When released from the vesicle, the zoospores move around in a swarm for a few minutes then form cysts which germinate by giving rise to a germ tube. The germ tube infects host tissue by



Fig. 6 Wilting and death of delphinium plants due to root and crown rot caused by *Sclerotium rolsfii*. Similar symptoms can be caused by *Sclerotinia* wilt and stem rot incited by *Sclerotinia sclerotiorum* (S. Wegulo © 2017. All Rights Reserved.)



Fig. 7 Black and rotted roots and crown of delphinium caused by *Sclerotium rolsfii* (S. Wegulo © 2017. All Rights Reserved.)

direct penetration. The mycelium can also produce a female oogonium and a male antheridium. The antheridium produces a fertilization tube which enters the oogonium. Nuclei of the antheridium move through the tube and fuse with nuclei of the oogonium to form a zygote, which becomes an oospore by producing a thick wall around itself. The oospore is resistant to adverse environmental conditions and is the survival and resting stage of *Pythium* spp. When environmental conditions become favorable,

oospores germinate in a manner similar to that of sporangia by directly forming a germ tube or by first forming zoospores and cysts. Rootlets can be infected at any stage of plant growth. Root rot progresses upward to the crown and stem. Disease development is favored by wet and waterlogged soils (Agrios 2005). Zoospores are disseminated by flowing water or splash dispersal. In the greenhouse, the primary means of introducing *Pythium* propagules are contaminated plant material and soiled hands, tools, and hose ends (Daughtrey et al. 1995).

Management

- ***Cultural practices*** for control include good water drainage. If the planting site is poorly drained, plant delphiniums in raised beds. Composted bark can be incorporated into the soil in the raised bed to improve aeration. Drainage tiles can be used to facilitate drainage and direct water away from the planting site. Do not reuse pots or trays from a previous crop for propagation. If pots or trays are reused, they should be sanitized or sterilized first.
- ***Fungicides*** registered for *Pythium* control such as sodium, potassium, and ammonium phosphites; etridiazole + thiophanate-methyl; mefenoxam; and etridiazole can be used to control Pythium crown and root rot (Pscheidt and Ocamb 2006).

Additional diseases caused by Pythium spp. *Pythium spp.* and *Rhizoctonia solani* can cause damping off (death of seeds or seedlings during or after germination) (Horst 2013).

Additional crown and root rot pathogens. Root and crown rot on delphinium can also be caused by the following pathogens: *Phoma* spp. (Forsberg 1975), *Phymatotrichum omnivorum* (Horst 2013), and *Rhizoctonia solani* (Horst 2013).

2.8 Powdery Mildew (*Erysiphe aquilegiae* var. *ranunculi*) (Formerly *Erysiphe polygoni*) and *Podosphaera delphinii* (Formerly *Sphaerotheca humili* var. *fuliginea*)

Geographic occurrence and impact. Powdery mildew has been reported in the USA (Anonymous 1960). It occurs wherever delphiniums are grown.

Symptoms/signs. White to gray powdery masses of mycelium and conidia form on leaf, stem, and flower surfaces (Figs. 8 and 9). Curling and stunting of young leaves and growing tips occur when infections are severe (Pirone 1978). Late in the growing season, minute, darkly colored ascocarps (sexual fruiting bodies) form on infected areas.

Biology and epidemiology. The causal fungi are obligate parasites. They overwinter by means of ascocarps (cleistothecia) in plant debris or mycelia in dormant plant tissue. During the growing season, conidia and ascospores released from ascocarps are dispersed by wind and infect healthy plants. Conidia are produced prolifically on



Fig. 8 Powdery mildew on leaves of delphinium (S. Wegulo © 2017. All Rights Reserved.)



Fig. 9 Powdery mildew on leaves, stems, and flowers of delphinium (S. Wegulo © 2017. All Rights Reserved.)

the surface of infected plant parts. They are blown by wind and cause new infections on the same or other plants. Disease development is favored by moderate to warm temperatures, high humidity, and shade. Unlike the case with other fungal pathogens, excessive water on the foliage inhibits infection and disease development (Jarvis et al. 2002). Powdery mildew fungi typically have a very narrow host range limited to a single or a few closely related plant species.

Management

Cultural practices – Grow delphiniums in full sun, ensure good water drainage, and space plants to allow sufficient air circulation. Destroy infested plant debris. Avoid planting highly susceptible cultivars.

Resistance – Cultivars with good resistance include Cameliard, Galahad, Blue Bird, Oriental Blue, and King Arthur (Wegulo and Vilchez 2008).

Fungicides – The fungicides myclobutanil and propiconazole achieved good control when applied in field trials (Wegulo and Vilchez 2006a, b). Many fungicides labeled for use on ornamental plants have good to excellent efficacy against powdery mildews. For effective control, fungicides should be applied preventively.

2.9 Downy Mildew (*Peronospora ficariae*)

Geographic occurrence and impact. Downy mildew on delphinium has been reported in California, USA (Tjosvold and Koike 2003).

Symptoms/signs. Tjosvold and Koike (2003) observed and described symptoms of downy mildew on delphinium. Initial leaf symptoms are light green patches that quickly turn dark green to blackish green and are often delimited by the central vein of the leaf (Fig. 10). A purple-gray sporulation of the pathogen occurs on the lower leaf surface. Symptoms are severer on older leaves. The lower leaves wither and dry up as the disease progresses, giving the plant a blighted appearance (Fig. 11).

Biology and epidemiology. *Peronospora* spp. are obligate parasites. Little is known about the biology and epidemiology of *P. ficariae* on delphinium. However, it is likely that the biology and epidemiology of this pathogen are similar to the biology and epidemiology of *Peronospora* spp. causing downy mildew on other plant species. In general, downy mildews are favored by wet, cool conditions and high relative humidity. Survival is by means of oospores and mycelia in infected



Fig. 10 Downy mildew symptoms on a delphinium leaf (S. Tjosvold © 2017. All Rights Reserved.)



Fig. 11 Withering and drying of lower leaves on delphinium caused by downy mildew (S. Tjosvold © 2017. All Rights Reserved.)

leaves, stems, sepals, and flower buds. Dichotomously branched sporangiophores arise from intercellular mycelium in the leaves and produce sporangia through stomata on the lower leaf surface. Sporangia are dispersed by blowing rain or splashing water. They germinate by means of germ tubes which penetrate leaf surfaces to cause new infections (Agrios 2005; Alexopoulos et al. 1996). For infection to occur, free water is required on the plant surface. The optimum temperature range for downy mildews is 5–21 °C.

Management

- ***Cultural practices*** – Remove and destroy infected plants. In the greenhouse, lowering humidity through ventilation and aeration and raising the temperature slows disease development. In the landscape or in the field, space plants to allow sufficient air circulation which lowers humidity and keeps leaf surfaces dry for longer periods of time.
- ***Fungicides*** – Applying fungicides labeled for downy mildew control is the most effective tactic for managing the disease. Examples of such fungicides are aluminum tris (O-ethyl phosphanate); sodium, potassium, and ammonium phosphites; fenamidone; dimethomorph; and mefenoxam.

Additional fungal diseases of delphinium. The following fungal diseases have been reported on delphinium in the USA: rust (*Puccinia delphinii, P. recondita*; Horst 2013; Pirone 1978), leaf and stem smut (*Urocystis sorosporioides*; Horst 2013), and white smut (*Entyloma winteri, E. wyomingense*; Horst 2013).

3 Bacterial and Phytoplasma Diseases

3.1 Black Leaf Spot (*Pseudomonas syringae* pv. *delphinii*)

Geographic occurrence and impact. Black leaf spot has been reported in the USA (Horst 2001). Significant losses can occur among seedlings (Forsberg 1975).

Symptoms/signs. Irregular tar-like black spots appear on leaves (Fig. 12), petioles, stems, and flower buds. On the lower surface of leaves, the spots appear brown. Under favorable environmental conditions for disease development, the spots enlarge and coalesce to form large blotches (Fig. 13) which can result in leaf distortion (Forsberg 1975; Horst 2001; Pirone 1978).

Biology and epidemiology. *P. syringae* pv. *delphinii* survives intercrop periods in the crowns on infected plants, in plant debris, or in the soil. During the growing season, bacteria are spread to new leaves by water splash and enter the leaves through stomata or wounds. Infections are more numerous following a hail or sand storm which creates wounds on the plants. Disease development is favored by cool, wet weather.

Management

- ***Cultural practices*** – Rotate fields to nonhost crops. Remove and destroy infected leaves or if infections are severe, entire plants. Space plants to allow sufficient air circulation. Avoid overhead watering. In the greenhouse, keep leaves dry through ventilation and aeration. After handling diseased plants, wash hands thoroughly



Fig. 12 Irregular tar-like black spots (*black leaf spot*) on the upper surface of a delphinium leaf caused by *Pseudomonas syringae* pv. *delphinii* (S. Wegulo © 2017. All Rights Reserved.)



Fig. 13 Symptoms of black leaf spot caused by *Pseudomonas syringae* pv. *delphinii* on the upper surface of a delphinium leaf incubated in a moist chamber for 3 days (S. Wegulo © 2017. All Rights Reserved.)

with warm water and soap before returning to crop production areas or handling healthy plants. Provide adequate nitrogen fertilization.

- ***Fungicides*** – Application of copper-based fungicides can provide some control (Forsberg 1975; Horst 2001; Pirone 1978; UC IPM 2014).

3.2 Soft Crown Rot and Black Leg (*Pectobacterium atrosepticum*, Formerly *Erwinia caratovora* subsp. *atroseptica*)

Geographic occurrence and impact. This disease has been reported in the USA (Horst 2013).

Symptoms/signs. Leaf yellowing, blackening of stem bases, and plant stunting are characteristic symptoms. Symptoms often appear at the flowering stage. In moist conditions, the stem is girdled quickly and a frothy, smelly ooze forms abundantly on the affected area on the stem. Stems may fall over due to rotting of the stem and crown. If diseased stems are split, mucilaginous masses of bacteria that ooze from the tissues are observed. Rotted tissues give off an offensive smell. Under dry conditions, the ooze may be scant or lacking and disease development is slowed. The leaves turn yellow and wilt, eventually becoming brown. In continued dry conditions, new shoots may form from the crown (Forsberg 1975; Horst 2001; Pirone 1978; UC IPM 2014).

Biology and epidemiology. *P. atrosepticum* survives in plant debris and soil and can contaminate seed. Infection occurs through cracks or wounds on the plant. Spread is by means of water. Disease development is favored by warm, wet weather.

Management

- ***Cultural practices*** – Avoid overhead irrigation especially during flowering. Plant delphiniums in raised beds. Avoid planting in low, poorly drained locations.
- ***Soil and seed disinfestation*** – Sterilize, heat, or fumigate soil used to raise seedlings. Hot water treatment of seed can reduce spread through contaminated seed.
- ***Fungicides*** – Application of copper-based fungicides can reduce losses.

Additional bacterial pathogens. Delphiniums are also susceptible to *Agrobacterium tumefaciens*, causal agent of crown gall (Horst 2013; Pirone 1978).

3.3 Aster Yellows ("*Candidatus Phytoplama* asteris")

Geographic occurrence and impact. Delphinium aster yellows has been reported on delphiniums in Japan, Lithuania, and the USA (Horst 2001; Navalinskiene et al. 2004; Tanaka et al. 2007). Severin (1942a) lists six states in the USA in which delphinium aster yellows was confirmed. According to Severin (1942a), the disease sometimes caused, during the second year, total failure of delphiniums grown from seed.

Symptoms/signs. The major symptoms of aster yellows on delphinium are plant dwarfing, short stems with a bunchy growth, general leaf yellowing, phyllody (flowers resembling leaves), and virescence (replacement of flower pigments with chlorophyll) (Forsberg 1975; Pirone 1978; Severin 1942a). Commonly, dense clusters of yellow shoots about 15 cm high develop from the crown and do not form flower spikes (Fig. 14).



Fig. 14 Aster yellows on delphinium. Note the short, yellowish dense clusters of shoots without flower spikes and the absence of flowers on the taller, yellowish plants (S. Wegulo © 2017. All Rights Reserved.)

Biology and epidemiology. The phytoplasma causing aster yellows in delphinium and other plant hosts is phloem limited and is vectored by the aster leafhopper (Agrios 2005; Severin 1942a). The phytoplasma survives in many plant species including perennial weeds, ornamentals, and vegetables. The leafhopper acquires the phytoplasma through its stylet while feeding on the phloem of an infected plant. Following acquisition, there is an incubation period in the leafhopper during which the phytoplasma multiplies in various organs and moves to the salivary glands. The leafhopper then transmits the phytoplasma when it feeds on a healthy plant. In delphinium, Severin (1942a) found an average incubation period (time from inoculation of plants by leafhoppers to first appearance of symptoms) of 19.5 days in seedlings, 43.5 days in adult plants inoculated before spikes appeared, and 45 days in adult plants inoculated after spikes developed. Female leafhoppers reared on aster yellows phytoplasma-infected plants have been shown to live longer and bear more offspring than those reared on healthy plants (Beanland et al. 2000).

Management. Remove and destroy infected plants. Control weeds in and around delphinium plantings. Control the leafhopper vector with insecticide sprays and exclusion strategies. Avoid planting delphiniums next to aster yellows–susceptible crops.

4 Viral Diseases

Delphiniums are susceptible to a number of viral diseases. Many of the viruses have a wide host range and cause a variety of symptoms including mottling, mosaic, or chlorosis on leaves (Fig. 15), stunting of the entire plant (Fig. 16), leaf curling, and ringspots and chlorosis on leaves. Different viruses may cause similar symptoms. Therefore, symptoms alone are not sufficient to identify the causal virus. A serological or molecular test is required for positive identification.



Fig. 15 Mottling and chlorosis on delphinium leaves caused by an unknown virus (S. Wegulo © 2017. All Rights Reserved.)



Fig. 16 Stunting of the delphinium plant shown in Fig. 15 (S. Wegulo © 2017. All Rights Reserved.)

4.1 Delphinium Ringspot Virus (Unidentified Virus)

Geographic occurrence and impact. Delphinium ringspot virus has been reported in California, USA (Horst 2013; Pirone 1978; Severin and Dickson 1942).

Symptoms/signs. Severin and Dickson (1942) described the symptoms of delphinium ringspot on delphinium. On naturally infected young leaves of delphinium, irregular, faint chlorotic rings measuring 1–5 mm in diameter enclose yellow or green centers with concentric chlorotic and green lines. Among these rings are scattered small, irregular chlorotic areas. On mature leaves, irregular chlorotic rings measuring about 10 mm in diameter encircle green areas, 1–2-mm-wide yellow bands, and 1–4-mm-wide irregular chlorotic areas. In addition, the areas near the margin of mature leaves show numerous small, concentric, chlorotic rings. Green and yellow lines encircling large circular or irregular chlorotic areas can be seen on older leaves.

Biology and epidemiology. There is no known vector of Delphinium ringspot virus. Severin and Dickson (1942) observed natural infection only on delphinium. They were able to mechanically transmit the virus to various susceptible host plants that included 11 species in 8 genera belonging to five families: Chenopodaceae, Malvaceae, Rananculaceae, Solanaceae, and Cucurbitaceae. In the greenhouse, symptoms appeared 32–42 days following inoculation. Thermal inactivation of the virus occurred at 65 °C for 10 min.

Management. Avoid planting delphiniums close to plants or crops known to be susceptible to Delphinium ringspot virus. In the greenhouse, sanitize tools between plants or cultivars. Wash hands with warm water and soap after handling infected plants.

4.2 Celery Calico [*Alfalfa Mosaic Virus* (AMV), Genus: *Alfamovirus*]

Geographic occurrence and impact. Celery calico has been reported on delphinium in the USA (Horst 2013; Severin 1942b). The causal agent, Alfalfa mosaic virus (AMV), has a wide host range and worldwide distribution.

Symptoms/signs. Severin (1942b) described the symptoms of celery calico on delphinium. In the field, in which the symptoms are very conspicuous, the lower and intermediate leaves show pale orange, amber, or lemon-yellow irregular areas. Younger leaves are symptomless. Patterns of symptoms on leaves vary considerably. Entire leaves may be pale-orange or lemon in color, or some leaves may show irregular chlorotic areas on some or all of the lobes. Small green areas may be scattered in the chlorotic areas of some leaves, whereas irregular discolorations may occur on other leaves of the same plant. Line or ring patterns frequently occur. The lines may be broken and contain a series of chlorotic dashes or dots. Alternating green and yellow lines may occur. Ring patterns may resemble ringspots composed of chlorotic dots encircling green areas. The chlorotic dots may be arranged in groups and the rings contain concentric, alternating green and yellow lines around green centers. Flowers are normal and breaking in flower color does not occur.

Biology and epidemiology. AMV is the sole member of the genus *Alfamovirus* in the family *Bromoviridae*. The virus has a very broad host range consisting of both cultivated crops and weeds. AMV is transmissible mechanically, by aphids, and by seed in some plant hosts. Several strains of AMV exist. The thermal inactivation point is between 50 °C and 65 °C (Smith 1972). Severin (1942b) determined the incubation period on delphinium to range from 11 to 178 days. Delphiniums may be symptomless carriers during the second year following infection. Under natural conditions, calico is often associated with aster yellows in delphiniums (Severin 1942b).

Management. In experimental inoculations, some delphinium varieties showed low infection rates (Severin 1942b), indicating that some varieties may have genetic resistance to calico. Where known, such varieties should be planted and varieties known to be very susceptible should be avoided. Avoid planting delphiniums close to susceptible weeds or crops. Use virus free seed or transplants. Control aphids through the use of insecticides and exclusion tactics such as fine mesh screening and reflective mulches where practical.

4.3 *Cucumber Mosaic Virus* (CMV, Genus: *Cucumovirus*)

Geographic occurrence and impact. Cucumber mosaic virus has been reported on delphinium in England, Israel, Italy, Lithuania, Taiwan, and the USA (Bellardi and Bertaccini 1993; Chen et al. 1995; Gera 1994; Horst 2013; Navalinskiene et al. 2004; Smith 1972). It has a very wide host range and occurs worldwide. Delphinium is very susceptible to the virus (Smith 1972). During 1991 and 1992 CMV occurred at

an incidence of 10–15% in delphiniums in Emilia-Romagna (Italy), and these outbreaks were attributed to seed infection by the virus (Bellardi and Bertaccini 1993).

Symptoms. Naturally infected annual larkspur plants are chlorotic, stunted, appear rosetted, and rarely produce flowers. Pale areas on the leaves follow the veins and a green mosaic mottle is present. Leaflets are chlorotic and curled downward and may be mottled with light green (Smith 1972). On young leaves in experimentally infected perennial delphiniums, Severin (1942c) observed circular, chlorotic areas that sometimes were distributed along the veins. These areas coalesced to form pale-yellow vein banding and later chlorosis. On lower leaves, green spotting in chlorotic areas, green vein banding, and a faint mottling or mosaic pattern were observed. Green blister-like elevations (a common symptom on many CMV-infected host plants) were rare.

Biology and epidemiology. CMV is the type member of the plant virus genus *Cucumovirus* in the family *Bromoviridae*. It is mechanically transmissible and can also be transmitted by aphids and through seed (Smith 1972). In experimentally infected delphiniums, it caused systemic infection (Severin 1942c). CMV has a very broad host range which includes many ornamentals, vegetables, and weeds.

Management. Aphid vectors can be controlled by use of insecticides and exclusion tactics such as the use of fine mesh screening and reflective mulch where practical. Information on delphinium cultivar resistance to CMV is lacking. When known, resistant cultivars should be planted. Because of the wide host range, control of hosts such as weeds is likely to be ineffective.

4.4 *Tomato Spotted Wilt Virus, Impatiens Necrotic Spot Virus* (TSWV, INSV, Genus: *Tospovirus*)

Geographic occurrence and impact. TSWV has been reported on delphinium in Australia (Latham and Jones 1997), England (Smith 1936), and the USA (Horst 2013). Another closely related tospovirus, INSV, was reported in this host in Italy (Dellavalle et al. 1999). Severin (1942c) stated that TSWV ranked next to aster yellows in seriousness as a disease of delphinium in California's coastal regions where in some areas plants in entire fields were observed to be infected by the virus.

Symptoms/signs. On experimentally infected delphiniums (Severin 1942c), the first symptom of TSWV on leaves consists of pale green, circular, elliptical, or irregular areas that may become surrounded by chlorotic rings. On some leaves, numerous double concentric rings varying in size and containing an outer green and an inner chlorotic ring occur. Chlorotic rings surrounding green centers may also be seen. Some leaves may show green or chlorotic banding of veins in large lemon-yellow blotches that may or may not have rings. In advanced stages of the disease, black rings varying in size surround chlorotic tissue which eventually becomes black on the lower leaves. Black patches spread on most of the lobes on the leaves, and

petioles and veins become necrotic. The lower leaves turn brown and dry with the blackened areas still visible.

Biology and epidemiology. TSWV is the type member of the genus *Tospovirus* in the family *Bunyaviridae*. It infects more than 800 plant species in more than 80 plant families. INSV has a somewhat narrower but still sizeable host range. These viruses are transmitted by several species of thrips. Transmission occurs only if the viruses are acquired during the thrips' larval stages. The viruses replicate within the thrips, and late 2nd instar larvae and adults can transmit them. Both the thrips and virus are spread when ornamental and vegetable transplants are transported. Weeds serve as overwintering and oversummering hosts. Seed transmission of TSWV has not been reported (Adkins et al. 2012).

Management. Use virus free transplants. Where known and available, plant resistant delphinium cultivars. Control thrips with insecticides – this may be more practical in the greenhouse than in the field. UV reflective mulches can be used in the field to reduce the numbers of incoming thrips. Remove and destroy symptomatic plants. Refer to ▶ Chap. 4, "Insect Management for Disease Control in Florists' Crops" for additional information (Table 1).

5 Nematode Diseases

5.1 Root Knot Nematode (*Meloidogyne* spp.)

Geographic occurrence and impact. Root-knot nematode has been reported in the USA (Horst 2013).

Symptoms/signs. In greenhouse trials, Kokalis-Burelle and Rosskopf (2013) and Wang and McSorley (2005) reported low levels of galling caused by *Meloidogyne* spp. on roots of *Delphinium*. In larkspur cultivar "Qis White," shoot weight was reduced by *M. incognita* race 1 (Wang and McSorley 2005). Low levels of galling were also observed in field trials (Kokalis-Burelle and Rosskopf 2013).

Biology and epidemiology. *Meloidogyne incognita*, *M. javanica*, and *M. hapla* have been reported to infect delphinium (Horst 2013; Pirone 1978). Low levels of galling were observed in greenhouse and field trials, leading to the conclusion that *Delphinium* is a poor host of *Meloidogyne* spp. Root-knot nematodes feed and reproduce within plant roots, inducing small to large galls known as root-knots. The adult females are sedentary in plant roots and have a round to pear-shaped body. Adult males are vermiform, leave the root, and become free-living in the soil. Adult females lay eggs in gelatinous sacs that are deposited on the surface of galled roots or may remain within the galls. The egg develops into a vermiform first juvenile stage (J1) which molts into a vermiform second juvenile stage (J2) that emerges from the egg into the soil. If the J2 (the only infective stage) encounters a susceptible host, it enters the root and becomes sedentary. It feeds on

Table 1 Additional viruses reported to naturally infect delphinium

Virus	Family, genus	Occurrence in delphinium	Transmission	Host range	Symptoms in delphinium	Management	References
Aconitum latent virus	Betaflexiviridae, *Carlavirus*	Japan	*	Domain Eucarya	Faint mosaic on leaves; spots on petals	*	Fuji et al. 2002
Arabis mosaic virus	Secoviridae, *Nepovirus*	UK	Nematodes (*Xiphinema* spp.); by seed in certain hosts; by dodder	Many species of wild and cultivated dicots and monocots	*	*	Ahmed and Bailiss 1975
Beet curly top virus	Geminiviridae, *Curtovirus*	USA	Beet leafhoppers	300 plant species in 44 families	*	Remove and destroy infected plants; control leafhoppers with insecticides; use resistant cultivars if available	Horn et al. 2011; Horst 2013; Pirone 1978; Severin 1942c
Cherry leaf roll virus	Secoviridae, *Nepovirus*	UK	Mechanically; Nematodes (*Xiphinema* spp.); by seed in certain hosts	Wide experimental host range	*	*	Ahmed and Bailiss 1975
Broad bean wilt virus	Secoviridae, *Fabavirus*	UK	Mechanically	>40 species in 14 mostly dicot families	Severe stunting and leaf deformation	*	Ahmed and Bailiss 1975

Delphinium vein clearing virus	Potyviridae, *Potyvirus*	USA	*	*	Vein clearing; chlorotic mosaic; stunting	*	Robertson and Brown 2009
Potato virus X	Alphaflexiviridae, *Potexvirus*	UK	Mechanically; certain grasshoppers; the fungus *Synchytrium endobioticum*	>240 species in 16 families	*	*	Ahmed and Bailiss 1975
Raspberry ring spot virus	Secoviridae, *Nepovirus*	UK	Nematodes (*Longidorus* spp.); by seed in certain hosts	Many species of wild and cultivated monocots and dicots	*	*	Ahmed and Bailiss 1975
Strawberry latent ringspot virus	Secoviridae, *Sadwavirus*	UK	Nematodes (*Xiphinema* spp.); by seed in certain hosts; by dodder	Many species of wild and cultivated plants	*	*	Ahmed and Bailiss 1975
Tomato ringspot virus	Secoviridae, *Nepovirus*	Lithuania	Nematodes (*Xiphinema* spp.); by seed in certain hosts	>35 dicot and monocot families	Leaf distortion mottling, light green or yellow ring spotting	*	Navalinskiene et al. 2004

*Information is lacking or insufficient

the root cells surrounding its head and grows, becoming sausage shaped. The saliva it secretes causes the root cells to enlarge and form galls. The J2 molts into a third stage juvenile (J3) which molts into a fourth stage juvenile (J4). Both the J3 and J4 are sedentary within the root. The J4 undergoes a final molt into a mature male which leaves the root or a mature female which remains sedentary in the root and lays eggs. Root knot nematodes are spread by contaminated equipment, infected plant material, or water.

Management. Sanitize equipment after use. Contaminated soil can be disinfested by fumigation, drenching with nematicides, or heat treatment with steam or solarization. Refer to ▶ Chap. 8, "Soil/Media Disinfestation for Management of Florists' Crops Diseases" for specific strategies.

Additional nematode pathogens. The leaf and stem nematode *Ditylenchus dipsaci* and the lesion nematode *Pratylechus pratensis* have been reported on delphinium (Horst 2013).

6 Nonparasitic Disorders

6.1 Association with *Peziza repanda*

Geographic occurrence and impact. An association between delphinium and the ascomycetous cup fungus *Peziza repanda* has been reported in California, USA (Wegulo et al. 2005).

Symptoms/signs. Wegulo et al. (2005) observed numerous fruiting bodies of *P. repanda* in two delphinium fields in southern California. Fruiting bodies were observed in association with bases of delphinium stems at the soil surface (Fig. 17). In approximately 1% of associations, fruiting bodies appeared to retard plant growth (Fig. 18), and in a few cases they caused plant death (Fig. 19).

Biology and epidemiology. *Peziza repanda* is an operculate ascomycete. It grows on and derives its nutrients from rotted wood, wood chips, or soil rich in decayed wood or covered with wood chips. Fruiting bodies (apothecia) are large (measuring 4–10 cm across), fleshy, brittle, single or clustered, and lack a stalk. They are cup shaped when young but spread and become wavy and irregular to nearly flat when mature (Figs. 17 and 19). Ascospores are forcibly discharged from mature apothecia. The fungus is common in nurseries that raise plugs on container media amended with wood material. Association of the fungus with stem bases of field-grown delphiniums is an indication that plugs were contaminated in the nursery with spores of the fungus. Retardation of delphinium plant growth or the resulting plant death due to association with *P. repanda* was most likely due to competition for space, water, and nutrients (Wegulo et al. 2005).

Management. Use steam-sterilized container media to raise plugs in the nursery.



Fig. 17 Apothecia of *Peziza reppanda* at stem bases of field-grown delphinium plants (S. Wegulo © 2017. All Rights Reserved.)



Fig. 18 A delphinium plant with retarded growth due to association with *Peziza reppanda* (S. Wegulo © 2017. All Rights Reserved.)



Fig. 19 Death of a delphinium plant due to its association with *Peziza reppanda* (S. Wegulo © 2017. All Rights Reserved.)

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Diseases of *Gerbera*

18

Elizabeth I. Brisco-McCann and Mary K. Hausbeck

Abstract

Gerbera daisy, *Gerbera jamesonii* H. Bolus ex. Hooker, is a spectacular cut flower popular around the world for its bold colors. Crop production is challenged by numerous diseases affecting petal quality and plant health. Powdery mildew, caused by *Golovinomyces cichoracearum* or *Podosphaera* sp., is the most common foliar disease of *Gerbera* daisies, but they are also plagued by Botrytis blight and Alternaria leaf spot. *Gerbera* is also susceptible to a host of root and crown rot diseases, including those caused by *Fusarium*, *Phytophthora*, and *Pythium* spp. Disease management strategies vary depending on the production system involved and can include cultural, chemical, and biological controls as well as the use of resistant cultivars.

Keywords

Alternaria alternata • *Botrytis cinerea* • *Fusarium oxysporum* • *Gerbera* • *Golovinomyces cichoracearum* • *Podosphaera* sp. • *Oidium* sp. • *Phytophthora cryptogea* • *P. drechsleri* • *P. palmivora* • *Pythium* spp. • *Pseudomonas cichorii* • *Cucumber mosaic virus* (CMV) • *Tospovirus* • *Tomato spotted wilt virus* (TSWV) • *Impatiens necrotic spot virus* (INSV)

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R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_18

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1 Introduction

Gerbera daisy, *Gerbera jamesonii* H. Bolus ex. Hooker, is an herbaceous perennial and important cut flower grown globally for its wide spectrum of colors and long-lasting blooms. *Gerbera* daisy is also known as Transvaal daisy or African daisy as it is endemic to the Transvaal region in southern Africa (Tourjee et al. 1994). In the United States, *Gerbera* cut flowers represent a US$31M industry with nearly 100 M stems sold in 2014 (Anonymous 2015a). *Gerbera* ranks second in cut flower production in the United States and Canada and fourth in Holland, China, and Mexico (Hanks 2015). *Gerbera* crop development began in the late nineteenth century when it was first introduced to Europe and the United States from Africa (Tourjee et al. 1994). *Gerbera* production is challenged by numerous diseases that can affect flower quality and quantity (Reddy 2016). In particular, powdery mildew, botrytis blight, and Alternaria leaf spot can destroy quality and marketability of *Gerbera* flowers with their unsightly symptoms (Bhat et al. 2013; Granke et al. 2012; Hausbeck and Moorman 1996). Crown and root rots caused by *Phytophthora*, *Fusarium*, and *Pythium* spp. can decimate crops by killing plants under severe disease pressure or, at a minimum, seriously limiting productivity (Martin and Loper 1999; Olson and Benson 2011; Pataky 1988). Production systems range from closed hydroponic systems to protected greenhouses to open fields, and propagation is primarily by seed.

Specific disease management information is provided in the following sections where data are available.

2 Fungal and Fungus-Like Diseases

2.1 Alternaria Leaf Spot (*Alternaria alternata*)

Geographic occurrence and impact. *Alternaria* leaf spot has been reported on *Gerbera* daisy in Australia, Barbados, Brazil, Bulgaria, India, Iran, Japan, the United States, Zambia, and Zimbabwe (Alfieri et al. 1984; Bitancourt 1938; Bobev 2009;

Farhood and Hadian 2012; French 1989; Mendes et al. 1998; Mirkova and Konstantinova 2003; Nishikawa and Nikashima 2015; Norse 1974; Raabe et al. 1981; Rao 1969; Riley 1956; Sen and Gupta 1996; Simmonds 1966; Whiteside 1966).

Symptoms/signs. Alternaria leaf spot first appears as small water-soaked spots on lower, older leaves. Spots become sunken and brown as they mature. They may also have a yellow halo or appear as concentric rings (Daughtrey et al. 1995). Lesions coalesce, leading to leaf chlorosis and defoliation. During severe infections, "shot holes" may also be produced (Nagrale et al. 2012). Affected *Gerbera* plants show reduced vitality and suppressed development and have fewer, smaller flowers that are distorted in shape (Farhood and Hadian 2012).

Biology and epidemiology. *Alternaria* is one of the most frequently occurring airborne fungal pathogens (Elad 2000). *A. alternata* is known to cause disease on a wide range of ornamental plant species including *Chrysanthemum*, *Dahlia*, *Hedera*, *Helianthus*, *Hibiscus*, *Pelargonium*, and *Petunia* in addition to *Gerbera* (Pegg et al. 2014). Spore dispersal is promoted by rapid increases in humidity or exposure to red light (Rotem 1994). Initial infection occurs by adhesion of conidia to the leaf cuticle, followed by spore germination, germ tube growth, and appressorium development (Troncoso-Rojas and Tiznado-Hernandez 2014). Conidia germinate quickly if moisture is present and can enter plant tissue through wounds, natural openings, and by direct penetration of the host cuticle (Troncoso-Rojas and Tiznado-Hernandez 2014). Conidia can germinate at temperatures between 10 °C and 35 °C (50 °F and 95 °F), but the pathogen requires longer wet periods for spores to germinate at the lower temperatures (Troncoso-Rojas and Tiznado-Hernandez 2014). *A. alternata* can overwinter as mycelia and/or conidia in soil, seed, or infected plant debris (Rotem 1994).

Management

- ***Cultural practices*** – Reducing relative humidity can help manage Alternaria leaf blight symptoms in *Gerbera* daisy (Bhat et al. 2013). This can be accomplished by limiting irrigation and spacing plants to provide better ventilation. Occurrence of Alternaria leaf spot during shipping and transport of *Gerbera* can be managed by minimizing temperature fluctuations and using containers that allow airflow during shipment (Daughtrey et al. 1995).
- ***Sanitation*** – Disease spread can be limited by immediate removal of diseased plants and good sanitation practices (Bhat et al. 2013).
- ***Fungicides and biocontrols*** – Fungicides including tridemorph, ziram (zinc dimethyldithiocarbamate), and mancozeb (zinc and manganese ethylene bisdithiocarbamate) have been found to be effective at controlling *Alternaria alternata* both in vitro and in pot culture experiments (Ghosh et al. 2002). Nagrale et al. (2012) reported that preventive sprays of tricyclazole (0.1%), Bordeaux mixture (0.6%), and iprodione + carbendazim (0.1%) were the most effective for the management of disease on *Gerbera* daisy followed by sprays of

difenoconazole (0.1%), propiconazole (0.1%), and hexaconazole (0.1%). However, resistant strains of *A. alternata* to fungicide classes including benzimidazoles, dicarboxamides, phenylpyrroles as well as Qo inhibitors and SDHI fungicides have been reported (Malandrakis et al. 2015). Sutar et al. (2012) found that the risk of *A. alternata* isolates from *Gerbera* developing insensitivity to carbendazim can be reduced by using it in combination with other fungicides including mancozeb, quinclorac, and copper hydroxide. Ghosh et al. (2002) looked at numerous plant extracts and biofungicides as control agents of Alternaria leaf blight of *Gerbera*. They found *Piper nigrum* and *Curcuma longa* extracts as well as *Trichoderma viridae*, *T. hamatum*, and *Aspergillus awamori* to be effective at limiting pathogen growth.

- ***Resistance*** – Resistance to *A. alternata* has been reported in different *Gerbera* cultivars in India. Kulkarni et al. (2009) found the cultivar Mammoth to be resistant and cultivars Shirley, Sangria, and Fireball to be moderately resistant to leaf spot. Nagrale et al. (2013) screened 21 varieties of *Gerbera jamesonii* and identified only a single cultivar, Tiramisu, as resistant to *Alternaria* blight.

2.2 Botrytis Blight (*Botrytis cinerea* Pers.:Fr.)

Geographic occurrence and impact. Botrytis blight has been reported in Australia, Bulgaria, China, Greece, Italy, Kenya, Korea, Mauritius, Portugal, and the United States (Alfieri et al. 1984; Anonymous 1960; Bobev 2009; Cho and Shin 2004; Dias and Lucas 1980; Holevas et al. 2000; Nattrass 1961; Preti 1937; Salinas et al. 1989; Shaw 1973; Shivas 1989; Zhang 2006).

Symptoms/signs. Botrytis blight can infect leaves, stems, petioles, and blossoms of *Gerbera* daisy. Blighting is the most common symptom of botrytis infection (Fig. 1), but it can also include leaf spots and damping-off of seedlings (Hausbeck and Moorman 1996). Small water-soaked spots on leaves, stems, or blossoms will coalesce rapidly, affecting large portions of these tissues. Infected blossoms may have prematurely senescing petals that will die and fall off, promoting further disease



Fig. 1 Blighting of *Gerbera* leaf due to *Botrytis* infection (Margery Daughtrey © 2017. All Rights Reserved.)

spread if these tissues fall onto and infect healthy leaves. Stem blight typically initiates in a broken or cut stem surface and progresses downward, leading to dieback of the entire stem and, in severe cases, extending into the base of the plant causing plant death (Hausbeck and Moorman 1996). Botrytis can also cause blights of cut *Gerbera* flowers during storage or transportation when temperature fluctuations result in high humidity and condensation on the flowers (Salinas et al. 1989).

Biology and epidemiology. *Botrytis cinerea* is generally dispersed by airborne conidia, although ascospores and mycelia can also initiate disease on plant tissue in the presence of free water such as condensation on leaves or flower petals (Kerssies et al. 1997). *B. cinerea* infection requires high relative humidity (>93%) and free water at the infection site within the canopy in order for infection to occur (Hausbeck and Moorman 1996). When relative humidity is low, conidia can survive as long as 14 months and still germinate on *Gerbera* flowers to cause lesions when water is made available (Salinas et al. 1989). Conidia are the most common means of propagating disease in the greenhouse. Dislodging conidia from germinating sclerotia, as occurs naturally during rainfall or irrigation, promotes further conidia production, thus perpetuating disease spread (Holz et al. 2007). Conidia invade healthy plant tissue via direct penetration of germ tubes into natural openings or through the cuticle (Van Kan et al. 1997). Temperature has little limiting effect on botrytis blight in *Gerbera* as petal spotting has been seen between 4 °C and 25 °C (39 °F and 77 °F), although higher incidence is associated with higher postharvest temperatures (Daughtrey and Benson 2005). *B. cinerea* is also a saprophyte on senescing and dead plant material (Holz et al. 2007).

Management

- ***Cultural practices*** – Reducing relative humidity can be very effective in managing botrytis blight outbreaks. Infrared heating systems or trickle, drip, trough, and flood and drain irrigation systems can assist in reducing the occurrence of free moisture (Hausbeck and Moorman 1996). Removing humid air from the greenhouse by venting and heating late in the day before sunset can also help to reduce relative humidity. Relative humidity can also be reduced within the plant canopy by increasing space between the plants and by arranging them in rows parallel to the air flow (Hausbeck and Moorman 1996). Reducing the plant canopy allows more light to penetrate which then reduces senescence of lower leaves and eliminates potential infection and sporulation sites (Hausbeck and Moorman 1996). Forcing heated air into the plant canopy can also flush humid air, reducing *B. cinerea* sporulation on senescent tissue and atmospheric conidial concentrations (Hausbeck et al. 1996).
- ***Sanitation*** – Soil solarization can be used to eliminate fungi, including *B. cinerea*, from soil and plant debris as a preplant treatment or following a disease outbreak (Hausbeck and Moorman 1996). Removing dead and senescing plant tissue reduces conidia and vegetative hyphae by limiting sites for pathogen colonization. In addition, infected plant debris should be disposed of immediately in covered containers to minimize inoculum sources and spread (Daughtrey and

Benson 2005). Plant debris should also be removed from benches and floors of growing and shipping areas as activity near these sources could cause the release of significant inoculum for adjacent newly established plants or cut flowers (Hausbeck and Moorman 1996).

- ***Fungicides*** – Fungicides can provide protection but are less effective than good sanitation and cultural practices at controlling botrytis blight outbreaks. Tank mixtures of two or more fungicides can provide longer and more effective protection than fungicides applied singly. Fungicide options are limited due to few modes of action or classes being available, but also because of resistance in *Botrytis* populations. Resistance has been reported to benomyl and other benzimidazole fungicides, dicarboximide, and diethofencarb. Fungicide classes that *Botrytis* has documented resistance to should be avoided as stand-alone treatments (Moorman and Lease 1992). In addition, low sensitivity has been detected to fenetrazole and fenethanil (Hausbeck and Moorman 1996). All chemical treatments should be used carefully in a way that limits selection pressure for resistance. Spray mixture additives may play a synergistic role with fungicides to provide more effective control of botrytis together than each treatment alone. Bourbos et al. (1994) found that the fungicides benzimidazole and dicarboximide gave less than 10% control and that highly refined horticultural oil provided 50% control when tested against a *Botrytis* strain that is resistant to both fungicide class. However, in combination, the oil plus either fungicide provided 88–100% protection. Caution must be taken when using horticultural oils for protection against botrytis blight as they are regulated as insecticides in the United States and must be registered for use on the crop to be treated. Fungicide application timing should maximize efficacy, such as immediately following flower harvest when new wounds will be created that could provide infection sites for the pathogen (Hausbeck and Moorman 1996). In greenhouse trials, applications of effective fungicides significantly reduced botrytis blight on *Geranium* compared to untreated plants, including treatments of chlorothalonil, fenhexamid, thiophanate-methyl, fludioxonil/cyprodinil, and pyraclostrobin/boscalid (Hausbeck et al. 2002; Hausbeck and Harlan 2010b; Webster and Hausbeck 2004). The biopesticide polyoxin D zinc salt has been shown to be highly effective in controlling botrytis in greenhouse crops (Hausbeck et al. 2002; Hausbeck and Harlan 2010b; Webster and Hausbeck 2004). Other biopesticides have shown varying degrees of efficacy; Webster and Hausbeck (2004) found that treatments of *Bacillus subtilis* significantly limited botrytis disease on *Geranium*.

2.3 Fusarium Wilt (*Fusarium oxysporum* f. sp. *chrysanthemi*, *F. oxysporum* f. sp. *tracheiphilum*)

Geographic occurrence and impact. *Fusarium* has been reported as causing disease in *Gerbera* in Africa, Australia, Brazil, Bulgaria, Greece, Italy, Korea, Malaysia, New Zealand, Poland, Spain, Thailand, and the United States (Alfieri et al. 1984;



Fig. 2 *Fusarium oxysporum* infection causing wilting and collapse of the *Gerbera* stem, leading to plant death (N. Selvaraj © 2017. All Rights Reserved.)

Bobev 2009; Cho and Shin 2004; French 1989; Garibaldi et al. 2004, 2007, 2008; Giatgong 1980; Holevas et al. 2000; Hopkins 1930; Kaewruang et al. 1987; Liu 1977; Mulenko et al. 2008; Pennycook 1989; Shivas 1989; Simmonds 1966).

Symptoms/signs. Diseased plants are initially stunted with chlorotic leaves. Disease symptoms often commence at the base of the stem and progress upwards, causing the leaves and flower heads to wilt, wither, and die (Fig. 2) (Pataky 1988). Brown streaks develop in the vascular system that eventually turn black. Brown discoloration also occurs in the vascular system of the crown and upper taproot. The leaves of affected plants can also occasionally turn red (Garibaldi et al. 2008; Troisi et al. 2010a).

Biology and epidemiology. *Fusarium oxysporum* Schlecht. is a ubiquitous soil-borne pathogen and the most common causal agent of Fusarium wilt disease. The *F. oxysporum* species complex comprises different formae speciales (f. sp.) that have specific host plant ranges (Dean et al. 2012). Specifically, Fusarium wilt of *Gerbera* is caused by *F. oxysporum* f. sp. *chrysanthemi* (Minuto et al. 2007) and *F. oxysporum* f. sp. *tracheiphilum* (Troisi et al. 2010a). A sexual cycle of *F. oxysporum* is unknown; therefore asexual spores and mycelia are responsible for plant disease (Dean et al. 2012). The pathogen can overwinter as mycelia and chlamydospores, either in dead plant parts or in the soil indefinitely in the absence of a host plant (Pataky 1988). Chlamydospores will germinate when stimulated by root exudates of host plants. The infectious hyphae will then penetrate the plant root system by directly growing into natural openings, such as wounds made when feeder roots emerge (Dean et al. 2012). Once it enters the plant, the pathogen will grow and multiply through its vascular system, spreading macro- and micro-conidia throughout the plant where they can germinate and affect new plant parts (Pataky 1988).

Management

- ***Cultural practices*** – Air and soil temperatures play an important role in disease and symptom development. Symptoms are usually absent or mild at temperatures below 21–24 °C (70–75 °F) and are most severe at constant temperatures of 29–32 °C (80–90 °F). Maintaining temperatures in these cooler ranges can minimize symptoms in infected plants and reduce disease spread (Pataky 1988). In addition, disease can be spread through infected transplants; therefore it is important to ensure transplants are disease-free before planting (Pataky 1988). For *Gerbera* plants grown in closed soilless systems, slow sand filtration, alone and in combination with the application of biocontrol agents, and a nutrient solution pH higher than 6.0 may significantly reduce *F. oxysporum* f. sp. *chrysanthemi* infections (Minuto et al. 2008).
- ***Sanitation*** – *F. oxysporum* can survive indefinitely in soil and soilless media (Palaky 1988); therefore clean media is essential to minimizing losses due to soilborne pathogens. Also, ensure soil is not carried from infested areas to clean areas by disinfesting structures, equipment, pots, etc. with an agricultural sanitizer (Pegg and Manners 2014). All infected plants must be removed and destroyed as they cannot recover from systemic infection (Pegg and Manners 2014). Soil solarization may not be an effective strategy for controlling *F. oxysporum* infestations (Kaewruang et al. 1989a, b). Irrigation water can also be an important source for the introduction of *Fusarium*. Water can be treated using chemical methods, heat (85 °C), UV irradiation, and slow sand filtration (Pegg and Manners 2014).
- ***Fungicides and biocontrols*** – Fungicides are not particularly effective against Fusarium wilt and should only be used prior to infection as protectants of healthy plant material. In research trials, azoxystrobin, which is upwardly systemic, has provided some protection against vascular diseases when applied as a drench and after diseased plants have been removed (Pegg and Manners 2014). Vermicompost has been shown to provide some reduction of disease incidence and spread when incorporated at 20% with or without chemical fertilizers (Rodríguez-Navarro et al. 2000). However, vermicompost should be used with caution as benefits are lost when it was incorporated at higher levels of 40% which produced higher disease incidence, lower yields, and smaller, chlorotic plants (Rodríguez-Navarro et al. 2000).
- ***Resistance*** – Minuto et al. (2006) screened 79 cultivars of *Gerbera* daisy and found that approximately 50–75% of the cultivars tested were resistant to highly resistant to *F. oxysporum* f. sp. *chrysanthemi* strains isolated from either *Gerbera* or *Chrysanthemum*.

2.4 Fusarium Root and Stem Rot, *Nectria* Blight (*Fusarium solani*, *Nectria haematococca*)

Geographic occurrence and impact. Fusarium root and stem rot has been reported on *Gerbera* in Australia, China, Japan, and North and South America (Deng and



Fig. 3 Blackening of the stem and crown of *Gerbera* affected with Fusarium root and stem rot caused by *Fusarium solani* (Penn State Department of Plant Pathology and Environmental Microbiology © 2017. All Rights Reserved.)

Harbaught 2006; Kaewruang et al. 1989a; Li et al. 2008; Rodríguez-Navarro et al. 2000; Takahashi et al. 1999). When isolated outbreaks occur, the disease can be devastating leading to major losses.

Symptoms/signs. Fusarium stem and root rot can resemble Fusarium wilt, but differs in the degree of root rot and is more frequently associated with blackening of the roots, crown, and stems (Fig. 3). In addition, when homothallic strains of the fungus are present, perithecia of *Nectria haematococca* can be observed sporulating on the base of the stems.

Biology and epidemiology. The pathogen's biology and epidemiology is very similar to that of *Fusarium oxysporum* with the one exception that ascospores can become another source of inoculum in the greenhouse.

Management. Management of Fusarium root and stem root is the same as for *Fusarium oxysporum*.

2.5 Phytophthora Crown Rot (*Phytophthora cryptogea; P. drechsleri; P. palmivora*)

Geographic occurrence and impact. Phytophthora disease has been reported globally. Specifically, *P. cryptogea* has been reported in Australia, Asia (China, Korea), North America (Canada, United States), Middle East (Iran), as well as in many countries throughout Europe – Bulgaria, France, Germany, Greece, the Netherlands, Poland, and Russia (Alfieri et al. 1984; Anonymous 1960; Blair et al. 2008; Bobev 2009; Cho and Shin 2004; Coelho et al. 1997; Cook and Dubé 1989; Erwin and Ribeiro 1996; French 1989; Grand 1985; Ho 2003; Holevas et al. 2000;



Fig. 4 Symptoms of Phytophthora crown rot: wilting and necrosis of leaves, darkening of leaf petioles due to infection by *Phytophthora drechsleri* (M.K. Hausbeck © 2017. All Rights Reserved.)

Hwang and Benson 2005; Joshi and Elmhirst 1998; Martin et al. 2014; Mostowfizadeh-Ghalamfarsa et al. 2010; Mulenko et al. 2008; Olson and Benson 2011; Pennycook 1989; Pirone 1939; Sarbhoy et al. 1971; Simmonds 1966; Tompkins and Tucker 1937; Vanev et al. 1993). *P. drechsleri* has been reported on *Gerbera* daisy in Australia, Mexico, Morocco, and the United States (Blair et al. 2008; Coelho et al. 1997; Erwin and Ribeiro 1996; French 1989; Martin et al. 2014; Olson and Benson 2011; Romero Cova and Solis Aragon 1996; Sampson and Walker 1982). *P. palmivora* has also been reported as causing disease in India (Erwin and Ribeiro 1996; Sarbhoy et al. 1971).

Symptoms/signs. *Phytophthora* spp. can cause devastating rot disease of both the crown and root of *Gerbera* daisy. Plants begin to wilt rapidly within 7–10 days of initial infection. Leaf symptoms include chlorosis and darkened midrib and veins followed by loss of turgor and desiccation (Benson and Parker 2011) (Fig. 4). Symptomatic plants will usually have significant root and crown discoloration and deterioration (Barnes 2000). *Gerbera* are susceptible at all stages of production, but infection appears more common after flowering (Arnold et al. 2000).

Biology and epidemiology. *Phytophthora* spp. can infect a wide range of hosts. Disease is associated most commonly with the release of biflagellate zoospores. Zoospore release requires free water for motility and infection. Therefore, disease spread is associated with lower temperatures that can enable longer periods of persistence of water on leaves and stems (Bush 2002). *P. cryptogea* can also survive in soil for long periods via resistant chlamydospore structures (Bumbieris 1979).

Management

- ***Cultural practices*** – Because *Phytophthora* spp. rely heavily on water for spore dispersal and infection, water management is crucial in controlling this disease. Overwatering can provide prolonged periods of free water for zoospore dispersal. Also, excessive irrigation as well as excessive drought can cause plant stress which can weaken plant roots making them more susceptible to infection (Barnes 2000). Transplants should not be planted with the crown below the media surface and, if possible, do not grow plants on the ground (Arnold et al. 2000). Soil compaction can significantly reduce drainage, thereby promoting disease spread (Barnes 2000). Also, lowering watering frequency and raising electrical conductivity of the nutrient solution have been found to inhibit *P. cryptogea* infection in *Gerbera* potted plants grown in ebb and flow systems with recirculation of the nutrient solution (Thinggaard and Andersen 1995).
- ***Sanitation*** – Sanitation is essential in all disease management programs. Removing diseased plants is the first step to limiting disease spread. Soil solarization has also been found to be effective in reducing *P. cryptogea* in *Gerbera* (Kaewruang et al. 1989a, b).
- ***Fungicides and biocontrols*** – Benson and Parker (2008) found fenamidone, mandipropamid, and fluopicolide to be effective controls of *P. cryptogea* on African daisy. Mefenoxam may provide some control but efficacy may vary (Benson and Parker 2008, 2009). Numerous fungicide drenches, including azoxystrobin, etridiazole, mandipropamid, fluopicolide, dimethomorph, dipotassium phosphonate/dipotassium phosphate, fenamidone, and mefenoxam, were found to completely prevent plant death caused by Phytophthora root rot (Hausbeck and Glaspie 2009a). Resistance to mefenoxam has been reported in *P. cryptogea* and *P. drechsleri* isolated from other floriculture crops and in *P. drechsleri* isolated from *Gerbera* daisy (Hwang and Benson 2005; Olson and Benson 2011). Fluopicolide, ametoctradin, fenamidone, and cyazofamid fungicide drenches were all found to effectively control mefenoxam-resistant *P. drechsleri* isolates in *Gerbera* (Benson and Parker 2010a). Alternating fungicides with different Fungicide Resistance Action Committee (FRAC) codes is recommended to avoid fungicide resistance developing in *Phytophthora* populations (Benson and Parker 2011). Grasso et al. (2003) reported that inoculation of the rhizosphere with some microbial strains may reduce incidence of root rot associated with *P. cryptogea* due to a protective microflora that prevents pathogen presence. However, Benson and Parker (2010b, 2011) found the biological did not prevent disease development and reported mortality of almost 100% for *Gerbera* daisy treated with drenches of the biologicals.
- ***Resistance*** – Yue et al. (2008) screened 13 *Gerbera jamesonii* cultivars to identify their resistance to root rot caused by zoospores of *P. cryptogea*. They found "Linglong" had the highest level of root rot resistance. "Nianhua," "Jintaiyang," "Liangfen," "Hongri," "Hanwang," and "Lvxinfen" had higher levels of resistance than "Dacheng," "Aishen," "Hongditan," "Dxueju," and "Redaicaoyuan." "Baimawangzi" had the lowest level of resistance to *P. cryptogea*.

2.6 Powdery Mildew (*Golovinomyces* (Syn. *Erysiphe*) *cichoracearum* DC.; *Podosphaera* sp.; *Oidium* sp.)

Geographic occurrence and impact. Powdery mildew (PM) caused by *Golovinomyces* (*Erysiphe*) *cichoracearum* has been reported in Argentina, Brazil, Finland, Israel, Italy, New Zealand, South Africa, Switzerland, and the United States (Alfieri et al. 1984; Amano 1986; Anonymous 1960; Boesewinkel 1979; Bolay 2005; Braun 1995; Crous et al. 2000; Delhey et al. 2003; French 1989; Grand 1985; Greuter et al. 1991; Mendes et al. 1998; Pennycook 1989; Preston 1945; Raabe 1966; Troisi et al. 2010b; Voytyuk et al. 2006, 2009). *Podosphaera* sp. has been reported as the causal agent of PM on *Gerbera* daisy in Australia, Brazil, China, India, Japan, Korea, and Romania (Baiswar et al. 2010; Cunnington 2003; Ito and Takamatsu 2010). Reports of PM caused by *Oidium* sp. have been made in Africa, Australia, Barbados, Bulgaria, Egypt, England, Greece, Jamaica, Kenya, New Zealand, Portugal, South Africa, Southern Africa, Tanzania, United States (Hawaii), and Zimbabwe (Amano 1986; Bobev 2009; Cook and Dubé 1989; Doidge 1950; Gorter 1977; Holevas et al. 2000; Hopkins 1932, 1939; Norse 1974; Raabe et al. 1981; Riley 1960; Shivas 1989; Simmonds 1966; Wallace and Wallace 1947; Whiteside 1966).

Symptoms/signs. The fungus forms a network of hyphae over the plant surface, and the leaves, flowers, and stems eventually become covered with white, talcum-like colonies (Granke et al. 2012) (Fig. 5). The disease may cause the lower leaves to drop, and white powdery growth may be visible on both stems and flowers, thus making plants or cut flowers unsightly and unmarketable (Daughtrey et al. 1995) (Fig. 5). PM is the most common foliar disease of *Gerbera* daisy and is considered the most destructive disease of commercial *Gerbera* production (Granke et al. 2012; Song and Deng 2013).

Biology and epidemiology. Environmental conditions most conducive for PM development include high relative humidity (80–90%) and moderate temperatures (20–28 °C/68–82 °F) (Daughtrey et al. 1995). In addition, cool night and warm day temperatures, closed canopy, and poor air circulation will promote disease development (Mueller et al. 2003). Initial infections are the result of airborne asexual spores (conidia) that land on the plant surface and germinate. Conidia will develop under conducive environmental conditions, and secondary spread can be the result of further movement of conidia through air currents and water splash (Granke et al. 2012).

Management

- ***Cultural practices*** – Reducing relative humidity in the greenhouse can prevent sporulation of PM pathogens. This can be accomplished by ensuring adequate ventilation and using wide plant spacing so that air can flow easily between plants (Granke et al. 2012). Overlapping leaves should also be removed (Mueller et al. 2003).



Fig. 5 Powdery mildew signs: white talcum-like colonies of conidia on leaves (*upper left* and *right*) and flowers (*lower left*) render the plants unmarketable (M.K. Hausbeck © 2017. All Rights Reserved.)

- ***Sanitation*** – Acidic electrolyzed oxidizing (EO) water has been shown to effectively reduce the percentage of PM when sprayed twice a week and when sprayed every other week, alternating with fungicides (Mueller et al. 2003). EO water is highly bactericidal and fungicidal because of its unique properties such as high oxidation–reduction potential, low pH, and the presence of hypochlorous acid (Mueller et al. 2003). EO water may need to be applied prior to or shortly after the onset of the disease in order to successfully control PM outbreaks. Multiple applications per week may also provide sufficient management; however, phytotoxicity has been seen when plants were sprayed with EO water more than once per week (Mueller et al. 2003).
- ***Fungicides*** – Frequent scouting and fungicide applications are the recommended methods of managing PM in *Gerbera* daisy (Granke et al. 2012). Fungicides have been found to be effective when used as either preventive or curative applications. Hausbeck and Glaspie (2009b) evaluated preventive PM applications in a greenhouse experiment and found myclobutanil, difenoconazole, triflumizole, and thiophanate-methyl/mancozeb to completely prevent PM infection. In a second greenhouse trial, Hausbeck and Glaspie (2009c) evaluated both experimental and

registered fungicides and found that all preventive treatments significantly limited foliar infection compared to the untreated control. Disease pressure in this trial was significant with nearly 47% of the leaf area colonized by PM on the untreated plants. Hausbeck et al. (2006a) had previously found myclobutanil and triflumizole to be highly effective in controlling PM as either preventive or curative treatments. When determining when to spray, curative fungicide applications, visual scouting for disease is more effective than spore sampling as a trigger to initiate treatment (Granke et al. 2012). Hausbeck et al. (2006b) also looked at application timing for control of PM and found that neither myclobutanil nor boscalid completely eliminated PM infection; however, all treatments were statistically better than the untreated control, and there was no difference when applications were repeated every 7, 14, or 21 days. More recently, pyraclostrobin/boscalid has been found to be as effective as myclobutanil and triflumizole (Hausbeck and Harlan 2010a).

- ***Biorational fungicides*** – Biorational fungicides have been evaluated for control of PM caused by *P. fusca* on *Gerbera* daisy. "Biorational" is used to describe products that are proported to be environmentally friendly in comparison to conventional products but are different from biofungicides as they do not contain living organisms (Elmer 2008). Acibenzolar-S-methyl, potassium silicate/polyoxyethylene, mono- and dipotassium salts of phosphorous acid, potassium bicarbonate, sodium tetraborohydrate decahydrate, and *Bacillus subtilis* all suppressed PM of *Gerbera* daisy compared with untreated controls under greenhouse conditions in Florida; however, no biorational fungicides were as effective as an azoxystrobin/myclobutanil fungicide program (Moyer and Peres 2008). Of the treatments evaluated in this trial, mono- and dipotassium salts of phosphorous acid, potassium bicarbonate, and sodium tetraborohydrate decahydrate were the most effective in suppressing disease, especially the former two products when treating the most susceptible cultivar, Snow White. Villavicencio (2010) has also reported success in preventing PM caused by *Oidium* sp. using a plant extract of *Reynoutria sachalinensis* in rotation or tank mixed with industry standard fungicides. Plant oils have also been effective in controlling PM caused by *Golovinomyces* (Syn. *Erysiphe*) *cichoracearum*: plant (rosemary, thyme, clove) oil-based fungicides were found to reduce PM development as effectively as triadimefon (Buck and Youmans 2014).
- ***Resistance*** – Significant progress has been made in breeding *Gerbera* daisies for resistance to PM. Initial studies reported resistance to *P. xanthii* in *Gerbera* as being controlled by a single dominant gene *Pmr1*, whose expression varied with genetic background and gene dosage, therefore suggesting the involvement of additional unknown genes in PM resistance (Kloos et al. 2005). However, Song and Deng (2013) reported resistance to PM as being controlled by two linked quantitative trait loci (QTL) that are involved in restricting fungal growth and sporulation. Two of the resistant genotypes identified in this study, UFGE 31–19 and its progeny UFGE 4033, have been used to successfully introduce this resistance into new *Gerbera* cultivars with high levels of PM resistance (Song and Deng 2013). These cultivars are UFGE 4141, UFGE 7014, UFGE 7015, UFGE 7023, UFGE 7032, and UFGE 7034, released in 2010, and UFGE 7031 and UFGE 7080, released in 2013 (Deng and Harbaugh 2010, 2013). UFGE 7031 and UFGE 7080 are available under the

names "Funtastic™ Tangerine Eye" and "Funtastic™ Golden Eye," respectively (Deng and Harbaugh 2013). Cultivar screening studies have identified additional resistant material including "Festival Light Eye Pink" and "Festival Semi Double Orange" (Sconyers and Hausbeck 2005). "Terrafame" has also been noted to be resistant to PM (Hausbeck et al. 2003).

2.7 Pythium Root Rot (*Pythium* spp.)

Geographic occurrence and impact. *Pythium* has been reported as causing disease in *Gerbera* daisy in Greece, Japan, Korea, Malaysia, and the United States (Alfieri et al. 1984; Cho and Shin 2004; Holevas et al. 2000; Liu 1977; Raabe et al. 1981; Suzuki et al. 2009).

Symptoms/signs. *Pythium* spp. are responsible for causing root and stem rots in mature plants as well as damping-off in seedlings. Seeds and seedlings are more susceptible than mature plants with reduced vigor and subsequent limited growth and productivity (Del Castillo Munera and Hausbeck 2016). *Pythium* attacks the root systems of the plant causing the above-ground tissue to wilt and die as the roots rot. Infected roots will turn various shades of brown to yellow, depending on the species of the pathogen, generally initiating at the root tips and progressing to the rest of the root system (Sutton et al. 2006).

Biology and epidemiology. Depending on the species of the pathogen, Pythium root rot can be spread by either zoospores, oospores, or mycelia (Moorman et al. 2002). Oospores of all *Pythium* spp. can be found in soil and other planting media and can survive in field soil for months to years (Martin and Loper 1999). Thick-walled chlamydospores can also form under adverse conditions as survival mechanisms, but only in a few species (e.g., *P. tracheiphilum* and *P. dimorphum*) (Schroeder et al. 2013). Inoculum can be introduced by dust particles, soil and planting media, residue on tools and equipment, footwear, and irrigation water (Sutton et al. 2006). *Pythium* oospores have also been detected in the digestive tracts and frass of fungus gnats (*Bradysia* spp.) and shore flies (*Scatella stagnalis* Fallen), demonstrating that *Pythium* can also be spread by insect vectors (Sutton et al. 2006). Oospores and sporangia of *Pythium* spp. do not germinate in the soil unless exposed to chemical stimulants present in root and seed exudates, plant debris, or organic matter (Martin and Loper 1999). In high moisture conditions, some *Pythium* spp. sporangia will produce motile zoospores instead of germinating directly (Martin and Loper 1999). Zoospores are particularly important for disease spread in hydroponic or ebb and flow systems (Sutton et al. 2006). Infection by zoospores is caused by direct penetration of unwounded root surfaces by penetration pegs, fine hyphae, and enzymatic action, although wounded tissues are also highly susceptible to *Pythium* spp. invasion (Sutton et al. 2006). Pythium root rot is highly correlated with high soil moisture and can be more severe under lower soil temperatures (Martin and Loper 1999).

Management

- ***Cultural practices*** – Pythium root rot requires high levels of free water for infection and spore dispersal; therefore, water management is crucial in controlling this disease. Overwatering can provide prolonged periods of free water for zoospore dispersal and is associated with increased disease severity (Martin and Loper 1999). Also, excessive irrigation as well as excessive drought can cause plant stress which can weaken plant roots making them more susceptible to infection (Barnes 2000). Transplants should be disease-free and not planted with the crown below the media surface and, if possible, do not grow plants on the ground (Arnold et al. 2000). Soil compaction can significantly reduce drainage, thereby promoting disease spread (Barnes 2000). The use of bottom heat and through-the-bench air movement can also reduce the incidence of *Pythium*-related diseases (Gullino and Garibaldi 2007). Composted pinebark-based mixes can suppress *Pythium* and other soilborne disease pathogens (Daughtrey and Benson 2005). Mounding plants may also limit disease by providing better drainage and warmer soil temperatures (Gullino and Garibaldi 2007). Recycling irrigation water should be avoided (Daughtrey and Benson 2005).
- ***Sanitation*** – Minimizing spread of contaminated material is essential for controlling Pythium root rot. Sanitizing all tools and materials can reduce spore quantities and spread. Chemical sterilants, including potassium monopersulfate and didecyldimethylammonium chloride, have been found to be only partially effective in destroying *Pythium* spp. in small-scale hydroponic systems (Sutton et al. 2006). Sodium hypochlorite was completely effective but can be corrosive.
- ***Fungicides and biocontrols*** – Limited fungicide options are available for treatment of *Pythium* root rot. Mefenoxam-resistant populations have been detected on *Gerbera* and other crops, and therefore this fungicide is no longer considered effective or recommended for treatment of *Pythium* root rot (Del Castillo Munera and Hausbeck 2016; Lookabaugh et al. 2015; Moorman et al. 2002). Treating resistant strains with mefenoxam may even promote disease, as seen in *Geranium* with higher levels of seedling damping-off with mefenoxam treatment (Lookabaugh et al. 2015). Other control options may include biocontrols. Jamwal and Jamwal (2012) investigated the use of *Trichoderma* spp. to manage root rots in *Gerbera* and found that *T. viride* effectively inhibited the radial growth of *P. irregulare*.

3 Bacterial Diseases

3.1 Bacterial Leaf Spot (*Pseudomonas cichorii*)

Geographical occurrence and impact. Bacterial leaf spot, caused by *Pseudomonas cichorii*, has been reported on *Gerbera* in Australia, Brazil, Greece, Japan, Lithuania, the Netherlands, and the United States (Alivizatos 1986; Anonymous 2015b; Janse 1987; Kawarabayashi and Suyama 1990; Malavolta Júnior et al. 1994; Miller and Knauss 1973; Snieškienė 1995).

Symptoms/signs. *P. cichorii* infection can cause a number of symptoms in *Gerbera* daisy. Small to large circular spots appear initially but will become irregular in shape and dark brown to black in color as the disease progresses (Reddy 2016). These spots may or may not also be associated with concentric ring patterns. Brown discoloration has also been noted along the main vein (Miller and Knauss 1973; Reddy 2016). In addition, large brown areas that extend from leaf margins and narrow as they reach the midvein have been noted (Miller and Knauss 1974). Disease symptoms can be variable in seedlings (Miller and Knauss 1973).

Biology and epidemiology. *P. cichorii* is a gram-negative bacterium that can infect and cause devastating symptoms on a wide range of host plants (Janse 1987). In addition to *Gerbera*, ornamental plant hosts can include *Hibiscus*, *Impatiens walleriana*, *Cyclamen persicum*, primrose, *Vinca*, and *Chrysanthemum* (Dicklow 2013; Janse 1987). Pathogen growth is favored by high temperatures and moist conditions (Janse 1987; Dicklow 2013). However, *P. cichorii* has been found to survive below freezing temperature for up to 12 months (Janse 1987). The bacterium can also overwinter in plant debris, as noted by Bazzi et al. (1984) who found surviving *P. cichorii* cells in lettuce debris after 115 days. The bacteria are easily spread by splashing water and require wounds or natural entry points in order to infect plant hosts (Dicklow 2013).

Management

- ***Cultural practices*** – Bacterial leaf spot infection and spread can be minimized by maintaining low relative humidity within the crop. This can be accomplished by using water management practices that keep foliage dry such as avoiding overhead watering (Reddy 2016; Miller and Knauss 1974). In addition, eliminating the source of future inoculum by removing diseased plants and residue can reduce disease spread (Miller and Knauss 1974). Propagation beds and media should also be pasteurized between crops (Moorman 2016).
- ***Chemical control*** – Antibiotic sprays such as streptocycline at 0.2 g/l or kasugamycin at 1.25 ml/l may provide some control of disease spread (Reddy 2016).

4 Viral Diseases

4.1 Bromoviridae

Geographic occurrence and impact. Only one virus of significance in the Bromoviridae is reported to cause disease in *Gerbera*: *Cucumber mosaic virus* (CMV, genus *Cucumovirus*) has been reported to cause disease symptoms in *Gerbera* daisy production in Australia and India (Finlay 1975; Verma et al. 2004).

Symptoms/signs. CMV symptoms vary depending on the plant host, environment, and other conditions. However, severe chlorotic mosaic patterns as well as floret and

flower deformations have been reported in CMV-infected *Gerbera* plants (Gautum et al. 2014). Color breaking has also been noted (Verma et al. 2004).

Biology and epidemiology. Viruses in the Bromoviridae family are transmitted by aphid vectors (Hemiptera: Aphididae). CMV is transmitted non-circulatively (non-persistently) primarily by green peach aphids (*Myzus persicae*) and cotton aphids (*Aphis gossypii*) (Verma et al. 2004). When the insect feeds on CMV-infected plants, the virus will attach to its mouthparts and then be spread when the insect moves to feed on neighboring uninfected plants. Once the plant has been inoculated by the feeding aphid, CMV, like other plant viruses, uses the phloem-assimilate pathway to systemically invade the rest of the plant (Jacquemond 2012). Seed transmission of CMV is unknown in *Gerbera*.

Management. Viral infections cannot be eradicated using chemical or cultural control measure; therefore it is imperative to use virus-free stock plants in *Gerbera* production systems. If viruses are suspected, diseased plants should be destroyed immediately. Insect vector populations should be monitored and controlled as needed. Insect populations can be tested for the presence of many viruses, including CMV. Refer to ▶ Chap. 4, "Insect Management for Disease Control in Florists' Crops" for additional virus management information.

4.2 Bunyaviridae

Geographic occurrence and impact. Bunyaviridae includes a number of viruses that cause disease in ornamental plants. Within this classification are the tospoviruses, two of which are important in *Gerbera* production: *Tomato spotted wilt virus* (TSWV) and *Impatiens necrotic spot virus* (INSV). TSWV is considered one of the most important plant viruses and has been linked to over 1 billion US dollars in crop losses globally (Mavrič and Ravnikar 2001). TSWV has been found to be the cause of up to 30% loss in *Gerbera* greenhouse crops in Serbia and Venezuela (Stankovic et al. 2011; Marys et al. 2014). TSWV has also been reported in Greece, Poland, Slovenia, and the Netherlands (Chatzivassiliou et al. 2000; Kaminska and Korbin 1994; Mavrič and Ravnikar 2001; Verhoeven and Roenhorst 1994). INSV has been reported in Hungary, New Zealand, and the United States (Elliott et al. 2009; Toth et al. 2007; Whipker 2014).

Symptoms. Viral infection symptoms can vary significantly depending on the host. Symptoms of TSWV infection in *Gerbera* initially appear as chlorotic oak-leaf patterns on leaves which are followed by necrosis and leaf distortion as the disease progresses (Stanovic et al. 2011). Flowers can also be distorted and discolored (Kaminska and Korbin 1994). INSV symptoms typically begin as a light green mottling with ring spots also possibly present. As the disease progresses, a distinct necrotic line pattern becomes evident on leaves (Whipker 2014).

Biology and epidemiology. Tospoviruses, like other members of the family Bunyaviridae, require an insect vector to transmit the disease. Most are vectored by *Thrips* species (Thysanoptera: Thripidae), a common polyphagous insect pest of most horticulture crops worldwide (Kazinczi et al. 2007; Moyer et al. 1999). TSWV and INSV are both transmitted primarily by the western flower thrips (*Frankliniella occidentalis*), although other *Thrips* species have been reported (German et al. 1992; Kaminska and Korbin 1994; Whipker 2014). These viruses are persistent, meaning that they survive and replicate within the gut of their vector host (Kazinczi et al. 2007). However, thrips can only acquire tospoviruses during the immature larval stage where it is transstadially transmitted to progressive life stages, but there is no evidence of transovarial transmission from one generation to the next (Kazinczi et al. 2007; Moyer et al. 1999). Seed transmission of tospoviruses is currently unknown, but the disease can be spread easily through infected propagation material (Kazinczi et al. 2007; Moyer et al. 1999).

Management

- ***Cultural practices*** – Prevention is key to managing *Tospovirus* diseases because *Tospovirus*-infected plants cannot be cured. If viral infection is suspected in *Gerbera* plants, samples should be sent to testing facilities to confirm the presence of the virus. Once the disease has been identified, the only management option is to discard infected plants (Whipker 2014). However, managing the vector of these viruses, the western flower thrips, can minimize spread. This can be done using strategies to physically exclude the pests such as installing fine mesh screens (mesh size <135 nm) on external openings to prevent entry of *Thrips* vectors into the greenhouse. Monitoring using indicator plants, such as petunia, or sticky cards can be helpful to provide early warnings of the presence of *F. occidentalis* (Allen and Matteoni 1991).
- ***Sanitation*** – Remove all plant debris as well as weeds and flowering plants growing near production areas as these can be sources of new infections and infestations (Kazinczi et al. 2007). Soil sterilization can also eliminate developmental stages of vector species. Refer to ▶ Chap. 4, "Insect Management for Disease Control in Florists' Crops" for additional virus management information.
- ***Biological controls*** – Biological controls can effectively control *Thrips* species when populations are low. The following species have been identified as predators of western flower thrips: *Euseius stipulatus*, *Metaseiulus occidentalis* (Nesbitt), *Amblyseius andersoni* (Chant), *Amblyseius barkeri* (Hughes) (*A. mckenziei*), *Amblyseius californicus* McGregor, *Amblyseius* (*Neoseiulus*) *cucumeris* (Oudemans), *Amblyseius* (*Iphiseius*) *degenerans* Berlese, *Amblyseius* (*Euseius*) *hibisci* (Chant), *Amblyseius limonicus* s.s. Garmon and McGregor, *Amblyseius scutalis* (Athias-Henriot), and *Amblyseius* (*Euseius*) *tularensis* (Congdon) (Hoddle 2015).
- ***Resistance*** – Resistance to tospoviruses is unknown in commercial *Gerbera* cultivars. However, Korbin et al. (2002) successfully introduced resistance to TSWV using *Agrobacterium*-mediated transformation. They integrated a

resistance-related nucleoprotein gene from the virus into *Gerbera* explants and found that these transgenic plants did demonstrate resistance when mechanically inoculated.

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Diseases of *Gypsophila*

19

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Abstract

G. paniculata L ("baby's breath") is a worldwide crop mainly used as a filler cut flower. It is commercially propagated vegetatively to preserve the ornamental characteristics of the plant. Among fungal diseases caused by soilborne pathogens, *Phytophthora* species and *Rhizoctonia solani*, due to their irreversible rotting of plants, are the most devastating *Gypsophila* disease worldwide. *Pythium* species and *R. solani* are especially destructive in the process of rooting cuttings. In adult plants, *Fusarium* species produce rotting of basal tissues resulting in death.

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R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_19

Powdery mildew caused by *Erysiphe buhrii* is important on aerial parts, producing in some countries nearly 100% of disease incidence at harvest. Crown gall has been reported in all continents, and few viruses and nematodes are cited.

Keywords

Gypsophila paniculata • *Phytophthora nicotianae* = *P. parasitica* • *Rhizoctonia solani* • Powdery mildew • *Erysiphe buhrii* • *Pythium* root rot • *Fusarium* stem rot • *Meloidogyne* spp.

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1 Introduction

Gypsophila genus belongs to the Caryophyllaceae family. The genus contains about 125 species of annual, biennial, and perennial plans that originated in Turkey, Ukraine, and Iran. *Gypsophila elegans* M. Bieb. (annual) and *Gypsophila paniculata* L. (perennial), both named "baby's breath," are worldwide crops mainly used as a filler cut flower. *G. paniculata* is the most widely used commercial species. It is very popular because of its airy and cloud-like appearance produced by the numerous single or double flowers, which develop in large inflorescences, which are diffusely branched, leafy-bracted, in panicle-like clusters. *G. paniculata* is commercially propagated vegetatively to preserve the ornamental characteristics of the plant. The original plant material comes from in vitro meristems, stem-segment explants, and leaf segment explants so that healthy material is obtained, free of bacteria and fungi. Cuttings are taken from the selected original plant material – the nuclear stock – and constitute the propagation stock, or the mother plants, which are grown in special aseptic conditions, using anti-insect meshes or bench beds, to obtain disease-free mother plants through vegetative propagation. These plants are also cultivated in aseptic conditions to obtain the herbaceous cuttings which are sold to the flower producers.

2 Fungal and Fungus-Like Diseases

2.1 Alternaria Leaf Spot [*Alternaria alternata* (Fr./Fr.) Keissl., *Alternaria gypsophilae* Neerg., *Alternaria juxtiseptata* E.G. Simmons, and *Alternaria subelliptica* E.G. Simmons]

Geographic occurrence and impact. Different species of *Alternaria* are reported on *Gypsophila* species: *A. gypsophilae* on *G. elegans* in Armenia (Simonyan 1981), *A. juxtiseptata* and *A. subelliptica* in Australia (Simmons 2007; Woudenberg et al. 2013), and *A. alternata* in Bulgaria and Israel (Margina et al. 1999; Elad et al. 2014) on *G. paniculata.*

Symptoms/signs. Symptoms on lower and middle leaves consist of small circular spots, with light brown centers and reddish purple margins. Development of numerous spots (which enlarge) and especially damage to the midrib leads to withering of leaflets (Margina et al. 1999).

Biology and epidemiology. Refer to the information on *Alternaria* spp. in ► Chap. 13, "Diseases of Carnation" (Sect. 2.2).

Management. Refer to ▶ Chap. 13, "Diseases of Carnation", Sect. 2.2 for information on management of *Alternaria.*

2.2 Botrytis Gray Mold [*Botrytis cinerea* Pers.:Fr., Teleomorph: *Botryotinia fuckeliana* (de Bary) Whetzel]

Geographic occurrence and impact. The pathogen has been reported worldwide on this host (Cho and Shin 2004; Engelhard et al. 1982; Garcés de Granada 1992).

Symptoms/signs. Little brown spots are observed on the leaves, stems, shoots, and flowers. Initially wet tan-colored blotches develop on petal tips which spread rapidly to produce a fluffy gray mold formed by fungal conidia and conidiophores (sign). Spots can coalesce, and plant tissues may blight. Injury can be produced at preharvest and postharvest stages (Engelhard et al. 1982; Fillinger and Elad 2016).

Biology and epidemiology. Senescent plant tissues, soil, and plant debris are natural reservoirs for the pathogen. The fungus produces a large number of conidia that are dispersed by airflows from affected crops, weeds, plant debris, and out- or indoor greenhouses. Under conditions of cool temperatures, high relative humidity (above 85%), with little or no air circulation or free water on the leaf surface, the spores land on the plant surface and germinate and penetrate the host through wounds.

Cutting stubs are particularly susceptible to gray mold infections. Symptoms will appear on infected plants within a few days. The fungus can also be a postharvest problem at temperatures of 0–10 °C.

Mangement. Refer to ▶ Chap. 13, "Diseases of Carnation", Sect. 2.4 for information on management of *Botrytis*.

2.3 Fusarium Stem Rot, Basal Rot, Crown Rot [(*Fusarium graminearum* Schwabe, *Fusarium culmorum* (W.G. Smith) Saccardo, *Fusarium proliferatum* Matsushima) Nirenberg, *Fusarium verticillioides* (Sacc.) Nirenberg = *Fusarium moniliforme* Sheld.]

Geographic occurrence and impact. Fusarium rots of *Gypsophila*, caused by several *Fusarium* species, have been cited in Argentina, Israel, Korea, and Poland (Elad et al. 2014; Lee et al. 2011; Orlikowski et al. 1991; Wolcan et al. 2007). Reported incidence of *Gypsophila* rots in Argentina and Korea ranged from 10% to 30% in affected greenhouses (Lee et al. 2011; Wolcan et al. 2007). In Argentina, stem rot was detected in the 1990s, and *F. graminearum* was identified as the causal agent (Wolcan et al. 2007). In South Korea, severe wilt symptoms were observed on cv. Bristol Fairy in greenhouses as a consequence of crown rot caused by *F. proliferatum* (Lee et al. 2011).



Fig. 1 Fusarium stem rot of *G. paniculata* caused by *Fusarium graminearum* (G.A. Lori © 2017. All Rights Reserved.)

Symptoms/signs. The symptoms and signs resemble those of Fusarium stem rot of carnation described by Nelson et al. (1975). Early symptoms include discoloration around the stem base and slight wilting. Late symptoms include a sunken crown rot, root rot, severe wilting, and dying plants (Fig. 1).

Fusarium proliferatum invades plants through the stem base, causing a crown rot that prevents the plant from taking up water and nutrients (Lee et al. 2011). Stem and crown rot occur on young and mature plants at all stages of growth. Leaves wilt and turn light pale, and in severe attacks the entire plant may collapse and die.

In the case of *F. graminearum* infection, internal tissues of the affected branches appear necrotic and blackish brown in color. Shoots appear stumpy and rot, and the wilted leaves remain attached to the stem. As a consequence of infection, the number of productive branches is reduced, and the profitable life cycle of the crop is shortened (Wolcan et al. 2007).

Biology and epidemiology. The disease is favored by high temperatures, moist conditions, and poor soil drainage. Outbreaks of stem rot and basal rot can occur during the spring-summer months when the greenhouse environmental is hard to control.

Fusarium graminearum, the main causal agent of Fusarium rots, has been found overwintering in crop and weed debris. The first 10 cm of the affected stem turns

whitish and is covered by pale orange sporodochia and, upon plant death, small black perithecia of the teleomorph *Gibberella zeae* Schwein (Petch) are formed (Wolcan et al. 2007). During spring and summer, the ascospores are discharged from the perithecia and easily spread by air currents and water splash in greenhouses. *F. graminearum* enters the plant through the wounds left by the harvest and the pinching of branches. From there, the lesion spreads toward the crown affecting the shoots produced from the basal buds. *F. graminearum* rarely affects the roots of *Gypsophila* (Wolcan et al. 2007).

Management

- ***Sanitation*** – Improve drainage by planting on raised beds. Do not moisture-stress plants, but do not overwater. Management measures aim at reducing the inoculum sources by removing the dead or senescent plants from inside greenhouses and controlling weeds and other plant hosts outside. It is also necessary to eliminate the substrates for the development of *F. graminearum* and *G. zeae* spores. Refer to ► Chap. 13, "Diseases of Carnation", Sect. 2.7 for more management information.

2.4 Powdery Mildew [*Erysiphe buhrii* U. Braun (Formerly *Erysiphe communis* Grev. f. *gypsophilae* Jacz l.c.), Anamorph: *Oidium dianthi* Jacz.]

Geographic occurrence and impact. The disease was reported in various genera of Caryophyllaceae in several countries, mainly in the Northern Hemisphere: on *G. paniculata* in Iran, Mongolia, Turkey, and the former USSR as *E. communis* (Amano 1986) and in Japan as *Oidium* sp. (Morita and Kikuchi 1996). Identified as *E. buhrii*, it was reported in Argentina, Armenia, Kazakhstan, Korea, Romania, and Ukraine (Braun 1995; Choi et al. 2014; Wolcan and Ronco 2003). Since 1995, the disease has occurred each year in main *Gypsophila*-growing area in Argentina and has increased in its economic importance (Wolcan and Ronco 2003). Powdery mildew produced nearly 100% disease incidence at the stage of harvesting in Iksan City (Choi et al. 2014) and is important in other provinces of Korea (Kang et al. 2009).

Symptoms/signs. Fungal growth first appears as circular to irregular white spots, which subsequently develop into an abundant growth of mycelia, conidia, and conidiophores (sign) on the upper surface of the leaves. The infection spreads to the lower leaf surface, stems, and peduncles (Fig. 2). In severe attacks, basal leaves die prematurely, and younger leaves become distorted. The disease occurs throughout the year, but mostly in autumn and spring.

Biology and epidemiology. Refer to the information on *E. buhrii* presented in ► Chap. 13, "Diseases of Carnation", Sect. 2.9.

Management

- ***Cultural practices*** – The same as explained for *E. buhrii* in ► Chap. 13, "Diseases of Carnation", Sect. 2.9.



Fig. 2 Severe infection of powdery mildew (S. Nakkeeran et al. Tamil Nadu Agricultural University, Coimbatore, Tamil Nadu, India © 2017. All Rights Reserved.)

- ***Biocompatible products*** – Among different products tested, the mixture of sodium bicarbonate with Tween #20 was the most effective in suppressing powdery mildew on *Gypsophila* (Kang et al. 2012). Also potassium bicarbonate is recommended by Gould (2012).
- ***Resistance*** – A new cultivar "Dream Pink," resistant to powdery mildew, was developed in Korea (Cheong et al. 2011).
- ***Fungicides*** – Matsura (1997) reported that triflumizole, triforine, sulfur, azoxystrobin, and thiophanate-methyl were effective in reducing powdery mildew caused by *Oidium* sp. on *Gypsophila* in Japan. Refer to ▶ Chap. 13, "Diseases of Carnation", Sect. 2.9 for additional information.

2.5 Phytophthora Crown Rot, Collar Rot, Root Rot, and Wilt [Current Name *Phytophthora nicotianae* van Breda de Haan (also Named *Phytophthora parasitica* Dastur and Formerly *Phytophthora nicotianae* var. *parasitica* (Dastur) Waterhouse and *Phytophthora parasitica* var. *nicotianae* Tucker)], *Phytophthora cryptogea* Pettyb. & Laff., *Phytophthora capsici* Leonian, *Phytophthora cactorum* (Leb. & Cohn.) Schroëter, and *Phytophthora drechsleri* Tucker

Geographic occurrence and impact. *Phytophthora nicotianae* was reported in Argentina, Australia (Queensland), Japan, Italy, Korea, Poland, South Africa,



Fig. 3 *G. paniculata* commercial plot with patches of dead plants and empty spaces due to infection by of *Phytophthora nicotianae* (S.M. Wolcan © 2017. All Rights Reserved.)

Taiwan, and the USA (Ann and Liu 1993; Engelhard 1974; Crous et al. 2000; Forsberg 1985; Garibaldi et al. 1990; Jin et al. 1998; Orlikowski et al. 1991; Sekiyama and Venatsu 1992; Thompson and Nauda 1992; Werner 1997; Werner and Antkowiak 2002; Wolcan et al. 2007). *P. cryptogea* was reported in Germany and Mexico (Kröber 1971; Ojeda and Zac 1993), *P. cactorum* in the USA (Wick et al. 1987), *P. capsici* in Taiwan (Ann and Liu 1993), and *P. drechsleri* in the USA (Abad et al. 1994).

The rapid and irreversible rotting of plants by *Phytophthora* species causes important production losses (Fig. 3). Ojeda and Zak (1993) reported an incidence of 25% of collar rot caused by *P. cryptogea* on *G. paniculata* var. perfecta when introduced into Mexico. Since the mid-1990s, all the greenhouses which produce *Gypsophila* in Argentina lose between 10% and 25% of the plants due to crown rot, mainly caused by *P. nicotianae* (Wolcan et al. 2007). In the USA, Engelhard (1974) reported up to 30% plant loss under commercial production, caused by *P. nicotianae*, during the first month after transplant.

Symptoms/signs. Infection can occur in every growing stage of plants. *Phytophthora* species produce rapid soft rot of roots and crown, causing wilting of the leaves and small stems. The decay of plants is very rapid (2 at 3 days) when conducive conditions exist including saturated soil, abundant surface moisture, and warm temperatures. Some of the leaves become chlorotic in the early stages of the disease while others simply wilt and die with little change in color (Fig. 4) (Ann and Liu 1993; Engelhard 1974; Wolcan et al. 2007).



Fig. 4 Wilt symptoms of plant with Phytophthora basal rot (*left*). Shoots from stubs are wilting due to the infection of *Phytophthora nicotianae. Right top* – healthy shoots (*right*) (S.M. Wolcan © 2017. All Rights Reserved.)

Biology and epidemiology. Refer to the information presented in ▶ Chap. 13, "Diseases of Carnation", Sect. 2.10.

The different *Phytophthora* species have similar life cycles and "fungal" structures. However, temperature requirements may differ, and formation of oospores may not occur in some species. The rate of *Phytophthora* root rot incidence on *G. paniculata* was higher in fields where the crop was overwintered than in newly planted sites, and the rate increased significantly in the overwintered sites with the start of the next cropping system. The cultural practice of cutting stems after harvest can serve as an entry for the pathogen disseminated by splashing, producing crown rot and decay of new shoots (Fig. 4). The disease is especially prevalent during warm months (Kang et al. 2009; Wolcan et al. 2007).

Management

- ***Cultural practices*** – Refer to ▶ Chap. 13, "Diseases of Carnation", Sect. 2.10. Improve drainage by planting on raised beds. Do not moisture stress plants, but do not overwater (Koike et al. 2009)
- ***Fungicides*** – Gould (2012) and Koike et al. (2009) recommend the use of etridiazole, mefenoxam, and metalaxyl applied as a soil drench at the time of transplant.

2.6 Pythium Root Rot, *Pythium* Basal Rot [*Pythium aphanidermatum* (Edson) Fitzp *Pythium irregulare* Buisman, *Pythium myriotylum* Drechs., *Pythium paroecandrum* Drechs, *Pythium sylvaticum* WA Campbell & JW Hendrix, and *Pythium ultimum* Trow]

Geographic occurrence and impact. *Pythium aphanidermatum* was recorded in Israel (Ben-Yephet et al. 1997; Vigodsky-Haas et al. 1979), while *P. irregulare* was recorded in Israel (Ben-Yephet et al. 1997) and Argentina (Frezzi 1956), and *P. paroecandrum*, *P sylvaticum*, and *P. ultimum* were recorded in Israel (Ben-Yephet et al. 1997; Gamliel et al. 1993). In Colombia *Pythium* spp. were reported causing losses >30% (Garcés de Granada et al. 1994).



Fig. 5 Pythium rot of cuttings. Soft rot is spread by contact of affected leaves (S.M. Wolcan © 2017. All Rights Reserved.)

Symptoms/signs. During rooting cuttings, the pathogen can cause partial or complete decay of the plantings resulting in an irregular and uneven rooting, affecting the quality of the cuttings (Fig. 5). Also, it has been found that the fungus in adult plants behaves as a minor pathogen, causing a reduction in plant growth and root activity with a decrease in yield and quality of flowers. Disease symptoms are more evident in soils with a history of continuous *Gypsophila* cropping (Galindo and Arbeláez 1995; Gamliel et al. 1993).

Biology and epidemiology. Refer to the information presented in ▶ Chap. 13, "Diseases of Carnation", Sect. 2.10.

A high cutting density in the rooting benches, overwatering, and high humidity in the propagative greenhouses favor the production and spread of basal rot in the cuttings or rooted cuttings (Fig. 5).

Management

- ***Cultural practices*** – Avoid high relative humidity and overwatering in the rooting trays as well as in production plots. Use well-drained soils. Control weeds inside and outside of greenhouses to keep better air circulation among plants and avoid possible alternative hosts of *Pythium* species. Also, avoid transplanting rooted cuttings with dark roots.
- ***Biological control*** – The application of *Trichoderma harzianum* isolate T17 increased the number of flower by 26.0% and 15.6% compared with an untreated control and commercial control, respectively (Galindo and Arbeláez 1995; Sivan et al. 1984). Some species of *Pythium* act as mycoparasites. *Pythium oligandrum*, *Pythium acanthicum*, and *Pythium periplocum* are well known as parasites of *P. irregulare* and *P. ultimum* (Van der Plaats-Niterink 1981).
- ***Fungicides*** – Mefenoxam is helpful when used in preventative programs (Koike et al. 2009). Gould (2012) recommends the use of etridiazole, mefenoxam, and metalaxyl applied as a soil drench during propagation and also at transplanting.

Metalaxyl produced a 11.4% increase in flower bunches compared with the untreated control (Galindo and Arbeláez 1995).

- ***Soil disinfestation – Solarization:*** Gamliel et al. (1993) used clear, 40–50-μm-thick polyethylene mulch to solarize both infested soils and substrates infested with *Pythium* spp. and *Rhizoctonia solani* for 40–55 d (max temperature 45–48 °C) and obtained increases in total yield of flowers (flower weight and number) and extended the cropping time. **Fumigation:** In the same experiment, metham sodium applied at a rate of 120 ml/m^2 via sprinkler irrigation produced the same results.

2.7 Rhizoctonia Rot and Rhizoctonia Stem Base Rot [*Rhizoctonia solani* Kühn Teleomorph: *Thanatephorus cucumeris* (Frank) Donk]

Geographic occurrence and impact. The disease has been reported in Argentina, Brazil, Israel, Korea, and Poland (Bueno et al. 2013; Elad et al. 2014; Kang et al. 2009; Werner and Antkowiak 2002; Wolcan et al. 2007).

Symptoms/signs. Refer to the information on *Rhizoctonia* in Sect. 2.12 of ► Chap. 13, "Diseases of Carnation".

In the field, symptoms of *Gypsophila* plants affected by *R. solani* may be mistaken for those produced by Phytophthora crown rot. In the case of *Rhizoctonia,* the stems are not easily pulled out. A dry and shredded appearance is observed when the rotted stem or the rhizome are split longitudinally (Fig. 6).



Fig. 6 Symptoms of dry and shredded rot in a rhizome of *G. paniculata* caused by *Rhizoctonia solani* (S.M. Wolcan © 2017. All Rights Reserved.)

Biology and epidemiology. Refer to Sect. 2.12 of ▶ Chap. 13, "Diseases of Carnation". *Rhizoctonia solani* populations are divided into anastomosis groups, and according to studies of Bueno et al. (2013) in Brazil, the pathogen from *Gypsophila* corresponds to the AG-4 HG III group.

Management

- ***Cultural practices*** – Refer to Sect. 2.12 of ▶ Chap. 13, "Diseases of Carnation".
- ***Fungicides*** – Gould (2012) recommends the use of iprodione, iprodione + thiophanate-methyl, and thiophanate-methyl + etridiazole applied by soil drench at the seedling stage and transplanting. Treat soil or the planting medium with granular PCNB before planting. Spray the base of plants before or after planting with iprodione or PCNB. Keep PCNB off foliage (Koike et al. 2009).
- ***Biological control*** – *Bacillus subtilis* var. *amyloliquefaciens* strain FZB24 is recommended by Gould (2012) to be applied as a soil and transplant drench.
- ***Soil disinfestation – Solarization:*** Gamliel et al. (1993) reduced disease caused by *Rhizoctonia* and *Pythium* and increased yield in *Gypsophila* using soil solarization (see Management of *Pythium* above). **Fumigation:** In the same experiment, metham sodium applied at a rate of 120 ml/m^2 via sprinkler irrigation produced the same results.

Additional fungal diseases – The following fungal pathogens of *Gypsophila* spp. have also been reported (see management of the same pathogen genera in the ▶ Chap. 13, "Diseases of Carnation"):

- ***Alternaria dianthi*****:** in Mexico (USDA, ARS, US National Fungus Collections (BPI) 1946). (http://mycoportal.org/portal/collections/individual/index.php?occid=1350477)
- ***Alternaria dianthicola*** **Neergaard:** in Poland (Werner and Antkowiak 2002).
- **Alternaria mold on flowers (*Alternaria axiaeriisporifera* E.G. Simmons & C.F. Hill)**. This species was recorded in New Zealand (Simmons 2007; Woudenberg et al. 2013).
- **Downy mildew (*Peronospora gypsophilae* Jacz., *Peronospora pulveracea* Fuckel).** *P. gypsophilae* was recorded in Israel (Ben-Ze'ev et al. 2006) and *P. pulveracea* in Ecuador (Puma Quinchuango 2010).
- **Phoma leaf spot – *Phoma gypsophilae* Hollós** was reported in Armenia (Simonyan 1981) and ***Phoma pinoggii*** **Servazzi** in Italy (Servazzi 1942).
- **Phyllosticta leaf spot** – ***Phyllosticta*** sp. was reported in the USA (Alfieri et al. 1984).
- **Rust** – ***Puccinia arenariae*** (Schumach.) G. Winter was reported on *G. paniculata* in Africa and on *G. elegans* in Chile, Germany, Portugal, and the UK (Farr and Rossman 2016).
- **White mold** – ***Sclerotinia sclerotiorum*** **(Lib.) de Bary, *Sclerotinia serica* M.A. Keay**. *S. sclerotiorum* was reported on *G. paniculata* in Israel and the USA (Elad et al. 2014; Koike et al. 2009), and *S. serica* was reported in the UK on *G. elegans* (Keay 1937).

3 Bacterial and Phytoplasma Diseases

3.1 Crown Gall [*Agrobacterium tumefaciens* (Smith and Townsend 1907) Conn (Formerly *Bacterium tumefaciens, Pseudomonas tumefaciens, Bacillus tumefaciens,* and *Phytomonas tumefaciens*)]

Geographic occurrence and impact. Crown gall is one of the most widely distributed bacterial diseases and has been reported from all five continents (Hayward and Waterston 1965; CMI 1976).

Symptoms/signs. Crown gall tumors are observed most often at the base of the stems and below ground on the main roots. Once initiated, tumors continue to growth into large, friable masses that dissociate into small pieces as the tumor ages and dries (Kado 2010).

Biology and epidemiology. *Agrobacterium tumefaciens* is a soilborne bacterium that infects dicotyledonous plants from over 90 different plant families. Crown gall tumors are the culmination of *A. tumefaciens* plasmid-mediated transformation of cells surrounding the infection court. Infection courts where bacteria proliferate are established mainly through injuries caused by mechanical trauma, grafting, freezing temperatures, and root-invading insects. High-moisture levels favor infections leading to transformation which take place in less than 3 h. Transformed cells proliferate into visibly abnormal tumors. As the tumors grow, *A. tumefaciens* is shed into the surrounding soil, particularly when rain or sprinkler irrigation occurs (Kado 2010). *A. tumefaciens* naturally resides on the rhizoplane of woody and herbaceous weeds. Its presence in soils originates from galls that were broken or sloughed off from infected plants during cultivation practices. Irrigation aids in further dissemination of the *A. tumefaciens* bacterial cells. *Agrobacterium tumefaciens* is also spread by infected and infested planting material originating from nursery stock from uncertified sources. Diagnosis is based on isolation in selective and semi-selective media (Schaad et al. 2001), bioassays, and PCR-specific detection (Haas et al. 1995).

Management. Refer to ▶ Chap. 13, "Diseases of Carnation", Sect. 3.4.

3.2 Fasciation [*Rhodococcus fascians* (Tilford 1936) Goodfellow 1984], also Called Leafy Gall

Geographic occurrence and impact. *R. fascians* is widely distributed and has been reported in Australia, Belgium, Canada, former Czechoslovakia, Denmark, Egypt, France, Germany, Hungary, Iran, Mexico, the Netherlands, New Zealand, Sweden, Russia, the UK, and the USA (Bradbury 1986; Putnam and Miller 2007).

Symptoms/signs. Excessive and abnormal proliferation of the leaves, flowers, and shoots, including gall formation on leaves and at wounds on the stems as a result of the hyperplastic growth of the affected tissues (Kado 2010).

Biology and epidemiology. Refer to ▶ Chap. 13, "Diseases of Carnation", Sect. 3.5.

Management. Refer to ▶ Chap. 13, "Diseases of Carnation", Sect. 3.5.

3.3 Gypsophila Gall, also Named Crown and Root Gall Disease of *Gypsophila* [*Pantoea agglomerans* pv. *gypsophilae* (Brown 1932), *Pantoea agglomerans* pv. *gypsophilae* (Formerly, *Erwinia herbicola* pv. *gypsophilae*, *Agrobacterium gypsophilae*, *Phytomonas gypsophilae*, *Pseudomonas gypsophilae*, and *Bacterium gypsophilae*)]

Geographic occurrence and impact. The host range of *Pantoea agglomerans* pv. *gypsophilae* is restricted to *Gypsophila* spp., and it has been reported in Israel, Italy, the Netherlands, Japan, Scotland, Taiwan, and the USA (Bradbury 1986; Kado 2010); whereas, *P. agglomerans* pv. *betae* is pathogenic on *Beta vulgaris* and *Gypsophila* (Manulis and Barash 2003).

Symptoms. Galls are formed, particularly, on the base of the stem. Symptoms resemble those of crown gall disease (Fig. 7). The galls may show water-soaked areas. The galls weaken the plants and cause defoliation and ultimately plant death (Manulis et al. 1998).



Fig. 7 Symptoms of Gypsophila gall on: cutting (*left*) and plants (*center* and *right*) (S Manulis-Sasson, Department of Plant Pathology and Weed Research, ARO The Volcani Center, Bet Dagan, Israel © 2017. All Rights Reserved.)

Biology and Epidemiology. The cut ends of stem cuttings are the sites of entry and colonization by *P. a*. pv. *gypsophilae*, f and gall formation. Galled *Gypsophila* cuttings are the primary sources of inoculum; bacteria are disseminated via cutting tools and splashing water (Kado 2010). Apparently the bacterium moves from the galls to the shoots via the xylem vessels (Manulis and Barash 2003). The bacterium can be reliably detected by Nested PCR or Bio-PCR in symptomless *Gypsophila* cuttings as reported by Manulis and co-workers (Manulis et al. 1998).

Management

- ***Cultural practices*** – The only control measure is by the production of pathogen-free planting material through culture indexing, followed by strict sanitation practices. The practice of placing freshly cut, bundled shoots in water effectively disseminates the pathogen to all cuttings (Kado 2010). Neither resistant clones nor effective chemical treatments are available (Manulis et al. 1998).
- ***Sanitation*** – Avoid recycling irrigation water because it can serve as a reservoir of the pathogen.

3.4 Phytoplasma Diseases

Geographic occurrence and impact. Aster yellows disease of *Gypsophila* has been reported in the USA (USDA 1960) and Canada (Northover 2007). Another phytoplasma disease affecting *Gypsophila* has been identified in Israel (Gera et al. 2007) and Poland (Kaminska et al. 1996). Phytoplasmas have a very broad and extensive host range covering at least 60 families (Kado 2010) indicating a lack of specificity.

Symptoms/signs. Aster yellows disease symptoms begin with a general yellowing of the foliage followed by growth distortions, such as the loss of apical dominance that results in shoot and floral proliferation into a witches' broom appearance. The yellowing leads to a general decline of the plant, culminating in death. Phyllody and alteration of tissue pigments (purple, yellow, red, and bronze) have been observed in some hosts (Kado 2010). Symptoms of stunting and shoot proliferation (witches' broom or "asparagus fern") and poor flower set have been observed in *Gypsophila* sp. in Israel (Gera et al. 2007) and Poland (Kaminska et al. 1996).

Biology and epidemiology. Phytoplasmas are members of the bacterial class *Mollicutes* which are Gram-negative pleomorphic cells without walls. Currently, they are not cultivable in vitro and are considered obligate parasites that reside in the plant phloem. Phytoplasmas are able to replicate in the plant host and in the insects that vector them. In the USA and Canada, *C.* phytoplasma spp. are vectored by the aster leafhopper or six-spotted leafhopper (*Macrosteles quadrilineatus* = *M. fascifrons*). In Israel, three leafhopper vectors of phytoplasmas were identified: *Orosius orientalis*, *Circulifer haematoceps*, and *C. tenellus* (Gera et al. 2007). The universal primers P1/P7 for PCR reaction have been developed for phytoplasma's detection (Deng and Hiruki 1991).

Management. Management of phytoplasma diseases is based on monitoring and reduction of insect vector populations through chemicals or biological means and vector exclusion through the use of fine mesh screening. Once a plant is infected, there is no curative treatment. Crop rotation is ineffective for phytoplasma management due to their wide host range. Market considerations must be made for phytoplasma disease management, and management strategies employed in certain crops will not necessarily be feasible in others.

4 Viral Diseases

4.1 Nepoviruses

Tomato ringspot virus (ToRSV)

Geographic occurrence and impact. The disease was reported in Lithuania (Samuitienė and Navalinskienné 2001).

Symptoms/signs. Infection reduces the plant growth, yield, and aesthetic quality. The main symptoms are plant stunting, malformation of leaves and flowers, chlorotic and necrotic spots and streaks, ring spots on leaves, vein necrosis, and shortening inducing leaf crinkling. When the infection is severe, plants may die early (Samuitienė and Navalinskienné 2001).

Biology and epidemiology. Transmission of ToRSV is by nematodes (Forer and Stouffer 1982) and vegetative propagation from infected plants. Also, pollen and seeds may disseminate the virus (Kahn 1956). Disease is widespread in field perennial flowers that may serve as reservoirs for viruses and a source of infection for other crops (Rosenberger et al. 1983).

Management. Propagation of selected healthy planting material, monitoring of plants for the symptoms, and elimination of affected plants. Control nematode populations by soil disinfestation through fumigation, soil solarization or steam treatment, or the use of chemical nematicides. Crop rotations can sometimes reduce nematode populations. The use of virus-free stock plants obtained by heat treatment and meristem culture ensures the start of a healthy plantation (Samuitienė and Navalinskienné 2001).

4.2 Cucumoviruses

Cucumber mosaic virus (CMV)

Geographic occurrence and impact. The disease was reported in Korea (Park and Choi 2009).

Symptoms/signs. Produces mosaic symptoms on *Gypsophila paniculata* (Park and Choi 2009).

Biology and epidemiology. The virus is transmitted by more than 60 species of aphids, notably *Aphis gossypii* and *Myzus persicae* (Francki et al. 1979). Seed transmission has been reported in more than 20 plant species, with varying efficiencies from <1% up to 50% (Palukaitis et al. 1992). The virus may be present in the embryo, endosperm, and seminal integuments, as well as in pollen. Seed transmission in weeds is of epidemiological significance (Rist and Lorbeer 1991).

Management. Infected plants cannot be treated and should be removed and destroyed. Suspect plants should be tested for CMV and other viruses. This is a virus with a very large host range. Good weed control, especially of perennial weeds is necessary. Aphid populations should be controlled/excluded to limit potential insect vectors (Michigan State University 2016).

Additional Virus Diseases

– **Tombusviruses.** *Carnation Italian ringspot virus* (CIRV) was isolated from *G. paniculata*, cherry trees, and surface water in Germany (Koenig et al. 2009).

5 Nematode Diseases

The number of reports and experiments devoted to nematodes is far less abundant in *Gypsophila* than in other florists' crops. Similar to another member of the Caryophyllaceae, carnation, *Gypsophila* is very susceptible to root-knot nematodes. Paradoxically, and in spite of its susceptibility, *Gypsophila* roots are high in saponins, and the potential use of these by-products for the environmentally friendly nematode control has been evaluated (Pensec et al. 2013).

5.1 Root-Knot Nematodes (*Meloidogyne* spp.)

Geographic occurrence and impact. *Gypsophila elegans* has been shown to be a very susceptible host of *Meloidogyne arenaria* race 1 (Mc Sorley 1994) under controlled conditions while *G. paniculata* was a susceptible host for *Meloidogyne incognita* race 2 (Wilcken and Ferraz 1998). In these assays, nematode reproduction rates were 4.96 and 2.73, respectively. Goff (1936), while rating many ornamental plants according to their susceptibility to root-knot nematodes, classified *Gypsophila* sp. as "very susceptible." No regular surveys or reports have been carried out about the occurrence of plant pathogenic nematodes on this crop. The only exceptions are those reported by Saira Shahzad et al. (2010), which found both *M. incognita* and *Meloidogyne javanica* on *G. paniculata* in Pakistan, and Goff (1936) which found severe galling by *Meloidogyne* in *Gypsophila* sp. in Florida.

Nevertheless, considering the wide distribution range of the three main root-knot nematodes species, it can be asserted that infection of *Gypsophila* spp. by *Meloidogyne* spp. is very likely to occur worldwide.

Symptoms/signs. Aboveground manifestations of heavy root parasitism result from the impairment in normal root functions and are similar as those mentioned for carnation (► Chap. 13, "Diseases of Carnation," Sect. 5.1). Infected roots display the typical galling.

Management. No particular research concerning either resistance or differences in susceptibility among cultivars has been carried out to date for *Gypsophila*. Control is based mostly on preplant soil disinfestation. For this purpose the same methods recommended for other crops are valid (see ► Chap. 13, "Diseases of Carnation," and ► 8, "Soil/Media Disinfestation for Management of Florists' Crops Diseases" for specific strategies).

Additional nematode pathogens. The following nematode pathogens of *Gypsophila* spp. have also been reported:

- ***Heterodera schachtii* (sugar beet cyst nematode)**. This nematode is distributed throughout Europe and also recorded in the Soviet Union, Turkey, Israel, the USA, Canada, Australia, and South Africa (Ferris 1999) and is cited as a parasite of *Gypsophila* sp. without specification of location (Southey 1993).
- **Root-lesion nematodes:** Wilcken and Ferraz (1998) tested the susceptibility of *G. paniculata* to *Pratylenchus brachyurus* and *P. coffeae* and found that it was a nonhost of these pathogens.

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Diseases of Lisianthus

20

Robert J. McGovern

Abstract

Lisianthus, *Eustoma grandiflorum* (Raf.) Shinn., is an important cut and potted flower that has been in increasing demand over the last three decades. Its profitability can be decreased by failure to maintain demanding propagation and production requirements and by numerous diseases. Particularly challenging lisianthus diseases are caused by *Botrytis cinerea*, *Fusarium* spp., and an ever increasing number of viruses, especially the tospoviruses. Management strategies for lisianthus diseases recently studied include chemical, biological, cultural, and physical control, host resistance, and their integration.

Keywords

Botrytis cinerea • *Fusarium* spp. • *Tospovirus* spp. • Integrated disease management

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R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_20

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1 Introduction

Lisianthus, *Eustoma grandiflorum* (Raf.) Shinn., commonly known as the "prairie gentian" or "Texas bluebell," is a flowering ornamental native to the Central and Southern USA (Shinners 1957). Beginning in the 1970s, Japanese plant breeders have been developing lisianthus cvs. with improved floral characteristics, and the plant was reintroduced into the USA cut- and potted-flower trade in the 1980s (Halevy and Kofranek 1984). Lisianthus ranks fifth in Japan, tenth worldwide, and eleventh in the USA in cut-flower value (Harbaugh et al. 2000; Kawakatsu and Fukuta 2012; USDA-NASS 2012). Although lisianthus can provide substantial profit, it is a challenging crop to propagate and grow and requires rigorous disease management (Harbaugh 2006). Propagation is exclusively by seed.

Where data are available, information on the management of specific diseases of lisianthus is presented in the following sections. General management strategies for diseases of florists' crops may be found in the introductory chapters on integrated disease management.

2 Fungal and Fungus-Like Diseases

2.1 Botrytis Blight (*Botrytis cinerea* Pers.: Fr.)

Geographic occurrence and impact. *Botrytis* blight is a ubiquitous problem for the production and postharvest shelf life of most florists' crops. The pathogen can cause serious losses in all flowering plants including lisianthus and has been shown to reduce the vase life of this crop by 50% compared to non-infected stems, or by up to 6 days (Vrind 2005).

Symptoms/signs. Botrytis blight, also known as gray mold, is characterized by prolific gray, fuzzy fungal growth consisting of masses of hyphae, conidiophores, and conidia. Symptoms on petals begin as small, bleached spots which rapidly enlarge to blight entire flowers. Senescing and abscising flowers and damaged tissue are very susceptible to *B. cinerea*, and the fungus commonly uses such infection sites to invade healthy tissue. Early symptoms on stems consist of brown lesions which may remain localized or enlarge and kill the entire plant. Localized lesions which may be somewhat concentric often girdle the stem and lead to wilting and death of tissue above the infection site (Fig. 1).



Fig. 1 Botrytis blight symptoms/signs: stem and leaf blight with profuse sporulation (*left*); flower and stem blight (*right*) (R.J. McGovern © 2017. All Rights Reserved.)

Biology and epidemiology. The fungus can survive as a pathogen or saprophyte in a wide range of plant hosts including many florists' crops (Daughtrey et al. 2000). Mycelium of the fungus was found to survive through the summer in plant debris in Israel (Yunis and Elad 1989), and some isolates produce small, black sclerotia (Ellis and Waller 1974). Conidia of *B. cinerea* are easily spread by air currents and water splash, making *Botrytis* blight one of the most commonly encountered and difficult-to-control diseases of lisianthus. All aboveground plant parts (stems, leaves, and flowers) are susceptible to *B. cinerea*, which is favored by cool, moist conditions. Blight severity in lisianthus was maximal at 15–22 °C/59–72 °F and 85–99% RH in greenhouse research conducted in Israel (Shpialter et al. 2009). Conidia of the pathogen must remain wet for 5–8 h for infection by *B. cinerea* to take place (Jewett and Jarvis 2001).

Management. Components of the integrated management of Botrytis blight include:

- ***Cultural practices*** – Reduction of RH and the duration of plant tissue wetness are critical for Botrytis blight suppression and may be aided by simultaneously heating and venting greenhouses in the evening in regions where such a practice is feasible (Daughtrey et al. 2000) and by avoidance of overhead irrigation. Greenhouse computer-controlled environment systems should be programmed to avoid RH above 85%. Where overhead irrigation must be used, watering should be scheduled early in the day to ensure rapid evaporation of moisture from plant surfaces. Shpialter et al. (2009) demonstrated that the use of polyethylene mulch versus bare soil and buried (20 cm) drip irrigation instead of surface drip irrigation significantly decreased the disease on cut stems of lisianthus following harvest in greenhouses in Israel. Increased plant spacing and blowing air inside the canopy of greenhouse-grown lisianthus, alone or in combinations, also suppressed Botrytis blight on stem bases and plant wounds (Elad et al. 2011). They attributed these results, in part, to a reduction of greenhouse RH and maintenance of a drier microenvironment. Supplemental calcium [$Ca(NO_3)_2$] applied through fertigation or as a spray also led to a moderate reduction in disease severity (Shpialter et al. 2009). Postharvest handling to minimize *B. cinerea* and other problems involves avoidance of plant injury and storage at 4 °C/39 °F (Sakata Ornamentals 2012).
- ***Sanitation*** – Removal of senescing and dead plant tissues that provide a ready substrate for *Botrytis* sporulation is important for the management of the disease. Flowering crops grown in overhead greenhouse spaces should be carefully monitored and properly maintained, as senescent infected tissue from these plants may serve as a source of *B. cinerea* for crops below.
- ***Fungicides and biocontrols*** – Using leaf disks removed from greenhouse-grown lisianthus in Australia, Stovold (1998) found that five fungicides (chlorothalonil, dichlorofluanid, fluazinam, prochloraz, and thiram) and two biocontrols (*Trichoderma harzianum* Rifai and a product containing a mixture of *Azotobacter*, *Bacillus*, *Pseudomonas*, *Rhizobium*, and *Chaetomium* spp.) were effective in reducing Botrytis blight severity when a benomyl- and iprodione-resistant isolate of the pathogen was inoculated 2 days after fungicide application; dichlorofluanid, fluazinam, pyrimethanil, and the mixed biocontrol were effective

7 days after fungicide application, but none of the treatments significantly reduced the disease 16 days after application. Interestingly, blight was significantly more severe 16 days after application of either benomyl or iprodione than the non-treated control when plants were inoculated with a benomyl- and iprodione-resistant isolate of *B. cinerea*. McGovern and Harbaugh (1999a) found in a growth chamber experiment that the fungicide fenhexamid at the highest rate + the spreader-sticker Latron B-1956 was superior to iprodione in reducing high levels of both Botrytis flower and stem blight severity in lisianthus and more effective than chlorothalonil in reducing flower blight severity. In a separate growth chamber trial, these researchers reported that a number of fungicides including various bicarbonate formulations, chlorothalonil + thiophanate methyl, copper sulfate pentahydrate, iprodione, and mancozeb produced equivalent reductions in Botrytis flower blight severity (McGovern and Harbaugh 1999b). In a field trial conducted in California, Wegulo and Vilchez (2004) found that all fungicides tested (azoxystrobin, boscalid, chlorothalonil, fenhexamid, fludioxonil, mancozeb, potassium bicarbonate, pyraclostrobin) and a biocontrol (*Bacillus subtilis* QST 713 strain) were equally effective in reducing a low incidence of the disease. Alternation of fenhexamid, iprodione, and pyrimethanil and fenhexamid and pyrimethanil alone were effective in reducing the disease in whole plants and plant stubs in greenhouse-grown lisianthus in Israel (Elad et al. 2008). However, only pyrimethanil was effective in reducing flower blight in lisianthus planted at a high density.

Widespread resistance to benzimidazoles (benomyl, thiophanate methyl) and less common resistance to dicarboximides (iprodione, vinclozolin) have been reported in populations of *B. cinerea* in the greenhouse flower industry in Europe and North America (Daughtrey et al. 2000). Stovold (1998) indicated that resistance to benomyl and iprodione in *B. cinerea* was also widespread in ornamental production facilities in Australia and that the level of resistance to benomyl was greater than to iprodione. In a survey of the fungicide sensitivities of *B. cinerea* from lisianthus in Israel, Elad et al. (2008) reported that isolates resistant to dicarboximide and benzimidazole fungicides were widespread at 65% and 27%, respectively, while isolates resistant to pyrimethanil and fenhexamid were found at lower incidences of 11% and 3%, respectively.

- ***Resistance*** – Wegulo and Vichez (2007) identified a number of commercial lisianthus cvs. that are moderately resistant to *B. cinerea* among which Magic Champagne was the most resistant. Other lisianthus cvs. reported to be resistant to the fungus are Piccolo 2 and 3 (Sakata Seed America 2015a, b).

2.2 Cercospora Leaf Spot [*Pseudocercospora eustomatis* (Peck) U. Braun (syn. *Cercospora eustomae*)]

Geographic occurrence and impact. To date, Cercospora leaf spot of lisianthus has only been reported in the Western Hemisphere (Argentina, Brazil, the Dominican Republic, and the USA) (Alfieri et al. 1994; das Chagas Oliveira Freire 2005; Wolcan 2005).

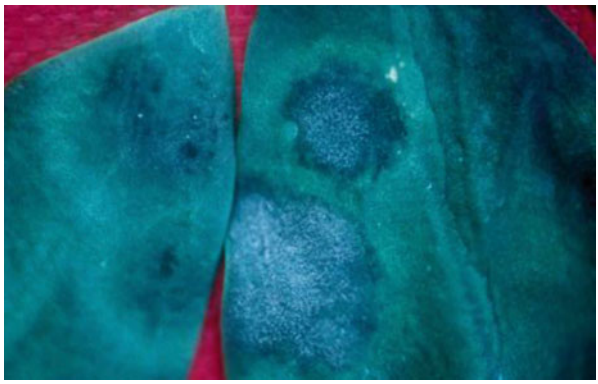


Fig. 2 Cercospora leaf spot symptoms/signs. Upper leaf surface (*left*), lower leaf surface (*right*) (note extensive sporulation) (R.J. McGovern © 2017. All Rights Reserved.)

Symptoms/signs. Leaf spots are at first yellow becoming dark brown, roughly circular, and mostly solitary but can become confluent as they enlarge. Spots generally have yellow halos, and mature lesions are covered with felty, olivaceous to dark gray masses of conidia, especially on the lower leaf surface (Fig. 2).

Biology and epidemiology. Specific information on the biology and epidemiology of *P. eustomatis* is not available. However, conidial germination of two related fungi, *P. macadamiae* and *P. fuligena*, was greatest at 26 °C (79 °F) and 28.5 °C (83 °F), respectively, and disease was enhanced by prolonged leaf wetness and high relative humidity (Mersha et al. 2014, 2010). *Pseudocercospora purpurea*, the causal agent of Cercospora spot of mango (*Mangifera indica*), survives in lesions in mature leaves which when wetted produce conidia which are spread by rain, irrigation, wind and insects (Marais 2004).

Management

- ***Fungicides*** – McGovern and Harbaugh (1999c) found that iprodione and thiophanate methyl + chlorothalonil were generally more effective than mancozeb and bicarbonate formulations in reducing low to moderate severity of Cercospora leaf spot on lisianthus in a greenhouse experiment in Florida.

2.3 Colletotrichum Blight (*Colletotrichum acutatum* J.H. Simmonds*)

Geographic occurrence and impact. The disease has been reported to occur in Japan, the USA, and Venezuela (Cedeño et al. 2007; Sato et al. 1997; Strandberg 2003). Stem cankers occurred at an incidence of 10% in 3-month-old potted lisianthus grown in a greenhouse in Venezuela (Cedeño et al. 2007). *[Based on genetic analysis, Sato et al. (2013) have re-identified the anthracnose pathogen of lisianthus in Japan as *Colletotrichum fioriniae*].



Fig. 3 Colletotrichum blight symptoms/signs: stem blight with black acervuli (*arrow*) containing conidia (Cedeño et al. 2007)

Symptoms/signs. *Colletotrichum acutatum* causes leaf, flower, and stem blight in lisianthus. Early leaf symptoms consist of minute, tan leaf spots which as they enlarge become the site of dark acervuli producing rose to orange conidial masses. The fungus often invades the main vein of leaves causing characteristic vein clearing in subsidiary veins. The fungus causes large blotches in opened flowers and complete flower blight at the bud stage. Tan lesions appear in the stem and may cause girdling and plant death (Fig. 3).

Biology and epidemiology. Conidia, produced in diseased leaves, flowers, and stems of lisianthus, are spread by rain or irrigation water to new infection sites (Strandberg 2003). Maximum growth of the fungus occurs between 26 and 28 °C/79 and 82 °F (Sato et al. 1997). In citrus, *C. acutatum* survives as dormant appressoria and hyphae in mature leaves (Zulfiqar et al. 1996). Survival of the fungus on alternate hosts is also a possibility. *Colletotrichum acutatum* can infect many fruit and ornamental crops, and isolates of the fungus from strawberry (*Fragaria* × *ananassa*) can survive without symptoms on a number of cultivated and weed plant species (Freeman 2008).

Management. Anthracnose in lisianthus was suppressed by UV-B radiation through pathogen inhibition and/or induction of host resistance (Satou et al. 2013).

2.4 Downy Mildew (*Peronospora chlorae* deBary)

Geographic occurrence and impact. This disease is prevalent in many countries including Argentina, Australia, Canada, Israel, Italy, Mexico, Norway, Poland, Taiwan, the UK, and the USA (Aloj et al. 1990; Auscher 1997; Cunnington 2008; Fucikovsky and Aranda 1994; Minchinton et al. 1997; OMAFRA 2003; O'Neil 2003; Orlikowski 2001; Toppe 2005; Wolcan et al. 1996; Yang and Hsieh 1998). Severe losses in lisianthus production have been reported; the incidence of downy mildew was 12–35% in a seedling bed in Taiwan (Yang and Hsieh 1998).



Fig. 4 Downy mildew symptoms/signs: leaf spots, upper surface (*left*); gray mass of sporangia and sporangiophores (*arrow*) in lesion on lower leaf surface (*right*) (Nelson 2005)

Symptoms/signs. *Peronospora chlorae* can affect both seedlings and mature plants. The pathogen rapidly infects the developing tissue of shoots and buds and causes stunting and distortion. Leaf blotches are also produced and begin as small yellow spots which enlarge and turn tan-brown. Gray masses of sporangia occur in mature blotches on the lower surface of leaves (Fig. 4).

Biology and epidemiology. *Peronospora chlorae* is an obligate biotroph; it can only survive by infecting a living host. It is fungus-like but not a true fungus related to the oomycetes *Pythium* and *Phytophthora*. Additional gentianaceous hosts of *P. chlorae* include *Blackstonia* spp., lesser centaury (*Centaurium pulchellum*), common centaury (*Centaurium erythraea*), and *E. roxburghii* (Hall 1994). Although infection of seed by other *Peronospora* spp. has been reported (Jang and Safeeulla 1990; Stegmark 1994), and seed infection of lisianthus by *P. chlorae* has been hypothesized (O'Neil 2003; Yang and Hsieh 1998), it has not yet been proven. Sporangia of the pathogen are dispersed by wind, rain, and irrigation. In the presence of abundant surface moisture, germination of sporangia occurs over a wide range of temperatures, 8–32 °C/33–90 °F (Yang and Hsieh 1998).

Management

- ***Cultural practices*** – As with other foliar diseases, minimizing the duration of leaf wetness is critical. Timing irrigation early in the day to allow for rapid foliar drying and the use of subirrigation systems can reduce foliar moisture. Careful greenhouse temperature and ventilation control especially during the night in cool climates may be used to prevent condensation on foliage.
- ***Fungicides*** – O'Neil (2003) reported that azoxystrobin and mefenoxam were effective in reducing downy mildew during propagation of lisianthus, while rotation of azoxystrobin with propamocarb hydrochloride and mefenoxam + mancozeb with

azoxystrobin, fosetyl-Al, and propamocarb hydrochloride were effective during production in greenhouse experiments in the UK. Yang and Hsieh (1998) found that cymoxanil +dithianon, fosetyl-Al, propineb + oxadixyl, propamocarb hydrochloride, and benalaxyl were the most effective fungicides in field trials in Taiwan.

2.5 Fusarium Crown and Root Rot [*Fusarium solani* (Mart.) Sacc. (Teleomorph: *Haematonectria haematococca* (Berk. and Broome) Samuels and Rossman (1999)]

Geographic occurrence and impact. The disease has occurred in Argentina, Costa Rica, Israel, Japan, South Africa, and the USA (McGovern unpublished data; Meir et al. 2010; Raabe 1991; Taubenhaus and Ezekiel 1935; Tomita et al. 2004; Truter and Wehner 2004; Wolcan et al. 2001a). Lisianthus mortality at a commercial production site reached 22% (Truter and Wehner 2004). Stunting of lisianthus attributed to *F. solani* infection of roots and the basal stem was observed in 100% of production greenhouses in Buenos Aires province, Argentina (Wolcan et al. 2001a). Losses of 50–100% were observed in cut-flower facilities in the USA (Texas) where successive crops of lisianthus were planted (Taubenhaus and Ezekiel 1935).

Symptoms/signs. The pathogen can attack seed, seedlings, and mature plants. Early symptoms include water-soaked areas in the stem at the soil line which become sunken and brown. The rot extends into the root system and the plant wilts and dies. Under humid conditions bright red perithecia of *Haematonectria haematococca* (sexual stage of *F. solani*) may be observed on infected stems (Fig. 5).

Biology and epidemiology. *Fusarium solani* produces three types of asexual spores, microconidia and macroconidia which are important in aerial dissemination, and thick-walled, stress-resistant chlamydospores which enable long-term survival. Survival by the fungus in association with plant debris similar to diseases in many other hosts



Fig. 5 Fusarium crown and root rot symptoms/signs: stem lesion (*left*, *arrow*); round red perithecia on stem (*arrow*, *right*) (R.J. McGovern © 2017. All Rights Reserved.)

caused by *F. solani* is very likely. Perithecia containing asci and ascospores of *H. haematococca* may or may not be present on the stems of declining/dead plants. The disease is favored by warm temperatures (~28 °C/82 °F) (Truter and Wehner 2004).

Management

- ***Biological control*** – Meir et al. (2010) observed a consistent though nonsignificant increase in lisianthus survival in soil infested by both *F. solani* and *R. solani* when plants were inoculated with the arbuscular mycorrhizal fungus *Rhizophagus intraradices* (formerly *Glomus intraradices*).
- ***Soil disinfestation*** – Fumigation with methyl bromide reduced the soil density of *F. solani* from 1.6×10^4 to 5×10^2 CFU/g and the incidence of stunting from 88% to 0.6%, respectively, compared with non-treated soil (Wolcan et al. 2001b). In a separate experiment, Wolcan et al. (2001b) demonstrated the equivalency of dazomet, metam sodium, and steam with methyl bromide in reducing the soil density of the pathogen.

2.6 Fusarium Crown and Stem Rot [*Fusarium avenaceum* (Fr.:Fr.) Sacc. (Teleomorph: *Gibberella avenacea* R. J. Cook)]

Geographic occurrence and impact. The disease has occurred in Ecuador, Israel, Italy, Japan, the Netherlands, Norway, and the USA (Elad et al. 2014; Horita and Kodama 1995; Koike et al. 1996; McGovern unpublished data; Pecchia et al. 2000; Toppe 2005; van der Burg and De Kreij 2003). During 1996–1997, losses of up to 70% were observed at field cut-flower production sites in California and Florida (McGovern et al. 1997a, 2003), and losses of 20–25% were reported at a cut-flower site in the Netherlands (de Werd 2003).

Symptoms/signs. Both seedlings and mature plants are susceptible. *Fusarium avenaceum* primarily attacks the crown and stems of lisianthus, but may also cause rot in the tap root and a few large feeder roots near the soil surface. The first aboveground symptom is a gradual loss of green coloration in leaves, followed by tan leaf flecks, browning of leaf veins, and tan desiccation of entire leaves. A brown stem rot and wilting occurs as the disease progresses, and infected plants rapidly die. Stem rot generally starts at or near to the attachment sites of the lowest leaves. Characteristic orange masses of conidia and white mycelial masses form at the base of rotted stems. Fusarium crown and stem rot may resemble Botrytis stem blight, but the two diseases can be differentiated based on their spore coloration: Fusarium crown and stem rot (orange) and Botrytis blight (gray) (Fig. 6).

Biology and epidemiology. *Fusarium avenaceum* produces numerous macroconidia but no microconidia; chlamydospores are not produced in the mycelium



Fig. 6 Fusarium crown and stem rot symptoms/signs: progression of symptoms (*left* to *right*) (*left*); mass of orange conidia (*arrow*) in stem lesion (*right*) (R.J. McGovern © 2017. All Rights Reserved.)

and rarely form in macroconidia (Booth and Waterston 1964). The sexual or anamorphic stage of the fungus, *Gibberella avenacea* R. J. Cook, has also been reported (Cook 1967), but rarely observed. Fusarium crown and stem rot is favored by cool temperatures, 14–23 °C/57–73 °F, and wounds (McGovern et al. 2003). The fungus has been reported to infect >150 plant genera and to survive as mycelium in plant debris (Booth and Waterston 1964). McGovern et al. (2003) indicated that *F. avenaceum* could survive on Styrofoam transplant trays and as an endophyte in the root systems of a number of ornamentals including wax begonia (*Begonia x semperflorens-cultorum*), carnation (*Dianthus caryophyllus*), exacum (*Exacum affine*), Gebera daisy (*Gerbera jamesonii*), Madagascar periwinkle (*Catharanthus roseus*), French marigold (*Tagetes patula*), pansy (*Viola tricolor* subsp. *hortensis*), petunia (*Petunia* x *hybrida*), rudbeckia (*Rudbeckia* sp.), salvia (*Salvia* sp.), and verbena (*Verbena* sp.). In addition, isolates of the fungus from several other hosts were pathogenic to lisianthus, indicating that *F. avenaceum* may be pathogenic on lisianthus regardless of its phylogenetic origin (Nalim et al. 2009). Seed infection by *F. avenaceum* has been reported in a number of hosts (Booth and Waterston 1964), but the fungus was not isolated from lisianthus seed (McGovern et al. 2003; Pecchia et al. 2000).

Short-distance aerial movement of macroconidia of *F. avenaceum* in a lisianthus transplant production house in California and two lisianthus cut-flower facilities in Florida was documented (McGovern et al. 2003; Seijo et al. 2000). The fungus is readily spread over long distances via infected transplants, and serious outbreaks of Fusarium crown and stem rot in cut-flower production facilities in the USA were attributed to infected transplants resulting from propagation in reused transplant

trays without prior disinfestation (McGovern et al. 2003). The fungus can also be spread by fungus gnats (*Bradysia* spp.), shore flies (*Scatella* spp.), and moth flies (*Psychoda* spp.) (McGovern and Harbaugh 1997; El-Hamalawi and Stanghellini 2005). Common feeding sites for fungus gnats in lisianthus are roots at the plant base which are covered by decomposing leaves closely appressed to the growing medium; these sites provide moist infection courts for *F. avenaceum*. The fungus may also be spread from plant to plant on cutting tools (McGovern et al. 2003).

Management

- ***Cultural practices*** – Use pathogen-free transplants. Avoid overwatering to prevent the buildup of fungus gnat and shore fly populations and control these vectors by biological or chemical means.
- ***Sanitation*** – Avoid reusing transplant trays without thorough chemical or steam disinfestation; an alternative is the use of disposable plastic inserts that fit within Styrofoam trays. Disinfest cutting tools between plants. Locate cull piles away and downwind from production facilities. Eliminate weeds. Disinfest soil between crops using chemical (fumigation) or physical measures (steam or soil solarization).
- ***Fungicides*** – McGovern et al. (2002a) reported that preventive application of azoxystrobin reduced disease incidence and plant mortality to ~20% and 0%, respectively, in a growth chamber trial; fluazinam, fludioxonil, myclobutanil, and thiophanate methyl were generally less effective. In a greenhouse experiment in the Netherlands, van der Wurff and Hamelink (2007) found that fludioxonil + cyprodinil and trifloxystrobin were effective in reducing *Fusarium* crown and stem rot.
- ***Resistance*** – Harbaugh and McGovern (2000) evaluated the susceptibility of 46 lisianthus cvs. under high disease pressure in growth chamber experiments and found that the lowest disease frequencies 55 days after inoculation with *F. avenaceum* occurred in "Ventura Deep Blue" and "Hallelujah Purple" (25%), "Bridal Pink" (23%), and "Heidi Pure White" (53%), representing the blue/purple, pink, and white flower color groups, respectively.

2.7 Fusarium Wilt (Fusarium oxysporum (Schlechtend):Fr. f. sp. eustomae)

Geographic occurrence and impact. Fusarium wilt of lisianthus has occurred in Ecuador, Israel, Italy, Japan, Korea, Poland, the Netherlands, the UK, and the USA (de Werd 2003; Elad et al. 2014; Hahm 1998; McGovern unpublished data; O'Neil and Green 2010; Orlikowski 2001; Raabe 1991; Rapetti et al. 2002; Tomita et al. 2004). Losses of 50–70% observed at a lisianthus cut-flower facility in the Netherlands were attributed to *F. oxysporum* f. sp. *eustomae* possibly in combination with *Pythium irregulare* (de Werd 2003). Losses of 10–50% and 100% were observed at cut-flower sites in Ecuador and the USA (California), respectively (McGovern, unpublished data). Fusarium wilt was reported to be widespread throughout the



Fig. 7 Fusarium wilt symptoms/signs: root and basal stem rot and white masses of conidia in sporodochia (*arrow*) on the stem of a wilted plant (R.J. McGovern © 2017. All Rights Reserved.)

ROK especially in alpine production locations where it occurred at incidences of 5–30% (Hahm et al. 1998). Up to 40% crop loss occurred in Poland (Orlikowski 2001).

Symptoms/signs. The *F. oxysporum* f. sp. *eustomae* infects and causes a dark discoloration of roots and the stem base. The pathogen moves upward through the xylem causing yellowing of leaves and wilting; vascular discoloration is visible when infected stems are cut lengthwise. As the disease progresses white masses of spores (sporodochia) are produced on stems, the leaves become tan, and plants wilt entirely and die (Fig. 7). The pathogen may also cause pre- and postemergence damping-off.

Biology and epidemiology. *Fusarium oxysporum* f. sp. *eustomae* produces three types of asexual spores: microconidia and macroconidia which enable aerial dissemination and thick-walled, stress-resistant chlamydospores which enable long-term survival. Survival by the fungus in association with plant debris similar to other *formae speciales* of *F. oxysporum* is very likely. The fungus has also been shown to survive as a symptomless endophyte in the roots of *Aubrieta* sp., rapeseed (*Brassica*

napus), and stock (*Matthiola incana*) (O'Neil 2006a). The sexual stage of *F. oxysporum* has not been reported. The disease is favored by warm temperatures (20–28 °C/68–82 °F) (Bertoldo et al. 2015; McGovern et al. 2002b). Genetic and virulence variability of the pathogen was reported in Italy (Bertoldo et al. 2015), and a PCR assay was developed which enabled its detection in the roots and stems of infected but symptomless plants (Li et al. 2010).

Management

- ***Biological control*** – A commercial formulation which combines *Bacillus* sp., *Pseudomonas* sp., *Streptomyces* sp., and *Trichoderma* sp. Consistently reduced plant mortality from *F. oxysporum* f. sp. *eustomae* in growth chamber experiments (McGovern et al. 2002c; McGovern unpublished data). The biocontrol *Trichoderma lignorum* significantly reduced the incidence of damping-off caused by *Pythium* sp. and *F. oxysporum* and increased grower profitability in research in commercial facilities in Ecuador (Sacoto Bravo 2009).
- ***Fungicides*** – In controlled environment experiments, fludioxonil and triflumizole were very effective in reducing both the incidence of and mortality due to *F. oxysporum* f. sp. *eustomae*; azoxystrobin, myclobutanil, thiophanate methyl, and trifloxystrobin were less effective, and thiophanate + chlorothalonil was ineffective (McGovern et al. 2002b, c). Chase (2011) reported that the severity of the disease was significantly reduced by the fungicide metconazole and the inducer of systemic acquired resistance, acibenzolar-S-methyl.
- ***Soil disinfestation*** – Preplant soil disinfestation is an important tool in the management of Fusarium wilt. Failure to fumigate the soil between successive lisianthus crops and inadequate fumigant distribution due to high soil compaction led to serious outbreaks of the disease in the USA (California) and Colombia, respectively (McGovern, unpublished data). O'Neil and Green (2010) reported that sheet steaming (>80 °C/176 °F for 10 h), a steam plough (>80 °C/176 °F for 1 h), chloropicrin applied via drip line irrigation, dazomet incorporated into the soil, and formaldehyde and metam sodium drenched onto the soil significantly reduced but did not eliminate the incidence of *Fusarium* sp. in buried, naturally infected roots and stems of lisianthus in commercial greenhouse experiments in the UK; calcium cyanamide incorporation was ineffective. Preplant steaming and application of metam sodium and dazomet provided acceptable control of *Fusarium* sp. for cut-flower production in Argentina (Salles et al. 2001).
- ***Resistance*** – Greenhouse trials conducted in Italy indicated that the lisianthus cvs. Mariachi Green and Echo Dream Yellow were partially resistant to the disease (Gilardi et al. 2006).
- ***Integrative strategies*** – O'Neil and Green (2007) found that the efficacy of sheet steaming in reducing the viability of *F. oxysporum* was not improved by combination with a number of biocontrols (*Gliocladium catenulatum*, nonpathogenic *F. oxysporum*, *Trichoderma harzianum*) or fungicides (calcium cyanamide, carbendazim).

2.8 Phomopsis Stem and Leaf Blight (*Phomopsis* sp.)

Geographic occurrence and impact. The disease occurred in the USA (Florida) where losses ranging from 4% to 80% were observed in two potted lisianthus production facilities (McGovern et al. 2000a).

Symptoms/signs. Stem necrosis is rapidly followed by leaf blight and the production of numerous dark pycnidia in diseased tissue. As the stem blight progresses, infected plants collapse and die (Fig. 8).

Biology and epidemiology. The fungus is spread by spores and infected plants and survives as pycnidia and mycelium in infected tissue.

Management. Refer to the introductory chapters on integrated disease management.

2.9 Powdery Mildew [*Leveillula taurica* (Lév.) G. Arnaud (Anamorph: *Oidiopsis taurica* (Lev.) E. S. Salmon)]; *Oidium* sp.

Geographic occurrence and impact. Powdery mildew of lisianthus has been reported to be caused by *L. taurica* (*Oidiopsis* spp.) in Brazil, Israel, Poland, Spain, Venezuela, and the USA and by *Oidium* sp. in Argentina and Japan (Cabrera et al. 2009; Cedeño et al. 2009; Elad et al. 2007; Koike et al. 1995; Melgares de Aquilar Cormenzana 1996; Okamoto et al. 2002; Orlikowski 2001; Reis et al. 2007).



Fig. 8 Phomopsis stem and leaf blight symptoms/signs. Note the production of numerous black pycnidia (*arrow*) in infected tissue (R.J. McGovern © 2017. All Rights Reserved.)

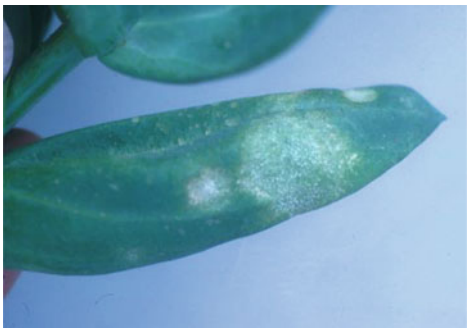


Fig. 9 Powdery mildew symptoms/signs: yellow blotches on leaves covered with profuse, white fungal growth (mycelium, conidiophores, and conidia) (R.J. McGovern © 2017. All Rights Reserved.)

Genetic analysis should elucidate the taxonomic relationships among the various powdery mildew pathogens of lisianthus.

Symptoms/signs. Symptoms and signs include yellow blotches on both the upper and lower leaf surfaces and stems which become covered with profuse white fungal growth (mycelium, conidiophores, and conidia). Floral infection and leaf distortion and premature abscission may also occur (Fig. 9).

Biology and epidemiology. Only the anamorphic stage of powdery mildew fungi has been observed on lisianthus; the role of the teleomorphic stage is currently unknown. The fungus infects the epidermis but, unlike most powdery mildew pathogens, it also colonizes mesophyllic tissue by means of stomata through which conidiophores subsequently emerge. Elad et al. (2007) found that conidial germination of *O. taurica* was optimal at 20 °C/68 °F and a relative humidity of 75–85% and that severe infection occurred at 15–20 °C/59–68 °F in pepper research in Israel; prolonged temperatures above 25 °C/77 °F decreased disease severity. Conidia of the fungus are spread by air currents.

It has been suggested that *L. taurica* is a composite species consisting of a number of host-specific races (Braun 1987, 1995). However, Correll et al. (1987) found that *L. taurica* isolates from a number of different genera and families could cause powdery mildew in tomato (*Solanum lycopericum*). Okamoto et al. (2002) demonstrated that an isolate of *Oidium* sp. from 4 o'clock flower (*Mirabilis jalapa*) could cause powdery mildew symptoms on both lisianthus and broad bean (*Vicia faba*). Reis et al. (2007) reported that in addition to lisianthus, nasturtium (*Tropaeolum majus*), calla lily (*Zantedeschia aethiopica*), impatiens (*Impatiens balsamina*), and balloon plant (*Asclepias physocarpa*) were hosts of *Oidiopsis*.

Management. The endoparasitic nature of *Leveillula taurica* makes it more difficult to control by contact fungicides than ectoparasitic powdery mildew pathogens which infect host tissue more superficially.

- ***Cultural practices*** – In greenhouse research on pepper (*Capsicum annuum*) in Israel, Elad et al. (2007) found that powdery mildew caused by *Leveillula taurica*

was decreased by increasing nighttime temperatures by heating and daytime temperatures by manipulation of the greenhouse vents.

- ***Fungicides*** – Numerous fungicides and a number of biocontrols are available for management of powdery mildew (refer to ▶ Chap. 7, "Fungicides and Biocontrols for Management of Florists' Crops Diseases"). Fungicides should be rotated by mode of action and applied preventively.

2.10 Pythium Root Rot (*Pythium* spp.)

Geographic occurrence and impact. Pythium root rot in lisianthus may be caused by a number of *Pythium* species including *P. irregulare*, *P. myriotylum*, *P.* spinosum, as well as unidentified species of the pathogen and has been reported in Ecuador, Israel, Japan, Norway, Spain, and the USA (Aegerter et al. 2002; Elad et al. 2014; McGovern unpublished data; Melgares de Aquilar Cormenzana 1996; Sacoto Bravo 2009; Tomita et al. 2004; Toppe 2005). Losses exceeding 75% occurred in greenhouse in the USA (California) (Aegerter et al. 2002).

Symptoms/signs. The root systems become darkly discolored and rotted, and infected plants rapidly wilt. The outer rotted and discolored layer of *Pythium*-infected roots is often easily pulled off leaving behind the central, lighter-colored stringy root fiber (stele). The pathogen may also cause pre- and postemergence damping-off (Fig. 10).

Biology and epidemiology. The genus *Pythium* is closely related to *Phytophthora* and also is fungus-like but not a true fungus. The pathogen is disseminated as



Fig. 10 Pythium root rot symptoms: discolored roots (*left*), rapid wilting (*right*) (R.J. McGovern © 2017. All Rights Reserved.)

zoospores through irrigation, surface water, and rain, and long-term survival is enabled by chlamydospores and sexually produced oospores. Pythium root rot is common in waterlogged soils with poor aeration; cycles of under- and overwatering which lead to root damage are very conducive to outbreaks of *Pythium*. Fungus gnats (*Bradysia* spp.) create infection sites through their root-feeding and may disseminate the pathogen. *Pythium*, depending on the species, may have a very broad host range.

Management

- ***Biological control*** – The biocontrol *Trichoderma lignorum* significantly reduced the incidence of damping-off caused by *Pythium* sp. and *F. oxysporum* and increased grower profitability in research in commercial facilities in Ecuador (Sacoto Bravo 2009).
- ***Fungicides*** – A number of isolates of *P. irregulare* recovered from lisianthus in the USA (California) were found to be resistant to the commonly used fungicide mefenoxam (Aegerter et al. 2002).

2.11 Rhizoctonia Crown and Stem Rot Blight [*Rhizoctonia solani* Kühn (Teleomorph: *Thanatephorus cucumeris* (A.B. Frank) Donk)]

Geographic occurrence and impact. Rhizoctonia root, crown, and stem rot is a major plant disease and has been documented in lisianthus in Israel, Japan, Spain, and the USA (Alfieri et al. 1994; Meir et al. 2010; Melgares de Aquilar Cormenzana 1996; Yoshimatsu 1993). Tomita et al. (2004) observed 2.8–31.6% losses due to damping-off in the field from *R. solani* in Japan.

Symptoms/signs. *Rhizoctonia solani* can infect the roots, stems, and foliage of lisianthus. The fungus most typically produces a dark discoloration and rot in stems at the soil line, leading to wilting and rapid collapse of the entire plant. Under humid conditions, mycelial growth of *R. solani* may rapidly envelop and blight leaves and shoots; this form of the disease is known as Rhizoctonia aerial blight. Discrete leaf lesions may have a concentric appearance. The pathogen may also cause pre- and postemergence damping-off (Fig. 11).

Biology and epidemiology. *Rhizoctonia solani* has a very large host range which encompasses most economically important cultivated plants including ornamentals and weeds; but some host specialization has been demonstrated (Mordue 1974). The fungus is very active at warm temperatures and effectively colonizes and survives as hyphae and sclerotia in plant debris, soil, and other growing media. Although *R. solani* does not produce spores, it can be spread by infected propagative material including seeds, water splash, and airborne particulate matter. The relationship of the sexual stage of *R. solani* to plant disease has not been well studied.



Fig. 11 Rhizoctonia crown and stem rot and foliar blight symptoms (R.J. McGovern © 2017. All Rights Reserved.)

Management

- ***Biological control*** – Meir et al. (2010) observed a consistent though nonsignificant increase in lisianthus survival in soil infested by both *F. solani* and *R. solani* by prior inoculation with the arbuscular mycorrhizal fungus *Rhizophagus intraradices* (formerly *Glomus intraradices*).
- ***Soil disinfestation*** – Chloropicrin applied by chemigation (through drip irrigation tubing) resulted in significant but minor reduction of *R. solani* soil densities in greenhouse research in the UK (O'Neil 2006b).

2.12 Thielaviopsis Black Root Rot [*Thielaviopsis basicola* (Berk. and Broome) Ferraris (1912)]

Geographic occurrence and impact. The disease has occurred in Canada, Switzerland, and the USA (Florida) (Joshi 2000; McGovern unpublished data, Michel 2015). A loss of ca. 70% occurred with one planting of a single cultivar in Florida.

Symptoms/signs. A black discoloration occurs in the small, feeder roots, or these roots are entirely absent. Infected plants are stunted, turn yellow, wilt, and die (Fig. 12).

Biology and epidemiology. *Thielaviopsis basicola* produces black, thick-walled, resistive chlamydospores (also referred to as macroconidia), and microconidia, which are involved in its survival and dissemination. The fungus is worldwide in distribution especially in cool, wet climates and infects a broad range of cultivated plants in many unrelated families. Black root rot is most severe in cool, wet soils (17–23 °C/62–73 °F) and at a soil pH above 5.5 (Shew and Lucas 1991).



Fig. 12 Thielaviopsis black root rot symptoms: note the black discoloration of small, feeder roots (R.J. McGovern © 2017. All Rights Reserved.)

Management

- ***Cultural practices*** – The disease can be reduced by lowering the soil pH to below 5.5 if feasible, and crop rotation.

Additional Fungal Diseases. The following fungal diseases of lisianthus have also been reported:

- Alternaria blight (*Alternaria* sp.) (Israel, Auscher 1997)
- Brown root rot [*Subplenodomus drobnjacensis* (Bubák) Gruyter, Aveskamp, and Verkley 2012] (Japan, Kondo et al. 2014)
- Curvularia leaf blotch (*Curvularia* sp.) (USA, Jones and Harbaugh 1995)
- Myrothecium leaf spot and blight (*Myrothecium roridum* Tode) (The Netherlands, Ludeking and Arkesteijn 2011)
- Penicillium root rot (*Penicillium* sp.) (Japan, Tomita et al. 2004)
- Phytophthora root rot and stem blight (*Phytophthora* spp.) (Japan, Uematsu et al. 1996)
- Phyllosticta leaf spot (*Phyllosticta* sp.) (USA, Daughtrey 2000)
- Sclerophoma stem blight (*Sclerophoma eustomis* Taubenhaus and Ezekiel) (USA, Taubenhaus and Ezekiel 1935)
- Sclerotium stem blight [*Sclerotium rolfsii* Sac. (teleomorph: *Athelia rolfsii* (Curzi) C.C. Tu and Kimbr.) (USA, Japan, McGovern et al. 2000b; Tomita et al. 2004)
- Sclerotinia stem rot [*Sclerotinia sclerotiorum* (Lib.) de Bary] (Wolcan et al. 1996)
- Stemphylium blight (*Stemphylium lycopersici* Wollr.) (Venezuela, Cedeño et al. 2011)
- White rust [*Albugo swertiae* (Bed. et Komm.) Wilson (Dingley 1969)] (New Zealand, McKenzie 1987)
- White blister rust (*Pustula centaurii* (Hansf.) Thines, C. Rost et Y. J. Choi, comb. nov, MB519564) (Australia, Tasmania, Ploch et al. 2011)

For an up-to-date list of fungi associated with lisianthus, access http://nt.ars-grin.gov/fungaldatabases/fungushost/FungusHost.cfm and enter "Eustoma" in the host name box.

3 Bacterial and Phytoplasma Diseases

3.1 Bacterial Crown Rot [*Burkholderia gladioli* (Severini 1913) Yabuuchi et al. 1993, comb. nov.]

Geographic occurrence and impact. The disease has been reported in Poland and the USA (Schollenberger and Zamorski 2008; Seijo et al. 2002).

Symptoms/signs. The pathogen can infect plants of various ages ranging from small seedlings to flowering plants and causes tan veinal necrosis in the leaves, rot in the tap root, crown, and stem, and wilting and death (Fig. 13).

Biology and epidemiology. *Burkholderia gladioli* (formerly *Pseudomonas gladioli*) can infect a number of plant hosts including gladiolus (*Gladiolus* x *hortulanus*), onion (*Allium cepa*), and mushroom (*Agaricus bitorquis*), and based on its host range was divided into three pathovars: pv. *gladioli*, pv. *alliicola*, and pv. *agaricicola*, respectively (Young et al. 1996). However, *Burkholderia gladioli* from lisianthus has not yet been characterized to the level of pathovar. The bacterium has also been isolated from the pulmonary tissue of cystic fibrosis patients (Beringer and Appleman 2000). The disease is favored by warm temperatures (28 °C/82 °F), and the bacterium can be seed-borne (Seijo et al 2002). Whitby et al. (2000) developed a species-specific PCR assay that distinguishes between *B. gladioli* and



Fig. 13 Bacterial crown rot symptoms: crown and stem rot and veinal necrosis (*left*) wilting and stem collapse (*right*) (R.J. McGovern © 2017. All Rights Reserved.)

a number of other closely related species such as *B. caryophylli* which causes similar symptoms in lisianthus.

Management
- ***Cultural practices*** – Use pathogen-free seed, and avoid unnecessarily wounding plants. Rapidly remove infected plants.

3.2 Burkholderia Wilt [*Burkholderia caryophylli* (Burkholder 1942) Yabuuchi et al. 1993, comb. nov.]

Geographic occurrence and impact. This disease has occurred in Japan (Furuya et al. 2000).

Symptoms/signs. The symptoms are similar to those caused by *Ralstonia solanacearum* (see next disease). The pathogen causes root rot and yellowing, wilting, and browning of the foliage through destruction of the water-conducting tissue; a cross section of infected stems reveals a typical tan to yellow-brown discoloration of the cortical tissue. The lower leaves are affected first.

Biology and epidemiology. *Burkholderia caryophylli* (formerly *Pseudomonas caryophylli*) also causes rot in carnation, onion, and statice (*Limonium sinuatum*) (Ballard et al. 1970; Jones and Engelhard 1984; Palleroni 1984). Shao et al. (2011) developed a real-time fluorescent PCR assay for specific detection of B. caryophylli.

Management
- ***Cultural practices*** – Rapidly remove infected plants.

3.3 Ralstonia Wilt (*Ralstonia solanacearum* (Smith 1896) Yabuuchi et al. 1996, comb. nov.)

Geographic occurrence and impact. Bacterial wilt caused by *Ralstonia solanacearum* (formerly *Pseudomonas solanacearum*) is one of the most important bacterial diseases of cultivated plants and has been reported to occur in hundreds of plant species representing more than 40 families (Buddenhagen and Kelman 1964). This disease of lisianthus has occurred in Japan and Taiwan (Chao et al. 1995; Tomita et al. 2004).

Symptoms/signs. Disease symptoms include wilting of lower leaves and then the entire plant, followed by browning of the foliage and plant death. In cross section, infected stems exhibit a characteristic brown discoloration in cortical tissue; infected stems also feel hollow when pressed.

Biology and epidemiology. Five groups (races) of *Ralstonia solanacearum* have been described based on host range (Buddenhagen 1986; Buddenhagen et al. 1962).

Thus far, all strains of the bacterium in Taiwan have been classified as race 1, including the strain that infects lisianthus (Lee et al 2001). Race 1 infects many solanaceous crops, ornamentals, and weeds and has a very high temperature optimum (35–37 °C/95–98 °F). The bacterium can survive in the soil in association with plant debris (Sugawara and Ishii 2009). In carnation the pathogen has been reported to move from one cutting to another in irrigation water during propagation, but plant to plant transmission appeared to be slow and limited to neighboring plants (Bradbury 1973). Lee et al. (2001) have developed a method to detect race 1 strains of *R. solanacearum* by PCR.

Management

- ***Cultural practices*** – Sugawara and Ishii (2009) recommended the avoidance of successive plantings of lisianthus, rapid removal of infected plants, disinfestation of soil and equipment, and improvement of drainage.

3.4 Phytoplasma Diseases

Geographic occurrence and impact. Diseases of lisianthus caused by phytoplasmas have been reported to occur in Brazil and Israel (Rivas et al. 2000; Weintraub et al. 2007). However, it is likely that additional reports of phytoplasma infection of lisianthus will be forthcoming given the global distribution of both host and pathogen.

Symptoms/signs. Symptoms caused by phytoplasmas may include yellowing of foliage, stunting and proliferation of shoots (witch's brooms), and the abnormal development of floral parts into leafy structures (phyllody) and of green coloration in flowers (virescence).

Biology and epidemiology. Phytoplasmas are prokaryotes that lack cell walls and are obligate plant parasites vectored by phloem-feeding insects in the Cicadellidae family and Fulgoroidea superfamily such as leafhoppers and planthoppers, respectively. Based on 16S rDNA analysis, the phytoplasma detected in lisianthus and those detected in other plants in Israel including apricot (*Prunus* sp.), cyclamen (*Cyclamen* sp.), grape (*Vitis vinifera*), papaya (*Carica papaya*), and pepper (*Capsicum annuum*) have been placed in the Stolbur phytoplasma group (Weintraub et al. 2007). Six confirmed leafhopper vectors and one suspected planthopper vector of phytoplasmas occur in Israel (Weintraub et al. 2007).

Management. Management of phytoplasma diseases, like those caused by viruses, is based on monitoring and reduction of insect vector populations through chemical or biological means, vector exclusion by physical barriers such as fine mesh screening, vector disorientation and repellence in field-grown crops through the use of reflective mulches, and elimination of, and avoidance of growing near, alternate hosts especially established, infected crops.

4 Viral Diseases

4.1 Bromoviridae

Geographic occurrence and impact. Viruses in the Bromoviridae commonly cause major worldwide crop losses. Two members of this group have been reported in lisianthus: *Cucumber mosaic virus* (CMV, genus *Cucumovirus*) and *Tobacco streak virus* (TSV, genus *Ilarvirus*) (de Freitas et al. 1996a; Fujinaga et al. 2006; Gera and Cohen 2007; Hahm et al. 1998; Providenti 1985; Rivas et al. 2000). The geographic occurrence of these viruses is indicated in Table 1. CMV was detected at an incidence of 8.6% in Japan (Nagano Prefecture) (Fuginaga et al. 2006) and 10% in alpine production areas in the ROK in combination with a *Fabavirus*, *Broad bean wilt virus* (Hahm et al. 1998).

Symptoms/signs. Symptoms caused by plant viruses, including the Bromoviridae, can be variable and depend on the host, environment, virus strain, and coinfection with other viruses; different Bromoviridae genera may produce similar symptoms. Symptoms caused by the Bromoviridae in lisianthus are indicated in Table 1.

Biology and epidemiology. Viruses in the Bromoviridae are acquired and spread by aphids (Hemiptera: Aphididae) or thrips (Thysanoptera: Thripidae) in a non-circulative (nonpersistent) manner; these viruses are mouthpart-borne and do not enter the insect's hemolymph. Spread of TSV-infected pollen by thrips has been demonstrated (Klose et al. 1996). Seed transmission of CMV has been reported in many plant species at incidences of less than 1% up to 50% (Garcia-Arenal and Palukaitis 2008). Seed transmission of TSV in various hosts has been observed at incidences of 0.7–90.6% (Kaiser et al. 1982).

Management. Virus management, including species in the Bromoviridae, is based on monitoring and reduction of vector populations through chemical and/or biological measures, vector exclusion by physical barriers such as fine mesh screens, vector disorientation in field-grown crops through the use of reflective mulches, and elimination of, and avoidance of growing near, alternate hosts especially established, infected crops. Cross protection, prior infection with an attenuated strain of CMV, was shown to reduce subsequent infection by a severe strain of the virus in cucumber, lisianthus, petunia, and tomato (Sayama 1996). Resistance to CMV has been introduced into a wide variety of vegetables through genetic modification. Additional information on integrated disease management of viruses may be found in the introductory (▶ Chap. 4, "Insect Management for Disease Control in Florists' Crops").

4.2 Bunyaviridae

Geographic occurrence and impact. Plant viruses in the Bunyaviridae and genus *Tospovirus* cause extensive losses in nearly all cultivated plants. Thus far seven

Table 1 Bromoviridae infecting lisianthus

Viruses	Occurrence in lisianthus	Transmission	Other natural hosts	Symptoms in lisianthus	References
Cucumber mosaic virus (CMV)	Brazil, Israel, Japan, the ROK, the USA	>80 aphid species including *Aphis gossypii* and *Myzus persicae* and by *seed*	A very large number of vegetable, ornamental, and weed species	Leaf mosaic and distortion, stunting, flower color-breaking, and malformation	Garcia-Arenal and Palukaitis (2008), Gera and Cohen (2007), Hahm et al. (1998), Providenti (1985), and Rivas et al. (2000)
Tobacco streak virus (TSV)	Brazil	*Frankliniella occidentalis*, *F. schultzei* *Microcephalothrips abdominalis*, *Thrips parvispinus*, *T. tabaci*, and by *seed*	A wide range of vegetables, ornamentals, and weed species	Irregularly shaped, necrotic streaks or ring spots on leaves; flower size reduction and premature senescence	de Freitas et al. (1996a), Kaiser et al. (1982), and Klose et al. (1996)

viruses in this genus have been detected in lisianthus including: *Chrysanthemum stem necrosis virus* (CSNV), *Groundnut ringspot virus* (GRSV), *Impatiens necrotic spot virus* (INSV), *Iris yellow spot virus* (IYSV), *Lisianthus necrotic ringspot virus* (LNRV), *Tomato chlorotic spot virus* (TCSV), and *Tomato spotted wilt virus* (TSWV) (Alexandre et al. 1999; de Freitas et al. 1996b; Fujinaga et al. 2006; Kritzman et al. 2000; McGovern unpublished data; McGovern et al. 1997b; Momonoi et al. 2011; Mumford et al. 2008; Shimomoto et al. 2014; Wolcan et al. 1996). Alexandre et al. (1999) found that three lisianthus plants in Brazil were co-infected with either CSNV + TCSV + GRSV or those three viruses + TSWV. The geographic occurrence of these viruses is indicated in Table 2.

CSNV was observed at an incidence of 10% in a commercial greenhouse in Brazil (Duarte et al. 2014). A very low incidence (<0.1%) of INSV occurred in commercial field in the USA (Florida) (McGovern et al. 1997b), while INSV and IYSV were observed at incidences of 42.9% and 17.1%, respectively, in a survey of greenhouses in Japan (Nagano Prefecture) (Fujinaga et al. 2006). The combined incidence of INSV and TSWV was estimated at 30–40% in two commercial lisianthus fields in the USA (California) (McGovern unpublished data).

Symptoms/signs. The range of symptoms caused by tospoviruses in lisianthus is presented in Table 2.

Biology and epidemiology. The tospoviruses are unusual plant viruses in that they are closely related to viruses that infect animals including insects; they are vectored in a circulative and propagative manner (they enter and replicate within the insect's hemolymph) by a number of thrips species (Thysanoptera: Thripidae) Table 2. Only the first and early second larval stages are able to acquire tospoviruses, and only immature thrips that acquire these viruses or adults derived from such immatures are vectors. Adult thrips remain viruliferous for life; but tospoviruses are not transovarial. Tospoviruses may be disseminated over long distances in vegetatively propagated plant material; seed infection has not been reported. Therefore, thrips population dynamics and the proximity of infected reservoir plants are the determining factors in *Tospovirus* outbreaks. Zen et al. (2008) detected IYSV in an onion field near lisianthus greenhouses in Japan and hypothesized that viruliferous *T. tabaci* dispersed after onion harvests spread the virus to lisianthus. Similarly, Kritzman et al. (2000) suggested that the source of IYSV outbreaks in lisianthus in Israel was infected onion crops.

Management

- ***Cultural practices*** – These practices include the use of virus-free transplants and trap plants/crops, nonhost barrier crops, and exclusion/repellence of thrips by fine mesh screening or reflective mulches and covers. In field trials conducted in the USA (California), reflective mulches and plant covers, alone and in combination, reduced the incidence of *F. occidentalis* in chrysanthemum (*Chrysanthemum morifolium*), golden rod (*Solidago* sp.), and roses (*Rosa* x *hybrid*) (Newman

and Robb 2010); however, reflective mulch effectiveness decreased with crop growth.

- ***Sanitation*** – Removal of weeds and other host plants of tospoviruses and their vectors from greenhouses and the surrounding area is essential. Weed removal is often impractical for controlling tospoviruses with large host ranges such as TSWV and INSV in field-grown crops. However, growing new crops near existing crops infected by these viruses should always be avoided. Rouging infected plants should be rapidly carried out in a manner that will prevent further virus spread by thrips such as by bagging or insecticide application before removal.
- ***Scouting*** – Monitoring thrips populations should be routinely done in the greenhouse and field through the use of yellow or blue sticky cards and/or tapping foliage over white paper. Virus indicator plants such as petunia (*Petunia* x *hybrida*) can be used in greenhouses to assess tospovirus inoculum pressure (Allen and Matteoni 1991).
- ***Insecticides*** – Resistance management of the limited number of insecticides effective against the two major tospovirus vectors, *F. occidentalis* and *T. tabaci*, is a significant challenge (Martin et al. 2003; Reitz and Funderburk 2012; Robb et al. 1995). Therefore, insecticides cannot be viewed as a stand-alone treatment for either thrips or virus, even if resistance management regimes such as rotation and limited usage are rigorously maintained. On the other hand, the incidence of necrotic ring spot disease in greenhouse-grown lisianthus in Japan caused by IYSV was decreased when applications of the insecticide acephate were timed with the emergence of viruliferous *T. tabaci* (Zen et al. 2010).
- ***Biological control*** – Biological control should be used preventively and not when thrips populations are high. Commonly used biocontrols for thrips management in greenhouses include predatory mites such as *Neoseiulus cucumeris* (formerly known as *Amblyseius cucumeris*) (Mesostigmata: Phytoseiidae), predatory bugs, *Orius* spp. (*Hemiptera*: Anthocoridae), and entomopathogenic nematodes such as *Steinernema feltiae* (*Rhabditida*: Steinernematidae) and *Thripinema nicklewoodi* (*Tylenchida*: Allantonematidae).

  In a greenhouse experiment in the UK, *N. cucumeris* was effective in controlling *F. occidentalis* on impatiens and decreased the spread and severity of TSWV (Bennison et al. 2001). Weekly foliar applications of *S. feltiae* reaching the growing medium reduced *F. occidentalis* populations, delayed TSWV infection, and reduced viral symptoms in greenhouse-grown chrysanthemums in the UK (Bennison et al. 2007). Infection of *F. occidentalis* by *T. nicklewoodi* reduced feeding, reproduction, and TSWV transmission by the insect in chrysanthemum in the USA (Texas) (Arthurs and Heinz 2003).
- ***Resistance*** – Partial resistance to TSWV was identified in chrysanthemum cvs. (van de Wetering et al. 1999); similar tospovirus screening in lisianthus could provide a useful virus management strategy and starting point for a traditional resistance breeding program in this crop. Using *Agrobacterium*-mediated transformation, Sherman et al. (1998) produced lines of the chrysanthemum cv. Polaris

Table 2 Tospoviruses infecting lisianthus

Viruses	Occurrence in lisianthus	Transmission[a]	Other natural hosts	Symptoms in lisianthus	References
Chrysanthemum stem necrosis virus (CSNV)	Brazil, Japan	*Frankliniella occidentalis*, *F. schultzei*	China aster (*Callistephus chinensis*), chrysanthemum (*Chrysanthemum morifolium*), tomato,	Necrotic streaks in stems and foliar necrosis (Fig. 14)	Alexandre et al. (1999), Momonoi et al. (2011), and Nagata et al. (1998)
Groundnut ringspot virus (GRSV)	Brazil	*F. gemina*, *F. intonsa*, *F. occidentalis*, *F. schultzei*	Coriander (*Coriandrum sativum*), cucumber (*Cucumis sativus*), eggplant (*S. melongena*), peanut (*Arachis hypogea*), pepper (*Capsicum annum*), tomatillo (*Physalis philadelphica*), tomato	Not determined – GRSV detected in mixed infections with other tospoviruses	Alexandre et al. (1999), Lima et al. (1999), Spadotti et al. (2014), and Webster et al. (2015)
Impatiens necrotic spot virus (INSV)	Japan, USA	*F. fusca*, *F. intonsa*, *F. occidentalis*	At least 300 species in 85 different plant families	Stunting, circular to irregularly shaped yellow-tan necrotic leaf spots/ring spots, and distortion of leaves and shoots, wilting, and plant death (Fig. 15)	Fujinaga et al. (2006), McGovern et al. (1997b), and Pappu et al. (2009)
Iris yellow spot virus (IYSV)	Israel, Japan, UK	*F. fusca*, *T. tabaci*	Amaryllis (*Hippeastrum hybridum*), iris (*Iris hollandica*), onion and related species, Peruvian lily (*Alstroemeria* sp.), spiny sow thistle (*Sonchus asper*), and 11 taxonomically diverse weed species	Necrotic spots and ringspots in leaves, systemic necrosis (Fig. 16)	Bag et al. (2014), Derks and Lemmers (1996), Doi et al. (2003), Kritzman et al. (2000), Mumford et al. (2008), Nischwitz et al. (2012), and Okuda and Hanada (2001)

Lisianthus necrotic ringspot virus (LNRV)	Japan	Not determined	Not determined	Necrotic spots and ring spots in leaves (Fig. 17)	Shimomoto et al. (2014) and Zen et al. (2008)
Tomato chlorotic spot virus (TCSV)	Brazil	*F. intonsa*, *F. occidentalis*, *F. schultzei*	15 species in six plant families including impatiens (*Impatiens* spp.); epidendrum orchid (*Epidendrum* spp.); phalaenopsis orchid (*Phalaenopsis* spp.)	Ring spots, necrosis, leaf deformation, stunting	Alexandre et al. (1999)and Polston et al. (2013)
Tomato spotted wilt virus (TSWV)	Argentina, Brazil, New Zealand, Spain, the USA	*F. bispinosa*, *F. cephalica*, *F. fusca. F. gemina*, *F. intonsa*, *F. occidentalis*, *F. schultzei*, *T. palmi*, *T. setosus*, *T. tabaci*	>900 species in >90 monocotyledonous and dicotyledonous plant families	Necrotic spots and ring spots in leaves (Fig. 18)	Alexandre et al. (1999), de Freitas et al. (1996b), McGovern (unpublished data), Melgares de Aquilar Cormenzana (1996), Pappu et al. (2009), Veerakone et al. (2015), and Wolcan et al. (1996)

[a]Riley et al. (2011) and Srinivasan et al. (2012)



Fig. 14 Symptoms of CSNV: stem and foliar necrosis (Momonoi et al. 2011)



Fig. 15 Symptoms of INSV: distortion and yellow spots in newly developing leaves (*left*) and yellow-tan and ring spots in mature leaf (*right*) (R.J. McGovern © 2017. All Rights Reserved.)



Fig. 16 Symptoms of IYSV: stem necrosis (*left*) and systemic necrosis (*right*) (Srinivasan et al. 2011)



Fig. 17 Symptoms of LNRV (Shimomoto et al. 2014)



Fig. 18 Symptoms of TSWV: yellow-tan ring spots (Florida Department of Agriculture and Consumer Services © 2017. All Rights Reserved.)

resistant to TSWV. Peng et al. (2014) produced transgenic tobacco plants (*Nicotiana benthamiana* Domin.) that were resistant to multiple tospoviruses including GNRV, INSV, IYSV, and TSWV. A method for transformation of lisianthus was developed and the TSWV nucleoprotein was introduced, suggesting that genetic engineering of this crop for tospovirus resistance is feasible (Semeria et al. 1996).

- *Integrative strategies* – Zen et al. (2010) reported that the combined use of acephate with reflective screening was very effective in reducing IYSV in lisianthus in greenhouses in Japan. The combination of UV-reflective mulch, the systemic acquired resistance activator acibenzolar-*S*-methyl, and insecticides (methamidophos, spinosad) was very effective in reducing TSWV incidence in field-grown tomato in the USA (Florida) (Momol et al. 2004).

  Additional information on integrated disease management of viruses may be found in the introductory (▶ Chap. 4, "Insect Management for Disease Control in Florists' Crops").

4.3 Geminiviridae Tomato Yellow Leaf Curl Virus (TYLCV, Genus *Begomovirus*)

Geographic occurrence and impact. The virus has become a limiting factor for lisianthus production in Israel; TYLCV incidences of near 100% have occurred in



Fig. 19 Symptoms of TYLCV: stunting, yellowing and upward curl of leaves, and flower bleaching in infected plant (*left*); healthy plant (*right*) (R.J. McGovern © 2017. All Rights Reserved.)

commercial facilities (Cohen et al. 1995). An incidence of 5% was reported in a greenhouse in the ROK (Kil et al. 2014).

Two other *B. tabaci*-vectored begomoviruses, *Ageratum yellow vein virus* and *Papaya leaf curl guangdong virus* were reported to infect lisianthus in Taiwan (Cheng et al. 2005; Chen et al. 2016).

Symptoms/signs. TYLCV symptoms in lisianthus include stunting, upward curling, veinal swelling (on the lower leaf surface), and yellowing of leaves, flower bleaching, and failure to flower (Fig. 19).

Biology and epidemiology. TYLCV comprises a virus complex that has become a worldwide limiting factor for production of tomato (*Solanum lycopersicum*). The virus is transmitted circulatively and non-propagatively by the whitefly *Bemisia tabaci* (Gennadius) (Hemiptera: Aleyrodidae). Once TYLCV is acquired, the whitefly remains viruliferous for life. Besides lisianthus and tomato, other natural hosts of TYLCV include a number of solanaceous plants [black nightshade (*Solanum nigrum*), chili pepper (*Capsicum chinense*), Jimsonweed (*Datura stramonium*), petunia (*Petunia* x *hybrida*), sweet pepper (*C. annuum*), and tobacco (*Nicotiana tabacum*)] and common bean (*Phaseolus vulgaris*) (Díaz-Pendón et al. 2010).

Management. Whitefly management through physical, chemical, and biological means and avoidance of locating lisianthus transplant and production facilities near established TYLCV-infected crops are essential for management of the virus.

Additional information on integrated disease management of viruses may be found in the introductory (► Chap. 4, "Insect Management for Disease Control in Florists' Crops").

4.4 Potyviridae

Geographic occurrence and impact. Viruses in the genus *Potyvirus* infect most cultivated plants and weeds. A number of potyviruses cause disease in lisianthus including: *Bean yellow mosaic virus* (BYMV), *Pepper veinal mottle virus* (PVMV), *Turnip mosaic virus* (TuMV), and *Watermelon mosaic virus* (WMV). The geographic occurrence of these viruses is indicated in Table 3.

Symptoms/signs. The range of symptoms caused by potyviruses in lisianthus is presented in Table 3.

Biology and epidemiology. Potyviruses are spread by aphids (Hemiptera: Aphididae) in a noncirculative (nonpersistent) manner. It was suggested that other ornamentals, such as freesia (*Freesia alba*) or gladiolus, known to be hosts of BYMV, may have provided a reservoir for outbreaks of the virus in lisianthus (Lisa and Dellavalle 1987).

Management. Uga et al. (2004) demonstrated that dwarf lisianthus plants that had been infected with an attenuated strain of BYMV (B-33) were protected from subsequent infection by a virulent isolate of the virus. Refer to the Bromoviridae *Management* section of this chapter and introductory (► Chap. 4, "Insect Management for Disease Control in Florists' Crops").

4.5 Secoviridae Broad Bean Wilt Virus (BBWV, Genus *Fabavirus*)

Geographic occurrence and impact. BBWV was detected in lisianthus in Japan (Nagano Prefecture) at an incidence of 5.7% (Fujinaga et al. 2006) and at 10% in combination with CMV in alpine production areas of the ROK (Hahm et al. 1998).

Symptoms/signs. Mosaic

Biology and epidemiology. BBWV can be found worldwide and infects many economically important vegetable and ornamental crops. It is transmitted in a noncirculative manner by a number of aphids including *A. gossypii* and *M. persicae* (Belliure et al. 2009).

Management. Refer to the Bromoviridae *Management* section of this chapter and introductory (► Chap. 4, "Insect Management for Disease Control in Florists' Crops").

Table 3 Potyviruses infecting lisianthus

Viruses	Occurrence in lisianthus	Transmission	Other natural hosts	Symptoms in lisianthus	References
Bean yellow mosaic virus (BYMV)	Brazil, Israel, Italy	At least 19 aphid species with *M. persicae* being the most effective	14 plant families including vegetables (legumes) and ornamentals (freesia, gladiolus, etc.)	Stunting, mosaic and leaf curl, flower color-breaking	Alexandre et al. (2005), Bos (2010), Gera and Cohen (2007), Lisa and Dellavalle (1987), and Swenson (1957)
Pepper veinal mottle virus (PVMV)	Taiwan	*Aphis gossypii*, *A. spiraecola*, *Myzus persicae*, *Toxoptera citricida*	Mainly infects cultivated and weedy solanaceous plants (black nightshade, pepper, petunia, tomato, etc.)	Stunting, yellow blotches, and ring spots on leaves	Cheng et al. (2009)and ICTVdB Management (2006a)
Turnip mosaic virus (TuMV)	Brazil, Taiwan	40–50 aphid species especially *M. persicae* and *Brevicoryne brassicae*	Chinese mustard (*Brassica campestris* ssp. *chinensis*), Chinese white cabbage (*B. campestris* ssp. *chinese var communis*), radish (*Raphanus sativus*), rape (*B. campestrisi*) and mustard (*B. juncea*), etc.	Stunting, systemic yellow spotting	Alexandre et al. (2005), Chao et al. (2000), and ICTVdB Management (2006b)
Watermelon mosaic virus (WMV)	Japan	>35 aphid species	>170 species in 26 mono- and dicotyledonous families including cultivated plants and weeds	Chlorotic or whitish ring spots on leaves	Inoue and Kasuyama (2001)and Lecoq and Desbiez (2008)

4.6 Tombusviridae

Geographic occurrence and impact. Three members of the Tombusviridae infect lisianthus including *Carnation mottle virus* (CarMV, genus *Carmovirus*), *Lisianthus necrosis virus* [LNV (a strain of *Eggplant mottled crinkle virus*), genus *Necrovirus*], and *Moroccan pepper virus* (MPV, genus *Tombusvirus*). The geographic occurrence of these viruses is indicated in Table 4. CarMV was detected at an incidence of 20% in field-grown lisianthus in Taiwan (Chen et al. 2011). About 85% of the lisianthus surveyed in greenhouses in the Pakdasht region of Iran exhibited symptoms of MPV infection (Beikzadeh et al. 2011).

Symptoms/signs. The range of symptoms caused by the Tombusviridae in lisianthus is presented in Table 4.

Biology and epidemiology. The Tombusviridae contains virus genera that persist in, and are spread by, soil, plant debris, water, beetles, chytrid fungi such as *Olpidium brassicae*, and mechanically. It has been suggested that plants may become infected when their roots come in contact with sloughed-off virus-infected root tissue. Tombusviridae generally have a limited host range compared to other virus groups infecting lisianthus. The mode of infection of lisianthus by mechanically transmissible viruses with no known vectors in the outbreaks of CarMV and MPV mentioned above remains unexplained. It is possible that these viruses were spread by cutting tools contaminated by use in another infected cut-flower crop or through the action of an unknown vector.

Management. Refer to the introductory (▶ Chap. 4, "Insect Management for Disease Control in Florists' Crops").

4.7 Virgaviridae

Geographic occurrence and impact. Three members of this virus group infect lisianthus: *Tobacco mosaic virus* (TMV, genus *Tobamovirus*), *Tomato mosaic virus* (ToMV, genus *Tobamovirus*), and *Pepper ringspot virus* (PepRSV, genus *Tobravirus*). The geographic occurrence of these viruses is indicated in Table 5.

Symptoms/signs. The symptoms of these viruses are presented in Table 5.

Biology and epidemiology. The Virgaviridae is a relatively new family of six rod-shaped plant virus genera including *Tobamovirus* and *Tobravirus* (Adams and Antoniw 2009). These viruses are transmitted mechanically and in some cases by seed, are resistant to degradation, and may persist in the soil.

Table 4 Tombusviridae infecting lisianthus

Viruses	Occurrence in lisianthus	Transmission	Other natural hosts	Symptoms in lisianthus	References
Carnation mottle virus (CarMV)	Taiwan	Mechanical, especially through asexual plant propagation	30 species in 15 plant families, including ornamentals such as calla lily (*Zantedeschia* spp.), carnation, geranium (*Pelargonium hortulanum*), hibiscus (*Hibiscus* sp.), narcissus (*Narcissus* sp.)	Systemic necrotic spots (Fig. 20)	Chen et al. (2011)and Qu and Morris (2008)
Lisianthus necrosis virus (LNV)	Japan, Taiwan	Mechanical and by the chytrid fungus *Olpidium* sp.	Calla lily, carnation, and a number artificially inoculated ornamentals such as petunia and zinnia (*Zinnia elegans*)	Initially yellow spots appear on upper leaves followed by systemic necrotic leaf lesions, ring spots and distortion, tip necrosis, and flower color-breaking (Fig. 21)	Chang et al. (2007), Chen and Hsu (2002), Chen et al. (2000), and Iwaki et al. (1987)
Moroccan pepper virus (MPV)	Iran, Japan	Mechanical	Jimsonweed, lettuce (*Lactuca sativa*), pelargonium (*Pelargonium zonale*), pepper, tomato	Stunting, necrotic leaf spots, distortion, and necrosis of leaves and stems (Fig. 22)	Beikzadeh et al. (2011), Fischer and Lockhart (1977), Okhi et al. (2014), Vetten and Koenig (1983), and Wintermantel and Hladky (2013)

Management. Use virus-free seed. Promptly rogue infected plants and have workers disinfest their hands before resuming regular duties. Disinfest cutting tools between plants. Areas where a high virus incidence has been observed should be scheduled last for cultural activities.



Fig. 20 Symptoms of CarMV: systemic necrotic spots (Y.-K Chen © 2017. All Rights Reserved.)



Fig. 21 Symptoms of LNV: systemic yellow turning necrotic leaf lesions and ring spots, leaf distortion, and flower color-breaking (Y.-K Chen © 2017. All Rights Reserved.)

5 Nematode Diseases

5.1 Root Knot (*Meloidogyne* spp.)

Geographic occurrence and impact. Root knot in lisianthus has been reported in Italy, Israel, and the USA (California and Florida) (Elad et al. 2014; Russo and di Vito 2005; Schochow et al. 2004; Wang and McSorley 2004).

Symptoms/signs. Yellowing of foliage, stunting, flower delay and number reduction, and root galls (Fig. 23).



Fig. 22 Symptoms of MPV: necrotic leaf spots (*left*); distortion and necrosis of leaves and stems (*right*) (Ohki et al. 2014)

Table 5 Virgaviridae infecting lisianthus

Viruses	Occurrence in lisianthus	Transmission	Other natural hosts	Symptoms in lisianthus	References
Tobacco mosaic virus (TMV)	Israel	Mechanical, seed	Very broad host range	Stunting, leaf mosaic	Gera and Cohen (2007)
Tomato mosaic virus (ToMV)	Italy, Taiwan	Mechanical, seed	Very broad host range	Stunting, mosaic, and necrosis of leaves	Jan et al. (2003) and Lisa and Gera (1995)
Pepper ringspot virus (PepRSV)	Brazil	Mechanical	Some vegetable species and ornamentals such as *Gerbera jamesonii*, *Gloxinia sylvatica*	Stunting, mosaic, and necrotic line patterns in leaves, flower color-breaking	Rivas et al. (2000)

Biology and epidemiology. Root knot in lisianthus can be caused by three species of *Meloidogyne* (Tylenchida: Heteroderidae): *M. hapla*, *M. incognita*, and *M. javanica*. Root-knot nematodes begin as eggs in root tissue or the soil and pass through four juvenile stages (J1–J4) before reaching maturity. The J2 stage is the only one capable



Fig. 23 Symptoms of root-knot nematode (R.J. McGovern © 2017. All Rights Reserved.)

of infecting roots, where they produce giant cells in the host in which they feed, enlarge, and undergo their final developmental stages. The mature females become globose and produce eggs, while the adult males migrate from the host and become free-living. Root-knot nematodes are primarily spread by water, equipment contaminated with infested soil, and in infected plant material in the case of vegetatively propagated crops.

Based on reproduction on cotton and/or tobacco, *M. incognita* was divided into four races (Taylor and Sasser 1978). Wang and McSorley (2004) reported that lisianthus "Avila Rose Rim" and "Echo Pink" were relatively poor hosts of *M. incognita* races 1 and 2. Schochow et al. (2004) found that lisianthus was a better host for *M. javanica* than for *M. incognita* and a poor host for *M. hapla*; but that all three species reduced flower number per plant.

Management. Prevention of nematode dissemination through disinfestation of equipment is critical. Disinfestation of soil in florists' crops production sites has utilized fumigation, chemical drenches, and heat treatment by steam or soil solarization. Refer to ▶ Chap. 8, "Soil/Media Disinfestation for Management of Florists' Crops Diseases."

Additional Nematode Pathogens. The following nematode pathogen of lisianthus has also been reported:

Needle nematode (***Longidorus* spp.**) (Israel, Elad et al. 2014)

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Diseases of Orchid

21

Prasartporn Smitamana and Robert J. McGovern

Abstract

Orchids are monocotyledonous plants that belong to the Orchidaceae family which has a diverse range of habitats from tropical to temperate zones. Orchids grow at different elevations from sea level to the very high mountainous level of the Himalayas. Due to great variation, orchids can be classified in many genera and can be infected with many of the same fungi, bacteria, viruses, and nematodes as other plants. In this chapter, economically important diseases of orchids and their management are described.

Keywords

Botrytis • *Phytophthora* • *Pythium* • *Fusarium* • *Rhizoctonia* • *Sclerotium* • *Acidovorax* • *Dickeya* • *Burholderia* • Viruses • Foliar Nematodes

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© Springer International Publishing AG 2018

R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_21

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1 Introduction

Orchid belongs to the Orchidaceae which is a highly diverse family and can be found worldwide, but most species are found in the tropics, especially in the tropical mountains (Dressler 1993). Orchidaceae is possibly the largest family of the flowering plants and consists of 899 genera that include 71,391 species, but only 27,135 are accepted species names (The Plant List 2013).

Among the orchids, 73% are epiphytes (Atwood 1986) and show stereotypical flower structure and remarkably diverse plant structure (Benzing 1986a, b). Orchids are economically important ornamental crops as potted plants and cut flowers but also provide raw materials for the fragrance and pharmaceutical industries. In some countries, orchids serve as the major source of income for growers. The commercial production of orchid began in natural habitats but has been adapted for large-scale industrial production. This change has made the environment conducive for the outbreak of many diseases. Due to vegetative propagation, the planting materials are the major source of inoculum, and pathogens are easily distributed, especially viruses. Major diseases of orchids and their control measures are listed below.

2 Fungal and Fungus-Like Diseases

2.1 Black Rot (*Phytophthora* and *Pythium* spp.)

Geographic occurrence and impact. Black rot is a common disease found in all orchid genera, especially after heavy rainfall or when in highly humid environments for extended periods, such as occur in poorly ventilated, crowded collections. Black rot is caused by one or both pathogens belonging to *Phytophthora* and *Pythium* spp. *Fusarium* can frequently occur as a secondary pathogen. *Pythium ultimum*, *Phytophthora cactorum*, and *Phytophthora palmivora* are commonly reported as the

major pathogens (Hine 1962; Burnett 1974; Uchida 1994; Uchida and Aragaki 1991a; Orlikowski and Szkuta 2006; Cating et al. 2013). *Phytophthora multivesiculata* was found in *Cymbidium* in New Zealand (Ilieva et al. 1998; Hill 2004).

Black rot is a common disease in all nurseries worldwide, especially in the tropics and subtropics, including Australia, Bulgaria, China, New Zealand, Poland, Taiwan, and the USA (Hawaii) (Burnett 1974; Chen and Hsieh 1978; Hall 1989; Duff 1993; Uchida 1994; Ilieva et al. 1998; Hill 2004; Orlikowski and Szkuta 2006; Tao et al. 2011).

Symptoms/signs. In seedlings and young plantlets, water soaked lesions and discoloration occurs at the base of the plant and roots during periods of high humidity. This condition can lead to damping off.

In ***monopodial type orchids***, e.g., *Vanda*, *Phalaenopsis*, and *Rhynchostylis*, the disease usually starts on new leaves as brown to black lesions (Figs. 1a, b and 2b).



Fig. 1 Black rot lesions surrounded by a yellowish region on *Vanda* (**a**) and *Rhyncostylis* (**b**) orchids; infected root velamen has dried out (**b**) (P. Smitamana © 2017. All Rights Reserved.)



Fig. 2 Black rot of *Catteleya* pseudobulb (**a**) and *Vanda* leaves (**b**) on which the fungal mycelia are visible under high moisture conditions (**a**, P. Smitamana © 2017. All Rights Reserved; **b**, Kamjaipai 1983)

The pathogens rapidly invade downward into the stem and roots. The infected shoot can easily be pulled out and, if unattended, the whole plant will die.

In the ***sympodial type orchids***, e.g., *Cattleya*, *Dendrobium*, and *Oncidium*, a light yellow-brown discoloration on the pseudobulbs is observed. After the disease progresses, softening and rotting of the pseudobulbs which turn brown to black occurs (Fig. 2a). The infected roots become darkly discolored as the velamen rots.

Leaf symptoms first appear on the underside as small, water-soaked, irregular spots that are yellowish brown in color, then turn brown or black with a yellowish margin. Under high temperature and humidity, the lesions rapidly enlarge and become soft with ooze if pressed. If the humidity decreases, the lesions dry and turn black into which other pathogens can easily invade. Pathogens can also infect the leaves via wounds.

In general, root symptoms initially appear as brown to black rot of root tips; the stem and/or pseudobulbs may rapidly rot. Infected roots can also be stunted, lose the velamen, and dry out. In *Cymbidium*, a dry rot of leaves can occur that has alternating horizontal light- and dark-brown zebra-like stripes. The pseudobulb tissue usually becomes wet and brown-black (Ilieva et al. 1998).

Biology and epidemiology. *Phytophthora* and *Pythium* are fungus-like, but not true fungi, and are favored by high humidity. Both species spread rapidly if free water is available giving rise to zoospores that swim freely to, penetrate, and infect plant tissue (Agrios 2005). Following infection, mycelia spread rapidly through the infected tissues. The lesions rapidly expand and exhibit brown-black necrotic regions. Motile zoospores produced in the infected lesions are spread easily in irrigation water and are splash-dispersed on uninfected tissue during watering (Uchida 1994).

Management

- ***Cultural practices*** – Carefully check newly acquired plants to be sure that they are free from disease. Separate new stock from other plants and monitor them for disease symptoms for least 6 weeks before moving them into the nursery. Elevating the plants 60–90 cm above the ground to avoid splashing from the soil below or keeping them on a solid surface can also help prevent infections. Growers should maintain good ventilation through adequate plant spacing to reduce duration of leaf wetness.
- ***Sanitation*** – If early symptoms of black rot are found, immediately excise infected tissue with a clean knife and discard it in order to prevent the spread of the disease on that plant or to other plants. Application of a fungicide (fosetyl-al, mefenoxam, etc.) may be effective when used immediately after. Use only clean water for watering orchids. Deep well water is safer than surface water unless surface water is disinfested. Discard or burn severely infected plants to prevent the spread of the disease.
- ***Fungicides and biocontrols*** – Use suitable fungicides (fosetyl-al, mefenoxam, promocarb hydrochloride, trifloxystrobin, etc.) as a preventative measure, particularly during hot humid periods. The highest rate of the harpin protein, which induces systemic acquired resistance, significantly reduced black rot in *Vanda*

caused by *P. palmivora* compared to the control; *Bacillus subtilis* + *Trichoderma harzianum* and etridiazole + quintozene were ineffective (Maketon et al. 2015). Growers should follow all label instructions.

2.2 Shoot Rot or Top Rot [*Phytophthora nicotianae* (syn. *Phytophthora parasitica*); *Phytophthora cactorum*]

Geographic occurrence and impact. This is a common disease found mostly on *Vanda* (upper stem) and *Cattleya* (leaf shoot) and is favored by high humidity and temperature, and poor ventilation due to high plant densities. This disease is caused by *Phytophthora nicotianae*, which can cause the death of the whole plant (*Vanda*); or in the case of *Cattleya* caused by *Phytophthora cactorum*, which causes rot of the shoots, leaves, pseudobulbs, rhizomes, and flower buds.

Like black rot, shoot rot is commonly found in humid and high temperature environments especially in the tropical and subtropical zones: Thailand, USA (Florida, Hawaii), Poland, and Australia (Daly et al. 2013; Kamjaipai 1983; McMillan et al. 2009; Orlikowski and Szkuta 2006; Uchida 1994; Uchida and Aragaki 1991a, b).

Symptoms/signs. A symptom of top rot includes the death of new leaves, which turn dark brown. This discoloration advances down the stem similar to the symptom caused by black rot. This disease may also start at the base of the stem and spread upward resulting in the same dark brown stem discoloration. Shoot rot was detected on *Cattleya* orchids in Darwin, Australia, and in USA (Florida) causing the rapid death of new side shoots, turning them almost black. The pathogen will spread back along the rhizomes to the next shoot causing the same symptoms. This disease has been reported on only *Cattleya* and *Vanda* but may affect others.

Biology and epidemiology. As stated before, *Phytophthora* is fungus-like and its infection and spread are favored by wet conditions. *Phytophthora cactorum* is a homothallic oomycete that produces a sexual spore, an oospore, for survival under unfavorable environments. In addition, this fungus also asexually produces survival spores called chlamydospores as well as sporangia. The pathogen spreads by means of the pear or lemon shaped motile zoospores produced by either oospores or sporangia under wet conditions. After encysting and germination, zoospores can penetrate the plant directly or enter through wounds to cause infection.

Management

- ***Sanitation and fungicides*** – Immediately cut the infected top with a clean knife, and discard severely infected plants to prevent the spread of the pathogen, and spray the plants with a fungicide (fosetyl-al, mefenoxam, etc.). Use suitable fungicides (see Sect. 2.1 above) as a preventative measure, particularly during hot humid periods, following label instructions. Improve ventilation to reduce leaf wetness.

- ***Biocontrol*** – Certain biocontrol products containing *Streptomyces lydicus*, *Bacillus subtilis*, *Trichoderma* species, and *Gliocladium virens* applied prior to the disease occurring in the growing area have been reported to help prevent some soilborne and aerial diseases (Bottom 2016). Spraying infected plants with a spore suspension of *Trichoderma harzianum* or *T. viride*, or in the case of *Vanda* directly injecting the solution into the infected shoot, may be helpful (Smitamana, unpublished data).

2.3 Botrytis Spot (*Botrytis cinerea*)

Geographic occurrence and impact. Botrytis spot is a fungal disease affecting many types of orchids, mostly during cool, damp weather and in nurseries with poor air circulation. *Phalaenopsis* and *Cattleya* are particularly susceptible to *Botrytis cinerea* infection, but the disease may be found in a wide range of orchid genera. This disease has been reported in the tropics and the temperate regions including the USA (Florida, Hawaii) and Thailand (Jones 2003; Kamjaipai 1983; Uchida and Aragaki 1991b).

Symptoms/signs. Older flowers are highly susceptible to infection. Brown necrotic or pale spots on orchid flowers are typical symptoms. The spots may enlarge and increase in number as the infection progresses and may be surrounded by a yellowish or pale pink margin depending on the background color of the flowers. Under cool and high moisture conditions, fungal mycelial webbing may be visible.

Biology and epidemiology. *Botrytis cinerea* is an airborne plant pathogen commonly found in nurseries. Hyaline conidia of the fungus are produced on grey, branching conidiophores. Sclerotia, highly resistant survival structures, can be produced on dying infected tissue. In the temperate zone, *B. cinerea* overwinters in dead and dying plant material and begins producing and dispersing spores during cool, damp weather in the spring and autumn. Spores are distributed by wind, rain, irrigation water, or any mechanical action. Temperatures in the range of 18–25 °C/64–77 °F and wet plant surfaces or at least 92% RH are conducive factors for infection, and the pathogen can quickly proliferate, infecting the surrounding healthy plant tissue in less than 14 h (Agrios 2005; Sumbali 2005).

Management

- ***Cultural practices*** – Growers should avoid wetting the flowers as much as possible. In tropical nurseries, the growers always spray or water the whole nonflowering plants early in the morning to make sure that the plants will be dry in the late afternoon or by nightfall. Water that remains on petals or leaves after a rain or watering promotes fungal growth. Avoid damp environments and poor air circulation that promote pathogen growth. Facilitate good air circulation through proper plant spacing and decrease the humidity during cool, damp

weather by heating and venting greenhouses in temperate climates, especially when plants are in the blooming stage.

- ***Sanitation*** – Remove any plant debris, fallen flowers, leaves, and infected tissue from the growing area in closed containers to reduce the infection potential and the possibility of spreading the fungus. Burning or burying all affected plant tissue is recommended. Eliminate unwanted residual ornamental crops and weeds in the growing area. Make sure that newly introduced plants are free of any disease; normally keep them in an isolated area for at least 2 weeks before bringing them into the nursery.
- ***Fungicides*** – Spray preventively with fungicides such as chlorothalonil, thiophanate methyl, iprodione, or vinclozolin. Resistance to thiophanate methyl and vinclozolin has been widely reported in *B. cinerea*; therefore, rotation of these fungicides with those of different modes of action is essential.

2.4 Petal Blight (*Curvularia eragrostidis, Alternaria alternata*)

Geographic occurrence and impact. This disease is found mostly in the *Dendrobium* hybrids whose flowers are most susceptible to the pathogens. Petal blight has been reported in Australia, Thailand, and the USA (Burnett 1957, 1965; Daly et al. 2013; Kamjaipai 1983).

Symptoms/signs. Small brown spots develop on the flower petals, then the spots enlarge and merge to form necrotic lesions; under high moisture conditions or after rainfall these spots will rot (Fig. 3a, b).

Biology and epidemiology. *Alternaria* and *Curvularia* belong to the fungal family Dermatiaceae which produces both dark hyphae and conidia. No organized fruiting bodies are formed, and the majority of these groups are saprophytic but some are plant pathogens (Sumbali 2005). Conidia of *Alternaria* are typically club or pear shaped and multicellular with both transverse and longitudinal cross walls. Conidia are easily detached and carried by air currents (Agrios 2005).



Fig. 3 Petal blight of *Dendrobium* flowers: water-soaked lesions turning brown or rust colored on white flower petals (**a**); and whitish yellow lesions on "Madame Pompadour" flowers (**b**). (Kamjaipai 1983)

Curvularia conidia are curved, clavate, broadly fusiform, obvoid, or pyriform with three or more transverse septa, pale or dark brown, with the end cells paler than the others (Ellis 1977). Both *Alternaria* and *Curvularia* can survive in plant debris, infected, or dry tissue, and the conidia are easily spread by air currents or water drops splashed off the orchid plants during overhead irrigation.

Management

- ***Cultural practices*** – Keep orchids in bloom under solid rooves in order to protect them from rain. Avoid damp environments caused by incorrect watering practices and poor air circulation that promote pathogen infection.
- ***Fungicides and biocontrols*** – The highest rate of the harpin protein, which induces systemic acquired resistance, significantly reduced rust spot in *Dendrobium* flowers caused by *Curvularia lunata*, compared to the control. Formulations of *Bacillus subtilis* + *Trichoderma harzianum* and mancozeb were intermediate in effectiveness (Maketon et al. 2015).

2.5 Sclerotium Stem Rot (*Sclerotium rolfsii*)

Geographic occurrence and impact. This disease is found mostly in *Ascocenda*, *Brassidium*, *Cymbidium*, *Dendrobium*, *Neofinetia*, *Phaius*, *Paphiopedilum*, *Phalaenopsis*, and *Vanda* orchids. Stem rot is commonly found in the tropics especially in nurseries that do not have roofs to protect the foliage from rainfall. Pots with poor drainage, such as those used to grow *Paphiopedilum*, are conducive to the disease.

Stem rot has been reported in both the tropics and temperate zones including Australia, India, Korea, Thailand, and the USA (Florida) (Bag 2004a, b; Cating et al. 2009a; Cating et al. 2013; Daly et al. 2013; Han et al. 2012; Kamjaipai 1983; Tara et al. 2003).

Symptoms/signs. The symptoms of this disease usually start on the lower shoots, pseudobulbs, and leaves which turn yellow, grow poorly, and then rapidly collapse (Fig. 4a). Rotting of the stem (Fig. 4c), roots, and leaf tissue gradually occurs, and in severe infections the entire plant wilts and dies. White fluffy mats of fungal mycelia and small yellow cream to brown spherical, mustard seed-size bodies called sclerotia appear on infected tissue (Fig. 4d). On *Dendrobium* plants, the pathogen can infect the leaves and basal part of pseudobulbs on which white mycelia are found; or in certain cases, the plants can be infected from the top causing rapid collapse and rotting of shoot apices (Fig. 4b).

On *Phaius flavus* and *Paphiopedilum venustum* the disease may be found on the base of the pseudobulbs and collar region, respectively, and causes rot and leaf yellowing or collapse, which spreads upward, until the entire plant turns brown to black and dies. Diagnostically characteristic white mycelia and small sclerotia are found on the infected areas.



Fig. 4 Infected *Vanda* basal shoot with white mycelia and small sclerotia (**a**), young *Dendrobium* plant with severe leaf rot and white mycelia (**b**), severely infected *Vanda* showing dry rot of stem (**c**), and sclerotia on the infected stem and leaves (**d**) (Kamjaipai 1983)

Biology and epidemiology. *Sclerotium rolfsii* is characterized by small cream to dark brown sclerotia produced on septate, white fluffy mycelia with hyphal clamp connections (Sumbali 2005). Sclerotia can be spread by water and act as sources of infection. The fungus has a wide host range and is favored by a warm, moist environment. In the temperate zone, the pathogen can overwinter in the sclerotia and starts infecting new plants when the temperatures become warm. Sclerotia can survive in soil for many years.

Management. Managing *Sclerotium* stem rot is quite difficult because of the pathogen's wide host range and long survival in soil via sclerotia. Infected plants should be immediately destroyed, and an appropriate fungicide such as PCNB should be applied when the disease is observed. Using biocontrol agents, such as *Trichoderma* spp., as an alternative measure for preventing and controlling the pathogen is also recommended.

2.6 Black Leg/Dry Rot and Slow Decline (*Fusarium* spp. *Rhizoctonia solani*)

Geographic occurrence and impact. *Fusarium solani*, *F. oxysporum*, *F. subglutinans*, and *F. proliferatum* have been associated with the disease; in addition, *Rhizoctonia solani* has also been involved in some areas. Susceptible orchids include *Dendrobium*, *Cattleya*, *Phalaenopsis*, *Vanda*, *Rhyncostylis*, and *Vanilla*. Black leg, dry rot, and slow decline have been reported in the tropics and temperate zones including Australia, Brazil, Indonesia, Malaysia, Taiwan, Thailand, the USA (Florida) (Daly et al. 2013; Dehgahi et al. 2014; Kamjaipai 1983; Pinaria et al. 2010; Su et al. 2010; Pedroso-de-Moraes et al. 2011; Wedge and Elmer 2008). If uncontrolled, Fusarium rot can affect up to 100% of an orchid crop (Kawate and Sewake 2014). *Fusarium* is considered a quarantine pest.

Symptoms/signs. This disease is mostly found in orchids grown in damp environments or waterlogged pots especially after heat stress and heavy fertilization (Wedge and Elmer 2008). Plantlets after transferring from tissue culture vessels to community trays or pots are most susceptible to infection. Infected plantlets show water soaked root tips followed by leaf yellowing in whole plantlets. In mature plants, the symptoms are mostly observed after transplanting when the pathogens can easily infect the plants through wounds. Infected plants show discoloration of the root tips and a gradual dry rot. Pseudobulbs become spongy and discolored, successive new shoots tend to get smaller in height, and stem thickness is also reduced. The leaves will yellow and drop off, one by one, until none are left and the plant dies (Fig. 5).

Biology and epidemiology. *Fusarium* and *Rhizoctonia* are both soilborne pathogens. *Fusarium* at first produces colorless mycelia but with age the mycelia color will change to cream, pale yellow, pale pink, or purplish. *Fusarium* produce three kinds of asexual spores: microconidia which have 1–2 cells; curved macroconidia which have 3–5 cells, and 1–2 cell, thick-walled resistive chlamydospores (Agrios 2005). The microconidia and macroconidia are dispersed easily by air currents and spread with rain and irrigation water. Fusarium can also be spread by fungus gnats.

Rhizoctonia produces sterile colorless mycelia which will change to yellowish or light brown with age. Hyphae consist of long cells, with branching at an approximate right angle to the main hypha with slight hyphal constrictions at the junction. Under certain conditions, *Rhizoctonia* produces sclerotia which function as survival structures (Agrios 2005). The fungus can overwinter as mycelia or sclerotia in or on



Fig. 5 Primary symptoms of *Vanda* plantlet rot caused by *Fusarium* spp. include root tip necrosis in the community tray (**a**); yellowish leaves and leaf drop, dark brown roots, and plantlet death (**b**); *Dendrobium* roots showing brown discoloration with white fungal mycelia, the root system totally dried out and the pseudobulb turning brown (**c**); *Paphiopedilum* showing decline symptoms including yellowish leaves and brown and rotten collars caused by *Rhizoctonia* sp. from contaminated coconut husks used as the planting medium (**d**) (P. Smitamana © 2017. All Rights Reserved.)

infected plants or by contaminating planting materials and containers, e.g., coconut husks and pots. The fungus spreads by rain or irrigation water.

Management

- ***Resistance*** – *Brassocattleya* "Orquidacea's Melody" and *Brassocattleya* "Orquidacea's Rare Bird," were resistant and moderately resistant, respectively, to Fusarium wilt caused by *Fusarium oxysporum* f. sp. *Cattleyae* (Pedroso-de-Moraes et al. 2011).
- ***Cultural practices*** – Growers should inspect newly introduced plants and keep them in an area removed from general production for 4–6 weeks to observe the health of the plants and make sure that they are free from any diseases. Transplant uninfected pseudobulbs into pathogen-free media and new pots. Adjust the pH of the irrigation water and medium to 6.0–6.5 to help reduce infection by *Fusarium*.
- ***Sanitation*** – For slightly infected *Vanda*, *Vanilla*, and *Rhyncostylis* with good aerial roots, cut above the diseased portion using a sterile cutting tool, pot in a fresh medium, remove and discard or burn infected tissue, and apply an appropriate fungicide or biocontrol (see below).

- ***Fungicides and biocontrols*** – Use biocontrol agents, e.g., *Trichoderma harzianum*, by incorporation into fresh media in order to prevent contamination by pathogens. If chemical control is needed, apply cyprodinil + fludioxonil, chlorothalonil, azoxystrobin, or thiophanate methyl when early symptoms are observed, being sure to rotate different classes of fungicides. Prochloraz and tebuconazole were very effective in reducing Fusarium bulb and root rot in *Cymbidium goeringii* (Jee et al. 2003). Quaternary ammonium salts, sodium hypochlorite, and other materials should be used to disinfest benches and containers (see ▶ Chap. 9, "Sanitation for Management of Florists' Crops Diseases" for more information).

2.7 Leaf Spots (*Fusarium*, *Phyllosticta*, *Guignardia*, *Septoria*, *Cercospora* spp.)

Geographic occurrence and impact. Leaf spots of orchids can be caused by many fungi. Symptoms in the diseased orchid are varied based on the causal agent and host. Many orchids are susceptible to these pathogens, e.g., *Brassolaeliocattleya*, *Cattleya*, *Cymbidium*, *Dendrobium*, *Epidendrum*, *Laelia*, *Laeliocattleya*, *Odontoglossum*, *Oncidium*, *Phalaenopsis*, and *Vanda*.

Leaf spots of orchids are found worldwide: Australia, Brazil, Japan, the Netherlands, Thailand, and the USA (Hawaii) (Daly et al. 2013; Ichikawa and Aoki 2000; Jones 2003; Kamjaipai 1983; Silva and Pereira 2007; Silva et al. 2008; Uchida and Aragaki 1980; Uchida 1994). Leaf spots are common in most nurseries and can lead to serious losses in shipping containers due to the high moisture and low light conditions which favor the growth and infection of the pathogens.

Symptoms/signs

***Fusarium* spp.** – Two spot types; yellow and black are caused by *F. subglutinans* and *F. proliferatum*, respectively, on *Cymbidium*. The yellow spot symptoms start as water soaked patches on the leaves then the lesions enlarge with sunken centers and turn reddish-brown surrounded with a yellowish swelling without a definitive boarder. In the case of black spot, small black speckles are found at the early stage, then the lesions enlarge to form irregular, angular black spots; some yellow halos are found in certain *Cymbidium* cultivars.

***Phyllosticta* spp.** – Symptoms may start anywhere on the leaf, flower, or pseudobulb. Tiny yellow and slightly sunken spots are initially observed on the leaves. As the lesions enlarge, they become more sunken with round or oval shapes (Fig. 6a). The fungal growth within the tissue turns the lesion tan or brownish as the disease develops. Severely infected leaves may drop prematurely and a black web-like pattern formed by the fungus is observable (Fig. 6b). Spot symptoms on flowers are small, faint, and commonly blue or lavender on purple cultivars.

***Guignardia* spp.** – Symptoms start as tiny, dark purple, elongated lesions which can be found on either leaf surface. With age, the lesions enlarge and run parallel to the veins and form elongated purple streaks. Large irregular lesions are formed by

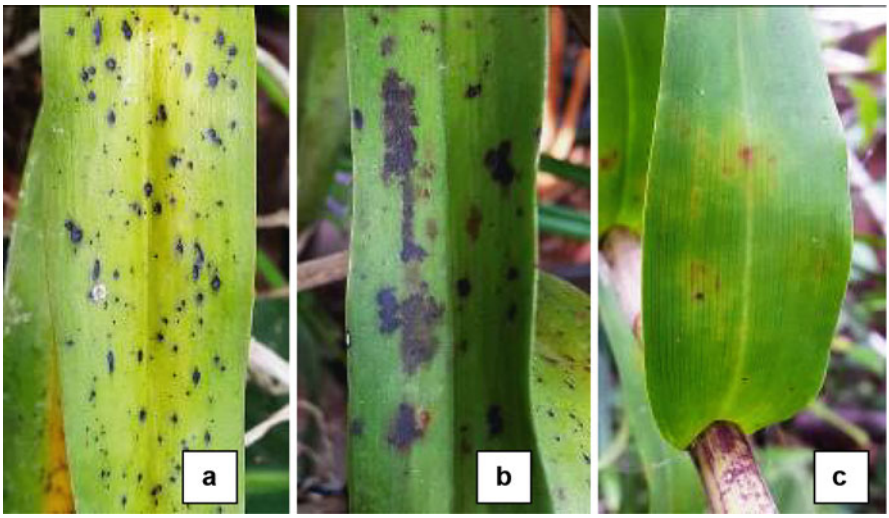


Fig. 6 Leaf spots caused by *Phyllosticta* sp. in *Vanda*: sunken lesions (**a**); merging lesions with black fungal growth in the leaf tissue (**b**); yellow green area with brown spots on *Dendrobium* leaf caused by *Cercospora* sp. (**c**) (P. Smitamana © 2017. All Rights Reserved.)

the merging of the spots, and the center of the lesions turns tan. Black fruiting bodies of the fungus develop in the affected area and can be seen as tiny raised black spots.

***Cercospora* spp.** – *Cercospora* attacks the underside of the leaf and induces small yellow spots. After the disease progresses, yellow green areas are observed on the top surface of the leaf (Fig. 6c). Subsequently, the spots enlarge and form irregular patterns with slightly sunken areas which turns brown to black. The necrotic lesions can enlarge to cover the whole leaf area and severely infected leaves may drop prematurely.

***Septoria* spp.** – *Septoria* can infect both sides of the leaf. Primary symptoms start from small sunken yellow lesions which gradually enlarge and form dark brown to black, circular, or irregular lesions. Lesions may merge to form irregular areas on the leaf which may prematurely drop.

Biology and epidemiology

Phyllosticta belongs to the Sphaeropsidales and produces asexual spores in pycnidia, small, black globose, elongate, or cup-like fruiting bodies. Pycnidia may be superficial or immersed in the tissue. Conidia are hyaline, single-celled, globose, or ovoid (Sumbali 2005). High light levels and dry conditions inhibit the growth and spread of the fungus, whereas low-light and high humidity, or rainy weather, promote *Phyllosticta* outbreaks (Jones 2003).

Guignardia is the perfect stage of *Phyllosticta* and belongs to the Ascomycetes, and produces eight single celled ascospores in perithecia (Silva and Pereira 2007).

Cercospora belongs to the Dermatiaceae and produces dark colored hyphae and long slender hyaline or dark, straight to curved, multicellular conidia on short dark conidiophores. Clusters of conidiophores arise from the plant surface through the stoma and form conidia on the growing tips (Agrios 2005). No fruiting bodies are formed (Sumbali 2005).

Septoria belongs to the Sphaeropsidales like *Phyllosticta* and produces long, slender, hyaline, generally curved conidia with one or more septa (Sumbali 2005).

Conidia of these fungi are easily detached from the conidiophores, and ascospores discharge from perithecia into the air and are carried great distances. Conidia are splashed from older diseased leaves to other leaves nearby and infect new plants.

Management

- ***Cultural practices*** – Good sanitation with adequate air movement is important to control these diseases. Discard severely infected plants. Collect all fallen leaves, old or dry pseudobulbs, and sheaths from pots, benches, and the ground and discard or burn them. Water the orchids early in the day to allow leaves to dry before dark.
- ***Fungicides*** – Spray with mancozeb, chlorothalonil, or another effective fungicide to kill the fungal spores and reduce the inoculum level (see ▶ Chap. 7, "Fungicides and Biocontrols for Management of Florists' Crops Diseases" for more information).

2.8 Anthracnose [*Colletotrichum gloeosporioides* (syn. *Glomerella cingulata*)]

Geographic occurrence and impact. This is a disease that commonly affects *Dendrobium*, *Vanda*, *Cattleya*, *Phalaenopsis*, and *Ceologyne* orchids. Anthracnose in orchids has been reported in Australia, India, Thailand, and the USA (Florida) (Bottom 2016; **C**howdappa et al. 2014; Daly et al. 2013; Jadrane et al. 2012; Kamjaipai 1983; McMillan 2011; Prapagdee et al. 2008; Prapagdee et al. 2012). This disease is considered only a minor pathogen and is usually a result of some type of injury to the leaf, whether it is mechanical, chemical, or insect damage. Nevertheless, anthracnose can result in reduced plant quality and growth; disease development may be sporadic as it is affected by levels of pathogen inoculum and environmental conditions.

Symptoms/signs. *Colletotrichum* infects the aerial portion of the plant of which leaves are most often attacked. Infected leaves show brown discolorations which are irregularly shaped sunken lesions. Leaf tips turn brown and the affected area gradually enlarges toward the base. With age, the lesions become dark brown or light gray patches, and sometimes brownish black concentric rings of fruiting bodies or numerous dark bands develop across the leaf (Fig. 7). Flower infection is characterized by water soaked spots and black or brown pustules which are usually raised

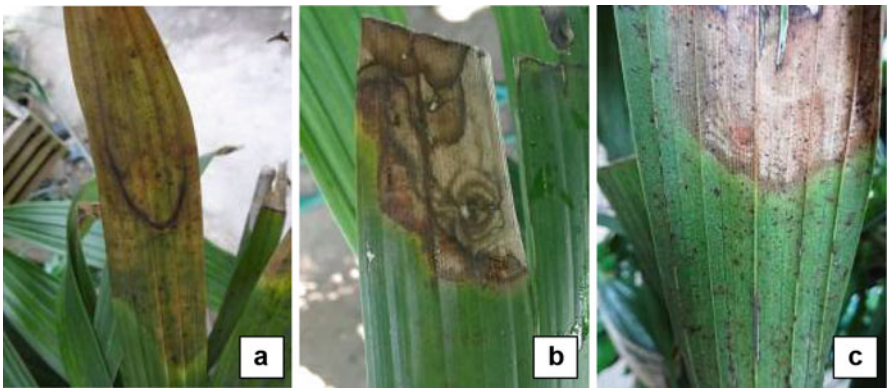


Fig. 7 Typical symptom of anthracnose on the black orchid (*Ceologyne pandurata*): the early symptom showing a brownish water-soaked area starting at the leaf tip (**a**); later stage lesions showing dark concentric rings on the upper leaf surface (**b**) and lower leaf surface (**c**) (P. Smitamana © 2017. All Rights Reserved.)

and occur on the underside of old sepals and petals. The spots may merge and cover the entire flower. In white flowered *Phalaenopsis*, the infected petals show anthracnose-like, sunken lesions which are surrounded by a ring of green tissue.

Biology and epidemiology. *Colletotrichum gloeosporioides* is a member of the Melanconiaceae and is the imperfect stage of the ascomycete *Glomerella cingulata*. The conidia are produced in acervuli and are single celled, hyaline, ovoid, cylindrical, typically elongated with round ends and slightly narrower in the middle. Large brown or black setae are formed among the conidiogenous cells (Agrios 2005; Sumbali 2005). The conidia are disseminated by air currents, wind, rain, or irrigation water. The pathogen is most active in warm weather when light is low and moisture is high. *Colletotrichum* can survive or overwinter on plant residue in soil or on infected plants. Anthracnose is common after orchids pass through a stress period, e.g., cold or hot temperature, mechanical injuries, or chemical damage.

Management

- ***Cultural practices*** – Good sanitation and air movement, lower temperatures (if possible), and increased light may help reduce the spread and severity of this disease.
- ***Fungicides and biocontrols*** – Alternation of protectant fungicides like mancozeb and systemic fungicides such as thiophanate methyl is recommended. The bacterium *Bacillus subtilis* was shown to reduce anthracnose caused by *C. gloeosporiodes* in *Paphiopedilum concolor* in Thailand (Kuenpech and Akarapisan 2014). Application of culture filtrates of the bacterium *Streptomyces hygroscopicus* prevented anthracnose on orchid (Propagdee et al. 2008).

2.9 Rust (*Sphenospora kevorkianii*)

Geographic occurrence and impact. *Sphenospora kevorkianii* has been found infecting orchids in Brazil and in the USA (Linder 1944; Pereira et al. 2002; Pereira and Silva 2009). Known hosts are *Cyrtopodium punctatum*, *Epidendrum difforme*, *E. secundum*, *E. xanthinum*, *Notylia lyrata*, *Pleurothallis mentigera*, *Prescottia sclerophylla*, *Stanhopea graveolens*, and *Zygostates lunata* which are epiphytes found in tropical forests.

Symptoms/signs. Rust fungi are very easy to identify from their rust colored spore masses on leaves and stems. The symptoms start out as flecks or spots and grow into bumps, and most commonly appear on the underside of leaves.

Biology and epidemiology. *Sphenospora kevorkianii* produces uredospores in ovoid, ellipsoid, or subspherical uredinia on the lower leaf surface (Pereira et al. 2002). The uredospores are easily detached and dispersed by wind, rain, and irrigation water.

Management. Cut off and discard or burn the infected parts. Discard or burn severely infected plants. Reducing nitrogen fertilizer application may help. If needed, spray with appropriate systemic fungicides (see ▶ Chap. 7, "Fungicides and Biocontrols for Management of Florists' Crops Diseases" for more information).

3 Bacterial and Phytoplasma Diseases

3.1 Leaf Spot (*Acidovorax avenae* subsp. *cattleyae*)

Geographic occurrence and impact. This disease has been reported in Australia, China, India, Italy, Korea, Taiwan, Poland, and the USA (Florida) (Borah et al. 2002; Cating and Palmeteer 2011; Ding et al. 2010; Hseu et al. 2012; Kim et al. 2015; Li et al. 2009; Lin et al. 2015; Pulawska et al. 2013; Scortichini et al. 2005; Stovold et al. 2001). *Acidovorax avena* is a pathogen of quarantine importance and causes serious losses to orchid production especially in *Cattleya*, *Cypripedium*, *Dendrobium*, *Oncidium*, *Phalaenopsis*, and *Vanda*.

Symptoms/signs. Symptoms usually start as small brown soft water-soaked spots. The spots turn black forming cavities in the parenchyma which quickly expand over the entire leaf. Eventually, the pathogen invades the growing point of the plant causing death (Fig. 8a, b). *Phalaenopsis*, due to its succulent leaves, is generally reported in many countries as the most susceptible genus.

Biology and epidemiology. *Acidovorax avenae* subsp. *cattleyae* is a gram-negative rod shaped, nonfluorescent bacterium, which moves using a single polar flagellum. The bacteria are easily spread by rain, irrigation, and contaminated tools commonly

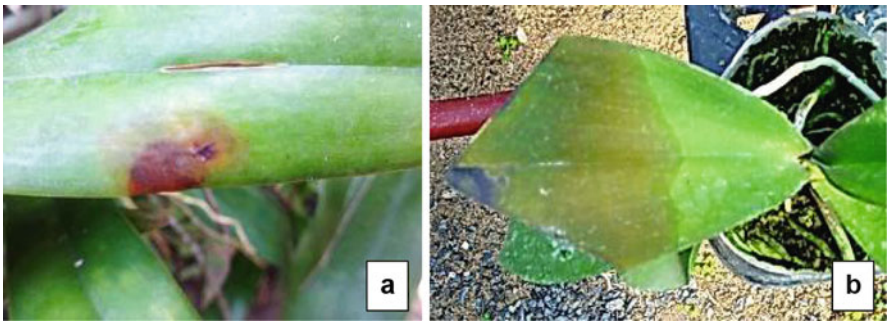


Fig. 8 Leaf spot of *Phalaenopsis* caused by *Acidovorax avenae*: brownish water soaked area which begins at a mechanical injury (**a**) and the soft rot of leaf (**b**) (P. Smitamana © 2017. All Rights Reserved.)

used in the nurseries. High moisture and temperature promote the growth of bacteria and accelerate the progress of disease development.

Management. Management of bacterial orchid diseases is challenging and must rely on an integrated approach.

- ***Cultural practices*** – Avoid overhead watering to minimize the dispersal of the pathogen in nurseries or water early in the morning to avoid long hours of leaf wetness. If the disease is found, reducing the use of nitrogen fertilizer and increasing potassium fertilizer has been associated with disease suppression.
- ***Sanitation*** **–** Growers should discard severely infected plants. Trim and discard diseased areas from slightly infected plants and keep them in an area removed from production for monitoring. Early detection of disease outbreaks is essential. Apply a bactericide as soon as the disease is observed (see below). Clean tools after each use by soaking in a 10% bleach solution.
- ***Bactericides*** – If the infection becomes widespread, growers may dip plants in a labeled quaternary salt material, orthophenylphenol, or natriphene, or spray with a copper compound. Formulations of hydrogen peroxide may help reduce the disease. If antibiotic use is allowed, streptomycin, tetracycline, or a mixture of both may be sprayed. Small-scale testing for phytotoxicity is recommended. However, reliance on chemical control alone for management of bacterial diseases of orchids is most often ineffective.

3.2 Soft Rot [*Dickeya chrysanthemi* (syn. *Pectobacterium chrysanthemi*); *D. dieffenbachiae*]

Geographic occurrence and impact. This disease mainly affects *Oncidium*, *Vanda*, *Tolumnia*, *Cattleya*, *Dendrobium*, *Phalaenopsis*, and *Miltonia*orchids and has been reported in China, Taiwan, and the USA (Florida) (Cating et al. 2008;

Cating et al. 2009b; Li et al. 2009; Cating et al. 2009b; Hseu et al. 2012; Keith and Sewake 2009; Zhou et al. 2012). This is an important quarantine disease that affects the export and import of orchids in many countries.

Symptoms/signs. The symptoms are typical of other bacterial soft rots and start with water-soaked tissue at the infection site. On *Oncidium*, the initial spots are commonly found at the base of the stem, and the lesions expand and become elongated. Gradually, the stem and leaf tissues become soft and watery. Bacterial ooze can be observed by cutting the edge of infected stem and leaf tissues and dipping them in a beaker or Petri dish containing water; bacterial streaming can be observed from the cut tissue by eye or under a stereomicroscope.

Infected *Phalaenopsis* plants initially show water-soaked, pale-to-dark brown pinpoint spots on leaves; some may be surrounded by a yellow halo. Under high humidity and temperature, the spots expand rapidly and extend over the leaf which turns a light tan color with a darker brown border in a few days. A typical odor of rotten tissue can be detected. When the bacteria invade the stem, plant death follows.

In *Miltonia*, *Cattleya*, and *Oncidium*, symptoms start with small water-soaked lesions which become a dark brown to black color and gradually expand throughout the leaves which subsequently rot. Some *Dendrobium* cultivars appear to be resistant to this disease and only small water-soaked spots are produced. In the susceptible types, the spots expand and coalesce to form larger necrotic areas; after that, the infected leaves dry out. If the bacteria invade the pseudobulbs, they turn soft and watery. In *Vanda*, infected areas show water soaked spots, some are brown color, and the leaves are rapidly rotted.

Biology and epidemiology. *Dickeya* spp. are gram-negative bacteria, facultative, anaerobic rods with peritrichous flagella. Infection by the pathogen normally starts in cutting wounds or other mechanical injuries. Drops of bacterial ooze from the infected leaves or stems are dispersed by rain, irrigation, and wind. The bacteria enter and colonize plants mostly at the basal part of leaves or pseudobulbs.

Management. Management of soft rot is the same as for Acidovorax leaf spot listed above.

3.3 Black Spot [*Burkholderia gladioli* (syn. *Pseudomonas gladioli*)]

Geographic occurrence and impact. *Burkholderia gladioli* was first characterized as the causal agent of leaf and corm disease in gladiolus. This pathogen is now known to cause serious losses in many plants especially orchids, e.g., *Phalaenopsis*, *Cattleya*, *Dendrobium Oncidium*, *Odontioda*, and *Miltonia*. This pathogen is found in tropical and subtropical zones of such countries as China, the USA (Hawaii), Indonesia, Taiwan, and Thailand (Chuenchitt et al. 1983; Hseu et al. 2012; Joko et al. 2014; Kamjaipai 1983; Keith et al. 2005; Lee et al. 2013; Takahashi et al. 2004; Uchida 1995; You et al. 2016).

Symptoms/signs. Infected leaves initially show small dark green to brown spots surrounded by a water-soaked area. Sometimes only small brown spots surrounded by yellow halos, usually circular, or in rare cases irregularly shaped occur. Under high humidity or after rain, the wet rot area rapidly expands and causes soft rot of the whole leaf, especially the succulent leaves of *Phalaenopsis*. The bacteria can invade the stem or pseudobulbs and sometimes cause defoliation. Under low humidity, the infected leaf may dry and crack in the center of the lesion. However, if the humidity increases, bacterial ooze can be seen on the cracks and the rotten tissues. Symptoms on the young plants spread rapidly and cause the whole plant to die.

Biology and epidemiology. *Burkholderia gladioli* is a gram-negative rod that may be straight or slightly curved. It is aerobic, catalase positive, urease positive, and non-sporeforming. Like other phytopathogenic bacteria, it is easily spread by water splash from rain, overhead sprinklers, and wind-blown water.

Management. Management of soft rot is the same as for Acidovorax leaf spot listed above.

3.4 Phyllody ("*Candidatus Phytoplasma*" sp.)

Geographic occurrence and impact. Only a *Sarcochilus* hybrid in Australia has been reported to be infected by a phytoplasma (Gowanlock et al. 1998).

Symptoms/signs. The infected *Sarcochilus* hybrid exhibited phyllody, the abnormal development of floral parts into leafy structures, and stunting.

Biology and epidemiology. Phytoplasmas are bacteria-like microorganisms lacking cell walls, but bounded by a unit membrane. Phytoplasmas are found located in the sieve tubes of phloem. Transmission of phytoplasmas is by phloem-feeding insects in the Cicadellidae family and Fulgoroidea superfamily such as leafhoppers and planthoppers, respectively.

Management. Avoid introducing diseased plants into nurseries. Remove and burn symptomatic plants.

4 Viral Diseases

Geographic occurrence and impact. Viruses are among the most damaging pathogens of orchids because of their debilitating effects on flower production and quality and extreme difficulty to control in a vegetatively propagated crop. Orchids can be infected by a very wide range of viruses. The most common and economically important orchid viruses worldwide are *Cymbidium mosaic virus* (CymMV) and *Odontoglossum ringspot virus* (ORSV), also known as *Tobacco mosaic virus*-orchid strain (TMV-O); and these two viruses are often found in mixed infections. Other

orchid viruses of note include: *Orchid fleck virus* (OFV), *Impatiens necrotic spot virus* (INSV), *Vanilla mosaic virus* (VanMV), and *Calanthe mild mosaic virus* (CalMMV). The geographic occurrence and orchid host range of these viruses are indicated in Table 1. Orchids have also been reported to host a large number of other viruses in the following genera:

Carmovirus – *Carnation mottle virus* (CarMV)
Closterovirus – *Dendrobium vein necrosis virus* (DVNV)
Cucumovirus – *Cucumber mosaic virus* (CMV)
Nepovirus –*Tomato ringspot virus* (ToRSV)
Potyvirus – *Bean yellow mosaic virus* (BYMV); *Ceratobium mosaic virus* (CerMV); *Clover yellow vein virus* (ClYVV); *Colombian datura virus* (CDV); *Dasheen mosaic virus* (DsMV)*; Dendrobium mosaic virus* (DeMV); *Diurus virus Y* (DVY); *Habenaria mosaic virus* (HaMV); *Pecteilis mosaic Virus* (PcMV); *Phalaenopsis chlorotic spot virus* (PhCSV)*; Spiranthes mosaic virus* 2 (SpiMV2); *Sarcochilus virus Y* (SVY); *Spiranthes mosaic virus* 3 (SpiMV3); *Turnip mosaic virus* (TuMV); *Vanilla necrosis virus* (VanNV)
Rhabdovirus – *Colmanara mosaic virus* (CoIMV); *Dendrobium ringspot virus* (DRV)
Tobravirus – *Tobacco rattle virus* (TRV)
Tombusvirus – *Cymbidium ringspot virus* (CymRSV); *Lisianthus necrosis virus* (LNV): *Tomato bushy stunt virus* (TBSV)
Tospovirus – *Capsicum chlorosis virus* (CaCV); *Tomato spotted wilt virus* (TSWV)

Symptoms/signs. Orchids can show variation in viral symptoms that depend on the virus, virus strain, mixed viral infections, and orchid variety. In addition, different viruses can cause similar symptoms in the same orchid, making diagnosis of virus infection by symptoms alone unreliable. Common symptoms of virus infection of orchids are presented in Table 1.

Biology and epidemiology. As obligate pathogens, viruses need a living host for their survival and replication. However, the molecular structure of Tobamoviruses, such as ORSV, makes them resistant to degradation for long periods on tools and in soil when they are released from host tissues. A number of important orchid viruses, such as CymMV, ORSV, VanMV, and CalMMV, are mechanically transmissible in plant sap. This characteristic is very important for virus spread in a vegetatively propagated crop such as orchid. In addition, potyviruses including VanMV and CalMMV are vectored in a nonpropagative (nonpersistent) manner on the stylets of aphids such as *Myzus persicae*. INSV, a tospovirus, is vectored in a circulative and propagative manner (it enters and replicates within the hemolymph of the insect) by a number of thrips species including the Western flower thrips, *Frankliniella occidentalis*. Only the first and early second larval stages are able to acquire tospoviruses, and only immature thrips that acquire these viruses or adults derived from such immatures are vectors. Adult thrips remain viruliferous for life; but tospoviruses are not transovarial. INSV also infects a large number of other

Table 1 Selected orchid viruses

Virus, genus	Geographic occurrence	Transmission	Orchid hosts	Symptoms	References
Cymbidium mosaic virus (CymMV), *Potexvirus*	Argentina, Brazil, China, Colombia, Cook Islands, Costa Rica, French Polynesia, Guam, India, Indonesia, Japan, New Caledonia, Netherlands, Niue, Palau, Puerto Rico, Reunion Island, Samoa, Singapore, Taiwan, Thailand, Tonga, USA, Vanuatu, Venezuela	Mechanical	A wide range including: *Aranthera*, *Arachnis*, *Calanthe*, *Cattleya*, *Cymbidium*, *Dendrobium*, *Gromatophyllum*, *Phalaenopsis*, *Phaius*, *Oncidium*, *Rynchostylis*, *Vanda*, and *Vanilla*	Chlorotic mosaic patterns, seen clearly on the youngest leaves in many orchid varieties, to black necrotic streaks, spots, or rings and sunken patches on *Cattleya* orchids. Flower size reduction with abnormal coloration and twisted young shoots with chlorotic streaks are common symptoms on young *Dendrobium* shoots (Fig. 9a, b)	Baker et al. 2007a; Cánovas et al. 2016; Davis and Ruabete 2010; Elliot et al. 1996; Farreyrol et al. 2001; Freitas-Astua et al. 1999; Gara et al. 1996; Hu et al. 1993; Inouye and Gara 1996; Jensen 1952; Kamjaipai 1983; Sherpa et al. 2003; Singh et al. 2007; McMillan et al. 2006; Sutrabutra 1989; Tanaka et al. 1997; Wong et al. 1994; Zheng et al. 2010; Zhou et al. 2004
Odontoglossum ringspot virus (ORSV), (also known as TMV-orchid strain and TMV-O), *Tobamovirus*	Argentina, Brazil, China, Colombia, Cook Islands, Costa Rica, Fiji, French Polynesia, India, Indonesia, Japan, Netherlands, New Zealand, Niue, Puerto Rico, Reunion Island, Singapore, Taiwan, Thailand, Tonga, USA, Vanuatu, Venezuela	Mechanical	A wide range including: *Aerides*, *Bulbophyllum*, *Calanthe*, *Cattleya*, *Cymbidium*, *Dendrobium*, *Oncidium*, *Phalaenopsis*, and *Vanilla*	Chlorotic or necrotic sunken lesions on leaves and flowers. In highly susceptible varieties, flowers may be smaller, distorted, and exhibit color breaking (Fig. 9c, d). Root tip necrosis is also a common symptom	Cánovas et al. 2016; Davis and Ruabete 2010; Elliot et al. 1996; Farreyrol 2001; Freitas-Astua et al. 1999; Hu et al. 1993; Inouye and Gara 1996; Kamjaipai 1983; McMillan et al. 2006; Pearson et al. 2006; Sherpa et al. 2006; Sutrabutra 1989; Tanaka et al. 1997; Thomson and Smirk 1967; Wong et al. 1994; Zhou et al. 2004

(*continued*)

Table 1 (continued)

Virus, genus	Geographic occurrence	Transmission	Orchid hosts	Symptoms	References
Orchid fleck virus (OFV), *Dichorhavirus*	Australia, Brazil, Costa Rica, Denmark, Germany, Japan, Korea, USA	False spider mite (*Brevipalpus californicus*)	*Cymbidium*, *Maxillaria*, *Renanthera*, *Phalaenopsis*	Yellow flecks and yellow or necrotic ringspot lesions on leaves (Fig. 9e). In *Dendrobium*, *Miltonia*, *Odontoglossum*, *Oncidium*, and *Paphiopedilum*, the chlorotic areas often with necrotic centers or rings	Brunt et al. 1997; Dietzgen et al. 2014; Freitas-Astúa et al. 2002; Kondo et al. 2003; Kitajima et al. 2001; Kubo et al. 2009
Impatiens necrotic spot virus (INSV), *Tospovirus* *Capsicum chlorosis virus* (CaCV), *Tospovirus*	China, Taiwan, USA Taiwan	A number of thrips species including the Western flower thrips (Frankliniella occidentalis) Mechanical	*Cymbidium*, *Dendrobium*, and *Phalaenopsis* and many other ornamentals *Phalaenopsis*	Large chlorotic or necrotic ringspots on leaves; leaf yellowing and distortion (Fig. 9f) Chlorotic spots with concentric necrosis	Bakardjieva et al. 1998; Baker et al. 2007b; Koike and Mayhew 2001; Zhang et al. 2010; Zheng et al. 2008a Zheng et al. 2008a
Phalaenopsis chlorotic spot virus *Potyvirus* *Vanilla mosaic virus* (VanMV), *Potyvirus*	Taiwan Cook Islands, Fiji, French Polynesia	Mechanical Mechanical; green peach aphid (*Myzus persicae*)	*Phalaenopsis* *Vanilla*	Chlorotic spots Chlorotic and necrotic flecks, mosaic and deformation in leaves; dieback of shoots	Zheng et al. 2008b Davis and Ruabete 2010
Calanthe mild mosaic virus (CalMMV), *Potyvirus* *Cucumber mosaic virus* *Cucumovirus*	Japan French Polynesia, Reunion Island	Mechanical; (*Myzus persicae*) Mechanical; aphid	*Calanthe*, *Phalaenopsis*, and *Tetragonia expansa* *Vanilla*	Mild leaf mosaic leaf and flower breaking Severe stunt, conspicuous stem and leaf deformation	Gara et al. 1998 Farreyrol et al. 2001; Farreyrol et al. 2010

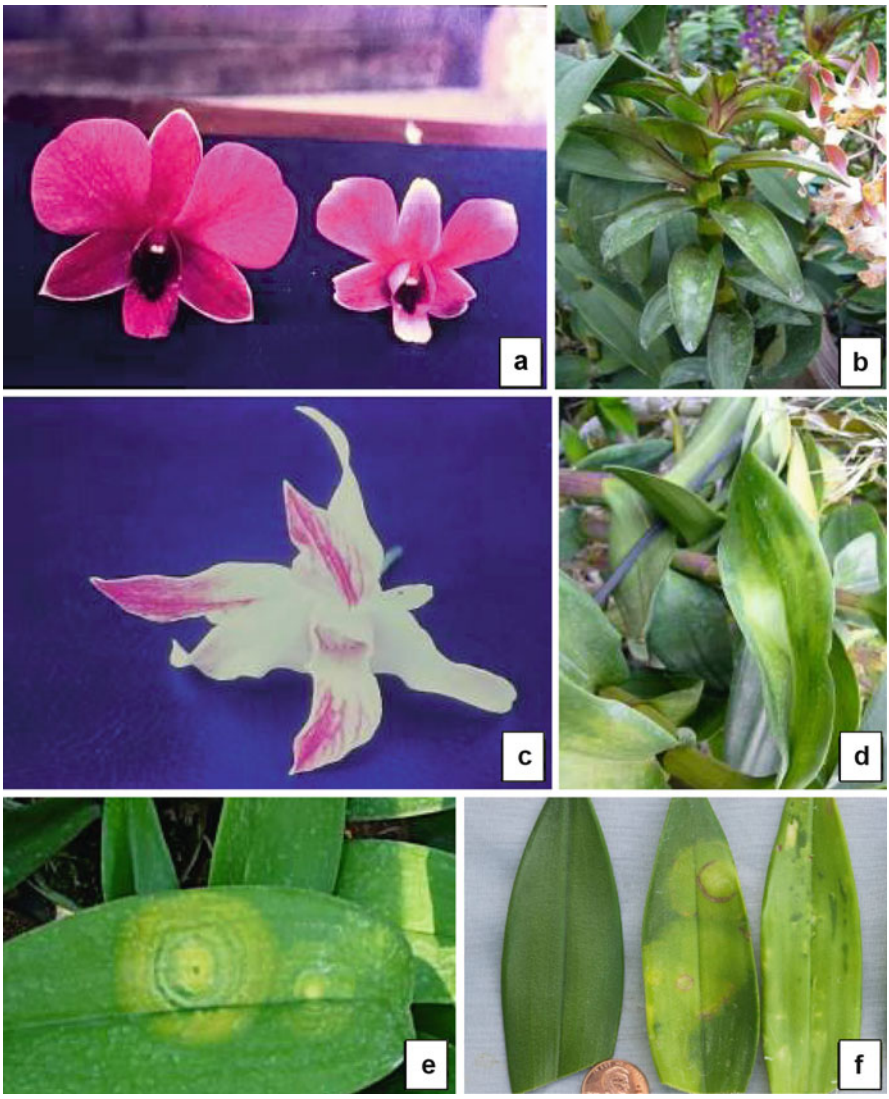


Fig. 9 Symptoms of orchid viruses. CymMV: small, discolored *Dendrobium* flower (**a**), twisted shoot with yellow streaks (**b**); ORSV: distorted *Dendrobium* flower with color-breaking (**c**), mosaic and yellow patches in *Dendrobium* (**d**); OFV: yellow concentric rings in *Phalaenopsis* leaf (**e**); INSV: chlorotic patches and necrotic ringspots and distortion in *Oncidium* leaves (**a-e**, P. Smitamana © 2017. All Rights Reserved.; **f**, S. Koike © 2017. All Rights Reserved.)

ornamentals which can serve as reservoirs of the virus. OFV is spread by the false spider mite, *Brevipalpus californicus*.

Management. Once infected by a virus, unlike with other pathogens, nothing short of complicated and exacting tissue culture procedures can rid plants of viruses, and

even these extreme measures are not always successful. Therefore, management of orchid viruses must be based on prevention of infection by using virus-free propagative material and good sanitation practices.

Isolate suspect plants and new introductions and monitor for symptoms for 6 weeks or until proven by a diagnostic test (ELISA, PCR, etc.) to be virus-free. Promptly discard or destroy by burning all confirmed diseased plants. Use only sterile cutting tools; soak the tools in a 10% bleach solution or saturated trisodium phosphate solution for 10 min; or use disposable razor blades when dividing plants and cutting flowers. Wear latex gloves when handling plants and discard those gloves when finished. Always change to new gloves when handling a new plant. Disinfest containers by first washing with soap to remove residual organic matter, then soak in a 20% bleach solution for 1 h, after that soak them for 1 h in a quaternary ammonium salt solution as per label instructions. Exclude insect-vectored viruses by using fine mesh screening, pesticides, and/or biological controls. Refer to chapter "▶ Chap. 4, "Insect Management for Disease Control in Florists' Crops" for additional information.

5 Nematode Diseases

5.1 Foliar Nematodes (*Aphelenchoides* spp.)

Geographic occurrence and impact. A number of *Aphelenchoides* spp. including *A. besseyi*, *A. fragariae*, and *A. ritzemabosi* have been reported to attack orchids in Singapore, Thailand, and the USA (Florida) (Esser et al. 1988; Kawate and Sewake 2014; Latha et al. 1999). Because warm, wet greenhouse environments are conducive to foliar nematode infection, the occurrence of these nematodes in orchids is undoubtedly more widespread. This disease affects many orchids including *Cattleya*, *Cymbidium*, *Dendrobium*, *Oncidium*, *Paphiopedilum*, and *Vanda*. Losses from uncontrolled foliar nematodes have been estimated at up to 90–95% in potted orchid production in Hawaii (Kawate and Sewake 2014).

Symptoms/signs. Foliar nematodes infect immature buds and prevent flower formation and development on *Vanda*; diseased buds abscise or adhere and become blackened. Symptoms on *Dendrobium* leaves include yellow-green blotches which become brown as the lesions age. On *Oncidium*, browning of foliar buds and dark streaks in pseudobulbs can occur (Uchida and Sipes 1998).

Biology and epidemiology. A film of water enables foliar nematodes to move up stems and over leaf surfaces to seek new infection sites made by wounds or natural openings such as stomates. Infection and symptom expression are, therefore, enhanced by warm, wet conditions. The *Aphelenchoides* spp. that attack orchids have a very broad host range that includes many ornamentals and other cultivated crops. Adults and 4th stage juveniles feed on mesophyll and epidermal tissue within leaves (endoparasitism). These tissues collapse and turn brown. Females lay eggs within green leaf tissue. Foliar nematodes also feed externally on stems, buds, and

flowers (ectoparasitism). Adults and 4th stage juveniles can survive in association with living and desiccated plant tissues for long periods. Foliar nematodes can be disseminated by splashing water, infected plant propagative material, and debris (Kohl 2011).

Management. Prevention of nematode introduction and good sanitary practices are the keys to managing foliar nematodes. Isolate suspicious plants and new introductions to monitor for symptom expression. Discard plant debris and infected plants. All surfaces coming in contact with plants (pots, stakes, tools, etc.) should first be cleaned with soap and water and surface-disinfested with a 10% solution of household bleach for 10 min after each use (Uchida and Sipes 1998). Growers should avoid wetting plant surfaces as much as possible. Increasing air circulation through increased plant spacing will help to dry plants. Only propagate from clean material. Hot water treatment of *Vanda* cuttings for 15 min at 46 °C (115 °F) or 5–10 min at 49 °C (121 °F) eliminated nematodes without injuring the plants (Uchida and Sipes 1998).

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Diseases of Peonies

22

Andrea R. Garfinkel and Gary A. Chastagner

Abstract

Herbaceous peonies (*Paeonia lactiflora* Pall.) are grown throughout temperate regions of the world for their use as high-value cut flowers. Despite their popularity, little information is available on diseases and disease management of this flower compared with other ornamental geophytes, such as tulips or lilies. From the literature, it is clear that the fungal diseases Botrytis gray mold and leaf blotch are among the most economically important diseases of peony; however, many other fungi, viruses, bacteria, and nematodes affect peonies worldwide. Management recommendations used for pathogens of peonies rely mainly on general disease management strategies and information on pathogen biology and epidemiology from other pathosystems.

Keywords

Botrytis • Cut flowers • Fungi • *Paeonia* • Plant pathogen

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R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_46

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1 Introduction

The vast majority of peonies produced for cut flowers are hybrids crossed with the herbaceous peony species, *Paeonia lactiflora* Pall. This species, native to China, comprises some of the oldest commercial cut flowers, rising first to popularity in Europe in the late 1700s (Rogers 1995). Hybridization of the herbaceous peony took place shortly thereafter and many of the still-popular cut flower types are relics of these nineteenth century breeders (Rogers 1995). The propagation of these cultivars is done exclusively through vegetative division of the root crown and fleshy storage roots. Peony is undergoing a resurgence in popularity, and cut flowers are available nearly year-round at the Dutch flower auctions as nontraditional flower-producing regions, such as the state of Alaska in the United States, are producing peonies at a rapidly increasing rate to fill gaps in availability (Auer and Greenburg 2009; Holloway and Buchholz 2013).

Despite the rise in production and consumption of cut peonies, the information about diseases and disease management is, at best, disjointed. This chapter will seek to consolidate the somewhat limited information available about peony diseases and their management. Where specific epidemiology and disease control methods are not known in peony, an attempt has been made to supplement with general information about the pathogen group or information about other pathosystems. Diseases of other *Paeonia* species not typically used for cut flowers, including tree peonies (*Paeonia suffruticosa*), will not be specifically discussed; however, a few mentions of diseases on tree peonies and other *Paeonia* species have been included when appropriate.

2 Fungal and Fungus-Like Diseases

2.1 Botrytis Gray Mold (*Botrytis cinerea* Pers. Fr. and *Botrytis paeoniae* Oud.)

Geographic occurrence and impact. Botrytis gray mold (also "Botrytis blight") affects peonies in nearly every region in which they are cultivated and can be considered, economically speaking, the most important peony pathogen (Whetzel 1939). *Botrytis* can infect every part of the peony plant including the foliage, stems, flowers, and the tuberous roots and root crown (Wallace et al. 1962; Whetzel 1939). Due to its ability to cause disease on floral tissues under storage conditions (Droby and Lichter 2007), *Botrytis* is both a pre- and postharvest concern for cut peonies.

Symptoms/signs. Early in the season, Botrytis blight can be seen on emerging shoots as a dark brown soft rot that causes toppling of stems (Whetzel 1939). Later in the season, lesions on expanded stems and foliage are dry and tough and exhibit dark and light brown zonation (Whetzel 1939). Lesions originating on the foliage can move down the petiole into the stem. Lesions at the base of the stem can girdle the stem and cause the upper foliage to become red and wilted (Whetzel 1939). *Botrytis* can colonize undeveloped or "blasted" buds causing them to turn black. Fully developed buds that become infected turn brown, and the petals rot inside causing failure of the flower to open (Whetzel 1939). *Botrytis* infection of the flower petals develops as brown spots that quickly coalesce, blighting the petals and causes them to drop off (Whetzel 1939). Infections of the flower or flower bud can move down the stem which will sometimes exhibit the zonation described above. Under moist conditions, conidia develop on the surface of lesions giving the tissue a gray fuzzy texture lending inspiration for the name "gray mold" (Fig. 1).

Biology and epidemiology. The source of a Botrytis gray mold infection on peony depends on which species of the fungus is the causal agent of disease. *Botrytis paeoniae* is thought to be a host-specific pathogen (causing disease only on peony); therefore, the inoculum from this pathogen will be from previous or nearby crops of peony (Holz et al. 2007; Whetzel 1939) or potentially infected planting stock. *B. cinerea* inoculum, on the other hand, could come from any of its over 250 hosts. Primary inoculum for both organisms is considered to be conidia. The first conidia of the season arise from sclerotia which overwinter on the surface of decayed peony foliage. Subsequent crops of conidia (secondary inoculum) are produced on infected plant material throughout the growing season. Conidia enter the host through natural openings, directly, or through wounds (Jarvis 1980). Colonized peony petals and other plant tissues can also serve as an important inoculum source, whereby mycelium directly infects healthy tissue from saprophytically colonized tissue (Daughtrey et al. 2000; Holloway personal communication 2015; Holz et al. 2007). Conidia are mostly dispersed by wind (Holz et al. 2007).



Fig. 1 Botrytis gray mold on peony foliage showing alternating tan and dark brown zones (*left*), a peony basal stem lesion showing zonation (*center*), and an infected flower bud with sporulation (*right*) (Andrea R. Garfinkel © 2017. All Rights Reserved.)

Disease development is typically considered to be favored by cool, wet weather conditions in which there is free moisture on the plant surface (Jarvis 1980). While *B. paeoniae* and *B. cinerea* are the most commonly cited *Botrytis* species to infect peony, preliminary investigations have identified several other *Botrytis* species infecting peony including the fenhexamid-resistant *B. pseudocinerea* (Garfinkel and Chastagner unpublished data; Muñoz et al. 2015).

Management

- ***Cultural practices*** – Reducing leaf wetness is one of the most important mechanisms for limiting Botrytis gray mold development (Daughtrey and Benson 2005). Therefore, wide plant spacing to avoid overcrowding of plants and allow for maximum air movement and sun exposure can help limit disease development (Whetzel 1939). Avoiding overhead irrigation can also help reduce disease incidence. Keeping flowers dry and reducing relative humidity and fluctuations in temperatures in storage areas will help decrease postharvest disease of peony (Whetzel 1939).
- ***Sanitation*** – Removal of all infected plant parts during the season, especially spent flowers (Pfleger et al. 2007) and other dead plant tissues (Daughtrey and Benson 2005), is an important way to reduce sources of secondary inoculum (Whetzel 1939). All foliage should be cut, removed from the field, and disposed of at the end of the season to eliminate plant material on which the fungus can survive and infect the next crop (Whetzel 1939). Planting of clean propagative material is also an important way to minimize in-field inoculum. Although there are no studies available on the effectiveness of hot-water treatment for controlling *Botrytis* on peony roots, this practice may be useful as it has proven effective in other geophyte crops.

- ***Fungicides*** – Fungicides are among the most important tools in an integrated *Botrytis* management program for many ornamentals (Daughtrey and Benson 2005). However, their efficacy is hampered by widespread resistance to fungicides, such as the dicarboximides and benzimidazoles (Leroux 2007). The prevalence of fungicide resistance in *Botrytis* populations on peonies is unknown. Fungicides typically effective against *Botrytis* include chlorothalonil, azoxystrobin, iprodione, mancozeb, fenhexamid (except *B. pseudocinerea*, see above), and copper-based products. Wallace et al. (1962) found that soil drenches containing dicloran were effective at controlling basal stem rot caused by infected roots.
- ***Resistance*** – Winters (1930) published a list of cultivars and their relative susceptibility to *Botrytis paeoniae*. The susceptibilities of some peony cultivars are described in Table 1. Itoh hybrids, crosses between the herbaceous *P. lactiflora* and the tree peony *P. suffruticosa*, which are gaining popularity in the cut flower market, appear to be more resistant to gray mold than other *P. lactiflora* types (Beckerman and Lerner 2009).

2.2 Leaf Blotch (*Graphiopsis chlorocephala* [Fresen.] Trail)

[Shubert et al. (2007) and Braun et al. (2008) conducted investigations into the genetics of herbarium samples of *Cladosporium paeoniae* Pass., syn. *Cladosporium chlorocephalum* (McKemy and Morgan-Jones 1991), casual agents of peony leaf blotch, and have suggested that this fungus be renamed *Graphiopsis chlorocephala*.]

Geographic occurrence and impact. Leaf blotch appears to affect many different species of peonies wherever they are grown (McKemey and Morgan-Jones 1991) and ranks perhaps only second in importance to Botrytis gray mold. Leaf blotch is not, as the name would suggest, limited to the leaves of the peony, but can impact all aboveground parts of the plant severely hampering marketability of stems, flowers, and foliage.

Symptoms/signs. Leaf blotch goes by many names (also Cladosporium leaf blotch, Cladosporium spot, Cladosporium red spot, red spot, or measles) that describe the various symptoms infected plants may display. Symptoms can range from purple-red, irregularly shaped flecks or streaks with diffuse margins on stems, foliage, flowers buds, and flower petals to large, expanding purple lesions on stems or foliage with dark centers (Fig. 2). The leaf spots as viewed from the underside of the leaves appear pale to chestnut brown. Infected stems and leaves maintain their succulence (Meuli 1937). The development of different symptoms appears to be correlated with leaf phenology; young, succulent tissue limits the infection to flecking, while large, expanding lesions develop on more mature tissues (Weiss 1940).

Biology and epidemiology. The most important source of initial inoculum, conidia, of *G. chlorocephala* arises from plant debris from the previous year in

Table 1 Susceptibility of peony cultivars to *Botrytis paeoniae* as determined by Winters (1930)

Susceptibility	Cultivars
Resistant	Akalu, Argus, Arthemise, Attraction, Avalanche, Balliol, Baroness Schroeder, Baron James Rothschild, Black Prince, Cavalleria Rusticana, Chalice, Christine Gowdy, Christine Ritcher, Comte de Nanteuil, Dorothy, Dorothy Echling, Dorothy E. Kibby, Ella Wheeler Wilcox, Eucharis, Eureka, Fragrans, Fulgida, General Bertrand, General Cavaignac, Gloire de Chenonceaux, Glorious, Flory of Somerset, Gretchen, Griff Thomas, Hermes, Hogioku, Itenshikai, King of England, Kumagaye, Lady Bellew, Lady Mayoress, L'étincelante, Lord Salisbury, Luetta Pfeiffer, Madame Lemonnier, Madame Schmidt, Maud L. Richardson, Meissonier, Monsieur Boucharlataine, Mrs. Gwyn-Lewis, Mr. L. van Leeuwen, Old Silvertip, Petite Renée, Plutarch, Princess Ellen, Purpurea Superba, Queen Wilhelmina, Red Bird, Ruigegno, Sarah Bernhardt, Speedwell, Sweet Home, Yeso
Moderately resistant	Admiral Dewey, Agnes Mary Kelway, Albâtre, Albert Crousse, Alfred de Musset, American Beauty, Archie Brand, Asa Gray, Augustin d'Hour, Aunt Ellen, Béranger, Bullock, Carnea Elegans, Carnot, Charles Binder, Charles Verdier, Charlotte Cushman, Clarisse, Comet, Conqueror, Couronne d'Or, Daubenton, Daybreak, Dorchester, Dorothy Kelway, Duchess of Portland, Duke of Devonshire, Edwin Forrest, Emile Lemoine, Enchantment, Eternal City, Etta, Faust, Favorite, Festiva Maxima, Flambeau, Frances Shaylor, Graziella, Grizzel Muir, Gypsy, Henry Avery, Hon. Mrs. Portman, Innocence, John Fraser, June Day, Jupiter (Calot), Kelway's Queen, La Coquette, Lady Alexandra Duff, Lady Somerset, La Fraicheur, Lake of Silver, La Perle, La Sublime, La Tulipe, La Vestale, L'étincelante (Dessert), Lord Lytton, Louis van Houtte, Mabel L. Franklin, Madame Coste, Madame de Guerle, Madame de Vatry, Mademoiselle Gaillant, Mafeking, Mary L. Hollis, Masterpiece, Mathilde de Roseneck, Mazie Terry, Monsieur Paillet, Monsieur Pasteur, Muchelny, Norfolk, Octavie Demay, Pallas, Phoebe Cary, Pierre Duchartre, Pink Enchantress, Princess Maud, Queen of Beauty, Rauenthal, Rhoda, Rubicunda, Ruth Brand, Simonne Chevalier, Sir Robert Gresly, Snowflake, Sosthenes, Sully Prudhomme, Torquemada, Triumphata, Trojan, Venus, Victoria, Ville de Nancy, Waterloo, Welcome Guest
Susceptible	Adam Bede, Agnes Barr, Amalthea, Armand Rousseau, Bertha, Camille Calot, Canariensis, Carlotta Grisi, Carnea Triumphans, Caul, Chrysanthemiflora, Comte de Cussy, Comte de Paris, Countess of Clancarty, Daniel d'Albert, Delachei, Duc de Cazes, Duc de Wellington, Eastern Beauty, Edmond Lebon, Etienne Mechin, Frances Shaylor, General Grant, Grandiflora, Jules Calot, Lady Beresford, Lutetiana, Madame de Verneville, Madame Emile Galle, Madame Hutin, Magnifica, Marie Lemoine, Marquise d'Ivry, Mathilde Méchin, Meadowvale, Monsieur Chevreul, Mrs. Lowe, Myrtle, Pottsi, Princess Beatrice, Pulcherrima, Queen's Perfection, Roem de Boskoop, Sappho, Sea Foam, Snowball (Hollis), Souvenir de Gaspard Calot, Strasbourg, Sunrise, Thomas S. Ware, Torch, Triomphe du Nord, Turana, Vicomtesse de Belleval, Virginie, Virgo Maria, Viscountess Folkestone, Whitleyi
Very susceptible	Antione Porteau, Armandine Méchin, Assmanshausen, Belle of France, Charles Toche, General Bedeau, Grandiflora Lutescens, Irma, Lutea Plenissima, Nivea Plenissima, Paradise, Territorial, Victoire Modeste



Fig. 2 Flecking on peony leaves and petiole (*left*), a severe infection on an unopened peony bud *(center*), and large leaf lesions on peony foliage (*right*) caused by *Graphiopsis chlorocephala* (Andrea R. Garfinkel © 2017. All Rights Reserved.)

which the fungus has overwintered as mycelium (McKemy and Morgan-Jones 1991; Meuli 1937; Weiss 1940). Ying and Guihua (1984) found that optimal growth and germination of *G. chlorocephala* conidia occurred at 20–24 °C and Weiss (1940) found that infection of peony could occur from 10 °C to 27.5 °C, but with significant increase in latent period at lower temperatures. Infection can occur on both wounded and unwounded tissues on tree peonies (Ying and Guihua 1984). Free moisture on the leaf is necessary for conidia germination and infection. Conidia can be produced on infected peony foliage throughout the year; however, its occurrence appears to be rare (Meuli 1937).

Management

- ***Sanitation*** – Removal of infected plant material from the previous year's foliage is effective at reducing leaf blotch severity in peony (Meuli 1937; Weiss 1940). Burning the plant debris in the field has also been shown to be effective (Meuli 1937).
- ***Fungicides*** – While testing products for *Botrytis* management on peony, researchers found that fungicides tebuconazole, fluoxastrobin, chlorothalonil, triticonazole, and copper hydroxide and fungicide mixes boscalid + pyraclostrobin and cyprodinil + fludioxonil reduced the severity of leaf blotch (Palmer and Vea 2014).
- ***Resistance*** – Itoh hybrids (see "Resistance" in "Botrytis Gray Mold" above) are reported to be more resistant to leaf blotch than *P. lactiflora* species peonies (Beckerman and Lerner 2009). Meuli (1937) reported that the cultivars Oshkosh White, Felix Crousse, and Livingstone are relatively susceptible to the disease, whereas cultivars Augustin d'Hour, Mathilde de Roseneck, Louis van Houtte, Edulis Superba, Jules Calot, Gigantea, and Humei carnea are more resistant.

2.3 Alternaria Leaf Spot (*Alternaria* spp.)

Geographic occurrence and impact. *Alternaria* spp. have been reported on peony in the United States (Anonymous 1960), Canada (Coulson 1920), China (Zhang 2003), Korea (Cho and Shin 2004), and Bulgaria (Bobev 2009). Most reports of *Alternaria* spp. on peony do not identify the organism to species; however, it appears that at least two species are associated with the herbaceous peony, *A. alternata* (Zhang 2003, China) and *A. infectoria* (Anderson et al. 2009, the Netherlands). Three additional species are reported on the tree peony in China, *A. suffruticosae*, *A. suffruticosicola*, and *A. tenuissima* (Zhang et al. 2008).

Symptoms/signs. The symptoms associated with this fungus are not well documented. At least two references question if the isolation of *Alternaria* from peony is due to a secondary infection by the fungus (Anonymous 1960; Coulson 1923) which is characteristic of *A. alternata* (Rotem 1998). The Texas Plant Disease Handbook (Anonymous n.d.) describes the leaf spots as irregularly shaped ranging in color from purplish-brown to reddish-brown. Later, leaves may yellow and fall off (Texas Plant Disease Handbook). Coulson (1923) suspects *Alternaria* as the causal agent of yellow leaf spots that were irregularly shaped with a clearly defined, dark border. *Alternaria* spp. have been isolated from peony leaves with light brown, irregularly shaped lesions in the US Pacific Northwest (Fig. 3, Garfinkel and Chastagner, unpublished). Leaf spots caused by the three *Alternaria* spp. infecting tree peony are described as circular or irregular, brown to black in color (Zhang et al. 2008).

Biology and epidemiology. No refereed information has been located on the biology and epidemiology of *Alternaria* spp. on peony. Generally, *Alternaria* spp. persist in plant debris or perennial plant structures as mycelium or spores, which are heavily pigmented (Rotem 1998). Conidia are primarily aerially dispersed, but have also shown to be dispersed with water and require free moisture on the surface of a



Fig. 3 Leaf spots on peony associated with an *Alternaria* spp. (Andrea R. Garfinkel © 2017. All Rights Reserved.)

plant to germinate (Rotem 1998). *A. alternata* is typically regarded as a weak pathogen, capable of infecting only weakened plants or plant tissues, and only causes infection on healthy plants in some pathosystems (Rotem 1998).

Management

- ***Cultural*** – Cultural management to prevent wounding of plant tissues and long periods of leaf wetness including wider plant spacing and avoiding overhead irrigation may help prevent Alternaria leaf spot (Rotem 1998). Removing, destroying, or burying plant debris will help to prevent the fungus from overwintering (Rotem 1998).
- ***Fungicides*** – Information on the effectiveness of fungicides in controlling Alternaria leaf spot is lacking, although there are many products registered for use for this pathogen in peonies in the United States including iprodione, thiophanate-methyl, propiconazole, trifloxystrobin, chlorothalonil, fluoxastrobin, azoxystrobin, pyraclostrobin, fludioxonil, and mancozeb.

2.4 Powdery Mildew (*Erysiphe* spp. and *Podosphaera* spp.)

Geographic occurrence and impact. Powdery mildew has been reported on peonies in Iran (Khodaparast and Abbasi 2009), the former Soviet Union (Amano 1986), Korea (Cho and Shin 2004; Shin 2000), China (Braun and Takamatsu 2000; Tai 1979; Zheng and Yu 1987), Japan (Kobayashi 2007; Takamatsu et al. 2006), Hungary (Braun 1995), and the United States (Anonymous 1960; French 1989).

Symptoms/signs. Powdery mildew is characterized by conspicuous white circles or a mat of mycelium on the surface of aboveground parts of the plant (Fig. 4). Later in the season, small, round, black, fruiting structures, called chasmothecia, can be seen on top of or buried within the white mycelium (Fig. 4). Infected peony tissues will sometimes turn purple underneath the mycelium in response to the presence of the pathogen.

Biology and epidemiology. Powdery mildews are considered highly specialized, obligate biotrophs which means that they are mostly host-specific and require living host tissue for growth and development. Powdery mildews can survive as mycelium or as chasmothecia, which contain sexually produced ascospores. Mycelium, conidia, or ascospores can serve as the source of initial inoculum in the spring. Asexual conidia are produced on the mycelium throughout the year and serve as a source of secondary infection. Powdery mildew spores, unlike many fungi, do not require free moisture to germinate.

Management

- ***Cultural*** – Removal of infected plant debris during and at the end of the year can help prevent inoculum buildup. Increasing plant spacing to provide as much sun



Fig. 4 A severe powdery mildew infection on a peony seed pod and foliage (*left*) and small black (mature) and orange (immature) chasmothecia among the mycelial mat on the lower leaf surface (*right*) (Andrea R. Garfinkel © 2017. All Rights Reserved.)

exposure to foliage as possible may also help reduce infection (Glawe and Grove 2015).

- ***Fungicides*** – Fungicides typically effective against powdery mildews include those that contain sulfur or petroleum-based oils, sterol-biosynthesis inhibitors (DMIs), or quinone outside inhibitors (QoIs) (Glawe and Grove 2015).

2.5 Phytophthora Blight and Phytophthora Root Rot (*Phytophthora cactorum* [Lebert & Cohn] J. Schröt)

Geographic occurrence and impact. Phytophthora blight on peonies has been officially reported in the United States (Anonymous 1960; Thurston and Orton 1921), Canada (Conners and Savile 1948), and Japan (Kobayashi 2007; Villa et al. 2006).

Symptoms/signs. Symptoms of Phytophthora blight can be easily confused with Botrytis gray mold as aboveground symptoms involve a dieback of stems and foliage. However, dieback caused by *Phytophthora* is sunken and black as compared to a *Botrytis* infection which is typically brown in color. Furthermore, incubation of the tissue in moist conditions does not result in the appearance of gray conidiophores; a sparse white mycelial mat can sometimes be observed. *Phytophthora cactorum* can infect the enlarged roots of the peony and will form chocolate brown lesions on the roots with defined margins (Fig. 5). Severely infected plants may fail to send up shoots in the spring.



Fig. 5 Lesion on a fleshy peony root caused by *Phytophthora cactorum* (Andrea R. Garfinkel © 2017. All Rights Reserved.)

Biology and epidemiology. *Phytophthora cactorum* is not a true fungus, but a fungal-like oomycete or "water mold." *P. cactorum* has over 200 plant host species and grows optimally at 25–30 °C (Erwin and Ribeiro 1996; Kim et al. 2003). Hyphae differentiate to produce sporangia that can either germinate directly or from which swimming zoospores emerge. These zoospores have been shown to be infective on the leaves of tree peonies (Kim et al. 2003). Some isolates can produce chlamydospores which can persist in soil and germinate in favorable conditions (Erwin and Ribeiro 1996). The organism is also reported to overwinter as mycelium and oospores (Erwin and Ribeiro 1996).

Management

- ***Sanitation*** – Plants with severe Phytophthora blight of the crown should be removed from the field along with surrounding plants and soil which may have become infected or infested by the pathogen.
- ***Fungicides*** – Fungicides can be applied to foliage or as a drench. Little information is available on the fungicide control of *Phytophthora* on peonies. General recommendations for fungicides to control *P. cactorum* include the use of metalaxyl or fosetyl-Al (Erwin and Ribeiro 1996) if registered for use on peony. Chemistries effective in managing *Phytophthora* spp. in other pathosystems include aluminum tris (O-ethyl phosphonate), captan, cyazofamid, dimethomorph, famoxadone + cymoxanil, fenamidone, fluopicolide, fluoxastrobin + myclobutanil, mandipropamid, mefenoxam, metalaxyl, pyraclostrobin, and fungicides containing phosphorous acids (Palmer and Vea 2010). Fungicide resistance can be a problem when using fungicides to manage Phytophthora diseases, so it is important to rotate

materials with different modes of action (see ▶ Chap. 7, "Fungicides and Bio-controls for Management of Florists' Crops Diseases").

- ***Cultural*** – Avoid the presence of standing water in the field which is favorable to pathogen development and movement of zoospores. Overhead irrigation should be avoided as splash can disperse the pathogen within a field.

2.6 Anthracnose (*Gloeosporium* spp. and *Colletotrichum* spp.)

Geographic occurrence and impact. Anthracnose has been reported on peonies in the United States (Anonymous 1960; Weiss 1940), Korea (Cho and Shin 2004; Korean Society of Plant Pathology 1998), Japan (Kobayashi 2007), and New Zealand (Pennycook 1989).

Symptoms/signs. The anthracnose fungi can cause lesions and cankers on all aboveground parts of peony. Cankers on the stems of peony are elongate and gray in color and often cause severe twisting or curling of the stem (Fig. 6). Pink conidomata can sometimes be observed in the cankers under favorable conditions. Foliar leaf spots and infections of flowers and flower buds are similarly gray in color and can cause irregular growth of plant tissues (Fig. 6) or interfere with flower development.

Biology and epidemiology. "Anthracnose" is a general term used to describe a disease that "appears as a black, sunken, leaf, stem, or fruit lesions, caused by fungi that produce their asexual spores in an acervulus" (Agrios 2005). Anthracnose fungi are generally thought to survive in leaf litter; therefore, infested debris can serve as an important source of initial inoculum. Anthracnose fungi on peony appear to



Fig. 6 Cankers (*left*) (Andrea R. Garfinkel © 2017. All Rights Reserved.) on and twisting of stem below bud (*right*) (Published with kind permission of J. Mullane © 2017. All Rights Reserved.) on peony caused by *Colletotrichum* spp.

sporulate under favorable conditions during the season; thus, secondary inoculum may play a role in the disease cycle.

Management. Follow general disease management strategies for fungi including cultural practices to reduce leaf wetness instance and duration (see Management of *Botrytis* above for specific tactics), and remove and destroy all foliage at the end of the season.

2.7 Crown Rot (*Sclerotium rolfsii* Sacc. and *Sclerotium delphinii* Welch)

Geographic occurrence and impact. Crown rot or southern blight has been reported on peonies in North America (Anonymous 1960; Parris 1959) and Japan (Kobayashi 2007). Limited information is available on the impact of this disease on peonies, but *Sclerotium rolfsii* (teleomorph *Athelia rolfsii*) and the closely related *S. delphinii* can cause extensive mortality on a number of ornamental geophytes.

Symptoms/signs. Symptoms will appear as small, water-soaked lesions at the base of a stem, quickly expanding to girdle the stem causing wilt and necrosis of the foliage above. As the disease progresses, a white mycelial mat can be observed on the stems and in the soil surface, within which, small orange-tan to brown sclerotia are formed (Fig. 7).



Fig. 7 Immature white to orange colored sclerotia (*arrow*) of *Sclerotium rolfsii* developing at the base of a bulbus iris shoot (Andrea R. Garfinkel © 2017. All Rights Reserved.)

Biology and epidemiology. The crown rot fungi persist in the soil on dead organic matter as mycelia or in the soil as sclerotia that germinate to produce mycelia. The mycelia directly infect crown tissues of the plant host. The production of basidiospores on infected tissues and on mycelium has been observed; however, it is thought that their production is rare (Mullen 2001). Thus, it is not common to practice management strategies based on secondary infection by basidiospores. Instead, spread is thought to occur by mycelial growth to adjacent plants and movement of infected plant material and infested soil. *S. rolfsii* and *S. delphinii* are favored by warm, humid conditions, with *S. rolfsii* specifically limited by low temperatures (Xu et al. 2008).

Management

- ***Fungicides*** – Preventing introduction of the crown rot pathogens by ensuring clean planting stock is an important element of disease management. Chemical treatment of planting stock can be an option to ensure clean planting materials; however, information on the chemical control of this disease on peonies is not available. Soil applications of azoxystrobin, flutolanil, PCNB, and/or preplant dips in PCNB have been used to control crown rot (Chastagner and Riley 2002; Chastagner et al. 1990). Soaking bulbs, such as iris, in hot water plus formalin is also recommended.
- ***Sanitation*** – Removal of infected plant material and surrounding soil is recommended to limit spread of the pathogen.
- ***Soil disinfestation*** – Preplant soil fumigation with metam-sodium has been effective in limiting disease development on other geophytes. Soil solarization or sterilization can be effective as preplant treatments if high temperatures and humidity can be maintained for a sufficient duration to kill sclerotia (Mullen 2001).

2.8 White Stem Rot (*Sclerotinia sclerotiorum* [Lib.] deBary)

Geographic occurrence and impact. White stem rot (also called "white mold" or "Sclerotinia stem rot") has been reported on peonies in the United States and Canada (Anonymous 1960; Ginns 1986). It has also been reported on tree peonies in Korea (Cho and Shin 2004).

Symptoms/signs. Plants infected with *S. sclerotiorum* initially display aboveground symptoms of wilt and necrosis. The pathogen can later be seen growing at the plant crown as a fluffy white mycelium. Large black sclerotia (white when immature) can be seen forming on the surface or inside the plant tissue. *S. sclerotiorum* is also able to cause lesions on foliage and flowers that turn brown and can become covered in a white, fluffy mycelial growth under moist conditions.

Biology and epidemiology. *S. sclerotiorium* overwinters as sclerotia formed on or inside plant tissue. Sclerotia germinate either to produce mycelium or apothecia which contain sexual ascospores that become airborne and can infect senescent plant

tissues. *S. sclerotiorum* does not produce conidia; thus, there is no repeating cycle throughout the year. However, healthy plants or plant parts can be infected if brought in contact with colonized host tissue or by mycelial spread on the soil surface.

Management

- ***Fungicides*** – Little information is available relating to fungicides that are effective in controlling white mold on peonies. On other crops, fungicides such as thiophanate-methyl, iprodione, boscalid, fludioxonil + cyprodinil, and fluazinam are recommended and can reduce the number of sclerotia in soil.
- ***Cultural*** – Sanitation to remove infected plant material and senescent plant tissues, especially flower petals, is crucial for preventing pathogen spread. Infected plants should be removed along with surrounding soil to remove any overwintering sclerotia. General fungal disease management strategies to decrease periods of leaf wetness (see Management of *Botrytis* above for specific tactics) can also be effective at reducing ascospore germination.

2.9 Fungal Root Rots (Multiple Species)

Geographic occurrence and impact. The fungal species reported to cause root rot of peony include *Armillaria mellea* (United States, Anonymous 1960), *Rhizoctonia* spp. (United States, Anonymous 1960; Canada, Ginns 1986; Korea, Cho and Shin 2004; Bulgaria, Bobev 2009), *Verticillium* spp. (United States, Anonymous 1960), *Thielaviopsis basicola* (United States, Anonymous 1960; French 1989), *Fusarium* spp. (United States, Anonymous 1960), and *Rosellinia necatrix* (Japan, Kobayashi 2007; United States, French 1989; Greece, Pantidou 1973).

Symptoms/signs. The specific information about these root-rotting pathogens on peony is limited. Aboveground symptoms of root rots typically first display as general wilting, chlorosis, or necrosis as a result of damage to root tissues. Many soilborne fungal pathogens will display characteristic disease patterns in the field with circular foci. Other symptoms are disease-specific. Roots infected with *Armillaria* often will have thick, black, specialized fungal structures called rhizomorphs, which resemble fine root hairs. Root lesions caused by *Rhizoctonia* are typically brown to red in color. *Verticillium* is diagnosed by discoloration or darkening of the vascular tissue in roots and stems. Roots infected by *Thielaviopsis* often show dark brown to black lesions due to the presence of pigmented chlamydospores that are visible with a microscope in the host tissue. In tree peony, Fusarium root rot causes reddish brown to dark brown lesions on the root (Guo et al. 2012). Infection by *Rosellinia* is characterized by a very dense, light colored mycelial mat growing up around the base of the stems on the soil surface.

Biology and epidemiology. While all are soilborne plant pathogens, the biologies of the root-rotting fungi are unique. *Armillaria* largely relies on underground spread through mycelial transfer by root-to-root contact and by rhizomorphs. Like

Armillaria, spores in *Rosellinia* seem to have little importance in the disease cycle with the majority of spread occurring underground by infected roots coming in contact with healthy roots. *Rhizoctonia*, *Verticillium*, and *Thielaviopsis* form persistent structures called sclerotia, microsclerotia, and chlamydospores, respectively. These structures germinate to produce mycelia which can infect healthy plant tissues. Root-to-root contact between infected and healthy roots can also spread these pathogens. Like most root-infecting pathogens, infected planting stock can play a large role in introducing the pathogen to a new field planting site.

Management. Ensuring clean planting material is a best first defense against root-rotting pathogens. For many of these pathogens, rogueing infected plants is recommended, as well as the closest adjacent soil and plant(s), which, although they may not be showing symptoms, could be infected and serve as a source of further spread of the pathogen. Cultural management strategies to alter the pH, temperatures, or saturation of the soils can be successful for some soilborne pathogens. Fungicide drenches or fumigants are available and effective for some, but not all, soilborne pathogens. It is important to always check the pesticide label for proper use.

2.10 Peony Red Spot (*Mycocentrospora acerina* [Hartig] Deighton)

Geographic occurrence and impact. This disease has only been officially reported in Chile (Gilchrist et al. 2015); however, the geographic distribution is likely much larger as it has also been found on peony in the continental United States and Alaska (Garfinkel and Chastagner unpublished data). As it has only recently been described on peony (Gilchrist et al. 2015), the impact of peony red spot is yet to be determined. The name "red spot," given to this disease by Gilchrist et al. (2015), is not to be confused with leaf blotch (see earlier in this chapter) which is sometimes referred to as "red spot" in older literature (Weiss 1940).

Symptoms/signs. *M. acerina* can cause disease on all parts of the peony except the flower, although infections of the first few petals of the flower bud have been observed (Gilchrist et al. 2015). On aboveground plant parts, symptoms are apparent as small (<1 cm), elongated red spots with light-colored centers, turning brown to black under cool, moist conditions. Multiple lesions may appear next to each other giving a streaked reddened look (Fig. 8), but lesions do not appear to coalesce upon close inspection. Infections on leaf margins cause twisting and cracking of the leaf during expansion. Lesions on the enlarged roots of the peony begin as orange, water-soaked spots and then turn black (Gilchrist et al. 2015).

Biology and epidemiology. There are no known studies on the biology and epidemiology of this disease specifically on peony. In caraway (*Carum carvi*), conidia of *M. acerina* have been shown to be primarily splash-dispersed at temperatures from 15 °C to 20 °C (Evenhuis et al. 1997). Due to the short distance of dispersal of this



Fig. 8 Closely packed red stem lesions on a peony stem caused by *Mycocentrospora acerina* (Andrea R. Garfinkel © 2017. All Rights Reserved.)

fungus in caraway, the authors suspect that infection likely comes from inoculum sources in the field, such as infected planting material or infested soil (Evenhuis et al. 1997). Since peony roots have been shown to be susceptible to this fungus (Gilchrist et al. 2015), it is possible, however unconfirmed, that it has the potential to move with rootstock. This fungus has many native hosts that could also contribute to initial inoculum in the field (George 2014). Gilchrist et al. (2015) were successful at initiating infections with mycelial plugs on both wounded and unwounded tissue; there are no reports of infections on peony initiated from conidia. The fungus can persist in soils and decayed plant tissues as chlamydospores which could represent important sources of initial inoculum once the pathogen has been introduced into a field (Wall and Lewis 1978).

Management

- ***Cultural*** – Spread can be facilitated by splash from irrigation (George 2014); thus, overhead irrigation should be avoided.
- ***Sanitation*** – Keeping weedy hosts out of fields can help reduce inoculum buildup of the pathogen.
- ***Prevention*** – Since the pathogen is known to infect roots, it may be important to ensure clean planting material as to not introduce the pathogen to a new field. Although there is no specific information available, treatments, including hot-water or fungicide dips, may be effective at managing rootstock infection.

2.11 Leaf Rust (*Cronartium flaccidum* [Alb. & Schw] Wint.)

Geographic occurrence and impact. Peony rust caused by *Cronartium flaccidum* has been reported from parts of Europe and Asia (Japan, Hiratsuka et al. 1992; China, Tai 1979; Poland, Mulenko et al. 2008; Korea, Korean Society of Plant Pathology 1998; Finland, Kaitera and Hiltunen 2011).



Fig. 9 Early season symptoms caused by *Cronartium flaccidum* on peony (*upper left*), leaves killed by *C. flaccidum* (*upper right*), uredinia with urediniospores of *C. flaccidum* on the underside of young peony leaves (*lower left*), and telia with teliospores of *C. flaccidum* on the underside of peony leaves (*lower right*) (Venche Talgø © 2017. All Rights Reserved.)

Symptoms/signs. In early season, brownish-purple spots occur on the upper leaf surfaces of peony (Fig. 9). In severe cases, the edges of infected leaves become curled, and the whole leaf may turn brown and die (Fig. 9). Signs of the fungus can also be seen at certain stages in the rust lifecycle. In spring, the underside of young peony leaves may become covered by yellow to light brown uredinia with urediniospores (Fig. 9). Later in the season, dark clusters of telia with teliospores form on infected leaves (Fig. 9) (Gjærum 1974; Toppe et al. 2007).

Biology and epidemiology. Like many rust fungi, *C. flaccidum* goes through multiple spore cycles and hosts to complete its lifecycle. Urediniospores are formed on peony in the spring, and then teliospores are formed later in the season on infected leaves. The teliospores germinate in the autumn to form basidia with basidiospores. The basidiospores spread by wind to newly emerged pine needles (or other alternate hosts such as *Melampyrum* spp. or *Vincetoxicum* spp.) where their germ tubes enter via the stomata. The fungus overwinters within the tissue of the alternate hosts. In the

spring, aeciospores emerge from infected host tissue and spread by wind to peony (Gjærum 1974; Toppe et al. 2007).

Management

- ***Sanitation*** – Removing alternate hosts, such as pine, in the vicinity of the peony may help, but will not guarantee the avoidance of rust attacks, as aeciospores appear to be capable of spreading long distances (Gjærum 1974; Toppe et al. 2007). Removal of infected peony leaves may keep the inoculum level down by reducing the amount of infection that occurs on alternate hosts (Gjærum 1974; Toppe et al. 2007).
- ***Fungicides*** – Chemical use of fungicides on newly emerged foliage may help protect against rust infection (Gjærum 1974; Toppe et al. 2007).
- ***Resistance*** – Some peony species and cultivars appear resistant against the fungus (Gjærum 1974; Toppe et al. 2007); however, there is no published list of resistant cultivars.

Other fungal diseases of peony include:

- **Leaf rust** (***Aecidium* spp.**) (United States, Farlow and Seymour 1888; China, Tai 1979)
- **Leaf spot** (***Cercospora* spp.**, ***Pseudocercospora* spp.**) (United States, Anonymous 1960; Canada, Ginns 1986; China, Tai 1979; Korea, Korean Society of Plant Pathology 1998)

For a current list of fungi that have been found associated with peony, visit http://nt.ars-grin.gov/fungaldatabases/fungushost/FungusHost.cfm and enter host name "Paeonia."

3 Bacterial and Phytoplasma Diseases

3.1 Bacterial Diseases (Multiple spp.)

Geographic occurrence and impact. *Agrobacterium tumefaciens* (Anonymous 1960), *Xanthomonas* spp. (Oliver et al. 2012; Putnam 2011), and *Pseudomonas syringae* (Putnam 2011) have been reported on peonies in the United States.

Symptoms/signs. Symptoms associated with *Pseudomonas* and *Xanthomonas* bacterial diseases of peony include red to purple leaf spots, which can progress to blight, from which bacterial streaming is evident under microscopic investigation. Symptoms of crown gall, caused by *Agrobacterium tumefaciens*, include tumorlike growths on the aerial stems of the plant.

Biology and epidemiology. Specific information on the biology and epidemiology of bacterial plant pathogens of peony is limited. Most plant pathogenic bacteria

survive in leaf litter or in perennial plant structures; however, some can survive in the soil, on harvesting or processing tools, or in association with their vector (Vidaver and Lambrecht 2004). Plant pathogenic bacteria can be disseminated by wind or water and enter through wounds or natural openings on plant tissues or by their insect vector (Vidaver and Lambrecht 2004). Plant pathogenic bacteria can also be introduced on propagative material. Environmental conditions for disease development are pathogen-specific.

Management. General disease management strategies for plant pathogenic bacteria include avoiding overhead irrigation; ensuring good air circulation within the plant canopy; exclusion by ensuring clean planting stock; using copper-based fungicide sprays or antibiotic treatments; burial, removal, or destruction of plant debris; and the use of antagonistic biocontrol agents (Anonymous 2016; Vidaver and Lambrecht 2004).

3.2 Phytoplasma Diseases (*Candidatus* Phytoplasma spp.)

Geographic occurrence and impact. "*Candidatus* Phytoplasma fraxini" has been identified in herbaceous peony in Chile (Arismendi et al. 2011). "*Candidatus* Phytoplasma solani" has been identified in tree peonies in China (Gao et al. 2013) and in *P. tenuifolia* in Serbia (Adamovic et al. 2014).

Symptoms/signs. Herbaceous peonies in Chile found to be infected with "*Candidatus* Phytoplasma solani" had malformed, rolled, necrotic leaves, green stripes on the petals, and desiccation of the developing flower bud (Arismendi et al. 2011). In tree peonies, the phytoplasma was found associated with plants that were stunted in growth and showed mottling or bleaching of the foliage (Guo et al. 2012) (Fig. 10). The phytoplasma in *P. tenuifolia* caused reddening of the lower foliage (Adamovic et al. 2014).



Fig. 10 Mottling on tree peony foliage caused by a phytoplasma (Gao et al. 2013)

Biology and epidemiology. Phytoplasmas are phloem-restricted, obligate bacterial plant pathogens and insect symbionts. Phytoplasmas are transmitted by phloem-feeding insects, especially leafhoppers. Phytoplasmas can also be transmitted through vegetative propagation. "*Candidatus* Phytoplasma fraxini" is most commonly known as the causal organism for ash yellows.

Management. Management of phytoplasmas is usually achieved by management of the insect vector. Since peonies are vegetatively propagated, care should be taken to avoid planting or dividing phytoplasma-infected plants.

4 Viral Diseases

4.1 *Tobacco rattle virus* (TRV; Family *Virgaviridae*, Genus *Tobravirus*)

Geographic occurrence and impact. Also referred to as "peony ringspot virus" or "peony mosaic virus," TRV has been reported in herbaceous peonies the United States (Anonymous 1960; Fisher 2012; Robertson et al. 2009), Japan (Chang et al. 1976), Lithuania (Samuitienė et al. 2009), and New Zealand (Jones and Young 1978; Pennycook and Fry 1989) and in tree peonies in China (Ningsheng and Yunfeng 1990). Peony ringspot symptoms have also been recorded in Italy (Pisi and Marani 1976), Korea (Korean Society of Plant Pathology 1998), and Denmark (Jensen and Dahl 1961); however, TRV has not been confirmed as the cause in these cases. TRV has a wide host range of over 400 plant species, including many ornamental cut flowers, and has been reported in many countries globally.

Symptoms/signs. TRV symptom expression in peony is highly dependent on cultivar and environmental conditions (Samuitienė et al. 2009). Symptoms can be expressed as concentric ringspots of alternating green and yellow (Fig. 11) or yellow mosaic, mottling, banding, or blotching (Fisher 2012; Samuitienė et al. 2009). Like many viruses, symptoms in TRV infected peonies can be transient, with infected plants showing no symptoms.

Biology and epidemiology. TRV is transmitted by nematodes in the genera *Trichodorus* and *Paratrichodorus*. The ability of these nematodes to transmit the virus appears to be strain-specific (Brunt 1995). Certain strains of TRV can also be transmitted mechanically. The strains infecting peonies are not known. The virus can be introduced to new plantings in rootstock.

Management. Management of TRV begins with clean planting stock and soil disinfestation. In areas where a nematode vector is present, rogueing of plants showing



Fig. 11 Ring spots on peony foliage caused by *Tobacco rattle virus* (Gary A. Chastagner © 2017. All Rights Reserved.)

symptoms is key to reducing spread to surrounding, healthy plants. Sterilization of pruning and harvesting tools may be important in limiting mechanical spread.

4.2 Lemoine Disease (Suspected Virus)

Geographic occurrence and impact. Reports of this suspected viral disease come from the United States (Anonymous 1960; Brown 1940); however, the disease was first noticed on plants originating from Europe (Hollingsworth n.d).

Symptoms/signs. Symptoms of Lemoine disease include irregular swellings or irregularities on the fleshy roots of the peony plant (Fig. 12). A cross section of the roots shows yellow inclusions in the swollen portions of the root. Diseased plants are also reported to have severe reductions in vigor and foliar growth and do not flower. However, the disease appears to have very little negative effects on the flowering or vigor of the peony cultivar Alice Harding (D. Hollingsworth, personal communication). The swellings produced by Lemoine disease are distinct from those produced by nematodes as nematodes generally affect the small feeder roots of the peony and the nematode females can be observed in a cross section of the root tissue.

Biology and epidemiology. The cause of Lemoine disease is unknown; however, some reports suspect it is caused by a virus (Anonymous 1960, 2015; D. Hollingsworth personal communication 2015). Older reports consider Lemoine disease to be a long-term reaction to former colonization of the roots by nematodes (Brown 1940).



Fig. 12 Swellings (*arrows*) on a peony root characteristic of Lemoine Disease (*left*) and cross sections of a healthy peony root (*top right*) and roots with Lemoine Disease showing irregular yellow inclusions (*bottom right*) (Andrea R. Garfinkel © 2017. All Rights Reserved.)

Observations suggest that Lemoine disease is spread mechanically through sap during root division, but there are no known vectors (Anonymous 2015; D. Hollingsworth personal communication 2015).

Management. Since the cause of this disease is unknown, any management strategies are based on speculation and personal threshold for risk. The most conservative growers will remove and destroy any plants showing symptoms of Lemoine disease. Others are careful to sterilize tools when transitioning between plants with Lemoine disease and those without symptoms. A report in the American Peony Society Bulletin (Garman 1923) suggests that Lemoine disease can be controlled by severe pruning of peony roots as to remove any enlarged portions of the root, leaving at least one bud per stem piece.

Other virus diseases reported on *P. lactiflora*-type peonies include:

Alfalfa mosaic virus (Italy, Bellardi et al. 2003)
Tomato spotted wilt virus (USA, Parrella et al. 2003)
Cucumber mosaic virus (France, Cardin et al. 2010)
Leaf curl (USA, unidentified virus, Brierley and Lorentz 1957)
Crown elongation (USA, suspected virus, Horst 2012)

5 Nematode Diseases

5.1 Root-Knot Nematode (*Meloidogyne* spp.)

Geographic occurrence and impact. Root-knot nematodes have been reported in Italy (Volvas et al. 2010), Korea (Choo et al. 1995; Park et al. 1998), China (Fei et al. 1990), the Netherlands (Anonymous 1978), Canada (Baker 1959), New Zealand (Knight et al. 1997), and the United States (Malek 1974). Root-knot nematodes are a quarantine pest in many peony-producing countries.

Symptoms/signs. Aboveground symptoms of peonies infected with root-knot nematodes include stunting, wilt, and chlorosis which can develop over a number of years. Plant roots infected with root-knot nematodes show round swelling, or galls (root knots), typically most noticeable on the small feeder roots. Female nematodes are visible under a microscope in cross sections of the root swellings. Root-knot nematode infestations will usually appear as circular foci in the field.

Biology and epidemiology. Root-knot nematodes develop within eggs that are born in gelatinous masses exuded from female nematodes who established successful infections on roots the previous generation. The nematode is released from the egg and enters the root tissue and establishes feeding sites, causing visible swellings on the roots. The female nematode becomes sedentary, mates, and dies associated with the host roots. A subsequent generation can be born in as few as 2 weeks, depending on species and environment (Mitkowski and Abawi 2003). Eggs can remain viable in the soil for a year (Mitkowski and Abawi 2003).

Management

- ***Physical*** – Hot-water dips of rootstocks have been shown to be effective at controlling root-knot nematode on peony (Nelson 1931; Taylor 1958). This practice is commonly used in the international peony rootstock trade.
- ***Nematicides*** – Chemical bare root dips of thionazin have shown to be effective treatment of rootstocks (Malek 1974). Some fumigants are effective as preplant treatments in areas known to contain root-knot nematodes (Mitkowski and Abawi 2003).

5.2 Foliar Nematode (*Aphelenchoides* spp.)

Geographic occurrence and impact. Foliar nematodes have been reported on peony in the United States (Handoo 2012), Japan (Yokoo and Matsusaki 1967), and Korea (Choo et al. 1995) and have been detected in the buds of shipments of peony from the Netherlands to Poland (Karnkowski 1999) and China to the United States (tree peony, Handoo 2012).

Symptoms/signs. Foliar nematodes cause purple, vein-delimited damage on foliage (Fig. 13). A severe infection can cause dieback of foliage that turns brown as the tissue decays.



Fig. 13 Angular, vein-delimited leaf damage (*left*) and severe dieback (*right*) to peony foliage caused by foliar nematodes (L. du Toit © 2017. All Rights Reserved.)

Biology and epidemiology. Adult foliar nematodes persist within dried plant debris in a desiccated state. In the presence of water, nematodes can become active and move in films of water. Movement can be further facilitated by splashing water. It is also suspected that foliar nematodes can be spread when infected tissues become dislodged and fall onto or are moved onto healthy plants (Kohl 2011). Foliar nematodes have been shown to move long distances on plant material, such as cut peony flower buds (Handoo 2012; Karnkowski 1999).

Management

- ***Cultural*** – Practices to reduce incidence and duration of leaf wetness and water splash help to limit nematode spread along the plant surface.
- ***Physical*** – Hot-water drenches have been shown to be effective against foliar nematodes in *Hosta* (Jagdale and Grewal 2004).
- ***Nematicides*** – Few pesticides are available for use on foliar nematodes, and those that are registered have limited efficacy against nematodes within the plant tissue (Kohl 2011). Experimental treatments of peroxyacetic acid and insecticidal soap in addition to a *Pseudomonas cepacia* biocontrol were shown to be effective at controlling foliar nematodes in *Hosta* (Jagdale and Grewal 2002).

Other nematodes reported on peony:

Root lesion nematode (***Pratylenchus*** **spp**.) (Knight et al. 1997; Park et al. 1998)

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Diseases of Proteaceae

23

Brett A. Summerell

Abstract

The Proteaceae are a southern hemisphere family of plants found predominantly in southern Africa and Australia. Included in the family are *Protea*, *Leucospermum*, and *Leucodendron* from southern Africa and *Banksia*, *Telopea*, and *Grevillea* from Australia all of which are important cut flower crops. All of these genera are susceptible to a range of diseases, especially root rot diseases caused by *Phytophthora*, *Armillaria*, and *Fusarium* which can limit production and leaf spot diseases which both limit production and impair the aesthetics of the cut flower. Management of these diseases is complex and includes chemical, biological, cultural, and physical control, as well as host resistance.

Keywords

Phytophthora spp • Leaf spots • *Fusarium* spp • Mycosphaerella leaf blotch • Integrated disease management

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R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_22

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1 Introduction

The Proteaceae is one of the most iconic groups of plants found in the southern hemisphere. It is widespread, reflecting its Gondwanic heritage (Barker et al. 2007), and is highly speciose adapting to diverse ecosystems throughout southern Africa, Australia, and to a lesser extent South America (Rebelo 2001; Weston and Barker 2006). Members of the family are also found in New Zealand, New Caledonia, Fiji, and Vanuatu, and there are a small number of species in Southeast Asia (Weston and Barker 2006). There are approximately 80 genera in the family, and these can range from ground cover trailing species through to very large rainforest trees (Rebelo 2001; Weston and Barker 2006).

Of the 80 genera only a relatively small number are grown for cut flower production, mainly species in *Protea*, *Leucodendron*, *Leucospermum*, *Banksia*, *Telopea*, and *Grevillea*. There are other species in some of the other genera that are cultivated, but these are rare and commercially insignificant despite being of interest from a plant pathological perspective. All of the species grown are essentially woody shrubs or small trees, perennial, and clonally propagated through cuttings or, occasionally, tissue culture (Crous et al. 2013). Due to the nature of the species, orchards are long lived, and hence ongoing disease management is extremely important and in some circumstances quite problematic (Greenhalgh 1981; Crous et al. 2013).

Where data are available, specific information on the management of diseases of Proteaceae is presented in the following sections. General management strategies may be found in the introductory chapters on integrated disease management.

2 Fungal and Fungus-Like Diseases

2.1 Leaf Spot Diseases (A Broad Range of Fungal Species)

Geographic occurrence and impact. These diseases have been reported in all regions where species of Proteaceae are grown and cultivated and in all regions where these plants grow naturally (Crous et al. 2000, 2008; Taylor et al. 2001a, b). They are a major constraint to cultivation of Proteaceae as they both reduce growth and reduce the esthetic quality of the cut flowers.



Fig. 1 Phyllosticta leaf spot on *Telopea* sp. (*left*); Mycosphaerella leaf spot on *Protea cynaroides* (*right*) (B.A. Summerell © 2017. All Rights Reserved.)

Symptoms/signs. Early symptoms on leaves consist of small necrotic spots which expand and coalesce potentially resulting in large areas of the leaf turning necrotic and dead. In some cases the fruiting structures of the fungus may be evident in the leaf spots as small black pinhead-sized dots. There are very subtle differences between some of the leaf spots caused by different fungal species, but generally the symptoms overlap considerably and generally the control approaches are consistent (Fig. 1).

Biology and epidemiology. The fungal species of major concern are *Teratosphaeria*, *Batcheleromyces*, *Brunneosphaerella*, *Coleroa*, *Pestalotiopsis* (Swart et al. 1999b), *Phyllosticta*, and *Ramularia*. Many of these species were formerly referred to under the broad heading of *Mycosphaerella* spp., and the older literature will refer to these pathogens by this name (Taylor and Crous 2000; Crous et al. 2009). A comprehensive account of the fungal species causing these diseases is provided in Crous et al. (2013).

Generally all of these fungi are favored by warm, humid conditions that allow sporulation and subsequent infection, although some species of *Teratosphaeria* appear to require a period of lower temperature (c. 15 °C/59 °F) in order for the infection process to occur. Infection propagules are a mixture of ascospores and conidia, with the latter often produced from pycnidia. It is presumed that persistence of the fungi occurs via ascocarps and mycelia in host tissue and trash in plantations.

Management

- ***Cultural practices*** – Reduction of relative humidity and the duration of plant tissue wetness are thought to assist in reducing opportunities for infection, and consequently drip irrigation is likely to be beneficial when used.
- ***Sanitation*** – Removal of senescing and dead plant tissue that provide a ready substrate for persistence of these fungi is likely to be important for the management of these diseases.

- ***Fungicides*** – Standard fungicide applications for the control of leaf spots in other florists' crops are likely to be effective depending on whether the fungicide is registered for use on Proteaceous crops (for additional information, refer to ▶ Chap. 7, "Fungicides and Biocontrols for Management of Florists' Crops Diseases").

2.2 Botryosphaeria Leaf Blight and Stem Canker (*Neofusicoccum* sp.)

Geographic occurrence and impact. Cankers and dieback of Proteaceae are among the most important and persistent diseases that afflict all of the cultivated Proteaceae around the world. Affected plants lose productivity, and the speed of the dieback can vary considerably depending on environmental conditions. Diagnosis is difficult because non-symptomatic plants can harbor the pathogen for some time without any obvious signs (Swart et al. 2000).

Symptoms/signs. Typically symptoms include cankers on limbs and branches of the bushes with a resultant dieback of the affected part of the plant. In some situations the whole plant may be affected and will die. There are indications, based on observations on Proteaceae but also from extrapolation from other perennial species, that pruning wounds and other damage facilitates entry into the plant.

Biology and epidemiology. There are a number of species that have been associated with these diseases on a range of Proteaceae, and the taxonomy of this group is complex. Many are referred to as *Neofusicoccum* species, but *Diplodia seriata* and *Lasiodiplodia theobromae* are also included in this group of pathogens on members of this plant family (Denman et al. 2003; Crous et al. 2006). All are considered wound pathogens that infect via pruning wounds, tears, and cracks and insect injury (there are a number of stem borers that affect Proteaceae species). Sporulation occurs in late spring and early summer depending on rainfall/irrigation frequency, and rain splash appears to be the most frequent method of dispersal. After infection the fungus may colonize parts of the plant and remains apparently dormant for some time causing no obvious symptoms. Stress events like drought stress or frosts appear to allow the fungus to begin colonizing the plant and eventually increase biomass and cause the characteristic symptoms.

Management

- ***Cultural practices*** – Cultivation practices that reduce the opportunities for the plant to be wounded are highly recommended. Given that the nature of the cultivation and harvesting requires cutting and wounding perennial woody material, this can be especially problematic in the Proteaceae. This includes ensuring that crop and plantation architecture are designed to allow rapid drying of cut surfaces and training harvesters to ensure that cuts are clean, cutting implements are clean, and stem bases and trunks are not damaged in harvest, weed control, and

other management routines. Practices that minimize the plants from being stressed are also critical. This can include irrigation frequency and management and use of mulches to prevent soil drying out and insect management.

- ***Sanitation*** – Removal of senescing and dead plant tissue that provide a ready substrate for these fungi to persist is important for the management of the disease. It is also important to ensure that dead and dying nonproductive plants are removed quickly and hygienically. Dead material, especially woody material, needs to be removed from the plantation or burnt to ensure there is no new inoculum available for reinfection. This may include woody material from other unrelated plant material that can potentially harbor the pathogen.
- ***Fungicides*** – Effective management of *Botryosphaeria* was reported using a range of fungicides (benomyl, captan, captab, mancozeb) (Benic and Knox-Davies 1983; von Broembsen and van der Merwe 1990). In South African field trials using *Protea magnifica*, the best control was achieved by treatments of prochloraz alternated with mancozeb (Denman et al. 2004). The occurrence of cankers was also significantly reduced by applications of bitertanol and fenarimol. However, the labeling of these materials for use on Proteaceae must be determined.
- ***Resistance*** – There is no information on the relative tolerance or resistance of the Proteaceae to these pathogens.

2.3 Botrytis Seedling and Flower-Head Blight (*Botrytis cinerea* Pers.: Fr.)

Geographic occurrence and impact. Botrytis blight is a ubiquitous problem for the production and postharvest shelf life of florists' crops. The pathogen occasionally attacks seedlings and the flowers of various Proteaceae when the conditions are humid and warm enough to favor both infection and proliferation of the fungus.

Symptoms/signs. Botrytis blight, also known as gray mold, is characterized by prolific gray, fuzzy fungal growth consisting of masses of hyphae, conidiophores, and conidia. On seedlings the fungus can cause a damping off and attack cuttings during propagation. On flowers the infection will initially begin on the styles and perianths and eventually attack the whole bloom (Fig. 2). Senescing and abscising flowers and damaged tissue are very susceptible to *B. cinerea*, and the fungus commonly uses such infection sites to invade healthy tissue; the nectar of Proteaceae flowers is produced in significant quantities and can provide a substrate to assist in proliferation of the fungus. New shoots are also susceptible to infection, and the symptoms can be evident as a shepherd's crook appearance which will eventually die-off. In extreme circumstance the whole bloom can be covered by a mass of grey *Botrytis* spores.

Biology and epidemiology. The fungus can survive as a pathogen or saprophyte in a wide range of plant hosts including many florists' crops (Daughtrey et al. 2000).



Fig. 2 Botrytis blight on *Telopea* sp. (B.A. Summerell © 2017. All Rights Reserved.)

Mycelium of the fungus was found to survive through the summer in plant debris in Israel (Yunis and Elad 1989), and some isolates produce small, black sclerotia (Ellis and Waller 1974). Conidia of *B. cinerea* are easily spread by air currents and water splash. All above-ground plant parts (stems, leaves, and flowers) are susceptible to *B. cinerea*, which is favored by cool, moist conditions. Conidia of the pathogen must remain wet for 5–8 h for infection by *B. cinerea* to take place (Jewett and Jarvis 2001).

Management. Components of the integrated management of Botrytis blight include:

- ***Cultural practices*** – Reduction of RH and the duration of plant tissue wetness are critical for Botrytis blight control (Daughtrey et al. 2000) and avoidance of overhead irrigation.
- ***Sanitation*** – Removal of senescing and dead plant tissue that provide a ready substrate for *Botrytis* sporulation is important for the management of the disease.
- ***Fungicides*** – Standard fungicide applications for the control of *Botrytis* in other florists' crops are likely to be effective depending on whether the fungicide is registered for use on Proteaceous crops.

2.4 Colletotrichum Anthracnose Disease Complex/ Colletotrichum Tip Dieback (*Colletotrichum* spp.)

Geographic occurrence and impact. The disease has been reported to occur in all areas wherever members of the Proteaceae are grown. It is more problematic in nursery situations, particularly in young plants, where both the environmental conditions favor the fungus, and the tissue of the younger plants are more susceptible to infection.

Symptoms/signs. Colletotrichum tip dieback is an important disease of Proteas in South Africa and Australia but only occurs in those regions where the environmental conditions favor the pathogen. Anthracnose on Proteas is typical of these types of diseases with symptoms that include leaf necrosis, sunken lesions, and cankers on stems and petioles which in some circumstances can girdle the stem. This can cause death above that point and may abort the flower. In extreme circumstances it may extend into the whole plant causing death of the plant. It is not uncommon for the disease to be quite prevalent in nursery situations causing damping off and seedling blight syndromes, and this can be difficult to control.

Biology and epidemiology. The specific identity of the species of *Colletotrichum* that are causing these diseases is poorly understood. In South Africa it was shown that *C. acutatum* and *C. gloeosporioides* isolates originating from *Protea* were the primary pathogens associated with Colletotrichum leaf necrosis, while *C. acutatum* was the primary cause of anthracnose and stem necrosis on the cvs. tested (Lubbe et al. 2006). It is likely that given both the geographic spread and the range of host plants, a complex of species of *Colletotrichum* is responsible for the disease. Conidia, produced in diseased leaves, flowers, and stems, appear to be spread by rain or irrigation water especially in nursery situations. This occurs very frequently when extended periods of warm, humid, and wet weather occur. It is likely that the fungus persists on infected plant material as acervuli and reinfection is initiated from conidia produced there.

Management. Components of the integrated management of Colletotrichum diseases in Proteaceae include:

- ***Cultural practices*** – Reduction of RH and the duration of plant tissue wetness are likely to reduce disease severity (Lubbe et al. 2006).
- ***Sanitation*** **–** Removal of infected, senescing, and dead plant tissue that provides a ready substrate for sporulation is critical for effective management of the disease.
- ***Fungicides*** – There were reports of effective management of the disease using a range of fungicides (e.g., benomyl, captan, mancozeb), but their labeling may be problematic.

2.5 Elsinoe Scab Disease (*Elsinoe* spp.)

Geographic occurrence and impact. Present on Proteaceae crops in South Africa, the USA (Hawaii), and Australia (Fosberg 1993). In some species, especially *Leucospermum* and *Leucodendron*, the disease can cause significant disease and crop losses. The scabby lesions can also reduce the esthetics and visual appearance on a range of species thus affecting the value per flower.

Symptoms/signs. Symptoms begin as small black specks on leaves, flowers, and the stem which expand to produce the distinctive scabby lesions. The scabs are more



Fig. 3 Elsinoe scab on *Protea magnifica* x *obtusifolia* (B.A. Summerell © 2017. All Rights Reserved.)

prevalent on leaves but can also be problematic on flowers. The lesions can coalesce and expand to disfigure large areas of the leaves. In some species there can be distortion of the stem and splitting of the stem may occur (Fig. 3).

Biology and epidemiology. There are a number of *Elsinoe* species involved in this disease, the most important being *E. leucospermi*, *E. proteae*, and *E. protearum* (Swart et al. 2001). Relatively little is known about the biology of these organisms, but it appears that infection only occurs on young actively growing tissue. The infection process is favored by rain or irrigation and moderate temperatures and is more prevalent on plants grown under shade cloth, presumably because leaf moisture persists longer. Microsclerotia may be formed by some species and may provide inoculum for the fungus to persist, but it is likely that small scabs on existing stems provide opportunities for the fungus to persist between favorable environmental conditions.

Management

- ***Fungicides*** – Fungicides (including prochloraz, captafol, and mancozeb) were effective for control of scab by spraying young active growth when conditions were favorable for infection in South Africa. However, availability of these fungicides may be limited, and alternatives have not been tested. Certain fungicides containing copper are registered for control of citrus scab caused by *Elsinoe fawcettii* in Australia, but their labeling for Proteaceae must be determined.
- ***Resistance*** – Cultivars and seedlings differ in susceptibility to the scab pathogens and even within species, there can be substantial variation in susceptibility to the disease amongst cultivars.

- ***Sanitation*** – Sanitation pruning is essential for control of this disease. Pruning should be focused on late summer when lesions are most evident and infection minimal; pruning should aim to remove stems and other material containing scab lesions.

2.6 Pyrenophora Blight (*Pyrenophora* spp.)

Geographic occurrence and impact. Present on Proteaceous crops in South Africa, the USA (Hawaii), and Australia and also reported in New Zealand, Canary Islands, and Madeira. Mostly associated with *Leucospermum* and *Protea* but also recorded on *Leucodendron*.

Symptoms/signs. The disease results in a severe blight of new leaves, shoots, and flowers as they are produced in growth flushes. Initial lesions on leaves are yellow with a red margin, and these expand and cause the blight. The lesions become necrotic with age (Fig. 4).

Biology and epidemiology. The disease is most commonly associated with *Pyrenophora biseptata*, with *P. leucospermi* also reported. Relatively little is known about the biology of these organisms, but it appears that infection only occurs on young actively growing tissue and that like other *Pyrenophora* pathogens is favored by moist conditions.

Management

- ***Fungicides*** – Information on fungicides for control of this disease is not available.



Fig. 4 Pyrenophora blight of *Leucospermum* sp. (B.A. Summerell © 2017. All Rights Reserved.)

- ***Resistance*** – *Leucospermum* species are most susceptible, and there appears to be some variability in sensitivity of different species and cultivars.
- ***Sanitation*** – Sanitation pruning is essential for control of this disease. Pruning should aim to remove stems and other material containing scab lesions.

2.7 Phytophthora Root and Collar Rot and Sudden Death Disease (*Phytophthora* spp.)

Geographic occurrence and impact. The disease is caused by a number of *Phytophthora* spp. but most especially *P. cinnamomi* and *P. nicotianae* but also by *P. citricola*, *P. citrophthora*, and *P. cryptogea* in South Africa and *P. multivora* in Australia (Burgess et al. 2017). Of these species *P. cinnamomi* is the most prevalent and the most destructive and is a pathogen of Proteaceae wherever they are cultivated, and indeed in many of the areas where they occur naturally.

Symptoms/signs. Symptoms include brown root rot, stunting, yellowing and purpling, and wilting of foliage – it is not uncommon for many species of Proteaceae to die from the impact of the disease – hence the description "sudden death disease" is provided. Since these symptoms overlap with those caused by other soilborne pathogens, a correct diagnosis is necessary for management. The symptoms will reflect the nature of the disease – root rot symptoms include necrosis and rot of the root system and depending on the susceptibility of the species will be restricted to the extremities of the root system or will advance through the root system eventually killing the plant. Infection at the collar will cause a collar rot that may eventually girdle the stem, also resulting in death of the plant. Infection of seedlings can result in damping off, often killing the young plants (Figs. 5 and 6).

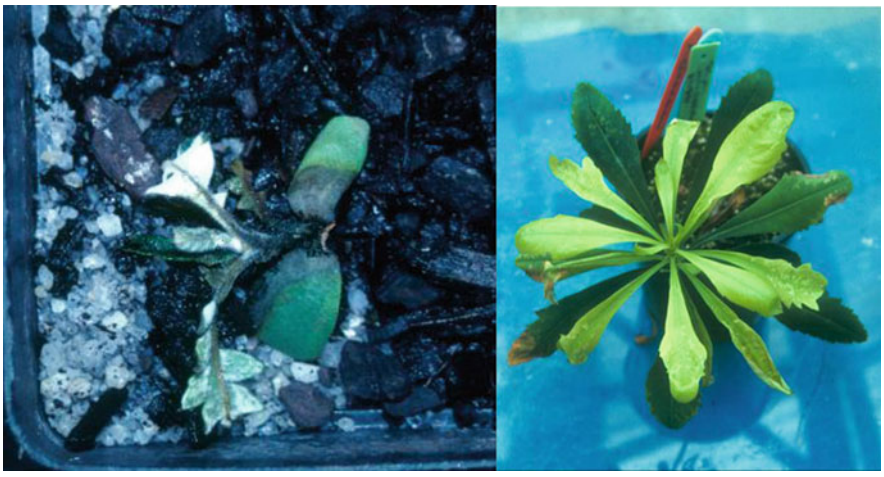


Fig. 5 Phytophthora root rot and blight on *Banksia grandis* seedling (*left*); yellowing of leaves of *Telopea* sp. caused by Phytophthora root rot (*right*) (B.A. Summerell © 2017. All Rights Reserved.)



Fig. 6 Purpling of the foliage of *Protea* sp. caused by Phytophthora root and collar rot (*left*); yellowing and dieback of the foliage and unthrifty growth of *Protea* sp. resulting from root and collar rot caused *by P. cinnamomi* (*right*) (B.A. Summerell © 2017. All Rights Reserved.)

Biology and epidemiology. Although the genus *Phytophthora* is fungus-like, it belongs to a different kingdom, the Chromalveolata. *Phytophthora* spp. are readily and rapidly disseminated as motile zoospores through irrigation, surface water, and rain; diseases incited by *Phytophthora* are favored by saturated soil. Long-term survival is by chlamydospores and sexually produced oospores. *Phytophthora,* depending on the species, has a broad host range.

Infection can occur in any part of the root system and occurs via zoospores that encyst on the root system and penetrate the roots.

Management. Refer to the introductory chapters on integrated disease management.

- ***Cultural practices*** – The use pathogen-free plant propagative material and media. In the field, plant in well-drained soil. Carefully monitor irrigation practices to avoid saturated soil and refrain from using flood irrigation which may spread propagules of the pathogen. Control weeds to increase air circulation and drying of plant surfaces especially the collar region. Control insects which may create infection courts for the fungus.
- ***Sanitation*** – Promptly remove and discard/burn infected plants. Avoid using untreated surface water which may contain inocula of *Phytophthora*. Disinfest tools, equipment, and shoes before traveling to other Protea production areas. Refer to ▶ Chap. 9, "Sanitation for Management of Florists' Crops Diseases," for information on disinfestation practices.
- ***Fungicides*** – Potassium phosphonate (phosphite)-based fungicides are widely used to reduce the impact of this disease on plantations. These can be applied via stem injection or via foliar applications as either a preventative or a curative.

- ***Resistance*** – In research conducted in South Africa with potted *Leucospermum*, the cv. Spider was tolerant to root and crown rot caused by *P cinnamomi* even when tested against a highly virulent isolate under disease-conducive conditions (Swart and Denman 2000). The researchers suggested the integration of tolerance, when known, with fungicide use.

2.8 Pythium Root Rot (*Pythium* spp.)

Geographic occurrence and impact. *Pythium vexans* was reported to cause disease in Proteaceae in the USA (California (Raabe and Hürlimann 1972), and *P. debaryanum* and *P. spinosum* were isolated from these plants in New Zealand (Robertson 1973). There are also a number of reports of *Pythium* spp. isolated from the root systems from a number of different species of Proteaceae in both South Africa and Australia, but little detailed information is available on the nature of the specific species of *Pythium* or the extent of disease caused. It is presumed that a number of different species are capable of causing damping off and root disease in nursery situations.

Symptoms/signs. The root systems become discolored and rotted, and infected seedlings rapidly wilt and may cause pre- and postemergence damping-off.

Biology and epidemiology. The genus *Pythium* is closely related to *Phytophthora* and is often recovered in association with species of *Phytophthora*. It is disseminated as zoospores presumably through irrigation, surface water, and rain, and long-term survival is enabled by chlamydospores and sexually produced oospores that may persist in potting mixes and soil.

Management. Following the management practices indicated above for *Phytophthora* should help to control *Pythium*.

2.9 Armillaria Root Rot (*Armillaria* sp.)

Geographic occurrence and impact. Armillaria root rot has been recorded on members of the Proteaceae in South Africa, Zimbabwe, Australia, New Zealand, and the USA. As the species of Proteaceae that are grown are woody perennials, the disease has time to develop and eventually causes significant problems in individual specimens. The disease is only sporadically considered an issue in cut flower plantations.

Symptoms/signs. Symptoms typically are dieback and death of part or all of the plant (Fig. 7). Diagnostic symptoms include splitting and cracking of the stem at ground level; white fungal growth may be obvious if the bark at ground level is lifted



Fig. 7 Dieback on *Protea cynaroides* caused by *Armillaria* (*left*); signs of *Armillaria* on wood: white fungal mycelium and black rhizomorphs (*arrow*) (*center*); fruiting bodies (mushrooms) of *Armillaria luteobubalina* on rotted roots and crown (*right*) (B.A. Summerell © 2017. All Rights Reserved.)

off. In severe cases the stem bases may become soft and decayed. Fruiting bodies of the fungus may appear on rooted wood.

Biology and epidemiology. Different species of *Armillaria* cause disease in different parts of the world. In South Africa *A. gallica* and *A. mellea* have been reported on *Leucodendron* and *Protea* species and appear to be incursions from the Northern Hemisphere. In Australia a broad range of Proteaceae species, both in natural ecosystems and cultivated circumstances, have been infected by the indigenous fungus, *Armillaria luteobubalina*. The fungus persists as mycelium on root systems and is the key inoculum for infecting plants; the fungus can persist on those root systems for extended period depending on the size of the piece of infected root or crown material. Infection occurs where an infected (or infested) root system comes into contact with uninfected root material. In the case *of A. mellea* and *A. gallica*, rhizomorphs may extend through the soil to infect root material; this does not happen with *A. luteobubalina*. The mycelium of the fungus colonizes the root system eventually killing that part of the roots and may eventually colonize and girdle the stem or crown of the plant thus killing the plant. The basidiomata are produced annually, usually in late autumn, but, while diagnostic, play a relatively little role in initiating new infections, but under some circumstances the basidiospores can initiate a new infection. Disease spread occurs via movement of the mycelium along the root system moving from plant to plant, through transplant or planting of infected material, or via the use of infested woody material as mulch.

Management. Once infected it is difficult to control the fungus in the plant. However in some circumstances, it is possible to expose infected areas by removing soil, and this allows the area to dry suppressing the growth of the fungus.

- ***Sanitation*** – Control depends on the removal of infected material from an area. This can be achieved by removal of as much of the root system as possible which will effectively result in much of the fungal material being removed thus stopping the infection cycle.

2.10 Rhizoctonia Root Rot (*Rhizoctonia* sp.)

Geographic occurrence and impact. Rhizoctonia root, crown, and stem rot is a poorly understood pathogen on Proteaceae but is believed to be common, particularly in a nursery situation. There is effectively no information of the nature of which species of *Rhizoctonia* or which of the anastomosis groups may be involved; however it is presumed that *Rhizoctonia solani* is involved. The disease has been reported in most regions where Proteaceae species are cultivated and across the broad range of Proteaceae species.

Symptoms/signs. The most often reported symptom is seedling rot and wirestem symptoms that may result in the death of the seedling. The pathogen may also cause pre- and postemergence damping-off.

Biology and epidemiology. *Rhizoctonia solani* has a broad host range that appears to include most of the Proteaceae that are cultivated. The disease appears to be more active and prevalent at warm temperatures, and the fungus effectively colonizes and survives as hyphae and sclerotia in plant debris, soil, and other growing media.

Management. There has been little research on control measures for this disease, but it is presumed that standard control measures for the disease in other crops would be effective.

2.11 Fusarium Wilt (*Fusarium oxysporum* Schlect.)

Geographic occurrence and impact. Fusarium wilt has been recorded on a number of South African species in the Proteaceae, most notably on *Protea*, *Leucospermum*, and *Leucodendron* and has been reported as the second most important root disease after Phytophthora root rot in South Africa (Swart et al. 1999a). The disease has also been reported in Colombia (Martínez Granja et al. 2014), and there are no substantial reports of Australian species of Proteaceae being affected by Fusarium wilt, although *F. oxysporum* may often be isolated from the roots of a range of Australian Proteaceae (Summerell et al. 2011; Summerell unpublished data).

Symptoms/signs. Plants that are infected will display early symptoms of leaf blackening which may coalesce resulting in leaf and shoot death. Most typically



Fig. 8 Fusarium wilt. Note brown discolored streak on the stem of the infected plant on the left (Swart et al. 1999a)

new shoot growth will wilt. Necrosis can be observed in the roots and will extend into the phloem and xylem with vascular discoloration evident (Fig. 8).

Biology and epidemiology. The etiology of the disease is typical of Fusarium wilt. Unassisted spread is slow but can easily be moved via movement of soil or in irrigation water. The pathogen can persist in soil as chlamydospores and in root tissue for extended periods of time. Typically infection occurs via roots and moves through the vascular system blocking the movement of water through the plant.

Management

- ***Sanitation*** – The use pathogen-free plant propagative material and media. Disinfest tools, work surfaces and containers before reuse as *Fusarium* can survive for long periods even on such inert surfaces. Promptly remove and discard/burn infected plants.
- ***Soil fumigation*** – Soil fumigation is reported to be effective in reducing the amount of inoculum in the soil, but little work has been done in determining effectiveness in disease control.
- ***Fungicides*** – No fungicides are available for control of this disease.

2.12 Ilyonectria Black Foot Rot [*Ilyonectria* spp. (formerly *Cylindrocarpon* spp.)]

Geographic occurrence and impact. The disease has been reported in South Africa and Australia on *Protea*, *Leucospermum*, and *Telopea* (Summerell et al. 1990). A range of species of *Ilyonectria* have been reported; this genus was



Fig. 9 Ilyonectria black foot rot: decline of newly planted cuttings in a field bed (*left*); root and black foot rot with vascular discoloration in the stem (*right*) (Lombard et al. 2013)

previously better known as *Cylindrocarpon*. Similar diseases are common in a range of perennial woody species, especially in grapevines.

Symptoms/signs. Disease symptoms include root, collar, stem, and crown rot, usually associated with blackening of these parts that slowly spreads up the stem of the infected plant (Fig. 9).

Biology and epidemiology. This pathogen is predominantly a problem of seedlings, particularly in nurseries and propagation facilities. If the plant is infected in this situation and then planted out, the disease can persist in older plants in plantations. In these situations the symptoms may not be obvious until an infected plant is stressed in some way and then symptoms can include death of the plant.

Management. Management of this disease has not received much attention, but it can be expected that nursery hygiene and ensuring that diseased plants are not used for establishing plantations is essential.

Additional Fungal Diseases. The following fungal diseases of Proteaceae have also been reported:

- Calonectria root rot (*Calonectria pauciramosa*) (South Africa, Crous 2002)
- Ceratocystis canker (*Ceratocystis albifundis*) (South Africa, Crous et al. 2000)
- Diaporthe stem canker (*Diaporthe saccarata*) (Mostert et al. 2001)
- Epicoccum brown stem canker (*Epicoccum sorghi*) (South Africa, Crous et al. 2013).
- Phloesporella canker (*Phloesporella protearum*) (Zimbabwe, Taylor and Crous 2000)
- Charcoal rot (*Macrophomina phaseolina*) (South Africa, Canary Islands, Espino et al. 2011, Knox-Davies et al. 1987)
- Rosellinia root rot (*Rosellinia necatrix*) (Canary Islands, Madeira Island, Espino et al. 2011, Moura and Rodrigues 2001)

3 Bacterial and Phytoplasma Diseases

3.1 Pseudomonas Leaf Spot [*Pseudomonas syringae* Pathovar "Pv. *Proteae*"]

Geographic occurrence and impact. The disease has been reported in Australia and the UK (Wimalajeewa et al. 1983) on *Protea cynaroides*.

Symptoms/signs. The pathogen can infect seedlings as well as older plants, and this can retard growth and reduce the esthetic quality of the cut flowers. The spots start as water-soaked lesions that expand, turn necrotic, and can be surrounded by a bright red halo. The entire leaf surface may become affected and wither and die.

Biology and epidemiology. Little is known of the disease, but it is presumed that spread of the pathogen occurs as a result of rain splash and movement of infected plant material. Disease development is, like many bacterial diseases, dependent on wet and humid conditions.

Management

- ***Fungicides*** – Copper sprays are reported to protect foliage against infection.

3.2 Phytoplasma Diseases: Witch's Brooms

Geographic occurrence and impact. Diseases of Proteaceae caused by phytoplasmas have been reported to occur in South Africa (Wieczorek and Wright 2003) and observed in Australia (Summerell unpublished data). The disease has been reported in species of *Aulax*, *Leucodendron*, *Leucospermum*, *Protea*, *Serruria*, and *Vexatorella* in South Africa and observed in species of *Banksia*, *Grevillea*, and *Hakea* in Australia.

Symptoms/signs. Symptoms caused by phytoplasmas may include yellowing of foliage, stunting, and proliferation of shoots ("witch's brooms"), the abnormal development of floral parts into leafy structures (phyllody), and of green coloration in flowers (virescence).

Biology and epidemiology. Phytoplasmas are prokaryotes that lack cell walls and are obligate plant parasites vectored by, in this case, a range of mites. Species in the genera *Aceria*, *Proctolaelaps*, and *Oxycarenus* were reported on Proteas in South Africa, but little is known as to whether these are the same species on other South African Proteaceae or associated with species in Australia.

Management. Management of phytoplasma diseases, like those caused by viruses, is based on monitoring and reduction of arthropod vector populations through chemical or biological means, vector exclusion by physical barriers such as fine

mesh screening, vector disorientation and repellence in field-grown crops through the use of reflective mulches, and elimination of, and avoidance, of growing near, alternate hosts especially established, infected crops.

4 Viral Diseases

No viral diseases of Proteaceae have been reported.

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Diseases of Rose

24

Jay W. Pscheidt and Tatiana Gomez Rodriguez

Abstract

Roses are one of the most popular flowering plants in the world and tend to have a few common disease problems such as black spot, Botrytis blight, downy mildew, powdery mildew, and rust. There are a dozen different fungal diseases, four bacterial problems, three nematodes, and many viral diseases that occur on roses. Several are root or root crown-related diseases, but most are concerns for canes, leaves, and especially flowers. Cultural management of these diseases is effective especially when combined with genetic resistance. Chemical management of important problems is useful for favored, susceptible rose cultivars.

Keywords

Rose • Black spot • Botrytis blight • Downy mildew • Powdery mildew • Rust • Diseases • Disease management • Rosarian

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© Springer International Publishing AG 2018

R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_23

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1 Introduction

Roses are one of the most popular flowering plants in the world grown as cut flowers in greenhouses, as whole plants in nurseries or in backyards. There tend to be a few common problems in each growing situation, such as black spot, Botrytis blight, downy mildew, powdery mildew, and rust. Some problems have become troublesome during the 2010s including downy mildew and rose rosette. Major



Fig. 1 Hydroponic production of roses in Sabana Bogotá. *Left* – 8-week-old plants in rice hull substrate. *Right* – same plants at 4 months old (Tatiana Gomez Rodriguez © 2017. All Rights Reserved.)

regional differences occur, such as rose replant disease, which is of concern in Europe but not recognized as a problem in North America. Powdery mildew, downy mildew, rust, and Botrytis blight are more problematic under greenhouse and tropical weather conditions found in production houses in Central and South America. Disease management and handling increases commercial production costs and limits rose production.

In Colombia, pest management has improved by switching from traditional production in the ground with drip irrigation to hydroponics. This change has resulted in better area usage, nutrition, and soil pest management, ultimately leading to better rose quality and productivity. Production takes place most commonly under plastic cover (in greenhouses) with varying degrees of environmental control. Although rose is a perennial, in this production system, it only has a useful life of 8–15 years. About 6.5 plants/m^2 or 380 plants per bed are used in a double row of plastic containers with nutrient film technique (NFT) subirrigation. In the hydroponic system, beds are covered by white plastic mulch or raw rice husks to improve soil moisture and facilitate weed management. It takes approximately 750–820 degrees days (equivalent to 70–80 calendar days), depending on the cultivar of rose, from the time of pruning to the first flower crop (Fig. 1).

An important consideration before implementing a disease management plan is the ultimate goal of growing roses. A backyard, non-gardener grower of a simple rose bush will have very different goals and concerns than the rosarian growing specific bushes for competitions or the nurseryman growing bare-root plants, plants in containers or cuttings. The grower's tolerance for various diseases will differ radically among these groups. We will cover many different management tactics. Choose the tactics most applicable for your situation.

2 Fungal and Oomycete Diseases

2.1 Armillaria Root Rot (*Armillaria mellea* (Vahl: Fr.) P. Kumm. 1871)

Geographic occurrence and impact. Worldwide, an occasional problem depending on the history of the growing area.

Symptoms/signs. Aboveground symptoms are general and not specific. Diagnostic mycelial fans can be observed beneath the bark of the root crown of infected plants. Dig down about 15 cm (6 in.) below the soil line, and use a pocketknife to remove thin layers of bark from the root collar. Mycelial fans are thick, white layers of the fungus that adhere to the inner root bark and/or the wood beneath the bark. The fungus also makes black, shoestring-like structures called rhizomorphs, which are occasionally found within the bark and/or extending into surrounding soil. Rhizomorphs may look like roots on the outside but have an entirely different structure when cut open in cross section.

Biology and epidemiology. The fungus, infects roots, killing the cambium and decaying the underlying xylem. Occasionally found on rose, this root pathogen is found in native vegetation where it occurs on the roots of many forest tree species and agronomic and ornamental hosts. The host range includes over 500 species of woody plants. The fungus spreads slowly, below ground, from root to root, and can survive on woody host roots for long periods. When infected plants are removed, infected roots that remain below ground serve as a source of inoculum for other plants placed in the same location. Infection occurs when rose roots come in direct contact with partially decayed tree roots and are colonized by mycelium. Infection can also occur when roots contact rhizomorphs that grow out from partially decayed roots and through the soil.

Management

- ***Cultural practices*** – Roses that are vigorous, as a result of favorable climate and sound cultural practices usually are more tolerant. Water deeply when watering is needed. Avoid surface watering, especially wetting the root crown and trunk root area. If using drip irrigation, move drip-line emitters away from the root crown and in between plants. Remove severely infected bushes and destroy them, being careful to remove small roots from the soil. Permanently removing soil from around the root crown and main root area has been effective in tree fruits grown in California and Australia and may be of benefit for managing infected bushes elsewhere (Schnabel et al. 2012).

2.2 Black Mold (*Thielaviopsis thielavioides* (Peyronel) A.E. Paulin, T.C. Harr. & McNew 2002)

Geographic occurrence and impact. Pacific Northwest of North America – rare, unknown how important in other regions. Fungus found in South America (Brazil), North America, Europe, Australia, Malaysia, and Indonesia.

Symptoms/signs. A white, granular, fungal growth that spreads rapidly over cut and bruised areas of stock and scion. Soon the growth turns black.

Biology and epidemiology. The fungus grows on the ends of "Manetti" cuttings (*Rosa chinensis* var. *manetti*) and on wounds left by disbudding, resulting in poor callus formation and, subsequently, poor root formation (Milbrath 1946). It develops under bud shields on field-budded "Manetti," thus preventing callus formation and causing the bud to die. The fungus also may damage rose roots if the plants are in storage too long before planting or if plants are overwatered after planting. The chief infection source is fungal spores in the dust and debris of storage houses where the plants are handled and cuttings prepared for planting.

Management

- ***Cultural practices*** – Practice strict sanitation in greenhouses, storage bins, and storage houses. Wash all dust and debris from the walls and floors before disinfecting storage house and bins. Use only uncontaminated stock for planting.

2.3 Black Spot (*Diplocarpon rosae* F.A. Wolf 1912)

Geographic occurrence and impact. Worldwide. Repeated defoliation from this disease leads to low vigor, inferior blooms, and high susceptibility to winter injury. Very common on plants grown outdoors.

Symptoms/signs. Circular black spots, frequently with fringed (diffuse or feathery) margins, on the leaf. Yellowing and defoliation are common in susceptible cultivars. Bare stems with few leaves attached near the top is a frequent symptom. In wet weather, spots may become very severe and coalesce to make large irregular spots covering a third of the leaf surface. In cases of severe infections, similar appearing lesions also form on the stems. May be confused with other fungal leaf spotting diseases (Fig. 2).

Biology and epidemiology. The fungus overwinters on living or dead plant tissue that was infected in the previous growing season. Newly emerging leaves are most susceptible to this fungus. Spores produced on the old plant material are splashed on young plant tissue by rains or by watering. After conidia land on a leaf, it takes at least 9 h of leaf wetness for the spore to infect.

Once the fungus is established in plant tissues, fungal fruiting bodies (acervuli) form in the spots in 11–30 days. A new crop of spores is produced and spreads to healthy portions of the plant. The cycle of infection is ready to begin again within 10–18 days. Rainy periods in any season encourage black spot, as long as the temperatures are between 10 °C and 26 °C (50–80 °F).

Leaves infected with black spot produce the plant hormone ethylene. In leaves, high ethylene content leads to leaf drop. As a result, rose plants infected with black



Fig. 2 Black spot leaf symptoms (*left*) and stem lesions with severe defoliation (*right*) (Jay W. Pscheidt © 2017. All Rights Reserved.)

spot are defoliated early and look bare. Leaves may drop after only one or two lesions. Dropped leaves around the base of the plant then serve as a reservoir of fungal spores, re-infecting the plant during subsequent rains or irrigation. The natural genetic variability of the fungus means roses found resistant in one region may be susceptible in another region due to the presence of different fungal strains. Also, resistant roses may become susceptible after a few years due to changes in the local fungal population.

Management

- ***Cultural practices*** – Plant cultivars known to be resistant in your area. **Note** that the cultivar Carefree Spirit was the first landscape shrub to survive for 2 years in All-American Rose Selections (AARS) tests without any fungicide and voted a winner. Avoid dense plantings and shaded areas. Avoid overhead watering that keeps plants wet for extended periods of time. Rake up and burn all leaves in a rose planting at season's end. Prune canes back to two buds if canes are infected. Remove and destroy diseased canes before bud break.
- ***Fungicides*** – Focus applications at the beginning of the season starting at bud break. Apply frequently in wet weather and occasionally in drier periods (1- to 2-week intervals). May become very difficult to manage once the disease gets started. Almost every fungicide on the market is registered in the USA for this disease on this crop, but only one or two materials are needed at any one time. Alternate or tank-mix materials with different modes of action to prevent developing resistant fungal populations.

Research in Alabama, USA, indicates that monthly dormant season applications of Fungicide Resistance Action Committee (FRAC) group 3 fungicides may help reduce or delay onset of this disease (Bowen and Roark 2001). Information developed by FRAC may be accessed at: http://www.frac.info/.

Bicarbonate-based materials are not recommended for use in high disease pressure regions as one will get poor control using these products (Pscheidt and Ocamb 2016). Chlorothalonil-based products may be a problem on "Knock Out" and "Double Delight" roses resulting in damaged foliage. Rosarians may not like the plant growth regulation effect (slight stunting) that may result from the use of FRAC group 3 fungicides. Always watch for warnings on labels of various products.

2.4 Botrytis Bud and Twig Blight (*Botrytis cinerea* Pers.: Fr. 1794)

Geographic occurrence and impact. Worldwide but mostly a problem in greenhouse cut flower production.

Symptoms/signs. Infected buds may fail to open, droop, and develop smooth, slightly sunken, grayish black lesions. A grayish brown mycelial growth may develop on the entire bud and extend into the stem. Petal tips may become brown, or numerous small flecks, circular brown spots, or blister-like patches may appear on petals (Fig. 3). Infected stubs or pruning wounds will develop into cane blights or cankers. Canes may be girdled, causing stem collapse.

Biology and epidemiology. The fungus is ubiquitous and survives well as a saprophyte on dead plant debris as mycelium or sclerotia. Conidia from these sources or from active infections initiate new infections. Common entry points include pruning wounds including those from normal winter pruning or from



Fig. 3 *Botrytis* sp. has infected the petals (Melodie Putnam © 2017. All Rights Reserved. (*left*) and Tatiana Gomez Rodriguez © 2017. All Rights Reserved. (*center* and *right*))

removing spent flowers (Pie and De Leeuw 1991). Buds, flowers, leaves, and stems can be infected directly when the fungus produces enzymes that degrade components of the cuticle. Healthy tissue can become infected through weakened or dead tissues that are colonized by the fungus. It may also be a secondary invader attacking plants already infected with another pathogen.

A moist, humid environment is ideal for pathogen sporulation and spread. Spore dispersal is stimulated by changes in relative humidity (Hausbeck and Moorman 1996). Concentrations peak in the greenhouse during irrigating, spraying pesticides, harvest, and shipping. Often the infection is imperceptible when cutting and packing flowers; however, moist conditions that occur during transport and/or storage favor disease development. Frost and cold injury to canes and buds can also contribute to disease development. Bare-root nursery stock held in moist, cold storage conditions also can develop Botrytis blight on canes and roots.

Management

- ***Cultural practices*** – Timely detection of the first signs of disease is essential. Collect and destroy infected plant debris and tissues from and around the flowers, stems, and leaves. Reduce greenhouse moisture by raising the temperature, venting, and/or increasing air circulation. Space plants for adequate air movement. Use drip rather than overhead irrigation. Use calcium nitrate, gypsum, or similar calcium source topdressed or incorporated into growing media.
- ***Fungicides*** – Use as a foliar spray, but focus on cultural controls. Alternate or tank-mix materials with different modes of action to prevent developing resistant fungal populations. Fungicides from the following groups are effective if resistance is not a problem: FRAC groups 1, 2, 7, 9, 12, 14, and 17. Good to mix or alternate with FRAC group M3 or M5 fungicides.

2.5 Brand Canker (*Coniothyrium wernsdorffiae* Laubert 1905)

Geographic occurrence and impact. Commonly found in North and South America, Europe, and Russia. Canker diseases can severely impact improperly grown roses leading to entire loss of the desired scion.

Symptoms/signs. Discolored (yellow to red) spots that gradually enlarge with the center turning light brown. In cut flower production, the cut stem turns yellow to black as the pathogen advances from the cut down to the base of the stem. Brand canker develops a reddish brown or purple margin that contrasts sharply with the normal green rose cane (Fig. 4). Small black fruiting bodies (pycnidia) develop toward the center of damaged tissue. Brand canker that develops under the snow line will at first in spring be black, but then it develops as described above after being uncovered several weeks. Cankers may girdle stems, causing stems and shoots above that point to wilt and die. The disease can be easily confused with common canker (*Paraconiothyrium fuckelii*) or brown canker (*Cryptosporella umbrina)*.



Fig. 4 Brand canker has gray lesions with a dark border that restricts water and nutrient movement in the cane (OSU Extension Plant Pathology Slide Collection © 2017. All Rights Reserved. (*left*) and Tatiana Gomez Rodriguez © 2017. All Rights Reserved. (*right*))

Biology and epidemiology. The fungus infects cane wounds caused by pruning, harvesting flowers, insects, thorn scars, or scratches (Intrama 1968). Contaminated cutting knives, scissors, or pruners are a major source of the pathogen. Cool winter temperatures and winter coverings that keep canes moist also favor brand canker.

Management

- ***Cultural practices*** – Avoid injuring canes. Prune canes with a disinfected, sharp knife or pruner immediately above a node. Avoid leaving long stubs above a node. Frequently clean and disinfect cutting tools (refer to ▶ Chap. 9, "Sanitation for Management of Florists' Crops Diseases"). Cut and destroy infected canes well below the affected area. Prevent irrigation from wetting the plant and/or use drip irrigation. Use a winter protecting mulch that does not hold much water such as sand, rock pumice, or coarse bark. Use a balanced fertilizer with moderate nitrogen.

2.6 Brown Canker (*Cryptosporella umbrina* (Jenk.) Jenk. & Wehm. 1935)

Geographic occurrence and impact. The disease is worldwide both in greenhouse and outside landscapes. Canker diseases can severely impact improperly grown roses leading to entire loss of the desired scion.

Symptoms/signs. Small red to purple spots appear on current season canes. Spots enlarge into whitish necrotic lesions with reddish purple boarders during the winter

months. Cankers may coalesce and can be 2.5–10 cm (1–4 in. long. Small black pycnidia can be seen in the cankered area. Cankers can cause wilt in and kill distal portions of the cane. It is easily confused with other canker diseases such as brand or common canker.

Biology and epidemiology. High moisture environments (such as hilling for winter protection) favor the disease (Horst and Cloyd 2007). Spores can penetrate uninjured tissue. Lesions can form after 10 days under favorable conditions.

Management

- ***Cultural practices*** – Cultural practices similar to common canker will be effective. Sanitation is very important. Prune canes with a sharp knife or pruner immediately above a node when first symptoms are noticed. Cut and destroy infected canes well below the affected area. Remove and destroy canes before sporulation occurs.

2.7 Cercospora Leaf Spot (*Pseudocercospora puderi* B.H. Davis ex Deighton 1976) (*Passalora rosicola* (Pass.) U. Braun 1995)

Geographic occurrence and impact. Worldwide. Misidentification of this disease on black spot resistant cultivars can lead to defoliation and loss of vigor.

Symptoms/signs. Numerous tiny maroon to purple oval lesions develop randomly across the leaf surface. The center of these spots then turns tan to gray, while the margins of spots remain maroon to dark purple. Heavily spotted leaves turn yellow and are prematurely shed. Typically, leaf loss begins at the base of the canes and gradually spreads upwards through the plant canopy towards the shoot tips. In the southeast USA, symptoms get worse through the summer and into early fall until many of the leaves are prematurely lost. Leaves are most often infected but stems, pedicels, fruits, and bracts can also have symptoms.

Biology and epidemiology. Damage appears to be greater on shrub and ground cover roses compared with hybrid tea and grandiflora roses. Spores produced in the spots are rain splashed to healthy foliage. Rainy weather favors the disease.

Management

- ***Cultural practices*** – Avoid dense plantings and shaded areas. Avoid overhead watering that keeps plants wet for extended periods of time. Rake up and burn all leaves at season's end.
- ***Fungicides*** – FRAC fungicide groups M5, 3, and 11 were found to be effective (Hagan and Akridge 2005).

2.8 Common Canker (*Paraconiothyrium fuckelii* (Sacc.) Verkley & Gruyter 2012)

Geographic occurrence and impact. Commonly found worldwide. Canker diseases can severely impact improperly grown roses leading to entire loss of the desired scion.

Symptoms/signs. Discolored (yellow to red) spots that gradually enlarge with the center turning light brown. Common canker has a darker brown margin and generally starts to develop at the cane's cut end. Small black fruiting bodies (pycnidia) develop toward the center of damaged tissue. Cankers may girdle stems, causing stems and shoots above that point to wilt and die. The disease is easily confused with brown or brand canker.

Biology and epidemiology. The fungus infects cane wounds caused by pruning, insects, thorn scars, or scratches (Intrama 1968). Common canker is favored by improper pruning, especially leaving long stubs above the last node. Common canker lesions developed more rapidly on young succulent stems than on older canes. High nitrogen fertility also increased common canker size.

Management

- ***Cultural practices*** – Avoid injuring canes. Prune canes with a sharp knife or pruner immediately above a node. Avoid leaving long stubs above a node. Cut and destroy infected canes well below the affected area. Use a winter protecting mulch that does not hold much water such as sand, rock pumice, or coarse bark. Use a balanced fertilizer with moderate nitrogen.

2.9 Cutting Rots (*Cylindrocladium scoparium* Morg. 1892 and *Fusarium* spp.)

Geographic occurrence and impact. Western North America and South America. A problem when trying to root new plants from soft wood cuttings.

Symptoms. General yellowing, wilting, and loss of lower leaves. Yellowing progresses upward from the base of the cutting. Cuttings show a basal stem rot where the base of the cutting becomes water soaked and sunken and turns dark brown.

Biology and epidemiology. The Oregon State University Plant Clinic has diagnosed cutting rots many times with the association of *Fusarium* spp., while Chase Ag Consulting, LLC, has reported that *Cylindrocladium* spp. are found in cutting rots of potted miniature roses (Chase 2015). Many fungi can be spread when

contaminated rooting solutions are used just before sticking cuttings in media. A few infected cuttings in a dipping solution can inoculate all the other cuttings that come in contact with the same solution. Disease is encouraged by warm wet conditions, which are typical when trying to root cuttings (Philley et al. 2001).

Management

- ***Cultural practices*** – Take cuttings from field plants above the splash zone. Rain and irrigation can splash soil particles as well as microorganisms onto plants. Do not take cuttings from obviously or subtly diseased plants. Take cuttings in the morning and/or after irrigation when they are more fully hydrated. Use sharp tools to remove cuttings, buds, or leaf tissue. Clean tools and soak in a disinfectant. Use two tools so a worker can trim or cut with one while the other soaks. When done with a batch, cultivar, etc., switch one tool for the other and allow the used one to soak. Saturate media 24 h before sticking. Make bottom cuts through nodes to help stimulate meristematic activity. Make all cuts of stems or leaves with clean sharp tools. Use disposable gloves and aprons for workers. Disinfect the storage facility with peroxide or quaternary ammonium products before cuttings come in. Disinfect the sticking area before work begins and frequently while sticking. Disinfect carts, benches, and any containers that come in contact with cuttings. Clean the floors at least once a day. Use as little misting as possible after sticking cuttings. Use only enough water to prevent cuttings from wilting. Mist should not wet the media until roots are formed. Adjust misting based on weather conditions such as sunny vs cloudy weather. Water cuttings after sticking and once on the bench. Maintain moderate media moisture while cuttings are forming callus. Start drying the media once callus has formed and reduce mist. Once roots form, dry media a little more but just enough to prevent wilting. Uniform moisture from top to bottom of media is important. Careful and frequent scouting for problems is important. Remove, diagnose, and destroy any diseased cuttings.
- ***Fungicides*** – Use products containing FRAC fungicide group 12 on the stock plants the day prior to cutting selection or just after sticking cuttings into media.

2.10 Downy Mildew (*Peronospora sparsa* Berk. 1862)

Geographic occurrence and impact. Commonly found worldwide and of major importance.

Symptoms/signs. Purplish-red to dark brown, irregular spots on leaves. Leaflets may yellow but contain “green island” areas. Major leaf veins often restrict fungal growth such that lesions become angular as they enlarge. A grayish spore mass may be on the leaf underside if humidity persists. However, the diagnostic sporulation of this fungus is not abundant. Defoliation also may occur in extreme cases. Small spots or long purplish areas may form on canes and may kill twigs. Some people may confuse this with black spot or even rust. In the early stages, it may get confused with



Fig. 5 Downy mildew can easily be confused for black spot or other problems (Melodie Putnam © 2017. All Rights Reserved. (*left*) and Tatiana Gomez Rodriguez © 2017. All Rights Reserved. (*center* and *right*))

nutrient deficiency or spray injury. In advanced stages, stem lesions may have white sporulation (Fig. 5).

Biology and epidemiology. This fungus-like organism can be systemic in roses. It overwinters as mycelium in stems, root crowns, and roots or as oospores in leaf debris and stems (Aegerter et al. 2002). Humidity above 85% and cool temperatures favor the disease. Infection usually occurs on young plant parts. Cuttings taken from infected stock plants will carry the disease. Infected stock may or may not show symptoms. Excess nitrogen favors disease development.

Management

- ***Cultural practices*** – Lower humidity in the greenhouse by raising temperatures and venting at key times of day. Rake leaves and prune out old flowers and stems. Burn or bury them or send them to a landfill. Take cuttings from plants with no history of the disease. Scout plants carefully for symptoms of the disease especially the undersides of leaves and any stock plants or plants held over from the previous year. Remove and destroy any tissue with symptoms of the disease. Entire leaves should be removed even if one leaflet has symptoms. Although hot water soaks have been effective, they have also damaged cuttings. Hot water soaks are not recommended until time and temperature durations have been investigated. Maintain adequate, not excessive, fertility. Silicon applications have been found to help strengthen tissue and minimize tissue damage in Colombia.
- ***Fungicides*** – Focus on cultural control methods first. Do not use mefenoxam-based products (FRAC group 4 fungicides) alone as they may not offer much control, and encourage resistance to develop. Chemical control tactics help prevent infection but may not be helpful once the disease starts. Fungicides from the following groups are effective if resistance is not a problem: FRAC groups 4, 33, and 40 and some FRAC group 11 materials.

2.11 Myrothecium Gall (*Myrothecium* sp.)

Geographic occurrence and impact. Tropical regions.

Symptoms/signs. A malformation or gall occurs at a cane node where a flower stem was removed. The gall is made of cortical tissue and can resemble the gills of a fish. The gall encompasses the cut surface and the lower portion of the new stem that developed from that node. The surface of the galled tissue becomes white to gray with orange yellow dots. Several galls can occur on the same stem. Small orange/pink fruiting bodies (sporodochia) develop toward the center of damaged tissue (Fig. 6).

Biology and epidemiology. The fungus infects through pruning wounds. Contaminated cutting knives, scissors, or pruners are a major inoculum source. The fungus is favored by temperatures between 21 °C and 27 °C with conditions of high relative humidity > 70%.

Management

- ***Cultural practices*** – Avoid injuring canes. Prune canes with a disinfected, sharp knife or pruner immediately above a node. Avoid leaving long stubs above a node. Frequently clean and disinfect cutting tools (refer to ▶ Chap. 9, "Sanitation for Management of Florists' Crops Diseases"). Cut and destroy infected canes well below the affected area. Avoid water splashes caused by irrigation, prefer the irrigation drip. Use a winter protecting mulch that does not hold much water such as sand, rock pumice, or coarse bark.

2.12 Petal Spots (Ghost Spot) (Many Fungi Possible Such as *Bipolaris, Botrytis, Cercospora*, and/or *Cladosporium*)

Geographic occurrence and impact. Worldwide. Damage makes flowers unmarketable.

Symptoms. Numerous small spots develop on petals as buds are opening through flowering. Spots may have colored or darkened margins depending on the cultivar and fungus involved.



Fig. 6 Myrothecium galls can start at pruning wounds (Tatiana Gomez Rodriguez © 2017. All Rights Reserved.)

Biology and epidemiology. Warm, humid/wet conditions favor disease development especially during rainy weather.

Management

- ***Cultural practices*** – Keep rainfall off blooms. Prune off symptomatic buds as soon as disease is noticed.

2.13 Powdery Mildew (*Podosphaera pannosa* (Wallr.: Fr.) de Bary 1870)

Geographic occurrence and impact. Commonly found worldwide on roses and other members of the rose family.

Symptoms/signs. White, powdery, fungal growth on young leaves, shoots, and buds. When scouting, check on the underside of leaves with good lighting conditions, as this is where it may first start. Distorted growth is common especially if young tissues are infected. If the disease is severe, foliage is stunted (Fig. 7).

Biology and epidemiology. The fungus overwinters in infected buds, rose leaves, twigs and branches. Infected buds grow in spring but are stunted and white with fungal conidia. Conidia are released in response to abrupt decreases in relative humidity. Wind blows conidia to healthy foliage where they start new mildew colonies. Germination and growth of conidia happen most readily on nights with high humidity or heavy dew and at temperatures near 21 °C (70 °F). Too much water, such as flowing water or rain, destroys spores by causing them to burst. The fungus does not grow in a leaf, but rather across the surface. Small anchor cells of the fungus, haustoria, remain in the leaf and take nourishment from the rose plant, but the main filaments and the multitudes of spores it produces are outside the leaf. Newly unfurled leaves are more susceptible to infection than mature leaves. Increasing day lengths to 20–22 h were shown to reduce conidial production on greenhouse roses while maintaining postharvest quality (Suthaparan et al. 2010). Brief exposure to red light during the dark period may also do the same. Small, black fruiting structures (chasmothecia) also allow the fungus to



Fig. 7 Powdery mildew symptoms are not generally seen on the flower petals but early infections can deform flower buds and affect the bloom (Jay W. Pscheidt © 2017. All Rights Reserved.)

overwinter on plant parts and debris. Ascospores from chasmothecia initiate new infections. The disease can develop under relatively dry conditions so long as the air is humid. Plants in shade tend to have more problems because that environment favors disease development. Multiple disease cycles occur during the growing season. Climbers, ramblers, and hybrid teas are susceptible. The natural genetic variability of the fungus means roses found resistant in one location may be susceptible in another due to the presence of different fungal strains. Also, resistant roses may become susceptible after a few years due to changes in the local fungal population.

Management

- ***Cultural practices*** – Plant cultivars known to be powdery mildew resistant in your area. Note that the cultivar Carefree Spirit was the first landscape shrub to survive for 2 years in AARS tests without any fungicide and voted a winner. Isolate susceptible cultivars. Space plantings for good air circulation between plants. Prune canes when dormant for an open habit also for good air circulation. Remove and destroy diseased canes. Rake and destroy fallen leaves. Use a high-pressure water hose to thoroughly wet all leaf and cane surfaces in the early afternoon so plants dry quickly. May need to do this two or three times a week. In Western Oregon, this reduced but did not eliminate powdery mildew. Briefly exposing plants to red light during the dark interval has some utility against powdery mildew in greenhouse production.
- ***Fungicides*** – Applying a dormant spray of lime sulfur at one part lime sulfur to nine parts water may help a planting that had severe powdery mildew the year before. Foliar applications of potassium salts have been found in Colombia to help reduce fungal sporulation. Apply foliar fungicides during the growing season, starting in early spring when young growth first appears. May need frequent applications, depending on the fungicide, to control the disease. Almost every fungicide on the market is registered for this disease on this crop, but only one or two materials are needed at any one time. Alternate or tank-mix products with different modes of action to prevent buildup of resistant fungal populations. Rosarians may not like the plant growth regulation effect (slight stunting) that may result from the use of FRAC group 3 or group 8 fungicides. Use of sulfur, which is a very effective organic material, can also stunt shoots, and frequent handling of sprayed foliage can irritate skin. Chlorothalonil-based products may be a problem on "Knock Out" and "Double Delight" roses resulting in damaged foliage. Always watch for warnings on labels of various products.
- ***Biological control*** – Various *Bacillus*-based products are available and may have efficacy beyond the use of water alone but will not be as effective as many synthetic materials.

2.14 Replant Disease (Many)

Geographic occurrence and impact. The problem appears to be important on roses in Europe but has not been recognized in the Pacific Northwest of North America.

Symptoms. The poor growth produced in replant situations may not be noticed unless you had a comparison. Roses planted into soil that has never grown roses (virgin soil) or on fumigated or sterilized soil would produce much more luxuriant, vigorous growth than when the same rose is replanted in an area that has grown roses for many years.

Biology and epidemiology. Replant diseases are complexes of biological and environmental factors that vary by geographic region (Pscheidt and Ocamb 2016). This is a real disease complex in the Pacific Northwest (PNW) of the USA for many crops in the rose family including pome fruit such as apple and pear and stone fruit such as cherry and peach. Similar problems exist for other crops such as strawberry (black root rot).

In the PNW, roses planted into grounds that have grown roses before appear to grow fine. The Washington Park International Rose Test Garden in Portland OR reports that they routinely remove a thousand plants every winter and replant a thousand new ones. They have not noted a replant problem.

Management

- ***Cultural practices*** – When replanting roses, follow correct planting practices. Correct any soil problems such as pH or drainage. You can also plant into new areas of your production site or rotate out of rose crops for several years before planting roses again on the same ground.

2.15 Rust (*Phragmidium mucronatum* (Pers.) Schltdl. 1824 and Many Other Species.)

Geographic occurrence and impact. Worldwide. Repeated defoliation leads to low vigor, inferior blooms, and high susceptibility to winter injury.

Phragmidium americanum (Peck) Dietel 1905 – Eastern North America and Japan
P. andersonii Shear 1902 – Northern North America; Asia; Europe
P. bulbosum (F. Strauss) Schltdl. 1824 – Africa, Asia, Europe
P. butleri Syd. & P. Syd. 1907 – Asia
P. fusiforme J. Schröt. 1870 – Temperate northern hemisphere
P. kamtschatkae (H.W. Anderson) Arthur & Cummins 1933 – Asia, Europe
P. montivagum Arthur 1909 – Temperate North America and Asia; USSR
P. mucronatum (Pers.) Schltdl. 1824 – Worldwide
P. occidentale Arthur 1901 – North America
P. rosae-arkansanae Dietel 1905 – Central North America
P. rosae-californicae Dietel 1905 – Western North America
P. rosae-pimpinellifoliae Dietel 1905 – North America; temperate Europe
P. rosicola (Ellis & Everh.) Arthur 1934 – Central North America
P. speciosum (Fr.) Burrill 1875 – North America
P. tuberculatum J. Müll. 1885 – Asia, Africa, Europe, North and Central America



Fig. 8 Note the two different rust pustules on this leaf, orange uredia and black telia (Jay W. Pscheidt © 2017. All Rights Reserved.)

Symptoms/signs. Small orange pustules (aecia) appear early in spring on both leaf surfaces. Later, the pustules enlarge and become more numerous on lower leaf surfaces (Fig. 8). Mottled and chlorotic areas may develop on upper leaf surfaces opposite the spots (uredinia) on the lower surfaces. In late summer and fall, the small pustules turn black (telia) and contain the winter spore stages of the rust. Stems occasionally are infected.

Biology and epidemiology. Many species of these autoecious fungi (fungi that complete their life cycle on one plant host) are found throughout the world. They overwinter on diseased leaves and stems. Wind blows spores to healthy foliage. They germinate and infect through the stomata when leaves are wet for 2–4 h. Mild, humid weather favors disease development.

Management

- ***Cultural practices*** – Rake up all dead leaves and prune out infected and dead wood during the dormant season. Plant resistant cultivars. Removing infected leaves early in the season may be effective in some small plantings.
- ***Fungicides*** – Focus applications during wet weather. Wetting agents will help with many of fungicides if allowed by the label. Many fungicides used for black spot will be helpful to control rust.

2.16 Spot Anthracnose (*Elsinoe rosarum* Jenk. & Bitanc. 1957)

Geographic occurrence and impact. Temperate regions. Repeated defoliation leads to low vigor, inferior blooms, and high susceptibility to winter injury.

Symptoms/signs. At first, the appearance of red spots that vary from brown or dark purple on the upper leaf surfaces occurs (Bagsic et al. 2015). Spots up to 0.5 cm in diameter may be scattered or grouped and sometimes overlapping. Chlorosis or

yellowing of the leaves may also occur. Later, spots become ashen white with a dark red margin. This tissue may fall off of the lower leaf surface, leaving a thin papery membrane or fall out entirely resulting in a shothole symptom. Symptoms occur on stems, hips, and pedicels as well as leaves. Can be easily confused with black spot or Cercospora leaf spot.

Biology and epidemiology. The fungus overwinters on infected leaves and stems with conidia continuously being formed in early spring and through the summer. Spore dispersal primarily occurs with the aid of water through rain and irrigation. There were 5 races of the fungus identified in Germany (Bagsic et al. 2015).

Management

- ***Cultural practices*** – Remove and destroy infected plant parts. Space or prune bushes to allow for good airflow.
- ***Fungicides*** – No specific chemical control has been reported. Fungicides in the FRAC group 3 used to control black spot, powdery mildew, or rust are also being used by growers to control this disease (Bagsic et al. 2015).

2.17 Verticillium Wilt (*Verticillium dahliae* Kleb. 1913)

Geographic occurrence and impact. Worldwide.

Symptoms. At first, leaves near the growing point of young canes wilt, and lower leaves yellow (Hammett 1971). Sometimes mature leaves become necrotic between veins while the veins remain green. Defoliation progresses from the base of canes to the tip. Permanent wilt, defoliation, and death also may occur. If only a few canes are infected, they may grow normally next season or dieback. Symptoms generally are more severe in the greenhouse than in the field. The characteristic vascular discoloration in other plants is not evident in rose.

Biology and epidemiology. This fungus survives a long time in soil and can infect a wide range of hosts. Rootstocks such as *Rosa odorata* and "Ragged Robin" are susceptible; *R. multiflora* and "Dr. Huey" have more resistance. *R. chinensis* var. *manetti* is very resistant. Under favorable growing conditions, plants may be able to tolerate infection.

The fungus grows into the xylem where it colonizes the plant through mycelial growth and conidial production. Fluid movement in the xylem passively transports the conidia. Once in the xylem, this fungus partially blocks water movement and produces toxins that result in wilt symptoms. Wilting occurs under periods of water stress such as midsummer heat and drought.

After diseased plant parts die, microsclerotia form and live several years in soil. Many weeds are susceptible and can help the fungus survive and disperse.

Send soil samples to any of various private and public laboratories to assay for *Verticillium* propagules. Nurseries may wish to test individual core samples to

determine the distribution in a particular field. The presence of any microsclerotia in the soil should be interpreted as a potential disease risk.

Management

- ***Cultural practices*** – Use resistant rootstocks. Avoid planting in old vegetable fields. In the greenhouse, use steam-sterilized soil or use a sterile soilless potting mix and/or hydroponics. Remove and destroy symptomatic or dead branches preferably before leaves fall and thus before new inoculum gets incorporated into the ground. Clean pruning equipment after use. Mulch with conifer-based products as conifers are resistant to infection. General yard waste mulch could contain infected plants and thus inoculum of the fungus.

2.18 Miscellaneous Fungal and Oomycete Diseases of Minor Importance or Considered Rare

Alternaria leaf spot – *Alternaria* spp.
Phytophthora root rot – *Phytophthora* spp. have been identified on rose samples with root rot from the PNW several times by the Oregon State University (OSU) Plant Disease Clinic. Also reported from Asia, Europe, and North America.
Septoria leaf spot – *Septoria rosae* Desm. 1831.
Southern blight – *Sclerotium rolfsii* Sacc. 1911.
Rhizoctonia root and stem rot – *Rhizoctonia* spp. – Japan.
Note: Fungal names based on Farr and Rossman (2015).

3 Bacterial and Phytoplasma Diseases

3.1 Bacterial Cane Blight (*Pseudomonas syringae* – Undetermined Pathovar)

Geographic occurrence and impact. Treasure Valley of Idaho, USA (Mohan and Bijman 2010).

Symptoms/signs. The symptoms usually start at the base of a vegetative bud or at leaf scars or wounds, as reddish-brown areas on the bark that later turn dark purple to black and necrotic (Fig. 9). The necrotic areas expand around and along the cane, often involving a major part or even the entire cane. Vegetative buds on the affected parts of the cane turn brown and dried. The surface of the necrotic areas of the bark is glossy, and the tissue beneath the epidermis is brown to dark brown and moist in the early stages. Often confused with winter injury.

Biology and epidemiology. This disease is different from the various symptoms on roses previously attributed to *P. syringae* and/or *P. syringae* pv. *morsprunorum*



Fig. 9 Glossy, dark purple to black, necrotic bark of canes with bacterial cane blight (Krishna Mohan © 2017. All Rights Reserved.)

(see below) in the literature. It has been an aggressive cane blight observed in the Treasure Valley of Idaho, USA, since 1996. Symptoms were common under cool, wet conditions in spring (March to May), and the level of incidence and severity of the disease varied from year to year. Several cultivars of climbing, floribunda, grandiflora, hybrid tea, hybrid perpetual, miniature, and shrub roses can show severe symptoms.

Management

- ***Cultural practices*** – The following is suggested in the absence of specific research to control this problem. Remove and destroy infected stems. Disinfect pruning shears before cutting more stems. Copper-based products are also recommended before fall rains begin and again when half the leaves have fallen.

3.2 Bacterial Leaf Spot and Blast (*Pseudomonas syringae* pv. *morsprunorum*)

Geographic occurrence and impact. Pacific Northwest of North America – rare.

Symptoms. Dark brown, sunken spots appear on leaves, flower stalks, and calyx parts. Flower buds may die without opening. Black streaks appear on 1-year-old stems.

Biology and epidemiology. The disease has been diagnosed only four times in the PNW from 1962 to 1992. Most common in cool, wet spring weather.

Management

- ***Cultural practices*** – Remove and destroy infected stems. Disinfect pruning shears before cutting more stems. Copper-based products are also recommended before fall rains begin and again when half the leaves have fallen. Repeat in spring to protect new growth.

3.3 Crown Gall (*Agrobacterium radiobacter* (Beijerinck and Van Delden 1902) Conn 1942 or Also Known as *Rhizobium radiobacter* (Beijerinck and Van Delden 1902) Young et al. 2001)

Geographic occurrence and impact. Worldwide and common.

Symptoms. Galls are often at or just below the soil surface in the basal or root crown region. They may frequently be on roots but less frequently on aerial plant parts such as stem and leaves. Galls are usually rounded with a rough, irregular surface (Fig. 10). They first appear as small protuberances on the plant surface. Young, actively developing galls are light green or nearly white, and the tissue is soft. As they age, galls become dark and woody. Outer portions can slough off with age. Sometimes galls have a rather smooth surface, which makes it difficult to distinguish between gall and callus growth, especially if the gall is at the plant base or at the graft or bud union. Plants can be stunted and have reduced vigor, poor foliage, and fewer blossoms. A single gall at the plant's base may be more detrimental than several galls on canes and roots. Symptoms may not develop for over a year if infection occurs when temperatures are below 15 °C (59 °F).

Biology and epidemiology. The bacterium enters plants through wounds, either natural or caused by pruning, grafting, mechanical injury from cultivation, heaving



Fig. 10 Galls can be seen above, below ground or starting at pruning wounds (Jay W. Pscheidt © 2017. All Rights Reserved.)

of frozen soils, chewing insects, or the emergence of lateral roots. Systemic populations that initiate disease do not seem to be as important in rose as they are in grape and walnut. After the bacterium enters a wound, a small piece of its DNA is transferred into the plant's DNA. The foreign DNA transforms normal plant cells in the wounded area into tumor cells. Once transformed, tumor cells proliferate automatically. The result is a gall: a disorganized mass of hyperplastic and hypertrophic tissue.

Pruning tools that cut through galls can become contaminated with the bacteria and spread them to cut surfaces of subsequently pruned plants. Galls breaking down in soil release bacteria, which can be transported by moving soil or water. In the absence of plant roots, bacterial populations gradually decrease; however, the pathogen may survive in soil for as long as 3 years.

Management

- ***Cultural practices*** – Use only pathogen-free nursery stock. Inspect new plants; do not plant any rose that has galls. Avoid wounding plants, especially at planting. If root pruning at planting, soak in the biocontrol agent listed below. Use plants with resistant rootstocks. Rootstocks differ widely in susceptibility. Prune off any galls on aerial parts of the plant. Disinfect pruning shears frequently. Clean shears and long soak times improve the disinfectant's efficacy. Remove and destroy badly affected plants.

  Preplant soil solarization has been effective against this disease for cherry nursery stock grown in Western Oregon and may be useful for roses. Place clear plastic (anti-condensation coating) on rototilled ground, irrigated to near field capacity, from mid-July to mid-September. Solarization is more effective on sandy loam soil. The technique may help after removing diseased plants from a bed in which roses will be planted again.
- ***Biological control*** – *Agrobacterium radiobacter* strain 84 has been used successfully with roses in Australia, New Zealand, and Spain but has not been effective in limited trials in the USA. Strain K 84 is preventive only. *Agrobacterium radiobacter* strain K1026 is a genetically modified strain of K84 that will help reduce the potential for development of resistant crown gall bacteria. Latent infections (symptomless) and existing galls are not controlled. A suspension of strain 84 may be used as a soak or spray. Thoroughly cover grafting wood, roots, and crown. Spray to runoff. To be effective, it must be applied a few hours after wounding.

3.4 Miscellaneous Bacterial Diseases of Minor Importance or Considered Rare

Hairy root – *Agrobacterium rhizogenes* (Riker et al. 1930) Conn 1942

4 Viral Diseases

4.1 Rose Leaf Curl ((A virus is suspected))

Geographic occurrence and impact. California.

Symptoms/signs. Spring leaves are small, leaflets detach easily, leaf epinasty, necrosis of shoot tips, and yellow vein flecking occur. Shoots are pointed with a broad base. Plants may recover in summer but show symptoms again in fall.

Biology and epidemiology. Little is known about this disease other than natural spread appears to be slow.

Management

- ***Cultural practices*** – Purchase stock that is clean and/or certified as tested and free of all known viruses. Remove and destroy infected plants. Heat-treat scion stock plants 4 weeks at 38 °C (100 °F) before grafting.

4.2 Rose Mosaic [*Prunus necrotic ringspot virus* (PNRSV), *Apple mosaic virus* (ApMV), *Arabis mosaic virus* (ArMV), and *Strawberry latent ringspot virus* (SLRSV)]

Geographic occurrence and impact. Worldwide.

Symptoms/signs. Symptoms may range widely depending on time of year, temperature, and type of virus(es) infecting the plant. Characteristic symptoms include chlorotic line patterns (zigzag pattern), ringspots, and mottles in leaves sometime in the growing season (Fig. 11). There may also be yellow net and yellow mosaic symptoms. Symptoms often are evident in spring and early summer but may not be on leaves produced in summer. Vein banding may be on leaves in long hot periods. Flower distortion, reduction in flower production, flower size, stem caliper at the graft union, winter survival, and early leaf drop and increase susceptibility to cold injury have all been reported. Some infected cultivars may not show any symptoms at all.

Biology and epidemiology. Several viruses are associated with the range of symptoms of rose mosaic, including *Prunus necrotic ringspot virus* (PNRSV), *Apple mosaic virus* (ApMV), *Arabis mosaic virus* (ArMV), and *Strawberry latent ringspot virus* (SLRSV). The disease does not spread naturally and has no known insect vector, but grafting transfers it to healthy plants. Transmission of ArMV and/or SLRSV could occur by Dagger nematodes, but this has not been extensively studied. Viruses can be in the rootstock or scion or both and may not show symptoms.



Fig. 11 Each of these leaves were from the same plant with rose mosaic (Jay W. Pscheidt © 2017. All Rights Reserved.)

"Madame Butterfly," "Ophelia," and "Rapture" are highly susceptible. Some report the disease does not spread; others indicate it may spread very slowly over many years. Root grafting between infected and healthy plants can also spread the disease (Golino et al. 2011).

Management

- ***Cultural practices*** – Purchase stock that is clean and/or certified as tested and free of all known viruses. Remove and destroy infected plants. However, the disease will not spread unless you propagate from or onto an infected bush. Heat-treat scion stock plants 4 weeks at 38 °C (100 °F) before grafting.

4.3 Rose Rosette (*Rose Rosette Virus*)

Geographic occurrence and impact. Widespread east of the Rocky Mountains of North America. Plants decline and die after a few years.

Symptoms. Rose rosette symptoms are complex and variable as plants of the same cultivar may have different symptoms at the same or different location(s). Infected plants may have foliage that is bright red throughout the summer rather than just in the spring and fall. Leaves may become unusually long and thin or strapped shaped.

Increased thorniness and flattening of stems (fasciation) is often but not always observed. Symptomatic foliage is often more susceptible to winter kill and/or desiccation.

Multiple shoots can emerge from a single node to form a witches' broom. Witches' brooms are easier to recognize in the winter months after most of the foliage has fallen. Unusually large masses of distorted flower buds may also occur that, in most cases, do not open. Infection can resemble herbicide (glyphosate) injury.

Infected bushes will decline and die in 3–5 years. Cane mortality is usually observed in spring when symptomatic canes fail to push out new foliage. Large commercial plantings or private rose gardens can be decimated by rose rosette if the disease is left unchecked.

Biology and epidemiology. All cultivated roses (shrub type, hybrid tea, floribunda, grandiflora, and miniature roses) are thought to be susceptible to the disease. The Knock Out rose cultivars are as susceptible to rose rosette as other types.

The virus is vectored by an eriophyid mite, *Phyllocoptes fructiphilus*. Although these mites do not fly, they may "balloon" in air currents, as do dust particles, and thus can be spread surprisingly long distances. The closer a healthy rose is planted to an infected rose, the more likely it is to become infected. In Tennessee, rose beds located near a source of the virus have a pronounced edge effect where roses nearest the source are more likely to become infected. Distribution of infected plants in a large rose bed will appear random if the plants were infected prior to planting or if there is a great distance between the rose planting and the inoculum source.

Management

- ***Cultural practices*** – Purchase stock that is clean and/or certified as tested and free of all known viruses. Roses should be inspected for symptoms before being purchased. Purchase plants from a nursery where all roses appear to be healthy. Inspected regularly for symptoms and rogue entire plants soon as possible when found. Do not just prune off symptomatic stems. Rogued plants should be bagged on site before removal.

  At this writing, preliminary studies have not demonstrated that miticides are effective. Also, it is not known if disinfecting pruning shears will also aid in disease management.

  Roses reported to be resistant include: *R. setigera*, *R. aricularis*, *R. arkansana*, *R. blanda*, *R. palustris*, *R. carolina*, and *R. spinosissima*.

4.4 Rose Spring Dwarf (*Rose Spring Dwarf-Associated Virus*)

Geographic occurrence and impact. California and Chile.

Symptoms/signs. A rosetting or balling of new growth (Salem et al. 2008). Leaves are curved, very short, and show vein clearing or a netted pattern. Leaves occur on

arrested shoots, which may not elongate for several months. Canes may grow in a zigzag pattern during the growing season.

Biology and epidemiology. Easily transmitted through vegetative propagation techniques. The virus has been found in aphids on affected plants but is not known if they are a vector of the disease or not (Rivera and Engel 2010).

Management

- ***Cultural practices*** – Purchase stock that is clean and/or certified as tested and free of all known viruses. Remove and destroy infected plants. Heat-treat scion stock plants 4 weeks at 38 °C (100 °F) before grafting.

4.5 Rose Yellow Mosaic (*Rose Yellow Mosaic Virus*)

Geographic occurrence and impact. North America.

Symptoms/signs. Symptoms associated with RoYMV infection include yellow mosaic, ring mosaic, premature leaf senescence, and necrotic dark brown rings on canes. Symptoms first appeared in new growth 4–6 weeks postinoculation. Necrotic cane symptoms are observed only in the cultivar Ballerina. Symptoms persist throughout the season (Lockhart et al. 2008).

Biology and epidemiology. Potyviruses such as RoYMV are known to be aphid transmitted.

Management

- ***Cultural practices*** – Purchase stock that is clean and/or certified as tested and free of all known viruses. Remove and destroy infected plants.

4.6 Miscellaneous Viral or Virus-Like Diseases of Minor Importance or Considered Rare

Rose flower break – England, New Zealand, and Australia – unknown causal agent
Rose Streak – eastern and mid-North America and Europe – suspect *Rose streak virus*
The following have only been associated with virus symptoms in rose and are not fully characterized; however, the four viruses commonly found in the rose mosaic complex were not present in plants infected with these viruses (Lockhart et al. 2008):
Rose yellow leaf virus
Rosa rugosa leaf distortion virus
Rosa multiflora cryptic virus
Rose chlorotic ringspot virus
Rose necrotic mosaic virus

5 Nematode Diseases (*Meloidogyne hapla*, *Pratylenchus Penetrans*, *P. vulnus*, and *Xiphinema diversicaudatum*)

Geographic occurrence and impact. Worldwide. More of a problem on sandy soils.

Symptoms/signs. Specific diseases have not been linked with specific nematodes; however, specific nematodes are associated with various root symptoms. In general, nematodes disrupt the root system, which will result in general aboveground symptoms that can include reduced vigor, poor growth or flowering, stunting, nutrient deficiencies, chlorosis, and/or wilting. These may be combined and considered a general plant decline.

Root swellings or galls occur from the feeding activity of root-knot nematodes (*Meloidogyne hapla*) or dagger nematodes (*Xiphinema* spp.). Dagger nematode form galls on the tips of feeder roots. Root-knot nematodes tend to form galls on smaller roots and, with magnification, the swollen females can also be seen. Excessive root branching may also be observed on root systems with root knot.

Small root lesions that become necrotic can be observed with an infestation of Root-lesion nematodes (*Pratylenchus* spp.). The severity of the symptoms will depend on the soil type and ability of the rose to tolerate various sized populations of root-lesion nematodes.

Biology and epidemiology. Nematodes that affect plant growth are classified by feeding behavior. Depending on the nematode, this will dictate what type of samples is needed for diagnosis: roots, soil, or both.

Root-knot nematodes (*Meloidogyne* spp.) are sedentary endoparasites, which tunnel into the roots, establishing permanent feeding sites from which they do not move. The feeding site causes the formation of giant cells, hyperplasia of cortical and vascular parenchyma, and retardation of meristematic activity in root tips (Fig. 12).

Root-lesion nematodes (*Pratylenchus* spp.) are migratory endoparasites that tunnel inside roots, feed inside roots, and freely move back into soil and on to new roots (Fig. 13).

Dagger nematodes (*Xiphinema* spp.) are migratory ectoparasites feed from outside roots, moving from cell to cell and piercing them to feed without entering root

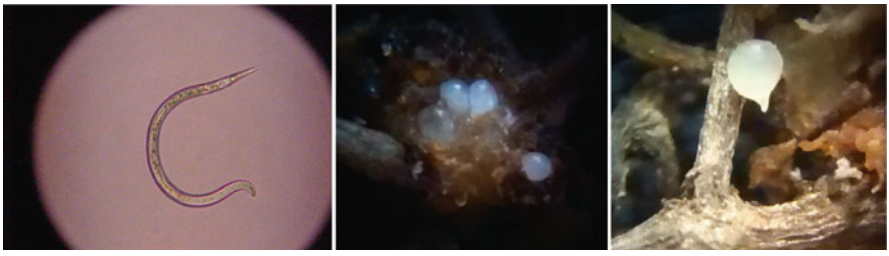


Fig. 12 Adult *Meloidogyne* sp. and egg mass (Tatiana Gomez Rodriguez © 2017. All Rights Reserved.)



Fig. 13 Adult *Pratylenchus* sp. and *Xiphinema* sp. (Tatiana Gomez Rodriguez © 2017. All Rights Reserved.)

tissue. The galls that are produced at this feeding site are caused by a hyperplastic response of cortical cells. The cells increase two to three times in size and meristematic activity is retarded. Although barely studied in rose, these nematodes can be vectors for some of the viruses in the rose mosaic complex such as *Arabis mosaic virus* (ArMV) (Fig. 13).

Soil types that are more sandy generally result in more nematode injury at lower population levels than less porous soils. Host ranges are wide for many of these nematodes making crop rotation impractical.

Management

- ***Cultural practices*** – Avoidance in floral production is accomplished by eliminating soil from the production system with soilless media and/or hydroponics. If soil must be used then steam treatment is necessary to eradicated nematodes from the soil. Although difficult, soil in planting beds can also be steam treated. Soil solarization is useful to reduce populations in the top foot of soil in order to get plants establish in infested soil. Thermal therapy after infestation of plants is also difficult but possible. Pretreat plants at 38 C for 24 h followed by immersing plants into hot water at 48 C for 35 min. Damage to rose roots with this treatment is possible. Use of resistant rootstocks is complicated by diverse reactions to different nematodes from or in different locations.
- ***Chemical practices*** – Preplant fumigation can be effective in the field, but available (registered) nematicides are becoming limited each year.

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Diseases of Snapdragon

25

Stephen N. Wegulo and A. R. Chase

Abstract

Snapdragons (*Antirrhinum majus*) are grown as bedding and container plants and as cut flowers. Diseases can be a limiting factor in the production of snapdragons. Fungal diseases commonly encountered in snapdragons include downy mildew, powdery mildew, Botrytis blight, and various leaf spots and root, crown, and stem rots. The main bacterial disease is a seedling blight caused by *Pseudomonas* spp. Impatiens necrotic spot virus (INSV) and tomato spotted wilt virus (TSWV) are the two major virus diseases affecting snapdragons. Root-knot and lesion nematodes can also attack snapdragons. Yield loss can be reduced and profitability increased by concerted efforts to manage these diseases. Management tactics include host resistance and cultural, chemical, biological, and physical control. Integration of as many of these tactics as practical will maximize the effectiveness of disease management. This chapter presents information on the occurrence, symptoms, biology, epidemiology, and management of individual diseases of snapdragon. Details are provided for diseases on which research has been done and published. Some diseases have only been observed and research has not been done or published. Such diseases are only briefly mentioned.

Keywords

Anthracnose • Blight • Leaf spot • Root and crown rot • Wilt • Mildew • INSV • TSWV

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© Springer International Publishing AG 2018

R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_24

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1 Introduction

Snapdragons (*Antirrhinum majus*) are popular plants that have been grown for centuries. They originated in the Mediterranean region (Maree and Wyk 2010; Rogers 1992), but are now grown in many parts of the world as cut flowers and garden, bedding, and potted plants. Cultivar groups differ principally in plant size. Dwarf types are used in gardens, whereas intermediate and taller types are grown for cut flowers. They display a range of beautiful flower colors from white to various shades of orange, yellow, peach, pink, red, and purple (Creel and Raymond Kessler 2007; Maree and Wyk 2010). In the USA, snapdragons were first included in the Agricultural Census of Horticultural Specialty Crops in 1959. At that time, they ranked seventh as the most valuable cut flower and made up 3.2% ($4.5 million) of the total wholesale value of flowers produced in the USA ($142.5 million), mostly in midwestern and northeastern states (Rogers 1992). In 2006, the wholesale value of snapdragon as a cut

flower in the USA for operations with sales worth $100,000 or more was $12.2 million or 3% of the total value ($411.3 million) of cut flowers (Jerardo 2007).

Diseases can significantly reduce marketability of snapdragons as plants or cut flowers. The most common diseases include *Botrytis* blight, powdery mildew, downy mildew, leaf spots, and root and crown rots. Viruses and nematodes also can attack snapdragons. The diseases found in a given location or geographical area will vary depending on environmental conditions. Losses due to diseases can be mitigated by integrating available management tactics which include cultural practices, physical control, chemical control, biological control, and host resistance.

2 Fungal and Fungus-Like Diseases

2.1 Anthracnose (*Colletotrichum antirrhini*, *C. destructivum*)

Geographic occurrence and impact. Snapdragon anthracnose has been reported in the USA (Stewart 1900; Horst 2013) and in Japan (Tomioka and Nishikawa 2011). It occurs most commonly on snapdragons grown in the field or landscape, but can also occur on greenhouse-grown snapdragons.

Symptoms/signs. The disease can occur on snapdragons at any growth stage. Leaf spots are circular and slightly sunken. They are initially yellowish green with indefinite borders, but soon become whitish with definite borders. Spots on stems are circular to elliptical and initially whitish with a narrow brown or reddish-brown border. Spots may enlarge and girdle the stem base causing death of lateral shoots. Spots on older, woody stems are sunken. Under high humidity, numerous acervuli form in diseased tissues on the leaves and stems, causing them to become smoky brown (Forsberg 1975; Nelson and Strider 1985; Pirone 1978).

Biology and epidemiology. Conidia are produced in acervuli in a sticky mass. They are dispersed by splashing water and may be carried by wind over long distances. Free water is necessary for conidial germination and infection. Disease development is favored by moisture and high humidity. Therefore, the risk for epidemics is higher in outdoor than in greenhouse snapdragons especially during wet growing seasons (Nelson and Strider 1985).

C. antirrhini is a synonym of *C. gloeosporioides* which has a wide host range (Farr et al. 1989).

Management. Adjust heating and ventilation to lower humidity and prevent condensation in the greenhouse. Avoid overhead watering. Apply fungicides labeled for anthracnose control on ornamental crops. Some of the most effective products contain a strobilurin (like pyraclostrobin or trifloxystrobin), a triazole (like triadimefon) or chlorothalonil. Combinations like pyraclostrobin and boscalid are especially effective on anthracnose diseases on ornamentals. Where available and known, plant resistant cultivars.

2.2 Botrytis Blight (*Botrytis cinerea*)

Geographic occurrence and impact. Botrytis blight on snapdragon has been reported in the USA (Horst 2013). It is most common on greenhouse-grown snapdragons, but occurs in field production of cut flowers and in the landscape.

Symptoms/signs. Often the first symptom observed is wilting of flower spikes, a result of girdling of the stem in the region of the lowest flowers. The pathogen also causes a soft decay of flowers, stems, and seedlings which is accompanied by wilting (Fig. 1). On mature plants, most infections start in the flowers and progress downward. A gray mold consisting of mycelia and spores of *B. cinerea* covers diseased plant parts. Sclerotia (compact masses of mycelia) may form inside or on the surface of infected plants (Agrios 2005; Forsberg 1975; Nelson and Strider 1985; Pirone 1978).

Biology and epidemiology. Botrytis blight is favored by high relative humidity and cool temperatures. The optimum temperature range for growth and sporulation of *B. cinerea* is 18–23 °C/64–73 °F. Free water on the plant surface is required for infection to occur. During favorable conditions, *B. cinerea* sporulates profusely, producing a mass of gray mycelium and spores. The fungus survives as mycelium or sclerotia in soil and plant debris. Spores are disseminated by air currents and will germinate and infect healthy plants if they land on wounds or senescent flowers. Insects such as moths, thrips, and fruit flies also can spread spores from infected to healthy plants (Forsberg 1975; Holz et al. 2004; Nelson and Strider 1985; Pirone 1978).

Management. Scouting for detection of diseased plants coupled with sanitation and good cultural practices can be effective in reducing damage and losses caused by *B. cinerea*. Removal and destruction of infected plants and infested plant debris can help to reduce the amount of inoculum. Good aeration keeps the plant surface dry,



Fig. 1 Wilting (*center*) of snapdragon caused by *Botrytis cinerea* (A.R. Chase © 2017. All Rights Reserved.)

which prevents spores from germinating. In the greenhouse, humidity can be reduced by ventilation and heating. Use of resistant cultivars where available is an effective and inexpensive means of managing *Botrytis* blight. Application of chemical and biological fungicides was shown to be effective in controlling *Botrytis* blight on lisianthus (Wegulo and Vilchez 2006a) and is likely to be effective on snapdragon as well. Rotation is critical since resistance to many previously effective active ingredients has been common for *Botrytis* in ornamental crops (see **Botrytis blight** in ▶ Chap. 20, "Diseases of Lisianthus").

2.3 Cercospora Blight (*Cercospora antirrhini*)

Geographic occurrence and impact. Cercospora blight has been reported on snapdragon in Guatemala (Muller and Chupp 1950), the USA (Bolick 1959), and Argentina (Nelson and Strider 1985). The disease is capable of causing significant economic losses.

Symptoms/signs. Symptoms of Cercospora blight on snapdragon were described by Jackson (1960). On leaves, lesions are subcircular and discrete to confluent with dull-white or gray centers surrounded by light-brown, slightly raised margins. They measure 0.5–7 mm in diameter. Under high humidity, rapid expansion of lesions may occur, resulting in irregular, poorly defined gray or tan necrotic areas measuring up to 15 mm in diameter with occasional faint concentric zones. The fungus sporulates profusely in the centers of lesions. Heavily infected leaves become chlorotic and drop. On stems, lesions occur mostly at the base near the soil line. They are depressed and dull white to gray with brown margins. Initially they are discrete, subcircular, oval, or elliptical. Eventually they become confluent, measuring up to 4 cm long. Cortical tissue on the stem becomes necrotic and small longitudinal cracks develop in it. Sporulation is sparse in stem lesions. Infected plants with leaf and stem lesions are chlorotic and stunted.

Biology and epidemiology. Conidia are disseminated by air currents, wind, or splashing water. Free water is necessary for spore germination and infection. Incubation period in greenhouse- and field-grown snapdragons inoculated at 8 weeks of age with a suspension of conidia and mycelial fragments was 21–30 days at an incubation temperature range of 16–32 °C/61–90 °F. Disease development was favored by warm temperatures (30 °C/86 °F) and wetness. The pathogen survived in dry leaf and stem tissues for at least 14 months and in leaf debris or in the soil for at least 3 months (Porter and Aycock 1967). Because seed transmission is common in *Cercospora* spp. plant host systems, it is possible that *C. antirrhini* can be seed transmitted. Farr et al. (1989) list *Antirrhinum* as the only host of *C. antirrhini*.

Management. Remove and destroy infected plants or plant parts. In the field or landscape, burying plant debris by plowing will reduce inoculum. Rotate with non-host crops. Use pathogen-free seed. Avoid overhead irrigation. Space plants to

allow adequate air circulation. In the greenhouse, keep humidity low through ventilation and aeration. Integrate these cultural tactics with spraying fungicides labeled for leaf spot control on ornamental crops. Thiophanate-methyl remains an excellent rotational choice in addition to strobilurins, triazoles, chlorothalonil, and fludioxonil.

2.4 Downy Mildew (*Peronospora antirrhini*)

Geographic occurrence and impact. Downy mildew on snapdragon has been reported in Ireland (Murphy 1937), England (Green 1937), Australia (Anon 1941), Italy (Garibaldi and Rapetti 1981), and the USA (Harris 1939; Kirby 1945; Yarwood 1947). The disease can be very destructive both in greenhouse- and field-grown snapdragons. Total loss can result from the disease (Yarwood 1947).

Symptoms/signs. Downy mildew affects primarily seedlings, but snapdragons can be infected at all growth stages. On seedlings, a downward curling of leaves and a reduction in the size of the plant and leaves occur. Infected leaves appear pale green on the upper surface and have a downy gray, lavender to white fungal growth on the lower surface (Figs. 2 and 3). This fungal growth can also occur on stems and upper leaf surfaces of young succulent plants. Systemically infected plants can be severely stunted, wilt, and eventually die. Seedling death progresses from the top down to the soil surface. On larger plants, symptoms of systemic infection include stunting (Fig. 4), pale green leaves (Fig. 5), and lack of flowering. Systemic infection can result in rosetting of growing points. Commonly, the shoots die and infected plants produce many secondary shoots from the base.

Biology and epidemiology. Snapdragon downy mildew is favored by high relative humidity and cool, wet conditions. Optimal temperatures for disease development range from 5 °C to 21 °C/41 to 70 °F. Local infection is characterized by pale round areas on leaves and is rarely destructive. Systemic infection can be very destructive



Fig. 2 Pale green appearance on the upper surface of a downy mildew-infected snapdragon leaf (S.N. Wegulo © 2017. All Rights Reserved.)



Fig. 3 The lower surface of a downy mildew-infected snapdragon leaf showing a gray to white fungal growth (S.N. Wegulo © 2017. All Rights Reserved.)



Fig. 4 Stunting and wilting of a snapdragon plant (front-center) due to systemic infection by downy mildew (S.N. Wegulo © 2017. All Rights Reserved.)

especially on seedlings. The incubation period can be as short as 4 days. Environmental conditions during the incubation period are most critical in determining how much infection occurs in a snapdragon planting or seedling tray (Yarwood 1947). *P. antirrhini* sporulates in cool, humid conditions. During sporulation, examination of the underside of infected leaves reveals a grayish downy growth consisting of sporangiophores and sporangia (spores). Sporangia are spread by wind, air currents, or splashing water. When they land on healthy plants, they germinate by means of a germ tube and cause new infections. In Michigan, Byrne et al. (2005) found that in field-grown snapdragons, dew periods of 6 h or longer were associated with large releases of sporangia, whereas consecutive days with short leaf wetness periods were associated with low sporangial concentrations in the atmosphere. Thick-walled resting spores (oospores) form abundantly in the roots, stems, and petioles of



Fig. 5 A snapdragon plant infected with downy mildew. Note the pale green color on the upper surfaces of leaves (S.N. Wegulo © 2017. All Rights Reserved.)

systemically infected plants and are the means by which the pathogen survives during unfavorable conditions. The host range of *P. antirrhini* is limited to *Antirrhinum* (Farr et al. 1989).

Management. Keep relative humidity in the greenhouse below 85% by balancing heat and ventilation. Keeping leaves dry using fans can be effective in preventing infections, but can also increase disease spread because spores can be disseminated by air currents. Avoid overhead irrigation. Thoroughly inspect all seedlings or plug trays for downy mildew before transplanting. Remove and destroy infected plants taking care not to spread the spores. After handling infected plants, thoroughly wash hands with soap before handling healthy plants. Some snapdragon cultivars have been shown in tests to have good resistance to snapdragon downy mildew (Byrne et al. 2004). Fungicides have been shown to be effective in controlling snapdragon downy mildew. Wegulo and Vilchez (2006b) found fenamidone, mancozeb, fosetyl-Al, and dimethomorph to have very good to excellent control of snapdragon downy mildew. To achieve effective control, apply fungicides preventively. The best management strategy for downy mildew is to integrate as many management tactics as practically and economically possible.

2.5 Phyllosticta Blight (*Phyllosticta antirrhini*)

Geographic occurrence and impact. Phyllosticta blight, also known as *Phyllosticta* leaf spot, has been reported in the USA (Guba and Anderson 1919). It is damaging primarily on snapdragons grown outdoors, but can also occur in the greenhouse (Nelson and Strider 1985).

Symptoms/signs. The pathogen can attack snapdragons at any growth stage. On leaves, initial symptoms are chlorotic spots that form green to black lesions. They



Fig. 6 Phyllosticta leaf spot on snapdragon (A.R. Chase © 2017. All Rights Reserved.)

enlarge and form yellow to brown concentric rings. The spots are visible on both sides of the leaf and become cream colored and slightly sunken in the center (Fig. 6). In wet weather, the spots continue to enlarge until the entire leaf collapses. Infection of young leaves causes them to become distorted and curled, and they may shrivel and die but remain clinging to the stem. Lesions on petioles are brown and elongated and may girdle the petiole causing the leaf to droop and die. From the petioles, lesions spread to the stem and cause infections at the leaf axil. Pinpoint size black pycnidia form in diseased tissues. On stems, lesions are dark green to brown with no definite margins. They may elongate up to 3 cm and girdle the stem. They turn dark brown, brittle, and dry and pycnidia eventually form in them. Damping-off occurs as a result of rapid wilting and dying of all aboveground parts of the plant (Forsberg 1975; Guba and Anderson 1919; Nelson and Strider 1985).

Biology and epidemiology. The pathogen overwinters as pycnidia on host debris. Moisture is required for conidial germination and infection. Conidia released from pycnidia are dispersed by splashing water. Spore germination was best at 25 °C/77 °F (Guba and Anderson 1919), indicating that moderate to warm temperatures

favor disease development. Disease development continues until the plant dies under wet conditions but is arrested or considerably slowed under dry conditions. In greenhouse infection experiments, lesions appeared on unwounded and wounded leaves of snapdragon plants 9 and 5 days after spray inoculation with conidia, respectively, indicating that wounding is not necessary for infection. Pycnidia began to appear on the fourth or fifth day after lesion appearance (Guba and Anderson 1919). In addition to *Antirrhinum*, *Penstemon* is also a host of *P. antirrhini* (Farr et al. 1989).

Management. Remove and destroy infected plants as soon as they are noticed. Remove and destroy plant debris at the end of the growing season. Avoid excessive and overhead watering. Space plants to allow good air circulation. Apply fungicides labeled for control of leaf spots on ornamental crops.

2.6 Phytophthora Stem Rot and Wilt (*Phytophthora cactorum, P. cryptogea, P. parasitica*)

Geographic occurrence and impact. Phytophthora stem rot and wilt has been reported in the USA (Gill 1960; Harris 1934). Gill (1960) cited reports of the disease in other countries including Rhodesia (now Zimbabwe), Madagascar, Mauritius, England, South Africa, India, and Australia. Losses of 50% or more in greenhouse-grown snapdragons were reported by Gill (1960), indicating the destructive nature of the disease.

Symptoms/signs. Symptoms of the disease as observed by Harris (1934) include wilting caused by girdling of the stem at or slightly above the soil line. Lesions initially appear as water-soaked areas on healthy white stem tissue. As the lesions enlarge, their older portions become yellow, brown, and eventually almost black. They enlarge, extending up and down, until the stem is girdled (Fig. 7). The outer portion of the stem may slough off exposing the hard woody xylem tissue. When



Fig. 7 Phytophthora stem rot and wilt on snapdragon (A.R. Chase © 2017. All Rights Reserved.)

initially infected, plants may wilt slightly during the day and recover at night. Severely affected plants wilt permanently within 2–3 days and later become dry and brown. Under humid and wet conditions, lesions may extend up the main stem onto the side branches.

Biology and epidemiology. *Phytophthora* spp. have a wide host range. They survive as oospores, chlamydospores, or mycelium in infected plant tissues or in soil. Long-term survival is by means of oospores and chlamydospores. Under favorable conditions, oospores and chlamydospores germinate by means of zoospores, whereas mycelium grows and forms sporangia which release zoospores. The zoospores swim in water and infect roots or are splash dispersed onto aboveground plant parts where they cause infections. Development of Phytophthora stem rot and wilt of snapdragon is favored by moisture and moderate to warm temperatures (Gill 1960).

Management. Harris (1934) observed differences in susceptibility of three snapdragon cultivars to Phytophthora stem rot and wilt, with disease incidence ranging from 20% in Cheviot Maid and 30% in Jenny Schneider to 75% in Roman Gold. Plant snapdragon cultivars are known to have good resistance to Phytophthora stem rot and wilt. Because the disease is favored by wetness, ensure good soil drainage. Avoid overhead watering. Keep plant surfaces dry by maintaining sufficient air circulation. Apply fungicides labeled for *Phytophthora* control on ornamental crops. Hausbeck and Glaspie (2009a, b, c) found the fungicides mandipropamid, fluopicolide, fenamidone, mefenoxam, and mono- and dipotassium salts of phosphorous acid to be effective in controlling Phytophthora root rot of snapdragons when applied as drenches.

2.7 Powdery Mildew (*Golovinomyces orontii* (Formerly *Erysiphe cichoracearum*); *Oidium* spp.)

Geographic occurrence and impact. Powdery mildew on snapdragon has been reported in England (Moore 1947) and the USA (Guba 1936).

Symptoms/signs. A powdery, white fungal growth appears on both surfaces of leaves, but mostly on the upper surface. Plants can be infected at any growth stage. Infections usually begin on the lower leaves, but can become severe on the upper leaves, stems, and flowers. Severely infected leaves may wilt and die.

Biology and epidemiology. Powdery mildew fungi are obligate parasites. Fungal growth occurs on the plant surface and is nourished by haustoria (special absorption structures that form in epidermal cells following infection). The fungal growth consists of mycelia and conidia which are dispersed by wind or air currents. When the conidia land on healthy plants, they germinate and cause new infections. Cleistothecia form in older infected plant tissues and produce ascospores. Disease

development is favored by shade and high relative humidity but not free water. These fungi typically have a very narrow host range and survive as mycelium in infected plants or as cleistothecia in plant debris.

Management. Space plants to allow good air circulation. Avoid planting snapdragons in shaded areas. In the greenhouse, reduce relative humidity by heating and venting. Apply fungicides labeled for powdery mildew control on ornamental crops. In trials conducted by Raabe et al. (1970), the systemic fungicide benomyl achieved complete control of powdery mildew on snapdragons when applied as a spray or a drench. Fungicides are most effective when applied preventively and especially include triazoles and strobilurins.

2.8 Pythium Root Rot (*Pythium* spp.)

Geographic occurrence and impact. Pythium root rot on snapdragons has been reported in the USA (Hanan et al. 1962).

Symptoms/signs. Snapdragons can be attacked at any growth stage. Infection of roots in the root hair zone and colonization of cortical tissues causes a brown, water-soaked rot. Basal stem tissues may also rot; however, stem tissues above the soil line are rarely colonized. Pre- or postemergence damping-off of seedlings may occur. Infected seedlings that survive and are transplanted and young plants infected after transplanting are stunted, chlorotic and wilt when exposed to sunlight. Infected plants have a reduced root system. Wilt collapse is a common symptom observed in mature plants (Fig. 8) especially at flowering when water demand cannot be met by the reduced root system. Older plants tend to be more resistant to symptom development than younger plants. In experiments conducted by Mellano et al. (1970), 15-day-old or younger seedlings infected with *Pythium ultimum* died within 6 days due to rapid and unrestricted colonization of host tissue. In contrast,



Fig. 8 Wilting caused by Pythium root rot (A.R. Chase © 2017. All Rights Reserved.)

25-day-old plants were tolerant to infection and host colonization. Wilting and stunting intensified by high temperature was observed in infected tolerant plants.

Biology and epidemiology. *Pythium* spp. have a wide host range. They live in the soil and produce a white mycelium that grows rapidly and gives rise to sporangia. A sporangium germinates directly by giving rise to a germ tube or by producing a short hypha bearing a balloon-like vesicle at its end. Within the vesicle, 100 or more zoospores are produced. When released from the vesicle, the zoospores move around in a swarm for a few minutes then form cysts which germinate by giving rise to a germ tube. The germ tube infects host tissue by direct penetration. The mycelium can also produce a female oogonium and a male antheridium. The antheridium produces a fertilization tube which enters the oogonium. Nuclei of the antheridium move through the tube and fuse with nuclei of the oogonium to form a zygote which becomes an oospore by producing a thick wall around itself. The oospore is resistant to adverse environmental conditions and is the survival and resting stage of *Pythium* spp. When environmental conditions become favorable, oospores germinate in a manner similar to that of sporangia by directly forming a germ tube or by first forming zoospores and cysts. Rootlets can be infected at any stage of plant growth. Root rot progresses upward to the crown and stem. Disease development is favored by wet and waterlogged soils (Agrios 2005). Zoospores are disseminated by flowing water or splash dispersal. In the greenhouse, the primary means of introducing *Pythium* propagules are contaminated plant material and soiled hands, tools, and hose ends (Daughtrey et al. 1995).

Management. Cultural methods of control include good water drainage. If the planting site is poorly drained, plant snapdragons in raised beds. Composted bark can be incorporated into the soil in the raised bed to improve aeration. Drainage tiles can be used to facilitate drainage and direct water away from the planting site. Do not reuse pots or trays from a previous crop for propagation. If pots or trays are reused, they should sanitized or sterilized first. Fungicides registered for *Pythium* control such as sodium, potassium and ammonium phosphites, etridiazole + thiophanate-methyl, mefenoxam, and etridiazole can be used to control root rot in snapdragons (Pscheidt and Ocamb 2016). Del Castillo Múnera and Hausbeck (2015) found that the efficacy of fungicides in controlling *Pythium* root rot on snapdragons depended on the species *Pythium*. Mefenoxam and fenamidone reduced root rot caused by *P. aphanidermatum* and *P. ultimum*; whereas fluopicolide and etridiazole reduced root rot caused by *P. ultimum*; and fenamidone, potassium phosphite, and the biological control agent *Trichoderma harzianum* effectively controlled root rot caused by *P. irregulare*. In the same study, differences in intensity of root rot caused by *Pythium* spp. were observed among snapdragon cultivars, although none of the cultivars was highly resistant.

2.9 Rhizoctonia Basal Stem Rot (*Rhizoctonia solani*)

Geographic occurrence and impact. Rhizoctonia basal stem rot on snapdragon has been reported in the USA (Baker and Sciaroni 1952) and Taiwan (Yang and Leu 1981).

Symptoms/signs. Snapdragons can be infected at any growth stage. They are most susceptible at the seedling stage and immediately following transplanting (Nelson and Strider 1985). Symptoms include pre- and postemergence damping-off. Infected seedlings that survive have red-brown lesions on the basal stem at the soil line. Wilting, collapse, and death occur if lesions girdle the stem. Affected tissues show a coarse brown mycelium of *R. solani* in wet and humid environments.

Biology and epidemiology. *R. solani* has a wide host range. It survives as mycelium or sclerotia in infected plants or plant parts, soil, or plant debris. It is spread by water, contaminated tools, or infected plants or propagative plant material. Basal stem rot on snapdragon is favored by high temperatures and moderately moist soils (Baker and Sciaroni 1952).

Management. Treat soil with heat (steam or solarization), fumigants, or fungicides labeled for control of *Rhizoctonia* in ornamental crops. Ensure good soil drainage and avoid excessive watering. Plant snapdragons on raised beds if the area is poorly drained. Use sterilized soil, trays, and tools to raise seedlings before transplanting. Transplant disease-free seedlings. In the greenhouse, keep relative humidity low by venting and heating. Space plants to allow good air circulation. Some of the most effective fungicides include fludioxonil, thiophanate-methyl, and strobilurins (like azoxystrobin and pyraclostrobin).

2.10 Rust (*Puccinia antirrhini*)

Geographic occurrence and impact. Snapdragon rust was first reported in the USA by Blasdale (1903) who had found it at Berkeley, CA, in 1895 (McClellan 1953). Peltier (1919) noted that the disease caused much loss in Illinois in the 4 years following its discovery in the state in 1913. McClellan (1953) echoed the seriousness of the disease by stating that "Of the numerous diseases of snapdragons, rust probably causes the most concern." Snapdragon rust occurs throughout the world wherever snapdragons are grown (Gawthrop and Brooks 1979; Nelson and Strider 1985).

Symptoms/signs. Seedlings, cuttings, and mature plants can be attacked by rust in the greenhouse as well as in the field. It is most severe on cuttings and on plants just before flowering (Peltier 1919). All above-ground parts of the plant except the florets can be infected. Initial symptoms on leaves are small, chlorotic swellings. These swellings increase in size until the epidermis ruptures, exposing red-brown uredinial pustules. Pustules are limited to the lower leaf surface. On the upper surface of the leaf above each pustule, a circular yellow area is apparent. The fungus continues to grow under favorable environmental conditions and in 4 or 5 days forms one or two concentric rings of smaller secondary pustules around the original pustule (Nelson 1962) (Fig. 9). In the presence of moisture and high relative humidity, the pustules are invaded by secondary microorganisms, mainly *Fusarium* spp., which cause irregularly shaped necrotic spots around the pustule (Dimock and Baker 1951). On stems, initial



Fig. 9 Rust pustules on the undersides of snapdragon leaves (A.R. Chase © 2017. All Rights Reserved.)

small pustules elongate, causing swollen cankers which serve as entry points for secondary invaders. These invaders cause lesions that girdle the stem, resulting in wilting and plant death. Severely affected plants have a scorched or brown appearance. Flowers open prematurely and are smaller than normal.

Biology and epidemiology. The host range of *P. antirrhini* is limited to *Antirrhinum* (Farr et al. 1989). Like other rust fungi, it is an obligate parasite. Urediniospores are spread by wind over long distances and by wind-blown rain or splashing water over short distances (Nelson and Strider 1985). They land on healthy plants and initiate new infections. Optimal conditions for infection are the presence of free water and a temperature range of 10–13 °C/50–55 °F. Disease development is optimal under a temperature range of 21–24 °C/70–75 °F and is slowed at higher temperatures (Dimock and Baker 1951). Under optimal conditions for disease development, new uredinia are formed within 8–16 days following inoculation (Peltier 1919), and the cycle of spore production is repeated. The urediniospore is the repeating spore of *P. antirrhini*. Teliospores form later in the growing season, but do not play an important role in the disease cycle. Under semiarid conditions, injury is primarily from desiccation through the rust pustules. In the presence of moisture and high relative humidity, severe injury is caused by secondary invaders, primarily *Fusarium* spp. (Dimock and Baker 1951). There are no known alternate hosts of *P. antirrhini* in the USA. The fungus survives as mycelium or urediniospores on snapdragon plants in the greenhouse or field.

Management. Cuttings are extremely susceptible (Peltier 1919). Therefore, only cuttings from rust-free snapdragons should be used. Otherwise start plants from seed. Allow good drainage and good air circulation. Keep temperatures in the greenhouse above 24 °C/75 °C for several days and not below 16 °C/61 °F at night (Pscheidt and Ocamb 2016). Avoid overhead watering. Integrate the above cultural management tactics with application of fungicides registered for rust control on

ornamental crops. Fungicides shown to provide good results on snapdragon rust include strobilurins (especially azoxystrobin) and triazoles which can show eradicant benefits at times. Mancozeb is also excellent but only as a true preventive application.

2.11 Verticillium Wilt (*Verticillium albo-atrum, V. dahliae*)

Geographic occurrence and impact. Verticillium wilt on snapdragons has been reported in the USA (Baker and Sciaroni 1952).

Symptoms/signs. The pathogens infect roots and colonize the xylem vessels, inhibiting water movement. Initially the lower leaves may become yellow, wilt, and drop. Later wilting and collapse of single branches may occur, causing the plant to appear as if wilting on one side (Nelson and Strider 1985; UC IPM 2014). The entire plant finally wilts, collapses, and dies. During periods when cool days or nights alternate with warm days, wilting and plant death can occur rapidly. Wilting often occurs after the onset of blossom development. Vascular tissues are discolored continuously from the base of the stem to the top of the plant. Black microsclerotia may form in diseased tissue.

Biology and epidemiology. *Verticillium* spp. are soilborne and have a wide host range. They survive over long periods of time as microsclerotia in the soil or plant debris. Introduction into non-infested fields or greenhouses is through microslerotia-contaminated soil or tools and mycelium in infected plant material such as cuttings. Disease development is favored by moisture and cool temperatures. Dutta (1981) showed that the severity of Verticillium wilt on snapdragon was higher in alkaline compared to acidic soil. Organic (chitin and green manure) and inorganic (ammonium sulfate, calcium nitrate, and combined NPK) soil amendments reduced disease severity, and this was attributed to boosting antagonistic microorganisms in the soil and direct nutritional effects on snapdragon plants (Dutta and Isaac 1979a, b; Isaac 1956). Excessive nitrogen fertilization increased disease severity (Isaac 1957).

Management. Treat soil with heat (steam or solarization) or fumigants before planting or transplanting snapdragons. In the greenhouse, sanitize trays, tools, and benches. Remove and destroy infected plants and infested plant debris. Use disease-free plants or cuttings. Avoid planting snapdragons in poorly drained soils.

2.12 Additional Fungal Diseases and Pathogens

The following fungal diseases and pathogens have been reported on snapdragon (Horst 2013): southern blight (stem rot, *Sclerotium rolfsii*), stem and crown canker (*Myrothecium roridum*), collar rot (*Rhizoctonia solani*), petal rot (*Bipolaris setariae*), charcoal rot (*Macrophomina phaseoli*), root rot (*Phymatotrichum*

omnivorum, *Thielaviopsis basicola*), and stem rot and wilt (*Fusarium* sp., *Sclerotinia sclerotiorum*, *S. minor*). In addition, seed blight (*Alternaria alternata*) has been reported (Harman et al. 1973).

3 Bacterial Diseases

3.1 Seedling Blight (*Pseudomonas antirrhini*)

Geographic occurrence and impact. Seedling blight on snapdragon has been reported in Japan (Takimoto 1920), Australia (Valder 1963), and the United Kingdom (Simpson et al. 1971). Chase (2001, 2004) listed *Pseudomonas* as a causal agent of *Pseudomonas* leaf spot on certain cut flower cultivars of snapdragon in the USA (Fig. 10), but did not specify the species.

Symptoms/signs. The disease is actually a leaf spot, but when severe it is better described as a seedling blight (Simpson et al. 1971). Leaf lesions are initially small, pale green, and less than 0.5 mm in diameter. They enlarge and become brown with a green halo. When fully developed, they are discrete, almost circular, and measure 4–5 mm in diameter. They appear sunken and papery brown with a defined dark brown margin. Water-soaked dark green zones may be seen around the lesions. As disease development progresses, the lesions coalesce, forming irregular blotches followed by leaf collapse. On dark-leaved cultivars, lesions are less discrete and the tissue surrounding the lesions may appear chlorotic (Simpson et al. 1971).

Biology and epidemiology. Simpson et al. (1971) observed that seedling blight appeared on snapdragon seedlings usually after the first transplanting and before reaching a height of 15–18 cm. Beyond this growth stage, the disease did not attack newly emerged foliage. Disease development during the susceptible growth stages is

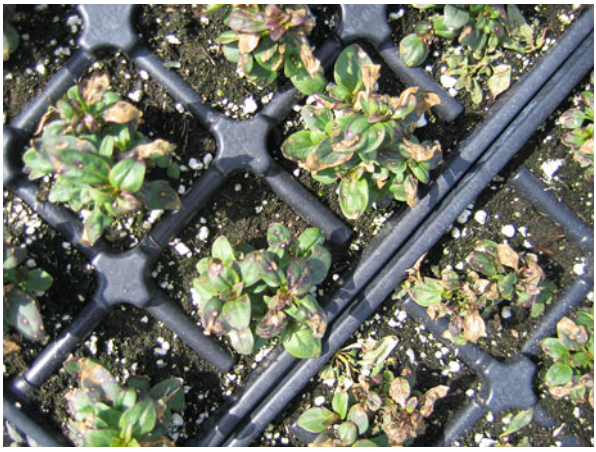


Fig. 10 Seedling blight caused by *Pseudomonas* spp. (A.R. Chase © 2017. All Rights Reserved.)

favored by moisture such as that from mist irrigation during propagation. The pathogen is spread by splashing water from rain or irrigation. Pathogenicity tests on 6 to 8-cm-tall seedlings showed that the incubation period was 10–12 days. Injury to leaf surfaces by an abrasive agent resulted in severe symptoms (Simpson et al. 1971). Seed transmission studies confirmed that *P. antirrhini* can be transmitted through seed. The host range of this pathogen is limited to members of the family Scrophulariaceae, of which snapdragon is the most susceptible.

Management. Use pathogen-free seed and propagative plant material. Avoid excessive moisture on the foliage of seedlings. Remove and destroy infected seedlings. Plant resistant cultivars where known and available. Preventively apply bactericides labeled for disease control on ornamental crops. Bactericides can only prevent or reduce infections; they cannot eliminate established infections.

3.2 Additional Bacterial Diseases and Pathogens

Crown gall (*Agrobacterium tumefaciens*) has been reported on snapdragon (Horst 2013).

4 Viral Diseases

4.1 Impatiens Necrotic Spot Virus (INSV) and Tomato Spotted Wilt Virus (TSWV)

Geographic occurrence and impact. Impatiens necrotic spot virus has been reported on snapdragon in the USA (Daughtrey et al. 1997; Horst 2013). Significant losses can result from infection by these viruses because symptomatic plants are not marketable.

Symptoms/signs. Symptoms of INSV on snapdragons can be variable and include brown to white spots on leaves, stunting, stem necrosis which causes tissues to turn black (Fig. 11), and plant death. Because symptoms are variable and resemble those caused by other diseases, they are not reliable for diagnosis. Confirmation is done by performing laboratory tests such as enzyme-linked immunosorbent assay (ELISA) or polymerase chain reaction (PCR).

Biology and epidemiology. INSV is one of two viruses that infect a wide range of plant species including ornamentals, vegetables, and field crops. The other virus is tomato spotted wilt virus (TSWV). Both belong to the genus *Tospovirus* in the family *Bunyaviridae* and are transmitted by the western flower thrip (Daughtrey et al. 1997). Spread of these viruses is through movement of infected plants or viruliferous thrips which feed on and transmit the viruses to healthy plants. Many plant hosts can be infected but show no symptoms. These plant hosts which include weeds serve as reservoirs for the viruses and their vectors.



Fig. 11 Necrosis on snapdragon caused by *Impatiens necrotic spot virus* (INSV) (A.R. Chase © 2017. All Rights Reserved.)

Management. Effective control can be achieved by virus exclusion and prevention of virus spread. Virus exclusion involves use of virus-indexed propagation material which can be purchased from certain suppliers. INSV and TSWV can also be excluded by preventing thrips from entering the greenhouse or propagation and storage areas. This can be achieved through the use of fine mesh screens. Weeds and other thrips and virus plant hosts growing in the vicinity of greenhouses should be controlled with herbicides or other means. Workers should take care not to introduce thrips into the greenhouse or propagation areas by changing clothes before entry and by not wearing clothes with colors such as yellow, green, or blue which attract thrips. Prevention of virus spread is achieved by controlling resident thrip populations with insecticides or biocontrol agents and by roguing and disposing of infected plants. Because virus-infected plants are more susceptible to the feeding and oviposition of thrips (Bautista et al. 1995), such plants should be disposed of as quickly as they are noticed.

4.2 Additional Virus Diseases

Cucumber mosaic virus, an unidentified mosaic virus, and an unidentified ring spot virus have been reported on snapdragon (Horst 2013).

5 Nematode Diseases

5.1 Root-Knot Nematode (*Meloidogyne* spp.)

Geographic occurrence and impact. Root-knot nematode has been reported on snapdragon in the USA (Horst 2013; Pirone 1978).

Symptoms/signs. Root galling and stunting.

Biology and epidemiology. Root-knot nematodes have a wide host range. They feed and reproduce within plant roots, inducing small to large galls known as root-knots. The adult females are sedentary in plant roots and have a round to pear-shaped body. Adult males are vermiform, leave the root, and become free-living in the soil. Adult females lay eggs in gelatinous sacs that are deposited on the surface of galled roots or may remain within the galls. The egg develops into a vermiform first-stage juvenile (J1) which molts into a vermiform second-stage juvenile (J2) that emerges from the egg into the soil. If the J2 (the only infective stage) encounters a susceptible host, it enters the root and becomes sedentary. It feeds on the root cells surrounding its head and grows, becoming sausage shaped. The saliva it secretes causes the root cells to enlarge and form galls. The J2 molts into a third-stage juvenile (J3) which molts into a fourth-stage juvenile (J4). Both the J3 and J4 are sedentary within the root. The J4 undergoes a final molt into a mature male which leaves the root or a mature female which remains sedentary in the root and lays eggs. Root-knot nematodes are spread by contaminated equipment, infected plant material, or water. Nematodes attack ornamental plants in the landscape or field; they do not seem to be present in greenhouse- or container-grown crops (Creswell 2009; Ismail 1980; McSorley 1994; McSorley and Fredeik 1994).

Management. Sanitize equipment after use. Contaminated soil can be disinfested by fumigation, drenching with nematicides, or heat treatment with steam or solarization. Refer to ► Chap. 8, "Soil/Media Disinfestation for Management of Florists' Crops Diseases" for specific strategies.

5.2 Additional Nematode Pathogens

The lesion nematodes *Pratylenchus* spp. have been reported on snapdragon (Baker and Sciaroni 1952; Horst 2013).

6 Additional Parasites

Dodder (*Cuscuta* sp.) has been reported on snapdragon (Horst 2013).

7 Nonparasitic Disorders

7.1 Tip Blight

A tip blight of unknown cause has been described on snapdragons (Forsberg 1975; Horst 2013). Leaves gradually wilt starting at the plant tip and the wilt progresses to affect petioles and stems. Sunken, water-soaked lesions may form on the stem and girdle it, causing the plant tip to wilt and die. This disorder usually occurs in winter when it is cloudy and disappears in spring when brighter weather returns.



Fig. 12 Phytotoxicity (second and third leaves from *left*) caused by an experimental fungicide on snapdragon. The first, fourth, and fifth leaves from left are from plots sprayed with commercial fungicides. The sixth leaf is from a non-sprayed check plot (S.N. Wegulo © 2017. All Rights Reserved.)

7.2 Phytotoxicity

Certain pesticides may cause injury to snapdragons. Figure 12 shows injury to snapdragon leaves following application of an experimental fungicide.

7.3 Additional Nonparasitic Disorders

Fasciation has been reported on snapdragon. It is probably a genetic abnormality (Horst 2013).

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Diseases of Stock

26

Steven T. Koike

Abstract

The ornamental plant stock (*Matthiola incana* R. Br.) is a popular and versatile flowering species that can be grown as a garden or bedding plant as well as a field grown cut flower. Profitability of stock production depends on the management of a number of diseases. As part of the Brassicaceae, stock is subject to a number of pathogens that infect vegetable crucifers as well. Major pathogens include *Sclerotinia*, downy mildew, two wilt pathogens (*Fusarium*, *Verticillium*), *Xanthomonas*, and *Turnip mosaic virus*.

Keywords

Fusarium • *Sclerotinia* • *Verticillium* • *Xanthomonas*

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R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_26

1 Introduction

Stock (*Matthiola incana* R. Br.) is also known as annual stock, column stock, garden stock, or tensweek stock. A closely related ornamental species is *M. longipetala* (Vent.) DC which is named night-scented stock or evening stock. Likely originating from the Mediterranean area, stock is a popular floral commodity that is strongly scented and is grown as a garden or bedding plant and also as a cutflower (Armitage and Laushman 2003). Depending on the species and region, stock can be grown as annuals, biennials, or perennials. As a member of the Brassicaceae, stock is susceptible to many of the diseases that affect commercial cole crops (broccoli, cabbage, cauliflower, etc.) vegetables (Koike et al. 2007).

2 Fungal and Fungus-Like Diseases

2.1 Cottony Rot (*Sclerotinia sclerotiorum*)

Geographic occurrence and impact. Cottony rot (also known as white mold and Sclerotinia crown rot) has been reported on stock in Australia, Greece, Korea, New Zealand, Scotland, South Africa, and the United States (California, Michigan, and Pennsylvania). The disease can occasionally be important on stock.

Symptoms/signs. Symptoms vary depending on the type of fungal inoculum that comes in contact with stock. The soilborne survival structure, the sclerotium, can germinate directly and the resulting mycelium can infect stock crowns at the soil line. Crown infections first result in a gray to brown lesion on crown tissues. Lesions expand, crowns become completely rotted, and the plant will suddenly collapse (Fig. 1). If conditions are moist enough, the diseased crown will support the growth of white mycelium and large (5–10 mm/0.2–0.4 in. in length), black, irregularly shaped, hard sclerotia (Fig. 2). A second type of inoculum produced by *S. sclerotiorum*, the ascospore, is airborne, lands on aboveground stock tissues, and causes a blight on stems, leaves, or flowers. To infect the host, the ascospore must land on tissue that is damaged or senescent. Once germinated, the ascospore produces mycelium that will colonize the compromised tissue and also spreads to adjacent healthy tissue, resulting in a brown, soft decay. White mycelium and black sclerotia also develop from these foliar infections.

Biology and epidemiology. Cottony rot is caused by *Sclerotinia sclerotiorum*. There is one report of another species, *S. minor*, causing a crown rot on stock in Australia. *S. sclerotiorum* survives and overwinters as sclerotia in soil. Sclerotia can germinate and form infective mycelium or alternatively will produce small, tan, mushroom-like structures called apothecia (Fig. 3). Apothecia emerge from the soil and release airborne ascospores that are carried by wind and infect susceptible stock leaves, stems, and flowers. When infected stock plants are plowed back into the soil,



Fig. 1 Stock plants infected with *Sclerotinia* species can suddenly wilt and collapse (Steven Koike © 2017. All Rights Reserved.)

sclerotia that had formed on infected parts are returned to the soil and enable the pathogen to persist. *S. sclerotiorum* has an extremely broad host range, so sclerotia and ascospores produced from other crops, such as gazania, gaillardia, cauliflower, lettuce, pepper, and many others, can provide inoculum that can infect stock.

Management

- ***Cultural practices*** – Avoid planting stock in fields having a history of cottony rot. Avoid planting stock adjacent to vegetable or other flower crops that are highly susceptible to *Sclerotinia* and which exhibit symptoms of the disease.
- ***Fungicides*** – Apply protectant fungicides to stock foliage prior to infection.

2.2 Downy Mildew (*Peronospora parasitica* = *P. matthiolae* or *Hyaloperonospora parasitica*)

Geographic occurrence and impact. This disease occurs worldwide on stock. Severe outbreaks of downy mildew can cause significant damage to the stock foliage and reduce flower quality and marketability.

Symptoms/signs. The first symptoms are irregular, light green to slightly yellow, diffuse patches on the top surfaces of leaves. As disease develops, the yellow color intensifies and large portions of the leaf can be affected (Fig. 4). Irregular dark specks can be seen within the yellow patches. Diseased leaves can be twisted and, in



Fig. 2 The hard, black sclerotia (arrows) of *Sclerotinia sclerotiorum* can form on the outside of stock stems (Steven Koike © 2017. All Rights Reserved.)



Fig. 3 Small, tan apothecia of *Sclerotinia sclerotiorum* will release spores that can infect stock foliage (Steven Koike © 2017. All Rights Reserved.)

advanced stages, the entire leaf can turn brown and die. White tufts of the pathogen are seen on the undersides of the lesions (Fig. 5). With a hand lens the tufts can be seen to consist of the branching, treelike structures that bear the downy mildew spores. If conditions are favorable, the sporulation can also occur on the top side of leaves. For some stock cultivars, extensive sporulation can develop well before any



Fig. 4 Stock plants infected with downy mildew will first develop yellow patches on leaves (Steven Koike © 2017. All Rights Reserved.)



Fig. 5 The fine, white growth of downy mildew can be seen on the surfaces of stock leaves (Steven Koike © 2017. All Rights Reserved.)

symptoms are seen on the top of the leaves (Koike 2000). Downy mildew has been known to kill stock seedlings if conditions allow seedling cotyledons to be infected early and severely.

Biology and epidemiology. Downy mildew is caused by the oomycete *Peronospora parasitica*. Researchers also refer to the pathogen as *Peronospora matthiolae* or *Hyaloperonospora parasitica* (Constantinescu and Fatehi 2002; Jafar 1963). Oomycete organisms are no longer considered true fungi but are more closely related to algae. This obligate pathogen forms mycelia and haustoria inside host tissues, and spore-bearing sporangiophores emerge through the leaf stomata. Some *P. parasitica* isolates and races form the sexual oospore stage, though this has not been observed on stock (Sherriff and Lucas 1989). There is considerable host specialization within the *P. parasitica* group of pathogens, and strains infecting stock apparently only infect this host (Sherriff and Lucas 1990). Cool (10 to 15 °C/50 to 59 °F), moist or high humidity conditions favor downy mildew sporulation. Spores,

which are short-lived, are dispersed via winds and splashing water. Germination of sporangia and infection usually require the presence of free moisture on the leaf surface. Lesion expansion is most rapid at 20 °C/68 °F.

Management

- ***Cultural practices*** – Irrigating early in the day, to enhance drying of the foliage, may reduce disease severity.
- ***Fungicides*** – Applying protectant foliar fungicides prior to infection is the best means of controlling downy mildew. Fungicides such as metalaxyl can be used as seed treatments for controlling early downy mildew on seedlings.
- ***Resistance*** – Differences in susceptibility may exist among different stock cultivars but resistant cultivars do not appear to be available.

2.3 Fusarium Wilt (*Fusarium oxysporum* f. sp. *mathioli*)

Geographic occurrence and impact. Fusarium wilt has been reported on stock in Germany (Gerlach 1975), Japan (Saito et al. 2008), South Africa, the United Kingdom, and the United States (Arizona, California) (Baker 1948). The disease has caused considerable damage in the United Kingdom (O'Neill et al. 2003, 2005; O'Neill and Mason 2014, Fig. 6).

Symptoms/signs. In some cases, if young seedlings are infected early, these plants will rapidly wilt and die. On larger plants, the lower leaves are affected first,



Fig. 6 Fusarium wilt can cause considerable damage to stock plantings (T.M. O'Neill © 2017. All Rights Reserved.)



Fig. 7 In advanced cases Fusarium wilt will cause all the leaves to dry up (T.M. O'Neill © 2017. All Rights Reserved.)

developing a clearing of the veinal tissue, followed by a general yellowing and then wilting. The yellowing and wilting symptoms may occur on only one side of the plant. Plants will be stunted and wilt during the warmer times of the day. In advanced cases, the leaves can dry up and the plant can die (Fig. 7). Examination of the stem and taproot vascular tissues will reveal a brown discoloration, though the root is not decayed (Fig. 8). Infected stock seed plants may exhibit seed pods that appear flattened and tan in color. On stock, Fusarium wilt symptoms closely resemble those of Verticillium wilt.

Biology and epidemiology. Like most Fusarium wilt pathogens, the stock pathogen (*F. oxysporum* f. sp. *mathioli*) is host specific to its original host and does not infect related species such as cabbage, kale, and radish. The report from Japan names the pathogen as *F. oxysporum* f. sp. *conglutinans* (Saito et al. 2008). This stock pathogen is typical of all *F. oxysporum* wilt isolates and forms four- to six-celled, fusiform, curved macroconidia, one- to two-celled oval- to kidney-shaped microconidia, and resilient chlamydospores. The pathogen can be seedborne and can survive via chlamydospores for long periods in the soil. The disease is most severe if the crop is grown during the warm summer and in regions where temperatures are high. The fungus is favored by temperatures around 25 °C/77 °F.



Fig. 8 Fusarium wilt causes a brown discoloration in stem vascular tissue (T.M. O'Neill © 2017. All Rights Reserved.)

Management

- ***Cultural practices*** – Avoid planting stock in fields known to be infested with the pathogen. Since the pathogen may survive in soil for 2 or more years, practice crop rotation with nonstock plants. Stock grown during the cooler winter or spring seasons and in cool coastal locations may escape the disease or experience less severe impacts from Fusarium wilt.
- ***Soil disinfestation*** – Preplant soil treatments such as fumigation and steaming can help reduce soil inoculum.

2.4 Rhizoctonia Foot Rot (*Rhizoctonia solani*)

Geographic occurrence and impact. While this pathogen is broadly distributed throughout the world, Rhizoctonia foot rot of stock has been reported only from Australia, Greece, the United Kingdom, the United States (California, Florida), and Zimbabwe. The disease has also been called Rhizoctonia root rot or wirestem, and the pathogen also is implicated in damping-off disease of emerging stock seedlings.

Symptoms/signs. The damping-off phase of the disease pertains only to young stock seedlings. For direct seeded crops, the fungus can attack seed or newly germinated seedlings and kill them prior to emergence (preemergence damping-off), or can attack roots and lower stem (hypocotyl) tissues shortly after the plant has emerged above ground (postemergence damping-off). For recently emerged plants, the stems in contact with soil become water soaked and later brown in color; these stems are often girdled and the plants fall over and die.



Fig. 9 *Rhizoctonia solani* infects the stock stem and causes the "wirestem" symptom (T.M. O'Neill © 2017. All Rights Reserved.)

The wirestem phase pertains to older seedlings and consists of browning and cracking of the stem epidermis that is in contact with soil (Dimock 1941, Fig. 9). These stem infections develop into lesions, with the outer tissue decaying away. When the outer stem layers deteriorate, only the fibrous inner xylem remains intact as a wiry strand, hence the name wirestem. Affected plants develop yellow foliage and later wilt. Seedling stems may break at the soil line, resulting in the plant falling over. Plants that survive will likely remain stunted and behind in development. Signs of the pathogen consist of coarse mycelium that can be observed, with a hand lens, in the lesion. The mycelium sometimes causes soil particles to adhere to and dangle from diseased stems. The pathogen is also known to cause a root rot on stock.

Biology and epidemiology. Rhizoctonia foot rot or wirestem is caused by the soil inhabitant *Rhizoctonia solani*. In culture, *R. solani* forms coarse, brown, relatively thick hyphae that are characterized by right-angle branching. Hyphae are constricted at branch points, and a cross wall with a dolipore septum is formed right after each branch. Older cultures form small, brown, loosely aggregated clumps of mycelia that function as sclerotia. The fungus is multinucleate and has a sexual phase, *Thanatephorus cucumeris* (Sneh et al. 1991), though this stage has not been reported on stock. This species is extremely diverse and can be divided into anastomosis groups

(AG) based on the process of hyphal fusion between compatible isolates. AG-2-1 is associated with Rhizoctonia diseases on Brassicaceae hosts (Keijer et al. 1997). While AG-2-1 isolates are pathogenic on plants in this family, these isolates may infect plants outside of the Brassicaceae. Likewise, non-AG-2-1 isolates may infect Brassicaceae hosts. Therefore, these AG pathogens are not absolutely restricted to certain plant groups. *R. solani* survives saprophytically in soil as mycelia or sclerotia; these fungal structures are the inocula for infecting stock seedlings and older plants. The pathogen is favored by warm soil conditions (25 to 30 °C/77 to 86 °F) but is capable of causing problems at much lower temperatures as well.

Management

- ***Cultural practices*** – When placing transplants in the field, avoid planting them too deeply in the soil as the hypocotyl stem tissue is the most susceptible part of the plant. Practice crop rotation so that nonhosts are included in the rotation (Henis et al. 1978). Do not plant stock in fields having undecomposed crop residues, since *R. solani* may be actively colonizing such residues.
- ***Fungicides*** – When direct seeding in the field, use seed that has been treated with a fungicide. If available, direct-spray fungicides to the base of young stock plants in the field.
- ***Sanitation*** – Practice thorough sanitation at nurseries to prevent contamination by *R. solani.* Clean and sanitize transplant trays and benches. Ensure that rooting media are not contaminated by infested soil or diseased plant residues.

2.5 Verticillium Wilt (*Verticillium dahliae* and *Verticillium zaregamsianum*)

Geographic occurrence and impact. Verticillium wilt has been reported on stock in Japan, New Zealand, and the United States (California, New York).

Symptoms/signs. Lower leaves are affected first, becoming yellow and then wilted. In some cases, the leaf interveinal tissue turns yellow first and the veins remain green longer. Plants will be stunted and wilt during the warmer times of the day. The yellowing and wilting of leaves advances up the stem until the stock plant becomes completely affected and can die (Raabe and Wilhelm 1958). Examination of the stem and taproot vascular tissues can reveal a light tan to brown discoloration (Fig. 10). On stock, Verticillium wilt symptoms closely resemble those of Fusarium wilt.

Biology and epidemiology. Verticillium wilt of stock appears to be caused by two *Verticillium* species (Inderbitzin et al. 2011). Most cases of stock Verticillium wilt are caused by *V. dahliae*. However, Verticillium wilt of stock in Japan is caused by *V. zaregamsianum*. This etiology is supported by inoculation experiments. For example, stock is susceptible to a tomato isolate of *Verticillium* which is the *V. dahliae* species; however, even though stock is a member of the Brassicaceae, stock is not susceptible to a *V. longisporum* isolate that infects Brussels sprouts and



Fig. 10 Internal stem tissues infected with *Verticillium* will turn light tan to brown (Steven Koike © 2017. All Rights Reserved.)

other brassica crops. In culture these stock pathogens form the typical *Verticillium* structures consisting of hyaline, verticillate conidiophores bearing three to four phialides at each node. Conidia formed from phialides are hyaline, oval, and single-celled. Older cultures form darkly pigmented, multicelled microsclerotia. The pathogen can survive as resilient microsclerotia for long periods in the soil.

Management

- ***Cultural practices*** – Avoid planting stock in fields known to be infested with the pathogen. Since the pathogen may survive in soil for many years, practice crop rotation with nonhost plants.
- ***Soil disinfestation*** – Preplant soil treatments such as fumigation and steaming can help reduce soil inoculum.

2.6 Additional Fungal and Fungus-Like Diseases

Alternaria leaf spot (*Alternaria japonica*) (Davis et al. 1949; Simmons 2007).
Club root (*Plasmodiophora brassicae*) (Samson and Walker 1982).
Damping-off (*Pythium* species) (Fig. 11).
Gray mold (*Botrytis cinerea*) (Baker et al. 1954).
Phytophthora root rot (*Phytophthora* species) (Fig. 12).
Powdery mildew (*Erysiphe cruciferarum*) (Braun and Cook 2012).
White rust (*Albugo candida*) (Choi et al. 2007; Garcia-Blazquez et al. 2006).

3 Bacterial and Phytoplasma Diseases

3.1 Bacterial Blight (*Xanthomonas campestris* pv. *incanae*)

Geographic occurrence and impact. Bacterial blight is a significant disease of stock that can cause significant losses in yield and quality (Fig. 13). The disease is



Fig. 11 *Pythium* infects stock seedlings early and results stunted growth and/or plant death (T.M. O'Neill © 2017. All Rights Reserved.)



Fig. 12 *Phytophthora* infections result in rotted roots and a reduced root system (T.M. O'Neill © 2017. All Rights Reserved.)

known to occur wherever stock is grown because it is seedborne (Minardi et al. 1988; Rahimian and Okhovatian 1989).

Symptoms/signs. Young seedlings can develop the disease and exhibit yellowing of the stem and leaves, followed by complete wilting of the foliage and plant death. On older plants, initial symptoms consist of yellowing and wilting of the lower leaves. Where lower leaves are attached, the stem develops water-soaked lesions that later turn dark brown to black and are sunken (Fig. 14). Lesions enlarge and can girdle the stem, resulting in complete wilting of the plant. These lesions also weaken the stem and are the points where the stem cracks and causes the plant to fall over (Fig. 15). Examination of the vascular tissue shows a darkened discoloration.



Fig. 13 Bacterial blight can cause significant losses in stock plantings (Steven Koike © 2017. All Rights Reserved.)

Biology and epidemiology. The pathogen, *Xanthomonas campestris* pv. *incanae*, is apparently host specific to stock and did not infect cabbage, cauliflower, or kale in inoculation experiments. In culture the pathogen is typical of most *X. campestris* pathovars and is characterized by slow growing, mucoid, yellow colonies. The pathogen is seedborne (Kendrick 1938) and can also persist in the soil in association with diseased stock plant residues. Disease is initiated by planting infested seed or by planting stock into soil containing plant residues of diseased stock. Once disease has developed, the bacteria are splashed to other plants via rain and overhead sprinkler irrigation. Cool, wet weather favors infection and disease development.

Management

- ***Cultural practices*** – Stock seed can be treated with hot water (50 to 55 °C/122 to 131 °F for 10 min) to disinfest seed; however, such treatments can result in loss of seed germination and need to be implemented carefully. Use drip or furrow irrigation instead of overhead sprinklers. Seed crops should be produced in regions where rain does not occur during seed production. Avoid planting stock in back-to-back plantings; a crop rotation of nonstock crops for 2–3 years likely results in eradication of the pathogen from soil. For stock grown in nurseries and containers, remove and discard symptomatic plants.
- ***Resistance*** – Resistant *M. incana* cultivars are not yet available, though resistance has been found in *M. aspera*, *M. longipetala*, and *M. tricuspidata* (Ecker et al. 1995).



Fig. 14 Stock plants infected with bacterial blight will first develop dark lesions on stems (Steven Koike © 2017. All Rights Reserved.)



Fig. 15 Bacterial blight infections weaken the stems of stock, causing them to crack and break (Steven Koike © 2017. All Rights Reserved.)

3.2 Blossom Blight (*Pseudomonas syringae* pv. *maculicola*)

Geographic occurrence and impact. Blossom blight has been reported only from Australia (Cother and Noble 2009). However, the blossom blight pathogen is widely distributed worldwide on cole crop and other crucifer vegetables (Cintas et al. 2001; Koike et al. 2007; Peters et al. 2004; Shackleton 1996).

Symptoms/signs. Disease first develops on flowers located on the lower portion of the spikes. Infected petals turn brown and later collapse (Fig. 16). Collapsed petals adhere to adjacent flowers. As disease develops, infections progress up the spike and in severe cases affect all the flowers (Cother and Noble 2009). Lesions can also develop where the flower base is attached to the spike. Foliar symptoms consist of small, angular, brown leaf spots that can be seen from both upper and lower leaf surfaces (Cother and Noble 2009, Fig. 17). This symptom has been referred to as



Fig. 16 Brown, collapsed petals caused by *Pseudomonas syringae* pv. *maculicola* (E.J. Cother © 2017. All Rights Reserved.)



Fig. 17 The blossom blight pathogen can also cause small, brown leaf spots that may be surrounded by yellow borders (E.J. Cother © 2017. All Rights Reserved.)



Fig. 18 A brown discoloration in stem tissue can be caused by *Pseudomonas syringae* pv. *maculicola* (E.J. Cother © 2017. All Rights Reserved.)



Fig. 19 Stock seedpods infected with *Pseudomonas syringae* pv. *maculicola* can develop dark brown lesions (E.J. Cother © 2017. All Rights Reserved.)

"pepper leaf spot" on vegetable crucifers (Koike et al. 2007). Leaf spots may be surrounded by yellow borders. A slight discoloration can be observed in stem vascular tissue (Fig. 18). For stock plants producing seed, seed pods may develop lesions (Fig. 19).

Biology and epidemiology. The cause of blossom blight is *Pseudomonas syringae* pv. *maculicola* (Cother and Noble 2009). The pathogen is an aerobic, Gram-negative bacterium that on standard microbiological media forms colonies that are cream to light yellow in color and smooth (Cintas et al. 2001). When cultured on King's medium B, strains that infect stock do not produce the diffusible pigment that fluoresces blue under ultraviolet light; this stock pathogen, therefore, is a non-fluorescing type of *P. syringae.* Consistent with *P. syringae* pv. *maculicola* isolates from crucifer vegetable crops, this stock pathogen can infect cauliflower and tomato (Cother and Noble 2009; Wiebe and Campbell 1993). The pathogen is seedborne in

vegetable hosts and appears also to be seedborne in stock. More details on disease epidemiology are not available.

Management. Disease control options are based on standard integrated disease control principles since the exact details are not available on how the disease develops. Resistant *M. incana* cultivars are not available.

- ***Cultural practices*** – Stock seed can presumably be infested with the pathogen and can be treated with hot water, though such treatments can result in loss of seed germination and need to be implemented carefully. Use drip or furrow irrigation instead of overhead sprinklers. Seed crops should be produced in regions where rain does not occur during seed production. Avoid planting stock in back-to-back plantings. This pathogen likely does not persist for long periods in soil; however, a crop rotation using nonbrassica and nonstock crops for 2 years is advisable. For stock grown in nurseries and containers, remove and discard symptomatic plants.

4 Viral Diseases

4.1 Turnip Mosaic Virus (TuMV, Genus: *Potyvirus*)

Geographic occurrence and impact. Stock is reported to be susceptible to a dozen or more viruses wherever the flower is grown. Some of the more familiar virus pathogens are *Alfalfa Mosaic Virus*, *Beet Curly Top Virus*, *Cauliflower Mosaic Virus*, *Cucumber Mosaic Virus*, *Tomato Spotted Wilt Virus*, and *Turnip Mosaic Virus.* It is not rare to have a stock plant infected with more than one virus (Alioto et al. 1994; Yoon et al. 1998). It appears that *Turnip Mosaic Virus* (TuMV) is perhaps the most commonly reported virus pathogen of stock and occurs worldwide (Bahar et al. 1985; Rosciglione and Cannizzaro 1983).

Symptoms/signs. As with most viruses, symptoms caused by TuMV vary depending on the strain of the virus, stock cultivar, age of plant when infected, and environmental conditions. Leaves will exhibit a range of symptoms consisting of mottling, mosaic, formation of dark green patches or "green islands," and clearing of veins. Leaves may also be curled, twisted, or otherwise distorted. The dark-colored (pink, red, purple) flowers that contain anthocyanin will show breaking, in which the petal coloration is broken up by streaks and sectors of nonpigmented tissue (Severin and Tompkins 1948, 1950; Tompkins 1939, Fig. 20). Cultivars with white and yellow colored flowers do not show flower breaking. Infected plants may be distorted.

Biology and epidemiology. The virus survives between stock crops in living plants such as cruciferous weeds and volunteer stock. Aphids feed on these reservoir hosts, move to stock plants, and inject the virus during feeding. The virus is borne on the stylet of aphids and is transmitted in a nonpropagative (nonpersistent) manner.



Fig. 20 If infected with *Turnip mosaic virus*, dark-colored stock flowers will "break" and develop white streaks and sectors (Steven Koike © 2017. All Rights Reserved.)

Management

- ***Cultural practices*** – Old and infected stock plantings should be disked and buried in the soil. Remove cruciferous weeds and volunteer stock from around fields.
- ***Vector management*** – Control the aphids by using insecticides and other IPM methods.
- ***Resistance*** – Some stock lines and cultivars are resistant to TuMV (Johnson and Barnhart 1956; San Juan and Pound 1963).

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Diseases of Sunflower

27

Thomas J. Gulya, Febina Mathew, Robert Harveson, Samuel Markell, and Charles Block

Abstract

Sunflower (*Helianthus annuus* L.) has become a popular cut flower in the United States and globally. Easy to grow, bright and cheerful large flowers, with ray flowers from white to yellow to orange to magenta, it is often a centerpiece in mixed bouquets. Sunflower and the rest of the *Helianthus* genus are native to North America, and, as such, there is a native population of disease organisms and insect pests that can be a production challenge. Fortunately, ornamental sunflower and oilseed sunflower are the same species, and thus genetic advances made with oilseed germplasm can be readily transferred to ornamentals, if the need arises. The major diseases posing threats to ornamental sunflower are the same as those

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© Springer International Publishing AG 2018

R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_27

threatening oilseed sunflower hybrids, namely downy mildew, rust, Phomopsis stem canker, Sclerotinia wilt, Verticillium wilt, and charcoal rot. However, greenhouse production presents a different environment, and thus, there can be diseases affecting cut sunflowers that are seldom seen under field production. General management strategies for diseases of florists' crops may be found in the introductory chapters on integrated disease management.

Keywords

Alternaria • *Botrytis* • *Fusarium* • *Phomopsis* • *Plasmopara halstedii* • *Puccinia helianthi* • *Phoma* • *Sclerotinia sclerotiorum* • *Verticillium* • White rust

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1 Introduction

Sunflower is a unique crop in that it is grown for ornamental use, as an oilseed crop, and for human consumption. All three uses dictate a different germplasm base, but all are *Helianthus annuus.* Additionally, wild *H. annuus* is native to the United States, and it occurs in almost all of the 48 contiguous states. Another unique feature of sunflower is that it is one of the few crops whose progenitors are native to the United States and was domesticated by Native Americans (Heiser 2008). There are 53 wild *Helianthus* species, of which all but one are found in the United States (Heiser et al. 1969; Schilling 2006). Among the 53 *Helianthus* species, 14 are annual species and 39 are perennial species. Many of these species are adapted to unique environmental niches and thus potential sources of genes, which can be transferred to cultivated sunflower through conventional breeding methods.

The popularity of ornamental sunflowers is relatively recent, and this has spurred university research in many areas of the country, addressing the challenges of many different environments. Oilseed sunflower, in comparison, has been a large and essential global oilseed crop for many decades, and there are numerous sunflower disease guides in many languages besides English (Chattopadhyay et al. 2015; Gulya et al. 1997) including French (CETIOM 2015), Portuguese (Leite 1997), Serbo-Croatian (Maric et al. 1987), Spanish (Alonso et al. 1988), and Russian (Artokhin and Ignatova 2013), whose information is just as relevant to cut flower production as for oilseed sunflower. In addition, the American Phytopathology Society has recently issued the *Compendium of Sunflower Diseases* (Harveson et al. 2016), which will have similar information to this chapter, but with extensive images which should aid with identification. In the United States, extension agencies of several universities have addressed the need for information on the production of cut and potted sunflowers, but few have detailed information on disease management (Garfinkel and Panter 2015; Gill et al. 2003; Schoellhorn et al. 2003; Whipker et al. 1998). The vast majority of public sunflower research in the United States is centered in Fargo, North Dakota, where both North Dakota State University and the USDA-ARS Sunflower and Plant Biology Research Unit are located. North Dakota State University and the USDA-ARS in Fargo have excellent research information available online, at www.ag.ndsu.ext/crops/sunflower and www.ars.usda.gov/main/site_main.htm?modecode=30-60-05-15, respectively. Lastly, there are two other organizations that foster sunflower research, namely the National Sunflower Association (NSA) and the International Sunflower Association (ISA), whose websites are www.sunflowernsa.com and www.isasunflower.org, respectively. The NSA website is publically accessible and contains archives of magazine articles, papers and posters presented at over 30 past "research forums," and a photograph gallery, one part of which deals with diseases and deformities.

2 Fungal and Fungus-like Diseases

2.1 Alternaria Leaf Spot and Blight (*Alternaria* spp.)

Geographic occurrence and impact. Alternaria leaf blight affects sunflower throughout the world and is a major defoliating pathogen in warm, humid climates. The fungus may also cause linear spots on the stems and water-soaked, sunken lesions on the back of sunflower head.

Symptoms/signs. Many *Alternaria* species can cause leaf spots on sunflower, but symptoms are similar for all, making field identification impractical. The primary symptom is dark brown lesions on leaves, and also on stems, petioles, and bracts. Initially the leaf spots are small, dark, and angular (Fig. 1), but with time, they coalesce into large, necrotic areas resulting in defoliation. Defoliation starts with the lower leaves where the microclimate is most favorable. Stem lesions typically are narrow (1–3 mm) black streaks up to 3 cm long.

Biology and epidemiology. Although *Alternaria helianthi* (Hansford) Tubaki and Nishihara, now reclassified as *Alternariaster helianthi* (Hansford) Simmons, is the major causal agent, there are eight other species reported on sunflower, including *A. zinniae* Pape, *A. helianthicola* Rao and Rajagopalan, *A. helianthiinficiens* Simmons, Walcz and Roberts, *A. leucanthemi* Nelen (syn. *Teretispora leucanthemi* (Nelen)

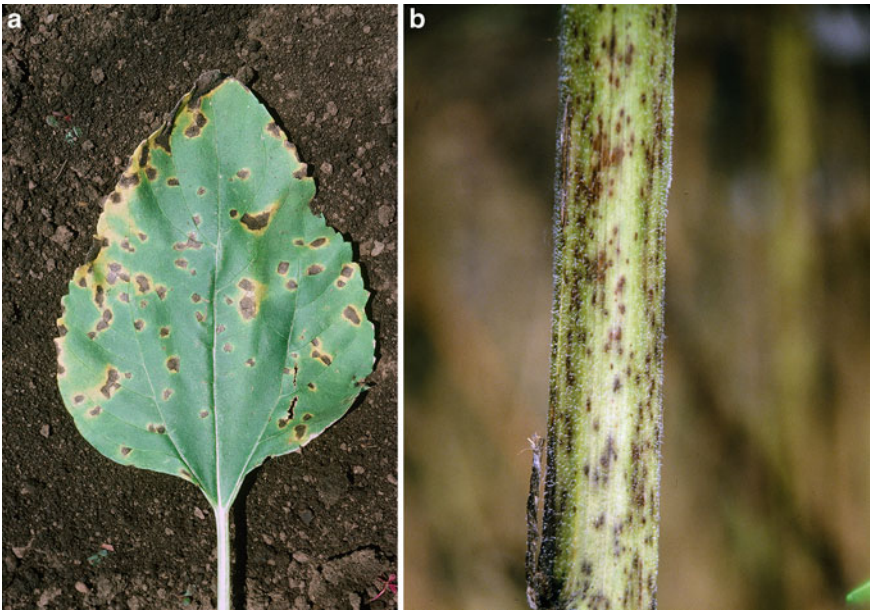


Fig. 1 (**a**) Alternaria leaf blight lesions. (**b**) Alternaria stem lesions (T.J. Gulya. USDA.)

Simmons), *A. protenta* Simmons, *A. roseogrisea* Roberts, *A. tenuissima* (Nees) Wiltshire, and *A. alternata* (Fries) Keissler. Most of these *Alternaria* spp. are specific for sunflower (except the opportunistic saprophyte *A. alternata*). However, *A. zinniae* has a broad host range that includes several *Asteraceae* genera, including weeds (*Bidens*, *Cardus*, *Eupatorium*, and *Xanthium*) and ornamentals (*Aster*, *Calendula*, *Chrysanthemum*, *Dahlia*, *Tagetes*, *Tithonia*, and *Zinnia*). Because symptoms caused by the *Alternaria* pathogens look very similar, field identification based on symptoms is difficult and may be inaccurate. Conidial morphology (Simmons 2008) combined with genetic analysis is the only sure means to delineate species.

As *A. helianthi* is the primary causal agent and most widespread, this section addresses just this species (Allen et al. 1983a, b, c). *Alternaria helianthi* overwinters on infected plant residue, but wild or volunteer sunflowers may also serve as reservoirs. All species including *A. helianthi* may also be seedborne. The conidia are windborne and spread by splashing water onto the lower leaves. They germinate best at temperatures >26 °C and require a minimum of 4 h of leaf wetness for sporulation. Disease progress is also heavily dependent on the duration of leaf wetness following initial infection, as the generation of new spores can occur within 2 days. Young seedlings are more susceptible than older plants, but senescing lower leaves on mature plants frequently are defoliated by *Alternaria* spp.

Management

- ***Cultural practices*** – including removal of wild and volunteer sunflowers, removal or incorporation in the soil of previous sunflower residues, and minimizing extended leaf wetness will all reduce disease potential.
- ***Fungicides*** – Seed treatments (captan, thiram, mancozeb) may offer some control (Jeffrey et al. 1985), but most growers rely upon multiple applications of fungicides containing chlorothalonil, iprodione, procymidone, or vinclozolin. More recent fungicide tests with ornamental sunflowers demonstrated that seed treatments with difenoconazole, prochloraz, pyrifenox or triadimenol were effective (Wu and Wu 2003). Consult government fungicide guides for specific products and rates.
- ***Resistance*** – Disease resistance has been found in oilseed sunflower and some attempts have been made to incorporate that into hybrids, but there have been no reports of resistance in ornamental cultivars.

2.2 Botrytis Blight or Gray Mold (*Botrytis cinerea* Persoon)

Geographic occurrence and impact. The fungus causing gray mold, *Botrytis cinerea* (syn. *Botryotinia fuckeliana* (de Bary) Whetzel), is found worldwide and impacts many floral, fruit, and vegetable crops. On sunflower, the pathogen causes mostly a head rot. However, it can also cause spots on leaves and petals and is most often observed in areas with cool summers accompanied by frequent rains. It is rarely seen outdoors in most areas of the United States but could be a problem in greenhouses where sunflower leaves remains wet for extended periods of time.



Fig. 2 Botrytis head rot (T.J. Gulya. USDA.)

Symptoms/signs. Gray mold first appears as sunken brown spots on the back surface of the head. During periods of high humidity or rainfall, these lesions will be covered with conidia, giving the infected tissue a gray color (Fig. 2). The gray color is an easy, macroscopic way to distinguish *Botrytis* head rot from other head rots. If heads are infected early and wet conditions persist, *Botrytis* may form small, black sclerotia within the sunflower head. *Botrytis* has also been observed to cause petiole infections in Egypt, India, and Pakistan, which can progress to the stem, resulting in lesions up to 10 cm long.

Biology and epidemiology. *Botrytis cinerea* is a ubiquitous fungus and a facultative pathogen with a wide host range encompassing most ornamentals, soft fruit, and monocots.

B. cinerea overwinters in the soil as sclerotia or as mycelium on infected plant debris and is also seedborne (Coley-Smith et al. 1980; Elad et al. 2007). Sclerotia germinate to produce conidia, which can colonize dead organic matter as well as living host tissue. Conidia are windblown and water splashed and require a wound or senescent tissue to gain entry into a plant. Optimal conditions for infection are 15–25 °C and 90% relative humidity. Under optimal conditions, *Botrytis* may produce a new crop of conidia every 5–7 days. *Botrytis* produces copious amounts of spores, which develop rapidly in decaying vegetation and on senescing flowers. Because of the wide host range, the inocula for sunflower infection may come from many other floral crops or weeds.

Management. For control of *Botrytis* on greenhouse or field grown ornamental sunflowers, fungicide applications coupled with cultural practices are useful.

- ***Cultural practices*** – Since spores readily form on infected plant tissue, elimination of infected plants is essential. Avoidance of overhead watering, especially prior to bloom, is important, or at least irrigate early in the day to promote drying. Wider spacing between plants will minimize the RH within the canopy, but this may not be practical under the intensive plant populations as seen in commercial

field operations. The fungus can develop at low temperatures, so cut flowers with no apparent infection may develop gray mold even under refrigeration. As free water is necessary for infection, minimizing conditions that lead to condensation in cold storage is critical (Gibson et al. 2014).

- ***Resistance*** – Oilseed germplasm with resistance to *Botrytis* has been identified, but to date these genes have not been incorporated into either oilseed hybrids or ornamental germplasm (Kanyion and Friedt 1993).

2.3 Charcoal Rot (*Macrophomina phaseolina* (Tassi) Goidànich)

Geographic occurrence and impact. Charcoal rot is a serious root-infecting disease that is found worldwide but is most severe in hot, dry climates. It is both soil- and seedborne (Raut 1983). The fungus has little effect on plants until they become stressed; the stress may be due to heat, lack of soil moisture, or seed filling. The disease may kill plants outright, but it also results in reduced head size.

Symptoms/signs. Damping-off may occur if *M. phaseolina*-infected seeds germinate in soil at or near 35 °C. Usually, symptoms appear on older plants after flowering, with a silvery-gray lesion at the base of the plant and eventually encircling the stem. Upon splitting open the stem longitudinally, one will notice pith disintegration, and often a characteristic compression of the pith into layers. Examination with a 5 to 10X hand lens will reveal tiny microsclerotia, which appear as pepper grains scattered within the pith and on the inside of the rind. Severe infections, especially on stressed plants, will cause premature death (Fig. 3a, b).

Biology and epidemiology. Charcoal rot is incited by *Macrophomina phaseolina*. This fungus has had 17 binomials as synonyms, of which *Rhizoctonia bataticola* and

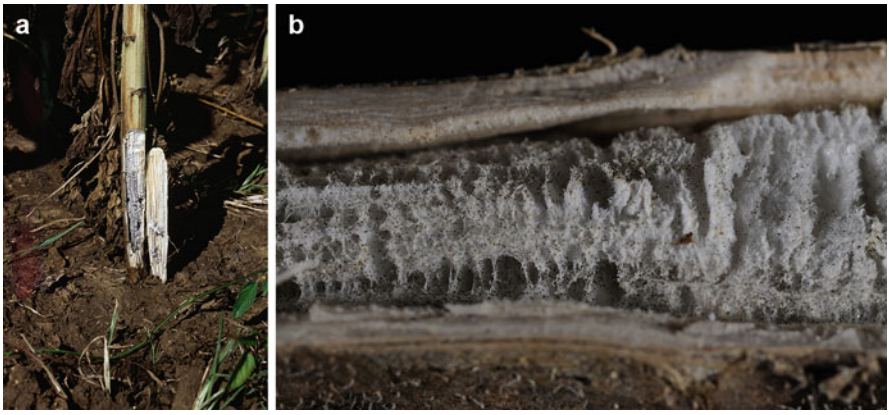


Fig. 3 (**a**) Charcoal rot, showing pith compression and microsclerotia. (**b**) Charcoal rot on sunflower, showing pith with microsclerotia (T.J. Gulya. USDA.)

Sclerotium bataticola are most commonly found in older literature. The fungus is characterized by producing black microsclerotia, 60–200 μm in diameter. Occasionally an isolate will be found which produces pycnidia (and pycnidiospores) in culture, but this is very rare in infected sunflower tissue. The fungus has a wide host range, but there is no evidence of isolates having host specificity.

Macrophomina phaseolina has an extremely broad host range, encompassing 500 species of both monocots and dicots (Dhingra and Sinclair 1978). Thus, nearly all ornamentals are hosts, as well as field crops, vegetables, and weeds. The fungus does not produce any conidia, but persists by forming small, irregular, black microsclerotia (60–200 μm in diameter) which are barely visible with the unaided eye. Microsclerotia in infected plant residue are released into soil as the host tissues decompose, and they can persist in the soil for at least 3 years. The fungus is unusual in its affinity or tolerance of high temperatures and grows best at soil temperatures >35 °C. Other conditions which favor disease development include low soil moisture, light sandy soils, as well as stress of the host plants due to herbicides or nematodes (Singh et al. 2012). Charcoal rot is frequently associated with Fusarium stalk rot. Additionally, the sunflower stem weevil has been shown to vector *Macrophomina phaseolina* in sunflowers (Yang and Owen 1982).

Management

- ***Cultural practices*** – Minimizing plant stress by lowering plant densities, and providing adequate soil moisture, will reduce the onset of the disease. However, since sunflowers grown for cut flowers are harvested in the late bud stage, charcoal rot should seldom be seen. Crop rotations will have little effect on the fungus due to its wide host range and the longevity of the microsclerotia.
- ***Fungicides*** – Seed treatments with fungicides will minimize the introduction of *M. phaseolina* (Raut 1983) and protect against early infection.

2.4 Damping-off (*Pythium* spp., *Phytophthora* spp., and *Rhizoctonia solani* Kühn)

Geographic occurrence and impact. Damping-off of oilseed sunflower is rarely noted in the literature, but is more likely to be observed with ornamental sunflowers due to their intensive management practices. Damping-off, as the name implies, leads to seedling death, whether pre- or postemergence, and thus can be quite devastating. Pathogens inciting damping-off are global in occurrence, although exact species may differ from region to region.

Symptoms/signs. Damping-off may be the result of seeds or seedlings rotting prior to emergence, or more frequently, the seedlings collapse as they emerge as the roots and/or stem at the soil line becomes necrotic and the plant collapses. When older seedlings are affected, the plant becomes stunted and dies later. *Pythium* spp.

generally attack the root tips, causing a dark brown to black rot, while various *Phytophthora* spp. may attack the fine roots including the tap root, crown, and all belowground parts (Banihashemi 1975). *Rhizoctonia solani* commonly causes a brown to black lesion encircling the stem at the soil line and may occur both on seedlings and older plants (Srinivasan and Visalakchi 2010).

Biology and epidemiology. All three pathogen groups have broad host ranges. *Pythium* species reported on sunflower include *P. aphanidermatum* (Edson) Fitzpatrick, *P. irregulare* Buisman, *P. debaryanum* Hesse, *P. rostratum* Butler, *and P. splendens* Hans Braun (Hendrix and Campbell 1973). *Phytophthora* species reported on sunflower include *P. cryptogea* Pethybridge and Lafferty, *and P. drechsleri* Tucker. *Rhizoctonia solani* isolates from sunflower belong to anastomosis groups AG-3 and AG-4.

Pythium spp., *Phytophthora* spp., and *R. solani* are all soilborne and found worldwide, and *Rhizoctonia* has been shown to be seedborne in sunflower (Lakshmidevi et al. 2010). They can survive for years in soil or infected crop residues with overwintering structures (sclerotia for *Rhizoctonia* and oospores for *Pythium and Phytophthora*). Sclerotia of *R. solani* germinate to form mycelium which attacks seedling roots. The oospores produced by *Pythium* spp. and *Phytophthora* spp. germinate to form motile zoospores that are chemotactically attracted to root exudates. Thus, water-logged soils are necessary for infection by *Pythium* spp. and *Phytophthora* spp., with longer periods of saturation leading to more damping-off.

Management

- ***Cultural practices*** **–** Crop rotation is generally of little use due to the broad host ranges and the longevity of overwintering fungal structures. At this time, disease management is aimed at prevention. Using disease-free planting media and avoiding overwatering are the easiest means of minimizing damping-off.
- ***Sanitation*** – Discard pots or flats with plants showing damping-off.
- ***Soil disinfestation*** – For field production in warmer climates, solarization has been reported to provide control.
- ***Fungicides and biocontrols*** – Fungicidal seed treatments, initially registered for control of downy mildew (metalaxyl and mefenoxam) will generally give control of *Pythium* and *Phytophthora*, while broad-spectrum strobilurin fungicides will help manage *Rhizoctonia*. Various biocontrol formulations of *Bacillus*, *Gliocladium*, and *Trichoderma* spp. are available that are applied as seed inoculations, incorporated into potting soil, or used as soil drenches. Fungicide soil drenches are also used in nonorganic production. Consult government publications for recommended products.
- ***Resistance*** **–** Although differences in susceptibility have been noted among oilseed sunflowers to these three pathogens, resistance has not been incorporated into either oilseed or ornamental sunflower.

2.5 Downy Mildew (*Plasmopara halstedii* (Farlow) Berlese and de Toni)

Geographical occurrence and impact. The pathogen causing downy mildew is found worldwide, with the exception of Australia. Downy mildew is one of the major diseases affecting oilseed sunflower, and can be devastating on ornamentals as well. The pathogen primarily causes systemic infection, which renders the plant unsalable thus affecting florists' trade.

Symptoms/signs. The most commonly seen symptoms are those caused by root infection of seedlings leading to a systemic infection (Friskop et al. 2009). Affected plants have chlorosis on the upper surface of leaves (either the entire leaf or the interveinal areas), with a white covering of spores on the underside of the leaves. Affected plants are severely stunted, and if they do not die within a few weeks, they will produce a head which is horizontal rather than vertical. The pathogen, by means of airborne spores, can also incite leaf spots typical of other downy mildews, with chlorotic spots on the upper surface, with a white coating of spores on the underside of each lesion. These "local lesions" do not lead to systemic infection and are of less impact as the affected leaves can be removed (Fig. 4a, b).

Biology and epidemiology. Downy mildew is caused by the obligate pathogen *Plasmopara halstedii* (Farlow) Berlese & de Toni (syn. *Peronospora halstedii* Farlow). The host range of the causal pathogen includes sunflower and all wild *Helianthus* species, plus a number of related Asteraceae weeds and native plants, including *Ambrosia*, *Bidens*, *Iva*, *Eupatorium*, *Silphium*, *Solidago*, and *Xanthium. P. halstedii* is also reported to cause downy mildew on some ornamental genera such as *Centaurea*, *Coreopsis*, *Dimorphotheca*, *Erigeron*, *Gerbera*, *Rudbeckia*, *Senecio*, and *Verbena*; however, cross-pathogenicity studies to prove isolates from these can infect sunflower, and vice versa, have not been done. *Plasmopara halstedii* exists as physiological races on sunflower, and there is evidence that there are biotypes infective on specific genera, even though morphologically the pathogen isolates are indistinguishable.

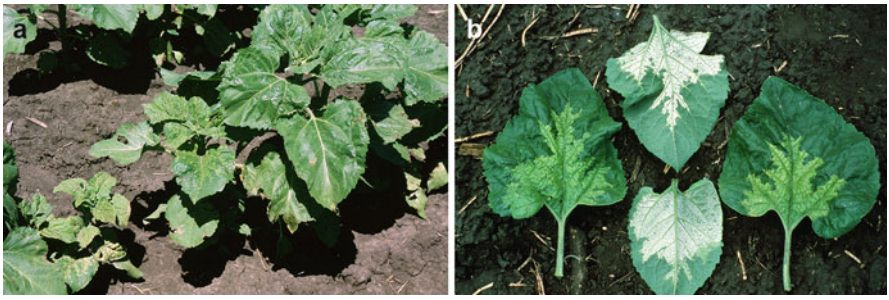


Fig. 4 (**a**) Downy mildew infected plants showing stunting and leaf chlorosis. (**b**) Downy mildew infected leaves showing chlorosis on upper surface and white sporulation on bottom surface (T.J. Gulya. USDA.)

Plasmopara halstedii overwinters as resistant oospores, which germinate in water-logged soils to produce motile zoospores. The spores swim towards sunflower roots and infect young seedlings to produce a systemic infection. Infection of older plants will produce a club root without leading to systemic infection. Once the fungus becomes systemic and produces spores on the undersurface of leaves, these spores can be blown some distance and can initiate foliar infection (i.e., local lesions). As the plants die, the fungus produces oospores which can remain viable for years in the soil. The fungus and disease development is fostered by cool (<15.5 °C) water-logged soils. Spore development takes place when there is overnight leaf wetness due to irrigation, dew, or rain. Leaf infection requires several hours of leaf wetness for spore germination and colonization of the leaf tissue. Once a plant is systemically infected, the impact of the environment is minimal.

Management. Downy mildew can be managed quite successfully through genetic resistance, fungicide seed treatments, and, to some extent, crop rotation.

- ***Cultural practices*** – Since the oospores are relatively long-lived, crop rotation is of limited effectiveness. Managing soil moisture, whether in a greenhouse situation or in field production, to limit water-logged soil will greatly curtail the germination of oospores and thus the initial infection. Managing watering to limit leaf wetness, especially overnight when temperatures are cool, will minimize the chances for local lesions to develop on leaves.
- ***Fungicides*** – Fungicidal seed treatments have been used quite successfully to prevent infection on both oilseed and ornamental sunflowers (Friskop et al. 2015a). However, *P. halstedii* has developed resistance to both metalaxyl and mefenoxam, rendering these two fungicides of minimal usage worldwide (Gulya et al. 1999). Strobilurin seed treatments are registered on oilseed sunflowers, but do not offer the complete control as previously displayed by metalaxyl and mefenoxam. Most broad-spectrum seed treatments are not effective against Oomycetes, the taxonomic group to which *P. halstedii* belongs.
- ***Resistance*** – Since the pathogen has multiple physiological races, genetic resistance must be chosen to combat those races present. Downy mildew is one of the few diseases for which breeders of ornamental sunflower have intentionally incorporated resistance, choosing genes which combat a wide range of races.

2.6 Fusarium Stalk Rot and Wilt (*Fusarium* spp.)

Geographic occurrence and impact. The complex of *Fusarium* spp. causing either stem rot or wilt are soilborne and ubiquitous around the world. On oilseed sunflower their impact is minimally acknowledged as the fungi are often found in association with other pathogens, and the exact role of *Fusarium* spp. is underplayed. However, in eastern Europe and Russia, Fusarium-incited stem rots

cause serious damage. While there are no reports in the literature of Fusarium on ornamental sunflowers, this does not preclude that ubiquitous and opportunistic *Fusarium* can cause problems on ornamental sunflowers as well.

Symptoms/signs. Fusarium stem rot has few distinct external symptoms, which culminate in premature wilting, leaf senescence, and ultimate plant death. Internally, many *Fusarium* species degrade the pith and, upon splitting the stems longitudinally, one may note a pink to reddish hue to the pith, starting at the soil line (Fig. 5).

Biology and epidemiology. Recent research has identified a large number of *Fusarium* spp. inciting stem rot on sunflower, including *F. acuminatum* Ellis and Everhart (synonym *Fusarium scirpi*, *F. gibbosum*), *F. avenaceum* (Fries) Saccardo, *F. anthophilum* (Braun) Wollenweber, *F. avenaceum* (Fries) Saccardo, *F. chlamydosporum* Wollenweber and Reinking, *F. culmorum* (Smith) Saccardo, *F. equiseti* (Corda) Saccardo, *F. graminearum* Schwabe, *F. sporotrichioides* Sherbakoff, and *F. proliferatum* (Matsushima) Nirenberg (Mathew et al. 2010, 2011; Nahar and Mushtaq 2007). Of these, *F. acuminatum*, *F. equiseti*, *and F. sporotrichioides* are the most aggressive (Mathew et al. 2010). Fusarium wilt, in contrast, has only two species associated with it, namely *F. oxysporum* Schlechtendal and *F. verticillioides* (Saccardo) Nirenberg (synonym *F. moniliforme*). See Leslie and Summerell (2006) to aid in identification of *Fusarium* spp.

Despite the multitude of *Fusarium* spp. associated with stem rot and wilt, their biology is very similar. The fungi survive in the soil within infected plant residues of their many hosts, either as mycelium or resting structures called chlamydospores.



Fig. 5 Fusarium stem rot with characteristic pink coloration of pith (T.J. Gulya. USDA.)

There are also reports of some species being seedborne. The fungi produce either micro- or macroconidia (or both) and these are dispersed by rain and wind. The conidia infect the root system and then colonize the internal tissues of the crown and lower stem. Symptoms generally are not noticed until the plants are subjected to stress, such as drought, lack of soil moisture, or seed filling. Many instances have been noted when *Fusarium* spp. act in concert with *Macrophomina phaseolina*, the causal agent of charcoal rot (de Barros 1985; Mathew et al. 2010).

Management. Controlling *Fusarium*-incited stem rots and wilts depends mainly upon sanitation and cultural practices.

- ***Cultural practices*** – Chlamydospores produced by *Fusarium* spp. can persist up to 4 years or longer in soil, so rotation is of minimal use for most *Fusarium* spp. However, the wilt-inducing *Fusarium* spp. exist as host-specific biotypes, so rotating with other plant genera should be of benefit.
- ***Soil disinfestation*** – Pasteurization of potting media and solarization of fields in warm climates have provided control of *Fusarium* and other soilborne fungi. Soil fumigation of raised beds has shown to be more effective than with ground beds.
- ***Resistance*** – While *Fusarium* resistance has been sought and bred into many other floral crops, there has yet to be any similar effort on ornamental sunflower, although resistance has been found in oilseed sunflower germplasm.

2.7 Leaf Smut (*Entyloma* spp.)

Geographic occurrence and impact. Leaf smut has only been reported on greenhouse grown sunflowers and thus is of limited occurrence (Dirac et al. 1999). As the disease disfigures the leaves, the impact of the fungus is lessened as most leaves are removed from cut sunflowers prior to marketing.

Symptoms/signs. Leaf smut on sunflower is characterized by small yellow-green spots that coalesce to form white lesions, delimited by veins. The white coating is both mycelium and spores, and occurs on both leaf surfaces. With age, the lesions turn necrotic and may result in defoliation (Fig. 6).

Biology and epidemiology. *Entyloma polysporum* (Peck) Farlow is the only confirmed *Entyloma* attacking sunflower, although *E. gaillardianum* Vánky and *E. compositarum* Farlow are reported on other Asteraceae ornamentals. Recent *Entyloma* isolates from sunflower grown in California may be a new undescribed species.

Leaf smut is favored by humid, cool, and shaded conditions and, as such, is more likely to be seen in greenhouse rather than field production. Spores are wind-borne and water splashed to healthy tissue. Under optimal conditions, 100% of the crop can be affected with significant losses. The fungus likely survives as the thick-walled teliospores. Systemic infections of young seedlings have been observed, suggesting the fungus may be seedborne. The fungus persists in the soil as teliospores or chlamydospores, of unknown longevity (Byrne 2006).



Fig. 6 Leaf smut (Entyloma) (H. Schenk © 2017. All Rights Reserved.)

Management. Leaf smut has yet to be observed in field-grown sunflower, and thus management is largely aimed at greenhouse production, where it is still rare.

- ***Cultural practices*** – Minimizing overhead irrigation to limit the duration of wet leaves will lessen the chance of infection and immediate rogueing of infected plants will minimize the spread of leaf smut.
- ***Fungicides*** – Foliar sprays with sterol-inhibiting systemic fungicides such as cyproconazole and difenoconazole have controlled leaf smuts on other floral crops (Ruegg 1990).

2.8 Phoma Black Stem (*Phoma macdonaldii* Boerema)

Geographic occurrence and impact. Phoma black stem has been reported from all sunflower-producing countries. It is the most commonly seen disease in the Northern Great Plains of the United States, but is rarely considered serious. In contrast, it is considered a yield-limiting factor in France and other European countries. Since the fungus is specific to *Helianthus*, it may be absent in areas where wild or cultivated sunflower has not been long present. While the stem lesions are usually superficial and seldom cause internal damage, if combined with stem-infesting insects, the combination can lead to pith rotting and premature death.

Symptoms/signs. The most characteristic symptom associated with this disease is a black, sometimes glossy, round to elliptical stem lesion centered on petioles (never internodal) (MacDonald 1964) (Fig. 7). The infection, however, starts as nondistinct leaf spots which coalesce and continue down the veins, onto the petioles, and thus onto the stem. Affected leaves quickly senesce, usually by the time the stem lesions appear. *Phoma* may also incite lesions on the bracts and back of the head. Girdling,



Fig. 7 Phoma black stem (T.J. Gulya. USDA.)

black lesions at the soil line may quickly lead to premature death (Donald et al. 1987). Phoma stem lesions rarely penetrate into the stem, but if stem-infesting weevils (such as *Apion* and *Cylindrocopturus* spp.) are present, this often leads to pith degradation and lodging. Mixed infections with Phomopsis stem canker have been observed, and the color of these lesions will be gradations of brown to black.

Biology and epidemiology. While in older literature the fungus was initially listed as *Phoma oleracea* var. *helianthi-tuberosi*, it has been reclassifed as *Phoma macdonaldii* Boerema (syn. *Leptosphaeria lindquistii* Frezzi; Boerema et al. 2004). *Phoma* infection may occur anytime in the growing season, but is usually noted as stem lesions appear, close to anthesis. The fungus overwinters as perithecia in infected crop debris which release ascospores. The ascospores are spread by splashing water and wind to initiate leaf infection. While the fungus infects over a wide range of temperatures (5–30 °C), it requires at least 24 h of leaf wetness for colonization and is thus often seen on lower leaves where the microclimate is favorable. Small, black pycnidia can form in stem lesions and can be seen with a 10X hand lens. In the United States, stem-infesting insects have been shown to vector *Phoma*, and they oviposit in the leaf axils. The subsequent larva carry *Phoma* as they tunnel in the pith, leading to large areas of rotted pith and resultant lodging (Gaudet and Schultz 1981).

Management. Cultural practices are the major means of control, although broad-spectrum fungicides may be of use.

- ***Cultural practices*** – Reducing plant densities, fostering better air movement, and lower nitrogen fertilization to minimize canopies will all aid in creating a microclimate less favorable to initial leaf infection (Debaeke and Peres 2003).
- ***Resistance*** – Resistance has been noted in oilseed germplasm, but there are no reports of Phoma resistance being incorporated into ornamental varieties.
- ***Insecticides and biocontrols*** – Controlling stem-feeding insects will preclude insect transmission.

2.9 Phomopsis Stem Canker (*Diaporthe helianthi* and Other *Diaporthe* spp.)

Geographic occurrence and impact. Phomopsis stem canker has become one of the most yield-limiting diseases worldwide on oilseed sunflower (Mathew et al. 2015c), but reports of the disease on ornamental sunflower are rare to date. On oilseed sunflower the fungus causes extensive pith rotting and lodging, and would be devastating on ornamental sunflower. The fungus *Diaporthe helianthi* is of "quarantine significance" in the EU and has not been recorded to date in Australia.

Symptoms/signs. The causal fungus *Diaporthe* spp., like *Phoma*, initiates a leaf infection starting at the leaf margin, and causes necrotic areas limited by veins, surrounded by a chlorotic margin (Masirevic and Gulya 1992). As the infection progresses down the main vein to the petiole, it reaches the stem where a large brown lesion (up to 15 cm or more) is seen (in contrast to the black lesion caused by *Phoma*) (Fig. 8). Lesions are always centered on the axil. The fungus penetrates the stem and causes extensive rotting of the pith, which often results in lodging and premature death. An easy way to distinguish *Phoma* from stem lesions of Phomopsis stem canker, apart from the color difference, is that light thumb pressure will usually crush a *Phomopsis*-weakened stem. As the plant matures, small black pycnidia, visible with a 10X hand lens, can be seen within the stem lesion.

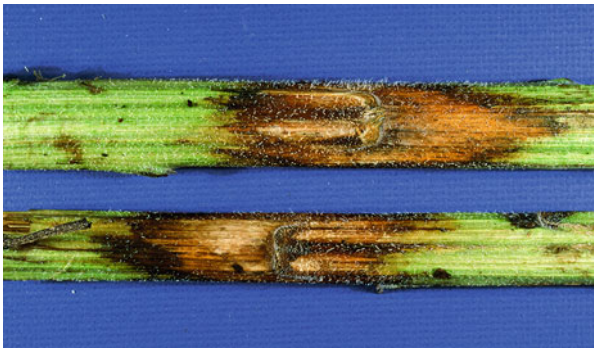


Fig. 8 Phomopsis stem canker (T.J. Gulya. USDA.)

Biology and epidemiology. Phomopsis stem canker was only recognized in the 1980s, when *D. helianthi* Muntañola-Cvetkovic, Mihaljcevic, and Petrov (syn. *Phomopsis helianthi* Muntañola-Cvetkovic, Mihaljcevic, and Petrov) was identified as the causal agent, first in Yugoslavia, later in Europe and the rest of the world. Several other new species of *Diaporthe* have been identified as stem pathogens of sunflower. Morphologically and symptom-wise they are almost indistinguishable, and thus molecular methods are necessary to speciate the causal fungus. A total of ten new *Diaporthe* species have thus been added as sunflower pathogens, including *D. gulyae* Shivas, Thompson, and Young; *D. kongii* Shivas, Thompson, and Young; *D. kochmanii* Shivas, Thompson, and Young; *Diaporthe masirevicii* Shivas, Thompson, and Tan; and *D. sackstonii* Shivas, Thompson, and Tan (Thompson et al. 2011, 2015). Of these, *D. gulyae* is the most aggressive, comparable with *D. helianthi*, and the former occurs in Australia, Canada, and the United States (Mathew et al. 2015b, c). No studies have looked into the *Phomopsis* complex that affects sunflower in other countries.

Diaporthe spp. survives on infected plant debris as mycelium and pycnidia, and also can be seedborne. Many of the species can cause infections on other crops and weeds, which thus serve as inoculum reservoirs (Thompson et al. 2015). The infected stems develop perithecia after overwintering, and they release ascospores following each rain event, to be dispersed by splashing water and wind. The ascospores germinate in guttation drops at the leaf margin, so free water is not absolutely essential for infection. Once the vascular leaf tissue is colonized, the mycelium progresses to the petiole and then the stem. Ascospore germination is favored by warm temperatures (20–25 °C), but rainfall is more critical for ascospore discharge and dispersal.

Management. In oilseed production, foliar fungicides had been used to prevent leaf infection, but current management practices rely more on genetic resistance and crop management. The remaining management tools are cultural practices and rotation.

- ***Cultural practices*** – Any practice that minimizes dense canopies, which foster long water retention on the leaves, such as reduced plant densities, drip versus overhead irrigation, not watering during the night, and lower nitrogen fertilization, will in turn minimize the possibility of *Phomopsis* infection (Debaeke et al. 2003). While *D. helianthi* and other sunflower pathogens were thought to be host specific, recent evidence shows they can infect and survive on weeds and other hosts, both on live and dead plants (Thompson et al. 2015). Rotation will thus be of limited use, but eliminating weeds and wild sunflowers from the field and margins will potentially cut down on inoculum. Burying plant residue, at least 5 cm deep, will hasten plant decomposition and expose *Diaporthe* to biodegration.
- ***Fungicides*** – Registered, systemic strobilurin compounds are likely to be most effective for control on ornamentals.
- ***Resistance*** – Resistance is widely available in oilseed germplasm, but as yet not incorporated into ornamental sunflowers.

2.10 Powdery Mildews (*Erysiphe* and Other Genera)

Geographic occurrence and impact. Powdery mildew is observed on sunflowers in all continents, with three primary genera being responsible. On oilseed sunflower the disease occurs late in the seed-filling period, and is generally not considered a serious threat in temperate regions, but more likely a problem in tropical or subtropical climates. On ornamental sunflowers where appearance is a primary concern, powdery mildew can be more of a problem. If infection is limited to the foliage, which is stripped prior to marketing, the impact is lessened, but powdery mildew can also occur on stems and the back of the heads.

Symptoms/signs. Powdery mildew caused by *Golovinomyces cichoracearum* (DeCandolle) Heluta and *Podosphaera xanthii* (Castagne) Braun & Shishkoff first appears as small, dull-white, circular spots on the upper surfaces of the lower leaves. The spots expand and eventually encompass the entire leaf. The infection spreads to upper leaves as they mature, and spots may also appear on stems, bracts, and the back of the head. Affected leaves often senesce and fall off prior to maturity (Fig. 9). As the leaves senesce, the cleistothecia appear as small black dots. Symptoms caused by *Leveillula taurica* (Léveillé) Arnaud differ slightly from that caused the other two fungi in that the initial spots are a pale green-yellow in color.

Biology and epidemiology. The major powdery mildew fungus seen worldwide on sunflower was previously known as *Erysiphe cichoracearum* DeCandolle, recently reclassified as *Golovinomyces cichoracearum* (DeCandolle) Heluta. In Asia, Africa, Europe, and South America, two other powdery mildew fungi have been recorded on sunflower: *Podosphaera xanthii* (Castagne) Braun and Shishkoff (syn. *Sphaerotheca fuliginea* (Schlechtendal) Pollacci) and *Leveillula taurica* (Léveillé) Arnaud. Identification of powdery mildew is usually based on the morphology of the cleisothecia, the sexual fruiting structure, and using molecular methods (Braun et al. 2002). In many instances, cleistothecia are not seen on infected plants, and thus identification relies on conidial morphology. In temperate climates, the three



Fig. 9 Powdery mildew on young sunflower plant (T.J. Gulya. USDA.)

taxa overwinter as cleistothecia in infected plant debris. In the spring, ascospores are released, which are wind-borne and infect leaf tissue. Powdery mildew fungi are unique in that the optimum relative humidity for infection is 75–95% and optimal temperatures between 15 °C and 25 °C. Free water can inhibit germination of the spores produced by the powdery mildew fungus. In subtropical or tropical regions, the powdery mildew pathogens survive between crops living on alternative hosts. After the initial infection, subsequent spread is by conidia, and their discharge is usually midday when wind speed is highest and humidity lowest. Under optimal conditions, there can be many cycles of infection, resulting in total colonization of the foliage. The host range of *G. cichoracearum* is restricted to the Asteraceae, while both *L. taurica* and *P. xanthii* have host ranges across several plant families, including Alliaceae, Solanaceae (*P. xanthii*), and Cucurbitaceae (both species).

Management. With oilseed sunflower, powdery mildew is managed by genetic resistance and/or foliar fungicides.

- ***Fungicides*** – Several fungicides provide very good control of *G. cichoracearum* including difenoconazole, penconazole, propiconazole, and triadimefon (Akhileshwari et al. 2012). Powdery mildew fungi have developed resistance to certain fungicides, so to be effective a spray program should include use of chemicals with differing modes of action.
- ***Resistance*** – Resistance to *G. cichoracearum* has been identified in many wild annual and perennial *Helianthus* species, and the USDA has released interspecific crosses (Jan and Chandler 1988; Reddy et al. 2013). This resistance, however, has not been transferred to ornamental cultivars.

2.11 Rhizopus Head Rot (*Rhizopus* spp.)

Geographic occurrence and impact. Rhizopus head rot has been reported from all sunflower-producing countries. It causes the most damage in areas with hot, dry growing conditions and where heads are damaged by insects and/or birds (Rogers et al. 1978; Shtienberg 1997). Thus in the United States, it is more severe in the southern Great Plains and California than in the cooler northern Great Plains.

Symptoms/signs. Rhizopus first appears on the back of heads as dark spots, followed by a watery, soft rot (Harveson 2013). In time the entire head is affected and the head turns brown and is "mumified." If the head is split open, the interior of the back of the head will have gray mycelium produced by the causal fungus interspersed with black sporangia the size of pepper grains. The head is seldom shredded as seen with *Sclerotinia* nor is there any characteristic odor as that associated with bacterial head rot. The face of the head may also be covered with a gray mycelium along with the black sporangia, but this is less common. The fungus also produces a toxin that will cause an extensive dark brown girdling lesion on the stem, extending down from the head (Fig. 10).



Fig. 10 Rhizopus head rot (T.J. Gulya. USDA.)

Biology and epidemiology. Several species of *Rhizopus* initiate head rot including *R. stolonifer* (Ehrenberg) Vuillemin, *R. oryzae* Went and Prinsen Geerligs (syn. *R. arrhizus* Fischer), and *R. microsporus* Tieghem. These *Rhizopus* spp. are differentiated on the basis of morphological characteristics and optimal growth temperature. All species are ubiquitous around the world and cause soft rots of fruits and vegetables and commonly referred to as "bread mold."

Rhizopus spores are found everywhere in soils and are widely disseminated in the air. The fungus, however, is categorized as an opportunistic pathogen as it cannot colonize healthy tissue. Any injury to the back of the head, whether caused by insects, birds, or hail, will provide a site for *Rhizopus* to colonize (Rogers et al. 1978). With sunflower, the most common insects affecting heads are larva of *Homeosoma electrum* (Klisiewicz 1979) and *Heliothis*. Pupa of a fly, *Melanagromyza splendida*, has been recently shown to cause pith decay and a stem lesion in California (Mathew et al. 2015a). The three *Rhizopus* species have different maximum temperatures for growth and thus head rot can develop at temperatures from 20 °C to 45 °C.

Management

- ***Cultural practices*** – Very few definitive management recommendations can be made for this disease. The primary techniques include avoiding mechanical damage to heads by controlling insect and bird feeding after flowering. Controlling weed sunflowers on field perimeters and ditch banks will also remove potential reservoirs of sunflower insects and pathogens.
- ***Fungicides*** – Dinitroaniline-based fungicides are effective against *Rhizopus* species.
- ***Resistance*** – No known resistant hybrids are available; however, research is underway to identify sunflower germplasm with potential resistance to the pathogen.

2.12 Rust – *Puccinia helianthi* Schwein.

Geographic occurrence and impact. Sunflower rust is found worldwide on oilseed, ornamental, and wild sunflowers (*Helianthus*). In epidemic situations, leaves wilt and senesce prematurely, which also leads to smaller head size. As the fungus also produces lesions on stems and bracts, rust can seriously damage the appearance of ornamental sunflowers.

Symptoms/signs. *Puccinia helianthi* completes its entire life cycle on *Helianthus*. Four different spore stages are macroscopically visible (Friskop et al. 2011). The first sign of rust infection are pycnia, which are yellow-orange spots (6 mm or less) on the upperside of the cotyledons or leaves that may be surrounded by a chlorotic halo. Aecia develop directly opposite the pycnia on the lower leaf surface, and they resemble a grouping of small orange cups. A few weeks later, uredinial pustules form, which are the most commonly observed stage of rust. Uredinial pustules are chocolate to cinnamon brown, and these spores are easily rubbed off. Pustules similar in appearance can also form on the bracts. As the plant senesces and the temperatures cool off, the uredinial pustules change to telia, which are filled with black spores, which do not readily rub off. *Puccinia helianthi* will also cause narrow, linear uredinial pustules on stems, looking like brown to black streaks, 1–2 cm long (Fig. 11a, b).

Biology and epidemiology. *Puccinia helianthi* is an obligate, autoecious, macrocyclic rust pathogen, which therefore completes its entire life cycle on cultivated sunflower or any of the *Helianthus* species. The fungus may also infect some wild Asteraceae such as *Encelia* and *Viguiera*, but no other ornamental Asteraceae are hosts (Cummins 1978). The fungus exists as different physiological races, and these

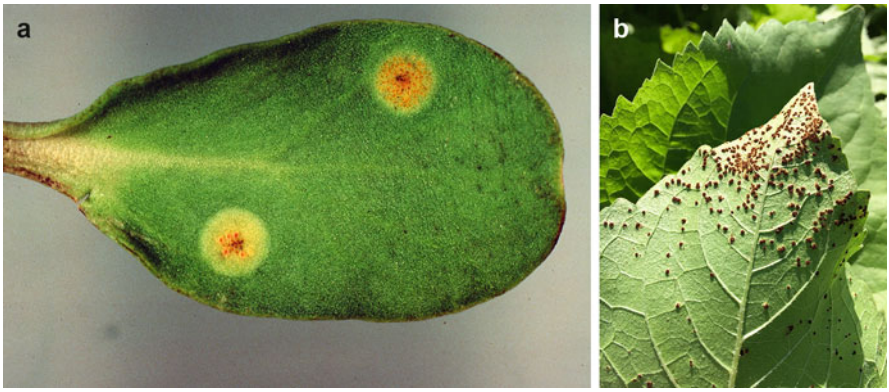


Fig. 11 (**a**) Rust aecia on sunflower cotyledon. (**b**) Rust uredinia on underside of sunflower leaf (T.J. Gulya. USDA.)

vary widely from country to country (Gulya and Markell 2009). *Puccinia helianthi* survives between growing seasons either as telia on dead plant debris or as uredinia on living plants where winter temperatures are above freezing. In areas of cold, subfreezing winters, the overwintering telia germinate in the spring to begin the entire rust cycle of spore stages. In areas with mild winters, the uredinia will continue to produce successive new generations of uredinospores, which can be spread hundreds of kilometers or more. Uredinospores germinate in the dark with a minimum of 4 h of leaf wetness and moderate temperatures (10–25 °C) (Sood and Sackston 1972). Under optimal conditions, the rust fungus can produce new crops of urediniospores every 7–10 days.

Management

- ***Cultural practices*** – Since wild sunflower is also a host for rust, removal of any wild sunflower in the vicinity will minimize the production of inoculum. Cultural practices to minimize the duration of leaf wetness (i.e., minimizing the canopy) such as lower plant densities, avoiding excessive nitrogen fertilization, and using drip or furrow rather than overhead irrigation will all help to manage sunflower rust.
- ***Resistance*** – As with many other diseases, genetic resistance is available within oilseed germplasm, and this has been obtained from many different annual and perennial *Helianthus* species (Gulya and Brothers 2000). This resistance, however, has not yet been incorporated into ornamental sunflower cultivars. Resistance is race specific; in order that resistance be effective, breeders must know which races are present in the area of interest and use the appropriate single, dominant genes (Gulya and Markell 2009).
- ***Fungicides*** – Many newer systemic fungicides are effective as both protectants and eradicants against rust (Friskop et al. 2015b). Timing of fungicide applications is of more impact than the type of fungicide used (Mueller et al. 2004).

2.13 Rusts – Other Minor Ones (*Puccinia* spp., *Coleosporium helianthi* Arthur)

Geographic occurrence and impact. While *Puccinia helianthi* is the main causal agent of sunflower rust, and is distributed globally, there are four other *Puccinia* species, primarily found in North America, which can cause rust on cultivated sunflower and wild *Helianthus* species. All four of these *Puccinia* spp. are very rarely seen and at present are of little economic threat to either oilseed or ornamental sunflowers. *Coleosporium helianthi* is another rust infrequently seen on *Helianthus* and, to date, is only recorded east of the Mississippi river in the United States. A related species, *C. asterum*, is commonly found on *Solidago* species but does not infect sunflower or any *Helianthus* species (Hedgecock 1928).

Symptoms/signs. Both *Puccina enceliae* and *Puccinia massalis* produce uredia and telia that are indistinguishable from those of *P. helianthi*. The only way to differentiate these three species is by teliospore morphology. All three species

produce small, cinnamon-brown uredia and blackish-brown telia. *Puccinia caniculata* produces small, raised pycnia on sunflower, followed by large (2–10 mm) clusters of typical orange aecial cups. Uredia and telia are found only on the alternate hosts, nutsedges. *Puccinia xanthii* is microcyclic and forms only telia, which are large (2–10 mm), raised, and chocolate brown, turning gray as the spores germinate in place. None of the species have been observed to cause pustules on sunflower stems or bracts, but this may be due to their scarce sightings. *Coleosporium helianthi* is considered a pine rust, with the uredial/telial stages on *Helianthus* and other hosts in the *Asteraceae*. Uredinia are bright orange and occur on the under surface of leaves in contrast to *Puccinia* where uredinia occur on both leaf surfaces (Fig. 12).

Biology and epidemiology. The "minor rusts" found on *Helianthus* include *Puccinia canaliculata* (Schweinitz) Lagerheim, *P. enceliae* Dietel and Holway, *P. massalis* Arthur, and *P. xanthii* Schweinitz (Cummins 1978). *P. enceliae* and *P. massalis* are full cycle, autoecious rusts like *P. helianthi. Puccinia canaliculata* is usually referred to as nutsedge rust, and *P. xanthii* is commonly termed cocklebur rust. *P. canaliculata* has been recorded on *Asteraceae* hosts in the Midwest United States (Gulya and Stegmeier 2002) and on *Cyperus* (sedges) (Callaway et al. 1985) through the United States and South America, Africa, and Asia. *Puccinia enceliae* is found in arid areas of the southwestern United States, Mexico, and Central America on *Helianthus* and some desert shrubs such as *Encelia*, *Enceliopsis*, *Simsia*, *Tithonia*, and *Viguiera. Puccinia massalis* has only been found in the Rio Grande river valley from New Mexico to the Big Bend in Texas. *Puccinia xanthii* is a cosmopolitan rust, found wherever *Xanthium* is present, and has rarely been recorded across the United States (Gulya and Charlet 2002), and in Asia, Africa, Europe, and Australia. On sunflower, however, it is recorded only from Australia, South Africa, and once in the United States *Coleosporium helianthi* Arthur, referred to as pine needle rust, is the only other rust likely to be seen on sunflower, and is only recorded in the eastern United States (Hedgecock 1928).



Fig. 12 Uredinia of *Coleosporium helianthi* (*orange*) and telia of *Puccina helianthi* (*black*) (T.J. Gulya. USDA.)

As the four *Puccinia* spp. are rarely seen, there is relatively little literature on them, with the exception of *P. caniculata* (of possible use as a bioherbicide to control sedges) and *P. xanthii* (since it occurs worldwide on weedy *Xanthium* spp.). Thus, we assume that epidemiological characters of these *Puccinia* species are similar to that of *P. helianthi. Puccinia canaliculata* is a full cycle, heteroecious rust whose uredial hosts are *Cyperus* (sedges). Aecial hosts of *P. caniculata* include sunflower, *Ambrosia*, *Cosmos*, *Senecio*, and *Xanthium*. Being heteroecious, the two hosts need to be in close proximity. *Puccinia xanthii* is an autoecious rust occurring only on *Xanthium*, *Ambrosia*, and sunflower. Recent research suggests that several *formae speciales* exist, and thus *P. xanthii* on other hosts may not readily infect sunflower. *Coleosporium helianthi* occurs on two- and three-needle pines (*Pinus banksiana*, *P. echinata*, and *P. virginiana*) indigenous to eastern North America (Nichols et al. 1968). In greenhouse inoculations, perennial *Helianthus* spp. were much more susceptible to *C. helianthi* than annual species or cultivated sunflower. Other *C. helianthi* hosts include *Iva* and *Silphium* spp.

Management. None of the four *Puccinia* species nor *Coleosporium* spp. have been observed to cause economic problems on cultivated sunflower. Cultural practices advised for *P. helianthi* would apply, and fungicides with efficacy versus *P. helianthi* should presumably be effective against the other *Puccinia* species. With the heteroecious rusts, ensuring that the alternate hosts were absent in the vicinity would minimize rust occurrence on sunflower.

2.14 Sclerotinia Stalk Rot and Head Rot (*Sclerotinia sclerotiorum* (Libert) de Bary and *Sclerotinia minor* Jagger)

Geographic occurrence and impact. Sclerotinia-incited stem and head rots are the leading cause of yield losses to oilseed sunflower worldwide, and they pose an equal threat to ornamental sunflowers. Their impact is not only upon the existing crop but the contamination of soil with long-lived sclerotia and their wide host range; an infestation of *Sclerotinia* can make it unprofitable or impractical to raise many other floral crops. *Sclerotinia sclerotiorum* is especially damaging to sunflower, as it causes both a wilt/stalk rot via root infection and a head infection, in contrast to other crops where the fungus only causes floral infections.

Symptoms/signs. Basal stem rot/wilt caused by *S. sclerotiorum* can occur any time after emergence but generally manifests itself just prior to flowering. Initial symptoms consist of a sudden wilting of the entire plant accompanied by a tan lesion girdling the stem at the soil line. The epidermis is easily sloughed off the stem, and under humid conditions the lesion may be covered with a white mycelium (Fig. 13b), and, eventually small irregularly shaped black sclerotia will form. If the lower stalk is split longitudinally, the pith degradation will be apparent, with masses of irregularly shaped black sclerotia, often larger than those found externally. Airborne spores may land on leaves, from which the fungus progresses to the stem, causing a tan

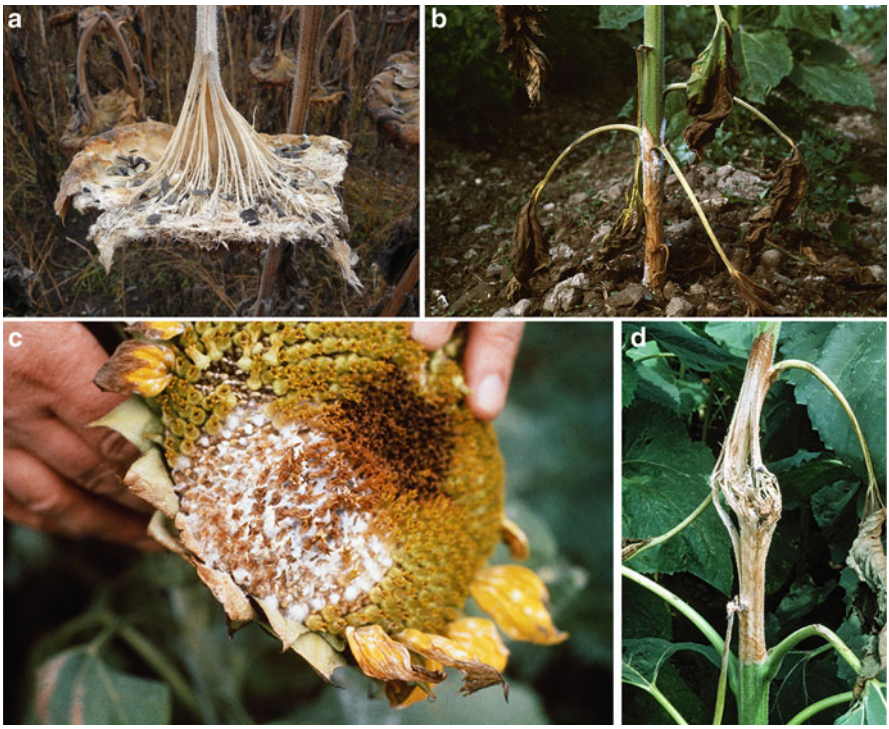


Fig. 13 (**a**) Sclerotinia head rot. (**b**) Sclerotinia stalk rot showing lesion and white mycelium. (**c**) Sclerotinia head rot showing white mycelium on face of head. (**d**) Sclerotinia mid-stem rot, often resulting in lodged stems (T.J. Gulya. USDA.)

lesion and rotted pith in the middle of the plant, known as mid-stem rot (Fig. 13d). With *S. minor* the symptoms are the same, and the sclerotia are also black but round and small (1–2 mm).

Sclerotinia head rot is only caused by *S. sclerotiorum.* Initial symptoms are tan, water-soaked small spots or areas of the back of the head, which eventually spread to encompass the entire head. With frequent rain or dews, the front of the head may be covered with a white mycelium. As the head rot progresses, the entire head becomes rotted, and large numbers of irregularly shaped, black sclerotia will form inside the head. On the surface of the head, the mycelium will convert to form a large netlike sclerotium which resembles a shower head as the seeds pierce causing openings in the massive sclerotium (often as large as the face of the head). In time, the entire head will rot, often dropping to the ground, leaving a bundle of vascular elements which resembles a straw broom (Fig. 13a–d).

Biology and epidemiology. *Sclerotinia sclerotiorum* (Libert) de Bary and *Sclerotinia minor* Jagger are both Ascomycete fungi. Neither produces any conidia and both form overwintering structures called sclerotia. The main feature

distinguishing the two species is the size of the sclerotia. With *S. minor*, the sclerotia are nearly round, black, and measure 0.5–2 mm. With *S. sclerotiorum*, sclerotia formed within the stem are black, irregularly shaped but generally long and thin, and can reach up to 5 cm long. When *S. sclerotiorum* infects sunflower heads, the sclerotia may form within the seeds and mimic the seed in size and shape, and when mycelium covers the face of the head, the fungus will form a huge, black, netlike sclerotium that, if intact, will reach 15–25 cm across. The two species also differ in that *S. sclerotiorum* produces apothecia which produce ascospores, while *S. minor* does not. Apothecia are small cup-shaped mushroomlike structures which are dark gray to brown in color, borne on short stalks and from 2 to 10 mm in diameter. There are other sclerotial forming pathogens which attack sunflowers, and these can generally be differentiated on the basis of sclerotial size, shape, and color (Schoen 1983). Both species quickly produce abundant white mycelium both in culture and on infected tissue, which gives them the common name of "white mold" or "cottony mold." *Sclerotinia sclerotiorum* is common in cooler, temperate climates, while *S. minor* is generally found in areas with hot, dry summers. The two species are not frequently seen together (Heffer Link and Johnson 2012).

Both *Sclerotinia* spp. overwinter as sclerotia, and these can remain viable for several years (Abawi and Grogan 1979; Purdy 1979). Deep burial of sclerotia decreases their survival. With *S. sclerotiorum*, the sclerotia may germinate in two ways, termed myceliogenic and carpogenic. Myceliogenic germination produces mycelium which grows through the soil, contacts sunflower roots, and initiates the root rot, stalk rot, and wilt phases of *Sclerotinia*. Soil moisture and soil temperature have less effect on myceliogenic germination than with carpogenic germination. With carpogenic germination, shallowly buried sclerotia produce tiny apothecia on the soil surface, and these in turn release airborne ascospores which, upon landing on senescent floral tissue, initiate the head rot phase. If dead pollen or wounds are present on leaves, this will provide the avenue for ascospores to infect leaves which will lead to a mid-stalk infection. Apothecial production is fostered by high soil moisture and ascospores can be produced at temperatures of 4–32 °C. However, optimal ascospore germination and penetration are at temperatures between 20 °C and 25 °C and long periods of wetness (>4 h). Thus, long periods of dew or fog producing noninterrupted periods of free water on the plant is more conducive to infection than short periods of rain, regardless of the amount. Temperatures > 25 °C and lack of free water are detrimental to ascospore germination/infection (Masirevic and Gulya 1992).

The ascospores can be dispersed short distances (100 m) by wind currents, as well as by splashing water. The fungus quickly produces abundant white mycelia in all infected plant parts, and then sclerotia, which can be found externally on plant roots and the lower stem, inside the lower stem, and on the head. *S. minor* sclerotia, in contrast, germinate only to form mycelium, which initiates root infection. Thus, *S. minor* does not produce head rot since it produces no airborne ascospores.

Sclerotinia sclerotiorum has an extremely broad host range, encompassing almost 400 plants in 278 genera of herbaceous plants with the Asteraceae, Cruciferae, Leguminosae, and Solanceaceae among the most important families. Important

crop hosts include lettuce, beans, soybeans, canola, and sunflowers, plus many Asteraceae ornamentals. *Sclerotinia minor* has a slightly less broad host range of 94 species in 66 genera. Important hosts include lettuce, *Brassica* crops, peanuts, and sunflower.

Management. Diseases caused by either of the *Sclerotinia* species are very difficult to control, and the best management is aimed at prevention.

- ***Cultural practices*** – Since infected plants can spread *Sclerotina* via root-to-root contact, lower plant densities will minimize wilt incidence (Nelson et al. 1989). To minimize head infection, anything which decreases foliage, increases air movement, or decreases head/leaf wetness will help, including lower nitrogen fertilization, lower plant densities, and furrow/drip versus overhead irrigation. Many different biocontrol/cultural practices have been shown to be partially effective at decreasing soil populations of sclerotia, and thus minimizing both root and head infections.

  Deep plowing, with a moldboard plow to completely invert the soil profile to >15 cm, will bury sclerotia into an aerobic environment where microbes should degrade the sclerotia (Mueller et al. 2002). However, there is conflicting data on this practice, and most experimental trials suggest that shallow burial leads to faster degradation of sclerotia (Ćosić et al. 2012; Subbarao et al. 1996, P. 48). Planting dense cover crops of cereal grains will produce the microenvironment conducive to apothecial formation, and if this crop is tilled under just prior to a susceptible crop, the net effect is to sap the sclerotia of energy/biomass sufficient to prevent production of more apothecia (Mueller et al. 2002).

  Rotation is of minimal use, since there are so many susceptible crops, but if several years of a nonhost monocot (for example, grasses or cereals) are planted, this also would hasten lowering the sclerotial soil population (Mueller et al. 2002). Once either stem rot or head rot are observed, there are no curative measures to save those plants. The best option at that point is to physically remove affected plants and dispose of them away from the field, thus minimizing soil contamination with more sclerotia.
- ***Biological control*** – Many commercial biocontrol products are available, based on fungi such as *Coniothyrium*, *Gliocladium*, and *Trichoderma*, and bacteria such as *Bacillus*, and these applied as soil drenches immediately following a Sclerotinia infestation will hasten sclerotial degradation and shorten the interval between planting another susceptible crop.
- ***Soil disinfestation*** – Preventative measures include using pasteurized soil, solarization, soil fumigation, or, where practical, flooding of fields for several weeks, all of which are aimed at decreasing the soil population of sclerotia. Biofumigation by planting *Brassica* cover crops and tilling them under will release isothiocyanates, which are toxic to a range of fungi (Griffiths et al. 2011).
- ***Resistance*** – In certain sunflower hybrids, some progress has been made in developing partial (incomplete) resistance or tolerance to *S. sclerotiorum*. There are no hybrids available with complete resistance to either of the two *Sclerotinia* spp.

2.15 Septoria Leaf Spot and Blight (*Septoria* spp.)

Geographic occurrence and impact. Septoria leaf spot is noted on sunflower throughout the world on every continent. The disease is most destructive in areas with heavy rainfall but is seldom observed in areas with drier climates. Under optimal disease conditions, premature leaf senesce and defoliation will result.

Symptoms/signs. Septoria blight is characterized by angular to circular necrotic leaf spots reaching 10–15 mm in diameter. Lesions are tan to gray with dark brown margins, and may be surrounded by a yellow halo. Symptoms may be observed on cotyledons and leaves of young seedlings, but it is more common to see severe symptoms on older plants. One characteristic feature which distinguishes *Alternaria* and *Septoria* leaf spots is the presence of pycnidia in the lesions produced by *Septoria* spp., which appear as small, black dots, barely visible with the naked eye. In addition, *Septoria* lesions are generally much larger than *Alternaria* lesions. Lesions coalesce in time, turning the leaf necrotic and defoliating the plant from the bottom leaves up (Fig. 14a, b).

Biology and epidemiology. Two species of *Septoria* are known to infect sunflower, *Septoria helianthi* Ellis and Kellerman found worldwide (Holliday and Punithalingam 1970) and *Septoria helianthina* Petrov and Arsenijevic, initially described in Yugoslavia (Petrov and Arsenijevic 1996). Both *Septoria* spp. are thought to be restricted to sunflower and wild *Helianthus* spp.

Septoria spp. overwinter as pycnidia on infected plant residue. Spores ooze from the pycnidia when wet and are spread by splashing rain and wind. Initial infections occur on lower leaves, where the microclimate is more conducive, and the upper leaves become affected later. Rapid disease development is fostered by abundant rain and moderately high temperatures. The development of disease is arrested with hot, dry weather but can resume with the return of favorable conditions.

Management

- ***Cultural practices*** – Any cultural practice such as crop rotation and tillage of crop residue will lessen the carryover of inocula.

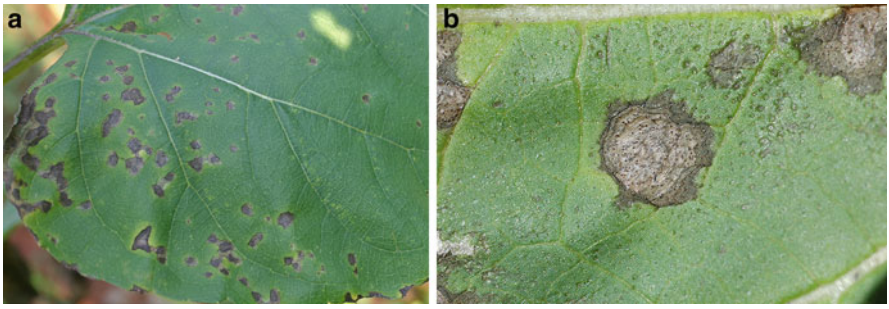


Fig. 14 (**a**) Septoria leaf blight (T.J. Gulya. USDA.). (**b**) Closeup (~10X) of lesion showing the fungal pycnidia, visible as tiny black dots (S. Markell © 2017. All Rights Reserved.)

- ***Fungicides*** – Broad spectrum fungicides commonly used to control foliar diseases on other ornamental crops should be effective against these *Septoria* spp.
- ***Resistance*** – Genetic resistance has been noted in oilseed germplasm (Block 2005; Carson 1985), but this has not been incorporated into commercial hybrids nor into ornamental varieties.

2.16 Sclerotium Blight (*Sclerotium rolfsii* Saccardo)

Geographic occurrence and impact. Sclerotium blight or wilt is found in Africa, Asia, Australia, Europe, and both Americas in areas that have tropical or semitropical climates (Punja 1985). In the United States, it is most frequently seen in Florida and the Gulf coast. Thus, ornamental sunflowers grown indoors in hot, humid conditions have the potential for significant losses due to this disease.

Symptoms/signs. The main symptom of Sclerotium blight or "Southern blight" is a girdling lesion at the soil surface, generally appearing on plants as they reach the bud stage. This tan to dark brown lesion may have alternating light and dark bands, corresponding to the diurnal growth of the fungus. Under moist conditions, a white mycelial mat may form over the lesion and around the base of the plant. Sclerotia will form on the exterior of the lesion. The sclerotia are uniformly round, 0.5–2 mm, and brown, in contrast to the black sclerotia formed by *Sclerotinia* spp. The fungus will rot the pith but the rind will be intact, and thus, there seldom is lodging (Fig. 15).



Fig. 15 Sclerotium blight (T.J. Gulya. USDA.)

Biology and epidemiology. *Sclerotium rolfsii* is the asexual stage of this pathogen; the sexual stage *Athelia rolfsii* (Curzi) Tu and Kimbrough is seldom observed in nature but can be produced in culture. The mycelium produced by this fungus is abundant, white, and cottony, and very similar to that produced by *Sclerotinia* spp. The sclerotia are the best means to distinguish *S. rolfsii* from the two *Sclerotinia* spp. *Sclerotinia sclerotiorum* produces sclerotia which are black, irregularly shaped but generally long and thin, and can reach up to 5 cm long. *S. minor* produces small round sclerotia (0.5–2 mm) black in color, while *S. rolfsii* sclerotia are similarly small (0.5-2 mm) but tan to dark brown.

Soilborne sclerotia germinate to form a mycelium which infects stems at or slightly below the soil line (Aycock 1966). Sclerotial germination is favored by moist, but not saturated soil at temperatures from 25 °C to 35 °C. From initially infected plants, mycelia can move plant-to-plant via root contact or even grow on the soil surface under wet conditions. Sclerotia can survive for several years in soil, but do not withstand alternating freeze/thaw cycles, so this fungus is thus not seen in colder climates. Like the *Sclerotinia* spp., this pathogen has a wide host range of nearly 500 dicot plants, but it also infects some shrubs and trees.

Management

- ***Cultural practices*** – Crop rotation with nonhosts (for example, cereals and grasses) and deep burial of sclerotia (>15 cm) will aid disease control (Hagan 2004). Early season planting may have less disease due to cooler soil temperatures.
- ***Soil disinfestation*** – Solarization or fumigation is more effective against sclerotia than soil-applied fungicides.
- ***Fungicides*** – Seed or soil-applied fungicides for control of the seedling phase of the disease can be effective.

2.17 Verticillium Leaf Mottle/Wilt (*Verticillium dahliae* Klebahn)

Geographic occurrence and impact. The fungus causing Verticillium wilt is found globally, and with its wide host range and longevity in the soil, it can be a serious problem not just for sunflower but for any other floral crops grown in the same field/soil.

Symptoms/signs. External symptoms of *Verticillium* infection are more accurately described as a leaf mottle (Sackston et al. 1957). Leaf symptoms generally appear on plants as they reach bud stage, with symptoms appearing on lower leaves and progressing up the plant. Interveinal areas become chlorotic and then quickly die to produce dark brown-black areas surrounded by a yellow margin. These symptoms are often mistaken for the initial leaf symptoms of Phomopsis stem canker. However, with Phomopsis stem canker, the necrotic areas are centered on the veins, while with Verticillium wilt, they are always between the veins. The entire leaf surface will be affected with this mottle pattern, and eventually the first affected leaves will senesce

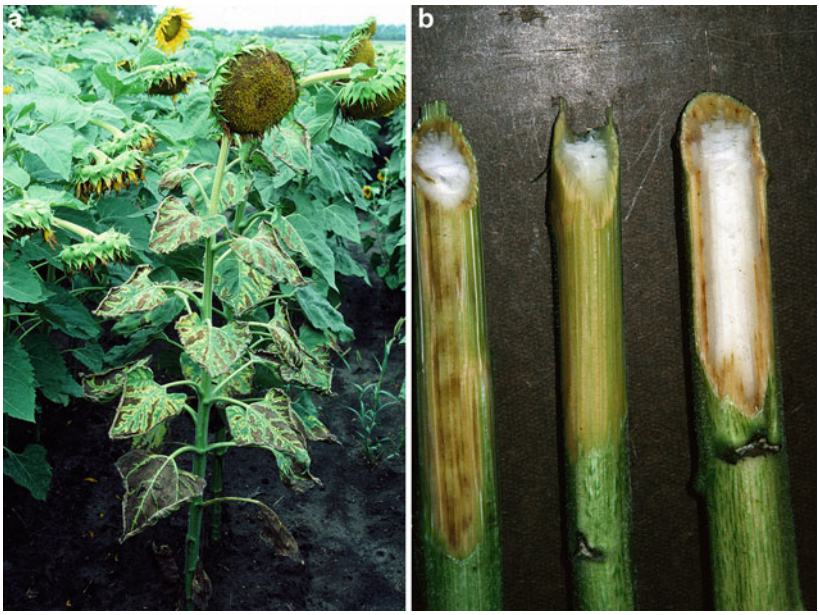


Fig. 16 (**a**) Verticillium leaf mottle/wilt. (**b**) Stem sections showing vascular browning caused by Verticillium (T.J. Gulya. USDA.)

and die. Wilting is not always observed, despite the inaccurate common name. Severely affected plants may die prior to maturity, producing small heads. Another way to confirm *Verticillium* infection is to cut the lower stem, either longitudinally or in cross section. The fungus will cause the vascular elements to be discolored brown. In severely affected plants, when the stem is cut open, the surface of the pith will be uniformly black, due to the presence of tiny microsclerotia (Fig. 16).

Biology and epidemiology. Older literature incorrectly identified the causal fungus as *V. albo-atrum*, "microsclerotial form" but *Verticillium dahliae* is the correct causal agent (Sackston et al. 1957). Another fungus, *Phialophora asteris* (Dowson) Burge and Isaac (syn. *Cephalosporium asteris* Dowson), produces symptoms in sunflower very similar to *Verticillium*, and its morphology in culture is also similar, making identification difficult (Hawksworth and Gibson 1976). *Verticillium dahliae* produces conidia, but the microsclerotia are the propagules that overwinter and are responsible for infection. *Verticillium dahliae* is very slow growing in culture, and isolations made from diseased plant tissue are often mixed with faster growing contaminating fungi, further complicating pathogen identification. *Verticillium dahliae* has a host range of over 400 species of herbaceous and woody plants, including trees, vegetables, flowers, and weedy species. Susceptible flower crops include aster, chrysanthemum, cineraria, dahlia, geranium, gerbera, marigold, rose, snapdragon, statice, and stock, among others. Among field crops, potatoes and mint are severely affected.

Verticillium dahliae overwinters in soil or plant residue as microsclerotia, and these remain viable for many years. Microsclerotia are stimulated to germinate by root exudates to form a mycelium which penetrates the roots, enters the vascular system, and eventually becomes systemic throughout the plant. Moist, but not waterlogged soil, and temperatures between 21 °C and 27 °C are optimal for sclerotial germination and root infection. The fungus produces a toxin that is translocated to the leaves, which induces the foliar symptoms. Thus, isolation from symptomatic leaves may yield no *Verticillium* as the symptoms are the result of the toxin produced some distance away. Wilting, when it occurs, is the result of fungal mycelia and polysaccharides plugging the vascular system. If the mycelium reaches the head, the seeds will become infected. The fungus produces copious amounts of black microsclerotia internally, which gives the pith a charred, blackened appearance. The microsclerotia are smaller than those produced by *Macrophomina phaseolina* (charcoal rot) and cannot be seen with the naked eye or even a 10X lens.

Management. As with other soilborne fungal pathogens forming sclerotia, management of *Verticillium* is challenging (Berlander and Powelson 2000). The sclerotia, despite their small size, are difficult to kill with fungicides; they exist in large numbers in the soil, and the fungus has a large host range. Since *Verticillium* is a serious problem on some high cash value crops like potatoes, there are commercial laboratories that assay soil samples to determine the presence and quantity of *Verticillium* microsclerotia.

- ***Soil disinfestation*** – Thus, prevention is the first aim of a management program, and this starts with clean seed and pathogen-free soil, via pasteurization, solarization, or fumigation. Flooding the soil for 20 days significantly reduces the number of microsclerotia, as does soil acidification with aluminum sulfate. As mentioned for Sclerotinia wilt, the practice of biofumigation with *Brassica* cover crops has been shown to decrease the number of microsclerotia and the severity of the disease.
- ***Cultural practices*** – While extra irrigation might be considered to help plants infected with a wilt pathogen, research has shown that frequent or excessive irrigation actually increases the severity of *Verticillium.*
- ***Resistance*** – Genetic resistance is available (Hoes et al. 1973) and widely deployed in oilseed sunflower germplasm. The fungus exists as several races (Gulya 2007) and vegetative compatibility groups (VCGs) (García-Carnero et al. 2014), and this needs to be known so that breeders can select the appropriate single, dominant resistance gene.

2.18 White Rust (*Albugo tragopogonis* (DeCandolle) Gray = *Pustula helianthicola* Rost and Thines)

Geographic occurrence and impact. Albugo or white rust is a disease historically found primarily in the Southern Hemisphere (Argentina, Australia, South Africa)

(Kajornchaiyakul and Brown 1976; Delhey and Kiehr-Delhey 1985) but more recently has been noted on both ornamental and oilseed sunflowers in Europe (Crepel et al. 2006; Thines et al. 2006), China (Chen et al. 2006), and, very infrequently, in the United States (Gulya et al. 2002b). The disease requires very specific environmental conditions, and the pathogen appears to be very host specific, and thus the disease is unlikely to appear year-in and year-out. While the disease is usually not devastating to the oilseed crop, the white blisters on the foliage detract from the value of ornamental sunflower.

Symptoms/signs. The disease is usually manifested by raised, small to fairly large (1–2 mm or 5–10 mm) pustules or blisters on the upper leaf surface, with white sporulation on the underside of the leaf (Kajornchaiyakul and Brown 1976). With age, the *Albugo* pustules become necrotic and the infected tissue may fall out, leaving a "shot hole" appearance. The causal fungus can also cause similar white blisters on floral bracts, and may also initiate faintly black, water-soaked lesions on petioles and the stem. Stem lesions are usually internodal and may be colonized by secondary fungi, leading to lodging in severe cases (Krüger et al. 1999; Van Wyk et al. 1995) (Fig. 17).

Biology and epidemiology. The fungus causing this disease is not a true rust and is actually more closely related to the downy mildews. The causal agent of white rust was classified as *Albugo tragopogonis* for over a century but was recently renamed *Pustula helianthicola* (Rost and Thines 2012). The fungus overwinters as oospores on infested plant debris and may also be seedborne (Viljoen et al. 1999). Oospores, the primary inoculum, are spread by rain and splashing water. They germinate to form motile zoospores that enter through leaf stomata. The white asexual sporangia produced on the underside of leaf pustules can be windblown to cause secondary infections on leaves, stems, petioles, and bracts. Oospores typically develop only in stem and petiole lesions, which cause the subtle, dark lesions. The disease is dependent on free water, from rain, dew, or irrigation. Optimum infection takes



Fig. 17 White rust on sunflower – leaves (T.J. Gulya. USDA.)

place at night between 10 °C and 15 °C, while optimal disease development is at temperatures between 20 °C and 25 °C. Thus, the disease is favored in environments with cool nights and warm days (Kajornchaiyakul and Brown 1976).

Management

- ***Cultural practices*** – Overhead irrigation at night will foster conditions conducive to foliar infection, and should be avoided.
- ***Sanitation*** – Removal of plant debris from previous sunflower crops, either in the same or nearby fields, will diminish the possibility of infection. Leaves stripped prior to shipping cut sunflowers, if infected, should be disposed of outside the field, and preferably composted or removed entirely.
- ***Fungicides*** – Seed-applied fungicides which control downy mildews (metalaxyl, mefenoxam, and strobilurins such as azoxystrobin) will provide early season control. Thereafter, foliar applications of these fungicides should provide protection against leaf infection (Lava et al. 2015).
- ***Resistance*** – In oilseed sunflower germplasm, resistance to white rust has been noted and is controlled by multiple genes, each governing resistance in different plant parts. No effort has been made to incorporate any type of white rust resistance into ornamental sunflowers, although it has been noted to occur in one older commercial cultivar ("Abendsonne"). Many annual and most perennial *Helianthus* species appear to be resistant (Lava et al. 2015).

3 Bacterial and Phytoplasma Diseases

3.1 Apical Chlorosis and Bacterial Leaf Spot (*Pseudomonas syringae* pv. *tagetis* and *Pseudomonas syringae* pv. *helianthi*)

Geographic occurrence and impact. Apical chlorosis was first identified on marigold in Australia (Trimboli et al. 1978) and later on sunflowers in the United States in the early 1980s (Gulya et al. 1982). The causal bacterium is found worldwide on many Asteraceae flowers and weedy species. Bacterial leaf spot is caused by a very closely related bacterium, and it also is found worldwide (Piening 1976). Less is known about its host range, and it may be restricted solely to *Helianthus*. Apical chlorosis is more likely to be observed and cause alarm to growers due to its spectacular appearance, but since its symptoms are confined to the foliage, which is stripped off for cut flower production, its impact is lessened.

Symptoms/signs. Apical chlorosis first manifests itself as a general chlorosis of the youngest leaf, either a portion or the entire leaf (Gulya et al. 1982). The affected area may be a pale yellow, or in extreme cases the leaf will be almost white. This symptom was initially confused with nitrogen deficiency, but the chlorosis is much more pronounced. The chlorosis will affect subsequent leaves as they develop, but the chlorotic leaves will not turn necrotic. When temperatures warm up, the newly emerging leaves will be normal green in color, but lower, chlorotic leaves will



Fig. 18 Apical chlorosis (T.J. Gulya. USDA.)

remain so (Fig. 18). Symptoms of bacterial leaf spot vary, depending upon the environment and sunflower cultivar. Lesions begin as small necrotic spots of varying size and shape and may be surrounded by chlorotic haloes. Under severe conditions the individual lesions coalesce to form large necrotic areas, giving the leaf a shot-hole appearance. Lesions may also form on petioles and stems, often leading to defoliation.

Biology and epidemiology. *Pseudomonas syringae* pv. *tagetis* (Hellmers) Young, Dye, and Wilkie is the causal bacterium for apical chlorosis, while *Pseudomonas syringae* pv. *helianthi* (Kawamura) Young, Dye, and Wilkie causes bacterial leaf spot (Piening 1976). Recent molecular studies suggest that both bacteria may be identical, differing only in a toxin production gene present in *P. syringae* pv. *tagetis*. *Pseudomonas syringae* pv. *tagetis* has a wide host range within the Asteraceae, affecting ornamental flowers such as *Tagetes* and *Zinnia*, as well as many weedy genera such as *Ambrosia*, *Cirsium*, and *Taraxacum* (Rhodehamel 1985). Less research has been done with *P. syringae* pv. *helianthi*, but its host range appears to

be restricted to *Helianthus* spp. Both bacteria can easily be identified as *P. syringae* pathovars based on the fluorescence under UV light of colonies grown on King's B medium.

Both bacteria survive in the soil and are seedborne. Apical chlorosis also persists on infected weedy hosts. Both are spread by splashing water and are typically seen early in the growing season during cool weather with wet soils. Apical chlorosis is typically seen in low areas of a field. Individual plants may be affected or there may be several affected plants in a row. *Pseudomonas syringae* pv. *tagetis* produces a toxin that interferes with chloroplast formation in developing leaves. In severe cases, the leaf may completely lack chlorophyll, but other pigments are not affected. Chlorotic leaves remain so throughout the life of the plant, but the appearance of new chlorotic leaves will cease as soils dry out and air temperatures warm up.

Bacterial leaf spot is quite common, and its appearance is not restricted to cool early season and wet soil (Arsenijevic et al. 1994). Infection generally starts on lower leaves where the microclimate is more favorable for both fungal and bacterial infection.

Management. Since there are no curative bactericides, there is no management option once symptoms are observed.

- ***Cultural practices*** – In field situations, limiting irrigation to prevent water-logged soils and water splashing, especially during cool weather, will likely preclude the appearance of apical chlorosis.
- ***Resistance*** – Differences in susceptibility to both bacteria have been noted in oilseed germplasm, but neither disease has been severe enough to warrant transferring that resistance into either oilseed hybrids or ornamental cultivars.
- ***Sanitation*** – As the symptoms of apical chlorosis will cease when temperatures warm up, the damaged leaves can be stripped off ornamental sunflowers, and similarly with bacterial leaf spot.

3.2 Bacterial Stalk Rot/Head Rot (*Erwinia* = *Pectobacterium* spp.)

Geographic occurrence and impact. The bacteria causing stem and head rot are ubiquitous in soils and are present in every continent. Stem infections, though uncommon, would render ornamental sunflowers unsaleable. Head rot, although potentially serious on oilseed sunflower, does not occur in the bud stage, so it is unlikely to be observed on cut sunflowers but could be present in potted plants or homegrown sunflowers.

Symptoms/signs. Bacterial stem rot and head rot can usually be recognized by smell as well as visual symptoms. The exterior of affected stems may have no discernible lesions or may have a blackened area, often centered around a petiole (Gudmestad et al. 1984). Upon splitting the stem open longitudinally, the pith will



Fig. 19 Bacterial stalk rot (T.J. Gulya. USDA.)

appear very water soaked, and if the infection has progressed long enough, there will be a strong odor of rotting potatoes. In time, due to gas produced by the bacteria, the stem will split open longitudinally and bacterial slime may ooze out, which often attracts flies and other insects. After this phase the stalks will either lodge or then may dry up and turn completely black, and the odor will be gone (Fig. 19).

The bacterial head rot phase is characterized by symptoms that initially are identical with those caused by many fungal incited head rots. Small, depressed, watery, soft rotted areas develop on the back of the heads that enlarge and turn from tan to brown. Again there is a distinct odor of rotting potatoes, and if one grasps such a head, the soft mushy condition will be evident. Head infections may also produce copious amounts of slime and exudate, which may smell either of rotting potatoes, or in some cases, of alcohol, due to the presence of other secondary organisms such as

yeasts. These exudates, combined with the bacterially produced gas, often look like foam on the head and stem. The smell and sugars in the exudates attract many insects, including flies, bees, and various beetles.

Biology and epidemiology. *Pectobacterium carotovorum* (Jones) Waldee and *Pectobaterium atrosepticum* (van Hall) Lehmann and Neumann are both responsible, alone or in combination, for soft rots of sunflower stems and heads. These "soft-rotting bacteria" can affect many plants, causing rots of flowers, soft fruit, and vegetables. A species from Mexico, *Pectobacterium cacticidum*, whose primary hosts are cacti, was recently isolated from sunflower (Valenzuela-Soto et al. 2015).

Both *Pectobacterium* spp. are soft-rotting bacteria that are found in soils and decaying plant debris (Charkowski 2015). They are very weak pathogens and require wounds to infect healthy tissue. These wounds can be mechanical damage, or caused by insects, birds, or hail. Free water and high temperatures speed the development of soft rot once the bacteria have colonized a wound. If insect larvae tunnel into the stems, they will not only vector the bacteria but will hasten its spread within a plant. Once the bacteria become established internally within the stem or head, the influence of air temperature and rainfall is negligible.

Management. Bacterial diseases, especially caused by ubiquitous organisms, are hard to control, and prevention is the best approach. Controlling insects and birds to minimize wounds through which the bacteria gain entrance is a sound first step (Chattopadhyay et al. 2015). Ground irrigation rather than overhead will minimize the free water necessary for infection. Once an infected plant is noted, it should be physically removed and disposed of outside the greenhouse or field, which will eliminate the inoculum that feeding insects might spread.

3.3 Aster Yellows (*Phytoplasma* spp.)

Geographic occurrence and impact. Aster yellows has been previously confused with virus diseases, then classified as caused by a mycoplasma (now known as phytoplasmas) (Bertacinni and Duduk 2009). The causal agent causes a "yellows" disease on over 200 crops and is the most widely seen "yellows" on nursery crops. The complex of phytoplasmas affecting sunflower has been reported from North and South America, Europe, and Northern Africa.

Symptoms/signs. Sunflower plants affected by "yellows" exhibit a range of symptoms including leaf chlorosis (of the entire leaf or in sectors), proliferation of secondary shoots, and various abnormalities of the head. There may be wedge-shaped sectors of the head showing hypertrophy, and bracts and ray petals may appear in the center of the head (Fig. 20).



Fig. 20 Aster yellows (T.J. Gulya. USDA.)

Biology and epidemiology. Aster yellows is caused by one or more closely related phytoplasmas. These organisms are smaller than bacteria, larger than viruses, and a few can, with difficulty, be grown in vitro. The phytoplasma causing aster yellows in sunflower and other crop is referred to as "*Candidatus* Phytoplasma asteris" (Harrison and Helmick 2008). Another phytoplasma causing similar symptoms has been studied in Argentina and tentatively termed "*Candidatus* Phytoplasma Sunflower Phyllody" (Guzmán et al. 2014), and a third one was characterized in Iran (Salehi et al. 2015).

Phytoplasmas are vectored by several genera of leafhoppers. The phytoplasma survives in perennial Asteraceae weed species and leafhopper vectors. Aster yellows multiplies within the body of the leafhopper and is transmitted through the insects saliva when feeding. The Aster yellows phytoplasma is believed to have a host range of more than 150 species, including many flower crops, vegetables (carrot, lettuce, potato, tomato), and even grains and grasses. Cool, wet weather seems to favor both the leafhopper vector and the disease.

Management

- ***Vector management*** – No chemicals are directly effective against phytoplasmas. Controlling the insect vectors, however, whether done by insecticides, biological control, or cultural practices (i.e., exclusion netting on greenhouse vents) will all but eliminate the disease. Removing weeds and volunteer sunflowers from the field and perimeter will reduce the inocula and vectors.
- ***Cultural practices*** – Some of the more susceptible floral crops include *Petunia*, *Tagetes*, *Viola*, *Chrysanthemum*, *Rudbeckia*, *and Salvia*, and thus it would be wise not to plant these in the vicinity of sunflowers, and to monitor all susceptible crops closely for symptoms and leafhoppers.
- ***Resistance*** – Aster yellows was once severe enough in Canada on oilseed sunflower to warrant research, and resistance was found within oilseed germplasm (Putt and Sackston 1970). Thus, it would be possible to transfer that level of resistance into ornamental sunflowers.

4 Viral Diseases

4.1 Sunflower Mosaic Virus

Geographic occurrence and impact. Sunflower mosaic virus is the only virus documented to occur naturally on sunflower in the United States, and currently is known nowhere else. Its range within the United States at the moment is restricted to southern Texas. As such it is classified as a quarantine pathogen by many importing countries, and while its impact upon ornamental sunflowers may not be great, the threat of restricted international movement of plants or seeds would be an economic hardship. Several other viruses have been confirmed on sunflowers in other countries, but their occurrence has usually been sporadic and limited to the specific countries. Thus, sunflower chlorotic mottle virus (SuCoMoV) has been found in Argentina (Giolitti et al. 2010), Pelargonium zonate spot virus (PZSV) also in Argentina (Giolitti et al. 2014), sunflower necrosis caused by tobacco streak virus (TSV) in India and Australia (Sharman et al. 2009; Prasada Rao et al. 2000), Bidens mottle virus (BiMV) in Florida (Wisler 1984) and Taiwan (Liao et al. 2009), plus several other instances of virus diseases of undetermined origin (Loebenstein and Thottappilly 2003).

Symptoms/signs. Sunflower mosaic is characterized by a mild mosaic pattern on leaves of young plants, becoming more pronounced, and then fading as the plant matures (Gulya et al. 2002a). The virus does not cause stunting, or any change in leaf or head morphology, nor does it cause ringspots (Fig. 21).

Biology and epidemiology. The disease is caused by *Sunflower mosaic virus* (SuMV), a *Potyvirus*. Virus particles are long, flexuous rods. Their long size,



Fig. 21 Sunflower mosaic virus (T.J. Gulya. USDA.)

along with characteristic pinwheel inclusion bodies, is diagnostic for Potyviruses. SuCoMoV is also a Potyvirus, while PZSV and TSV are Ilarviruses, with small, isometric virus particles.

The Rio Grande river valley of southern Texas, where the disease is endemic, is subtropical, so that both wild sunflower and insect vectors can be found year-round. The virus was vectored experimentally by aphids (*Myzus persicae* and *Capitphorus elaegni*), both of which are common greenhouse inhabitants. Sunflower plants are optimally susceptible for 1 month, after which they become progressively less receptive to the virus. The virus was found to be seed transmitted. SuMV has a very narrow host range, consisting of *Helianthus*, *Zinnia*, *and Sanvitalia*. Other sunflower viruses, such as SuCoMoV, TSV, and PZSV, have much broader host ranges.

Management. While resistance to SuMV and SuCoMoV has been noted, it has not been transferred to commercial oilseed hybrids or to ornamental cultivars (Gulya et al. 2002a; Lenardon et al. 2005). The most effective management for these sunflower viruses include the use of virus-free seed, minimizing insect vectors (aphids and thrips) through insecticide sprays or cultural methods, and removing infected plants. The use of silver reflective mulch in field plantings has been shown to repel aphids, as well as conserving soil moisture.

5 Nematodes

5.1 Root-Knot Nematodes (*Meloidogyne* Species)

Geographic occurrence and impact. Root-knot nematodes are found worldwide on many hosts but are especially serious on sandy soils and in warm climates. Countries where root-knot nematodes have had the greatest impact on sunflower are Egypt, Mozambique, South Africa, and Zambia in Africa (Bolton et al. 1989; Lawn et al. 1988); Pakistan and India in Asia (Ravindra et al. 2015); and Greece and Italy in Europe (Tzortzkzkis et al. 2014; Zazzerini et al. 1997). In the United States, root-knot nematodes have been documented on sunflower in Florida and Texas (Rich and Dunn 1982; Isakeit 2011). There are currently 30 *Meloidogyne* species in the United States, of which five are known to parasitize sunflower experimentally. *M. hapla* is the most widespread, found in every state except Alaska, while *M. incognita*, *M. arenaria*, and *M. javanica* are most common in southern and coastal states, and *M. chitwoodi* is only found in the western states of WA, OR, CA, CO, UT, and NM, and in Mexico (Walters and Barker 1994). *M. incognita* appears to be most serious on sunflower. Lack of confirmation of root-knot nematodes on sunflower in a particular state may be due to the lack of nematologists to confirm their presence.

Symptoms/signs. Symptoms of root-knot nematode infection may not be apparent aboveground, or the plant may display stunting, chlorosis, and daytime wilting. Affected plants may also have fewer and smaller leaves and flowers. Root galls are



Fig. 22 Root-knot nematode damage on sunflower hybrid "954", with smaller galls caused by *Meloidogyne hapla* (*left*) and larger galls caused by *M. arenaria* (*right*) (M.K. Beute © 2017. All Rights Reserved.)

the belowground symptom (Fig. 22). The extent and size of root galls or "knots" depends upon the nematode population density, the species of *Meloidogyne*, and the host cultivar. Among ornamentals and field crops, sunflower is rated as highly susceptible (Crow 2014; Ferris et al. 1993). In the absence of galls, only a soil analysis in a nematology lab will confirm *Meloidogyne* as the causal agent, and be able to pinpoint the exact species.

Biology and epidemiology. There are five *Meloidogyne* species documented to parasitize sunflower: *M. arenaria* (Neal) Chitwood; *M. chitwoodi* Golden, O'Bannon, Finley and Santo; *M. hapla* Chitwood; *M. incognita* (Kofoid and White) Chitwood; and *M. javanica* (Treub) Chitwood. Root-knot nematodes are sedentary endoparasitic nematodes, meaning they spend most of their life cycle within plant tissue. They hatch from eggs, stimulated by both temperature and exudates from plant roots. The juvenile nematodes migrate through the soil, penetrate the tips of fine roots, and the nematode's head becomes embedded in the vascular bundle where it begins feeding and becomes immobile. Root cells around the nematode enlarge and form galls, which inhibit root function, leading to the aboveground symptoms.

Female nematodes can produce eggs parthenogenetically, and up to several hundred eggs in a gelatinous mass can be found on the root surface. Generation time can vary from 20 to 45 days, allowing for multiple infections in a growing season.

Management

- ***Cultural*** – Depending on the root-knot species present, rotation with nonhosts (arugula, onion, sesame, sorghum-sudangrass) will decrease the nematode population but will not completely eliminate *Meloidogyne* (Dover et al. 2004; El-Ghonaimy et al. 2015; Kokalis-Burelle et al. 2013). Conversely, planting of "trap crops" of susceptible hosts, such as cowpea, and removing them completely, has also been shown to decrease damage on subsequent sunflower crops. Selecting preceeding crops in a commercial nursery which are least susceptible will minimize damage to sunflower (Creswell 2000). Solarization, in climates where feasible, will reduce nematode populations.
- ***Chemical*** – While no nematicides are registered for use by home owners, several fumigants are commonly used in high-value annual crops and the nursery trade which are effective against all soilborne nematodes, including chloropicrin, 1,3-dichloropropene, metam sodium, and the nonfumigant ethoprop (Stapleton et al. 2014).
- ***Genetic Resistance*** – Evaluations of oilseed sunflowers have demonstrated that there is considerable variation in susceptibility to *Meloidogyne* spp., but to date there have been no public releases of root-knot tolerant or resistant germplasm (Fabiyi and Atolani 2013; Rehman et al. 2006).
- ***Biological*** – Several nematode-trapping fungi and antagonistic bacteria have been identified and shown to reduce, but not eliminate, populations of *Meloidogyne* spp. (Karssen et al. 2013; Duncan & Moens 2006). At least two mycopesticides, *Myrothecium verrucaria* (DITERA), and *Paecilomyces lilacinus* (MELOCON) are registered for use on ornamentals in the United States (Stapleton et al. 2014; Crow 2014).

5.2 Root-Lesion Nematodes (*Pratylenchus* Species)

Geographic occurrence and impact. Root-lesion nematodes, of the genus *Pratylenchus*, have a very wide host range and are found throughout the world, ranging from cool temperate to hot tropical climates (Davis and MacGuidwin 2005). They rank third behind root-knot and cyst nematodes as the nematodes of greatest economic impact on all crops worldwide. Their small size (0.4–0.7 mm long) relative to other plant parasitic nematodes enables them to survive in almost any texture soil. At least eight species have been found on sunflower worldwide, with *P. penetrans* the most widely distributed and important species. It interacts with wilt-inciting fungi like *Fusarium* and *Verticillium*, thus augmenting the physical damage it causes (Castillo and Vovlas 2007).

Symptoms/signs. Aboveground symptoms due to root-lesion nematodes are nonspecific, and like root-knot damage, are manifested by stunting and chlorosis, and wilting during hot days. Nematode damage in fields often occurs in

random patches which are associated with higher populations of the nematodes. Root-lesion nematode feeding will cause brown lesions on the exterior of roots, while feeding inside roots will result in cracking and eventual rotting.

Biology and epidemiology. *Pratylenchus* species documented on sunflower include *P. alleni* Ferris, *P. brachyurus* (Godfrey) Filipjev and Schuurmans Stekhove, *P. crenatus* Loof, *P. hexincisus* Taylor and Jenkins, *P. penetrans* (deMan) Filipjev, *P. scribneri* Steiner, *P. thornei* Sher and Allen, *and P. zeae* Graham. *Pratylenchus* spp. are usually migratory endoparasites, and remain vermiform throughout their life (Castillo and Vovlas 2007). Males only occur in some species, and thus reproduction is mostly by parthenogenesis. The life cycle can be completed in 28–56 days, depending upon the species, plant host and environment, so several generations can occur during a growing season. As with all plant-feeding nematodes, there are four juvenile life stages following the egg, and culminating in the adult. All juvenile and adults feed on roots, burrowing into the cortical tissue, and they can enter and leave roots during any life stage. Reports on how far they may travel range from 2 cm up to 1–2 m in the soil to reach other plants. Movement of soil and/or infected plant material provides a much wider dispersion. *Pratylenchus* overwinter in infected plants, including weedy hosts, in any life stage. Eggs can also dehydrate and become dormant and remain viable for over 2 years.

Management. Cultural, biological, and chemical management recommendations for root-lesion nematodes are much the same as for root-knot nematodes, covered previously (Creswell 2000; Stapleton et al. 2014; Thompson et al. 2008). Differences in susceptibility to *Pratylenchus* in oilseed sunflower have been noted (Bolton and De Waele 1989), but there have been no reports of resistance in any ornamental sunflower germplasm.

5.3 Other Nematodes

Many other nematodes have been identified as causing damage on sunflower. These include dagger (*Xiphinema*), pin (*Paratylenchus*), spiral (*Helicotylenchus*), sting (*Belonolaimus*), stubby-root (*Paratrichodorus*), and stunt (*Tylenchorhynchus*) nematodes, among others (Bridge and Starr 2007). Identification of the causal nematodes always depends upon soil analysis by a nematology lab, as the aboveground symptoms are not nematode specific.

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Diseases of Zinnia

28

Dorota Szopińska

Abstract

Modern garden zinnias are widely cultivated ornamentals produced for cut flowers and flowerbeds. Diseases often reduce the quantity and quality of zinnia flowers, causing their production to be unprofitable. Three major, widespread diseases occurring on zinnias are: Alternaria blight (*Alternaria zinniae*), powdery mildew (*Golovinomyces cichoracearum,* formerly *Erysiphe cichoracearum*), and bacterial leaf spot (*Xanthomonas campestris* pv. *zinniae*). Other pathogens, such as *Botrytis cinerea*, *Cercospora zinniae*, *Fusarium* spp., *Rhizoctonia solani*, aster yellows phytoplasma, and several viruses may also be harmful to zinnias; however, their economic impact on zinnia production is less significant. Various management strategies for zinnia diseases currently studied include chemical, biological, and physical control and integration of these methods.

Keywords

Alternaria zinniae • *Golovinomyces cichoracearum* • *Xanthomonas campestris* pv. *zinniae* • Integrated management

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R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_28

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1 Introduction

The genus *Zinnia* (family Asteraceae) consists of 19 species of annual or perennial plants originating in North and South America and particularly in Mexico (Spooner et al. 1991; Stimart and Boyle 2007). Among them common zinnia (*Zinnia elegans* Jacq., syn. *Zinnia violacea* Cav.) is the most widely cultivated species. Variation in ray floret morphology and color, plant height, and habit has led to development of many cultivars useful for landscape borders or cut flowers. The other two commonly planted species, *Zinnia angustifolia* Kunth (syn. *Zinnia linearis* Benth.) and *Zinnia haageana* Regel, are less conspicuous, characterized by a large number of small inflorescences and often with single whorl of ray florets (Linderman and Ewart 1990; Spooner et al. 1991; Stevens et al. 1993).

The common zinnia is highly susceptible to three pathogens: *Alternaria zinniae* Pape (Alternaria blight), *Golovinomyces cichoracearum* (DC.) V.P. Heluta (powdery mildew), and *Xanthomonas campestris* pv. *zinniae* Hopkins and Dawson (bacterial leaf and flower spot), which may cause severe epiphytotics in zinnia, resulting in plant losses, decrease of ornamental value, or both. *Zinnia angustifolia* on the other hand is highly resistant to these pathogens and has been used as a source of

resistance genes for *Zinnia* breeding programs (Boyle and Wick 1996; Jones and Strider 1979; Spooner et al. 1991). Management of the most important zinnia diseases is presented in this chapter. General management strategies, especially referring to widespread pathogens causing diseases of numerous crops, may be found in the introductory chapters.

2 Fungal and Fungus-Like Diseases

2.1 Alternaria Blight (*Alternaria zinniae* Pape)

Geographic occurrence and impact. The pathogen is widely distributed. The disease has been reported in North and South America (Dimock and Osborn 1942; Gombert et al. 2001; Judd 1976; Palacios et al. 1991), Europe (Beaumont et al. 1958; Christova et al. 1964; Dias and Lucas 1978; Gambogi et al. 1976; Imre 1974; Łacicowa et al. 1979, 1991; Mowsiesjan 1976; Szopińska 2013, 2014a, b; Szopińska et al. 2012), Asia (Rao 1971; Wu and Yang 1992), and New Zealand (Dingley and Brien 1956).

Symptoms/signs. The most conspicuous symptom of Alternaria blight is spotting of the foliage. Characteristic reddish-brown spots, sometimes with grayish-white centers, may also appear on stems and blossoms. The spots on the leaves are first circular and vary in size from 2 to 10 mm in diameter but soon enlarge and become irregular. The affected leaves become brown and desiccated. On blossoms and stems, the spots are relatively smaller, rarely more than 2 mm across. On the ray flowers, sporulation on the spots is often profuse and secondary infection may be abundant leading to darkening and withering of petals. On stems, internodal spots frequently show elongation and are usually superficial. Nodal lesions do not remain superficial and the distal parts of the affected stems may be killed by girdling at the nodes. Dark brown to black cankers with sunken centers may also appear near the soil line. Infected roots may turn dark gray, rot, and slough off resulting in wilting and death of the plant. Seedlings may also wilt and collapse (damp-off) (Fig. 1a, b).

Biology and epidemiology. *Alternaria* spp. usually overseason as mycelium or spores in plant debris and seeds. Fungal sporulation and infection are enhanced by moderate temperatures. In the case of *A. zinniae*, the optimum temperatures for conidial germination in vitro and plant infection are 20–24 °C and 22–26 °C, respectively. Free moist is required for infection, but the pathogen survives better under dry conditions. The fungus can persist in the seeds for up to 6–7 years, although in plant debris usually not longer than 2 years (Rotem 1994). Seeds are a very important source of inoculum for Alternaria blight. Deeply seated, severe seed infections may lead to preemergence death of seedlings, while superficial seed infections usually cause diseases on plants after emergence (Gambogi et al. 1976).

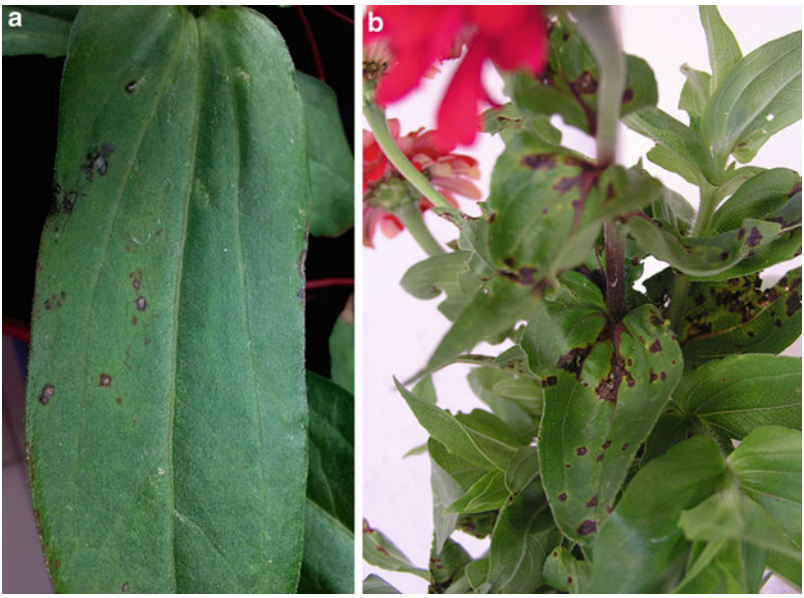


Fig. 1 Alternaria blight symptoms: first spots (*left*), irregular patches (*right*) (D. Szopińska © 2017. All Rights Reserved.)

Management

- ***Cultural practices*** – The seeds and transplants used for propagation should to be free of *A. zinniae*. Rotation to nonsusceptible crops for 2 years, control of susceptible weeds, plant spacing ensuring good air circulation, and proper time and type of irrigation are the most important cultural control measures. Overhead irrigation as well as late afternoon and evening irrigation prolong the time during which the plants remain wet, and therefore favor spread of the disease (Gombert 1998).
- ***Sanitation*** – Crop debris from the planting area should to be removed and destroyed after harvest.
- ***Physical treatment*** **–** To eradicate the pathogen, the seeds can be treated with hot water at 51.5 °C for 20 min (Beaumont et al. 1958; Lamboy and Call 2001). A high risk of germination percentage reduction, especially if older seeds are treated, is a reason why this approach is not widely use by the ornamental seed industry (Daughtrey and Benson 2005).
- ***Fungicides*** – The plants should be treated with protective fungicides at regular intervals (7–14 days), especially during warm wet weather or during warm dry weather if overhead irrigation is applied. Fungicides which are known to control Alternaria diseases include mancozeb, iprodione, chlorothalonil, copper, and triflumizole (Hagan 2009).
- ***Organic treatment*** – Szopińska (2013) observed that treating zinnia seeds with 1.0% and 2.5% lactic acid for 30 min decreased the percentage of seeds infected

with *A. zinniae* from 56.0% to 33.0% and 23.0%, respectively. Significant reduction of zinnia seed infestation with this pathogen was also observed if the seeds were treated with 6% hydrogen peroxide (H_2O_2) for 60 min and 12% H_2O_2 for 20 min. These treatments decreased seed infestation from 76% to 28.0% and 27.5%, respectively (Szopińska 2014b).

- ***Resistance*** – All *Z. elegans* selections are moderately to highly susceptible to Alternaria blight (Gombert et al. 2001), while *Z. angustifolia* is highly resistant to this disease (Hagan 2009). Therefore, colchicine-induced amphiploids have been developed in breeding zinnia plants resistant to *A. zinniae* (Terry-Lewandowski and Stimart 1983, 1985; Terry-Lewandowski et al. 1984). A new species of *Zinnia marylandica*, an artificial hybrid between *Z. angustifolia* and Z. *elegans*, was described and illustrated by Spooner et al. in 1991. Plants of this species exhibit high levels of resistance to *A. zinniae* and *G. cichoracearum* and moderate to high levels of resistance to *X. campestris* pv. *zinniae* (Terry-Lewandowski and Stimart 1983).

2.2 Botrytis Blight, Gray Mold, Stem Canker (*Botrytis cinerea* Pers ex Pers)

Geographic occurrence and impact. The pathogen is distributed worldwide and affects over 200 plant species in temperate and subtropical regions (Williamson et al. 2007). *Botrytis cinerea* has been reported on zinnias in Canada, Great Britain, Poland, the USA, and Venezuela (Bolton 1976; Kiecana and Mielniczuk 2010; Moore 1959; Palacios et al. 1991; Ruhl et al. 1987).

Symptoms/signs. Large areas of petals, leaves, and stems turn brown. The infected plant parts develop a dusty gray mycelium during humid conditions (Fig. 2a, b).

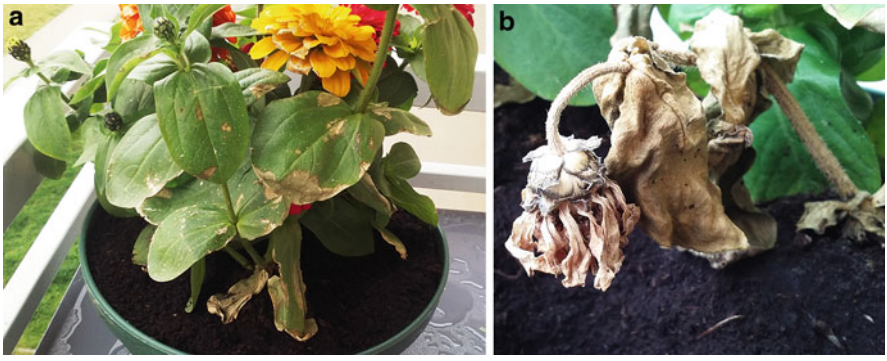


Fig. 2 Botrytis blight symptoms/signs: leaf blight (*left*) flower and stem blight with profuse sporulation (*right*) (D. Szopińska © 2017. All Rights Reserved.)

Biology and epidemiology. The pathogen can survive as mycelia and conidia or for extended periods as sclerotia in crop debris (Williamson et al. 2007). *Botrytis cinerea* produces tremendous quantities of airborne spores, which are moved around by wind or splashing water onto blossoms or young leaves. Cool temperatures (7–15 °C) and high humidity (≥93%) favor spore germination; however, the disease may occur within wide range of temperatures. The pathogen invades host plant tissues directly or through natural openings or wounds (Daughtrey et al. 2000).

Management

- ***Cultural practices*** – Crucial for controlling *B. cinerea* is reducing free water on plant surfaces and lowering humidity (Daughtrey and Benson 2005).
- ***Sanitation*** – Strict sanitation is required to prevent the disease from spreading. The dead or dying tissue from the plants and from the soil surface as well as old blossoms and leaves should to be removed and destroyed.
- ***Fungicides and biocontrols*** – There is a high risk of resistance arising in *B. cinerea* if a chemical product is applied repeatedly. Since 1980s, there have been numerous reports about the resistance of *B. cinerea* to benzimidazoles (e.g., benomyl, carbendazim, thiophanate-methyl) and dicarboximides (e.g., iprodione, procymidone, vinclozolin) (LaMondia and Douglas 1997; Northover and Matteoni 1986; Vali and Moorman 1992; Yourman and Jeffers 1999). This phenomenon has also been recorded to a lesser extent in the case of the anilinopyrimidines (e.g., cyprodinil, pyrimethanil), the hydroxyanilide fenhexamid, and some multisite fungicides and fluazinam (Leroux 2004; Leroux et al. 2010). Therefore, application of multisite fungicides such as chlorothalonil, copper hydroxide, copper sulfate pentahydrate, and mancozeb as well as mixed spray programs have been advised (Daughtrey et al. 2000; Williamson et al. 2007). Fungicide resistance has also led to the intensive evaluation of microbial antagonists for control of gray mold. The greatest potential for control of *B. cinerea* has been shown by the fungi *Trichoderma harzianum*, *Gliocladium roseum*, and *Ulocladium oudemansii*; the bacteria *Bacillus subtilis*, *Pseudomonas syringae*, *and Streptomyces griseoviridis*; and the yeasts *Candida oleophila* and *Pichia pastoris* (Elad and Stewart 2004).

2.3 Cercospora Leaf Spot (*Cercospora zinniae* Ellis & G. Martin)

Geographic occurrence and impact. The pathogen has been reported on *Z. elegans* in India, Jamaica, Mauritius, Thailand, the USA, and Venezuela (Felix 1960; Palacios et al. 1991; Pande 1975; Pereira 2008; Phengsintham et al. 2013; Wehlburg 1969).

Symptoms/signs. Cercospora leaf spot closely resembles Alternaria leaf spot, and symptoms of both diseases often occur together on the same leaf. Leaf symptoms include relatively large, round, reddish brown or dark purple spots, with light gray



Fig. 3 Cercospora leaf spot symptoms (R.J. McGovern © 2017. All Rights Reserved.)

centers, which become ashen gray, papery, and brittle. Severely infected leaves become brown and desiccated. The dead tissues often crack and tear (Fig. 3).

Biology and epidemiology. The fungus is favored by warm weather and high humidity. Conidia of *C. zinniae* germinate within 6 h after inoculation of the zinnia leaf surface. Germ tubes penetrate leaf tissue through the stomatal pores. Spots on the leaves appear 5–7 days after inoculation. Development of the first conidia begins on the spots at 9 days after inoculation. The spores are dispersed in the field by wind, insects, splashing water, and humans (Pereira 2008).

Management

- ***Cultural practices*** – Avoiding overhead irrigation and proper plant spacing are important to prevent spread of this pathogen. Crop rotation with hosts not affected by *Cercospora zinniae* is recommended.
- ***Fungicides*** – Under field conditions mancozeb (0.2%) followed by carbendazim (0.1%) and chlorothalonil (0.2%) reduced Cercospora leaf spot percent disease index from 53.62 (water spray) to 5.94, 13.36, and 18.26, respectively (Yadahalli et al. 1994). Similar results were recorded by Raghavendra Rao and Chacko (1986) and Madhumeeta and Shyam (1989).

2.4 Fusarium Root Rot (*Fusarium* spp.)

Geographic occurrence and impact. Several species of *Fusarium*, e.g., *F. avenaceum*, *F. equiseti*, *F. culmorum*, *F. oxysporum*, and *F. solani* have been isolated from diseased zinnia plants in Poland. *Fusarium equiseti* showed the highest pathogenicity towards zinnia seedlings (Kiecana and Mielniczuk 2010; Łacicowa et al. 1979).

Symptoms/signs. Disease symptoms include reduction of growth, necrotic streaks on the hypocotyls, necrotic spots with yellow halos limited to the edge of leaves, and root rotting of severely infected plants. *Fusarium* spp. may also cause pre- and postemergence damping-off.

Biology and epidemiology. *Fusarium* root rot is favored by high air and soil temperatures that exceed 23 °C. At lower soil temperatures, disease symptoms may not occur, and even though the plant is diseased, it may appear to be healthy. The pathogens survive in the soil associated with plant debris but also directly in the soil as mycelium, spores, and, except in the case of *F. avenaceum*, as chlamydospores (especially in cooler regions). They spread by means of water and contaminated equipment. Growing mycelia invade the root system of healthy plants through wounds or at the point of formation of lateral roots.

Management. The fungi are so persistent and widespread that control measures, such as crop rotation and soil sterilization, are of limited value. Some biological methods were proposed but chemical control is currently the only practical way to manage *Fusarium* spp. effectively in zinnia.

2.5 Pythium Root Rot (*Pythium* spp.)

Geographic occurrence and impact. *Pythium* spp. belongs to the kingdom Chromista, class Oomycetes. These pathogens have been reported on zinnia in the USA (Lewis et al. 1996; Lumsden and Locke 1989).

Symptoms/signs. The pathogens attack roots which become dark and rot. Infected plants wilt very quickly. *Pythium* spp. may also cause pre- and postemergence damping-off.

Biology and epidemiology. The pathogens from genus *Pythium* are distributed as zoospores with water, and long-term survival is enabled by chlamydospores and sexually produced oospores. Wet soil with poor aeration favors *Pythium* growth and development. *Pythium* spp. have wide range of hosts worldwide.

Management

- ***Biocontrol*** – *Gliocladium virens* controlled damping-off of zinnia caused by *P. ultimum* and *R. solani* in a nonsterile soilless mix. Disease control efficacy lasted for at least 2 m when G. *virens* was introduced with the pathogen inoculum and the mix was planted at intervals with zinnia seeds (Lumsden and Locke 1989). Alginate prills, formulated with the isolate GI-22 of *G. viren*s and various food bases (wheat bran, corn cobs, peanut hulls, soy fiber, castor pomace, and chitin) also significantly reduced the damping-off of zinnia in a soilless mix caused by these pathogens (Lewis et al. 1996).

2.6 Phytophthora Root and Crown Rot (*Phytophthora* spp.)

Geographic occurrence and impact. *Phytophthora* spp. also belong to the kingdom Chromista and class Oomycetes. These pathogens infest a wide range of crops, including zinnia (Moore 1959).

Symptoms/signs. Symptoms include rot of roots and lower stems and damping-off of seedlings. Infected plants wilt and die.

Biology and epidemiology. *Phytophthora* spp. are disseminated as zoospores with water through irrigation and rain. Wet soil favor incidence of this disease. Long-term survival is by chlamydospores and sexually produced oospores.

Management. Susceptible crops should be planted in pathogen free soil. Crop rotation with resistant or less susceptible crops is advised.

2.7 Powdery Mildew [*Golovinomyces cichoracearum* (DC.) V.P. Heluta (Formerly *Erysiphe cichoracearum* DC. ex Merat); *Euoidium* sp.]

Geographic occurrence and impact. Powdery mildews are common and widespread diseases, usually more abundant in semiarid regions than in areas of high rainfall (Kamp 1985). The disease caused by *G. cichoracearum* has been reported on zinnia in Egypt, India, Japan, New Zealand, the Republic of South Africa, the USA, and Venezuela (Amano 1986; Baker and Locke 1946; Dingley 1965; Gombert et al. 2001; Hegazi and El-Kot 2010a, b; Husain and Akram 1995; Kamp 1985; Linderman and Ewart 1990; Mir et al. 2012; Palacios et al. 1991; Ruhl et al. 1987; Schmitt 1955; Terry-Lewandowski and Stimart 1983; Tesfagiorgis 2008). A powdery mildew caused by *Euoidium* sp. has been reported on zinnia in Japan (Hoshi et al. 2013). High incidence and severity of powdery mildew on zinnias in some

regions of Asia and North America appears to be a major factor for the declining commercial value of these plants (Mir et al. 2012; Spooner et al. 1991).

Symptoms/signs. White fungal growth forms on the upper surface of leaves and may form on flower petals. Leaves die from base of plant upwards. Affected plants lose vigor and cease growth.

Biology and epidemiology. The fungi causing powdery mildews are obligate parasites. The mycelium of most powdery mildew fungi grows only on the surface and produces usually short conidiophores. When environmental conditions become unfavorable, the fungus may produce sexual structures called cleistothecia, containing one or a few asci. The spores can be released, germinate, and cause infection even if there is no film of water on the plant surface as long as the relative humidity in the air is moderately high (Agrios 2005). Baker and Locke (1946) revealed that seed transmission of the pathogen is possible; however, primary inocula of *G. cichoracearum* for zinnia infections are likely to come from adjacent wild or cultivated Asteraceae (Boyle and Wick 1996).

Management

- ***Cultural practices*** – For powdery mildew, unlike Alternaria blight and bacterial leaf and flower spots, drip irrigation should to be avoided, because it may produce greater powdery mildew severity than overhead irrigation (Gombert 1998).
- ***Fungicides*** – Currently, the only practical method for controlling powdery mildew on zinnias in commercial production is by applying synthetic chemicals. Fungicide applications must be started when the first sign of the white powdery colonies on the lower leaves appears and should to be reapplied according to the specifications on the fungicide label. Such fungicides as tebuconazole, chlorothalonil, triforine, propioconazole may be use to control powdery mildew (Hagan 2009).

  Kamp (1985) demonstrated that the use of a polymer-based antitranspirant significantly reduced powdery mildew on *Z. elegans*. The author suggested that the epidermal coating may interrupt pathogen development on the leaf surface by repelling the film of free water from the leaves and disorientation of pathogen germ tubes by changes in surface topology. Locke et al. (2006), on the other hand, observed delay of expression of powdery mildew on zinnia grown hydroponically in Hoagland's solution fortified with silicon. Tesfagiorgis (2008) found that zinnia plants have the ability to accumulate high levels of silicon in the leaves, which may support an active mechanism of defense within the host plant.
- ***Biological control and organic treatment*** – Hegazi and El-Kot (2010a) sprayed zinnia plants four times at 1 week intervals with 25 and 50% culture filtrates of *Trichoderma harzianum, Epicoccum* sp., *Streptomyces endus*, as well as two plant extracts, miswak *(Salvadora persica*) and henna (*Lawsonia inermis*) to

control powdery mildew. All applied treatments significantly decreased disease incidence and severity; however, *Epicoccum* sp. and *T. harzianum* at 50% concentration followed by henna and miswak extracts inhibited spread of disease to the highest extent. The authors also observed a significant decrease of powdery mildew incidence and severity as well as improvement of plant growth parameters (i.e., plant height, number of branches per plant, leaf area, fresh and dry weights of shoots and roots, root length) when zinnia plants were treated with cinnamon, clove, and ginger oils at a concentration of 0.1 ppm (Hegazi and El-Kot 2010b). In organic farming use of preparations based on copper, organic, and mineral oils (e.g., neem oil, paraffinic oil) and *Bacillus subtilis* strain QST 713 are recommended (Hagan 2009).

- ***Resistance*** – There are only a few *Z. elegans* cultivars that are resistant to *G. cichoracearum* (Gombert et al. 2001). Artificial hybrids between *Z. angustifolia* and Z. *elegans*, on the other hand, are highly resistant or immune to powdery mildew (Terry-Lewandowski and Stimart 1983, 1985; Terry-Lewandowski et al. 1984). This disease has rarely been noted on the Crystal and Star series as well as the hybrid Profusion series zinnias (Hagan 2009).

2.8 Rhizoctonia Root Rot [*Rhizoctonia solani* Kühn (Teleomorph: *Thanatephorus cucumeris* (A.B. Frank) Donk)]

Geographic occurrence and impact. Rhizoctonia diseases occur worldwide and cause losses in almost all crops. Rhizoctonia root, crown, and stem rot have been documented in zinnia in Australia, Poland, and the USA (Łacicowa et al. 1979; Lumsden and Locke 1989; Schisler et al. 1994).

Symptoms/signs. The first sign of infection in seedlings is damping off. In older plants, the infection begins on the stem as dark-brown lesion below the soil line and then a cottony, white mycelium grows upward in the plant. Root discoloration and rot can also occur. Numerous small black sclerotia may be produced on all infected tissues.

Biology and epidemiology. The pathogen overwinters as mycelium or sclerotia in the soil and in some species, including zinnia, may be carried in the seeds. Very young seedlings may be killed before or after emergence. The fungus spreads with rain and irrigation, tools contaminated with soil, and with infected seeds. High temperatures (about 35 °C) and moderately wet soil favor infection.

Management

- ***Cultural practices*** – Disease-free seeds should be planted.
- ***Biocontrol*** – *Gliocladium virens* controlled damping-off of zinnia caused by *Pythium ultimum* or *R. solani* in a nonsterile soilless mix (Lewis et al. 1996; Lumsden and Locke 1989).

2.9 Sclerotinia Stem Rot [*Sclerotinia sclerotiorum* (Lib.) de Bary]

Geographic occurrence and impact. The pathogen has been reported on zinnias in Argentina, Great Britain, Poland, and the USA (Grabowski and Malvick 2015; Kiecana and Mielniczuk 2010; Kiehr et al. 2010; Łacicowa et al. 1979; Moore 1959).

Symptoms/signs. *Sclerotinia sclerotiorum* causes crown rot, bulb rot, stem rot, wilt, and death of a wide variety of plants. The typical early sign of *Sclerotinia* disease on zinnia is the appearance on the infected plant of a white fluffy mycelium in which variable sized sclerotia soon develop. The mycelium first develops on the base of stem. When the fungal infection develops and the stem rots, the foliage above the lesion wilts and dies. The sclerotia of *S. sclerotiorum* may be also formed inside the stem. Moreover, the pathogen is frequently responsible for damping-off of zinnia seedlings (Łacicowa et al. 1979).

Biology and epidemiology. The pathogen overwinters as sclerotia in the soil and as sclerotia and mycelium on or within infected tissues of dead plants. The sclerotia germinate in the spring or early summer producing apothecia which discharge a large number of ascospores into the air. The spores reaching available sources of food (e.g., old plant parts) start to germinate and cause infection. Infection may also start from the soil when sclerotia produce mycelial strands which invade young plants (Agrios 2005). The pathogen may cause pre- and postemergence damping-off.

Management

- ***Cultural practices*** – Susceptible cultivars should be planted in well-drained soil. Weeds as a potential hosts should to be removed. Because sclerotia may survive in the soil, at least a 3-year rotation with non-susceptible crops is recommended.

2.10 Sclerotium Stem Blight, Southern Blight [*Sclerotium rolfsii* Sac. (Teleomorph: *Athelia rolfsii* (Curzi) C.C. Tu & Kimbr.)

Geographic occurrence and impact. *Sclerotium rolfsii* has been reported on zinnia in India, Japan, and New Zealand (Boesewinkel 1977; Ishii and Abiko 1997; Ramakrishnan 1930; Thakur 1969).

Symptoms/signs. The pathogen attacks the stem bases, leaves turn yellow, the plant initially wilts, and then dies. A large number of white turning tan, mustard seed-sized sclerotia are formed on the infected tissue.

Biology and epidemiology. Wet and warm weather favor disease progress. Optimal temperatures for fungal growth and disease development are 25–35 °C. High relative humidity is required for germination of sclerotia (Mordue 1974). Sclerotia in the soil are the main source of this disease.

Management

- ***Cultural practices*** – Management strategies include a 3- to 4-year rotation with non-susceptible crops, raising the soil pH, and removal of crop debris (Mordue 1974).

Additional fungal pathogens of zinnia. The following fungi have also been reported as pathogenic to zinnia:

***Alternaria carthami* S. Chowdhury** – in Taiwan (Wu and Chou 1995)
***Colletotrichum acutatum* J. H. Simmonds** – in India (Kulshrestha 1976)
***Colletotrichum falcatum* Went.** – in Venezuela (Palacios et al. 1991)
***Nigrospora sphaerica* (Sacc.) E.W. Mason** – in the USA (Meepagala et al. 2015)
***Sclerotinia minor* Jagger** – in Argentina (Kiehr et al. 2010)

3 Bacterial and Phytoplasma Diseases

3.1 Apical Chlorosis [*Pseudomonas syringae* pv. *tagetis* (Hellmers) Young, Dye & Wilkie, syn. *Pseudomonas tagetis* (Hellmers)]

Geographic occurrence and impact. This disease has been reported on zinnia in Australia and the USA (Gulya et al. 1982; Jutte and Durbin 1979; Trimboli et al. 1978).

Symptoms/signs. Leaf spots, sometimes with chlorotic haloes, and apical chlorosis are typical symptoms of this disease (Fig. 4).

Biology and epidemiology. *Pseudomonas syringae* pv. *tagetis* is a Gram negative aerobic rod. The pathogen overseasons in infected plant debris in the soil. In sunflower, this pathogen is seed-borne, but it is not known if this type of transmission occurs in zinnia. Weeds as well as cultivated crops within the family Asteraceae may provide natural sources of inoculum. Wet weather favors spread of this disease.



Fig. 4 Apical chlorosis symptoms caused by *Pseudomonas syringae* (T.J. Gulya. USDA.)

Management. A 3-year or longer rotation is recommended to avoid building up large soil populations of the bacterium. Plants should to be watered in a manner that keeps the leaves dry to inhibit spread of this disease.

3.2 Bacterial Leaf Spot (*Xanthomonas campestris* pv. *zinniae* Hopkins and Dawson)

Geographic occurrence and impact. The disease has been observed in many countries including Australia, Brazil, Bulgaria, Hungary, India, Italy, Korea, Malawi, Pakistan, Rhodesia, Sierra Leone, and the USA (Akhtar and Khokhar 1988; Bertus and Hayward 1971; Boyle and Wick 1996; Deighton 1957; Dimitrov and Tsoneva 1977; Hopkins and Dowson 1949; Jones and Strider 1979; Myung et al. 2012; Nannizzi 1929; Peregrine and Siddiqi 1972; Rangaswami and Gowda 1963; Robbs 1954; Sahin et al. 2003; Schwarczinger et al. 2008a, b; Sleesman et al. 1973; Strider 1973, 1976, 1979a, b, 1980; Terry-Lewandowski and Stimart 1983).

Symptoms/signs. The pathogen can attack all aboveground parts of zinnia plants, causing necrotic lesions of leaves, stems, and flowers. The first symptoms of the disease are small (1–2 mm), dispersed, transparent spots encircled by broad yellowish haloes. Under wet conditions, the lesions slowly enlarge. The spots become angular to irregularly circular and develop a reddish center. The lesions may merge to form irregular dead areas (0.5–1.0 cm long), which often crack as they dry. During very humid weather, small brown spots may appear on the ray flowers. Severely infected flower heads are seriously disfigured and may completely decay (Fig. 5).

Biology and epidemiology. The pathogen has been reported to overseason in diseased plant residue in the field and in or on seeds (Strider 1973, 1979a). Infected seeds are known to be the major means of long-distance dispersal and primary inoculum source of the pathogen (Strider 1979b). *Xanthomonas campestris* pv. *zinniae* is capable of spreading rapidly through warm, humid, and rainy weather.



Fig. 5 Bacterial leaf spot symptoms (R.J. McGovern © 2017. All Rights Reserved.)

Infected zinnias develop characteristic spots on the leaves within 2 weeks. Jones and Strider (1979) observed that plants at a 29–31/21–23 °C (max/min) temperature regime developed the most severe disease symptoms.

Management

- ***Cultural practices*** – The use of pathogen free seeds and transplants has been recommended for control of bacterial leaf spot (Strider 1979a, 1980). Whenever possible, plants should be irrigated in a manner that keeps the leaves dry to inhibit spread of this disease.
- ***Sanitation*** – Plants with leaf spots should be discarded and diseased plant debris should be removed from the growing area. Washing hands after handling diseased plants or soil is recommended. Handling of wet foliage should be avoided.
- ***Chemical control*** – Chemical control of bacterial leaf spot on zinnia is difficult if not impossible to obtain. Copper hydroxide may provide some protection from this disease. To be effective, treatments have to be applied on a weekly schedule, beginning when the spots first appear on the leaves (Hagan 2009). Bactericides are only marginally effective in controlling *Xanthomonas* spp. Strider in 1980 recorded the efficacy of captan as a bactericide for control bacterial leaf spot on zinnia.
- ***Resistance*** – Most common zinnia cultivars as well as *Z. haageana* "Persian Carpet", *Z. angustifolia* "Pixie Sunshine", *Z. tenuifolia* "Red Spider", and *Z. pumilia* "Cut and Come Again" are susceptible to *X. campestris* pv. *zinniae* (Hagan 2009; Gombert et al. 2001). However, hybrids of *Z. elegans* and *Z. angustifolia* exhibit moderate to high levels of resistance to this pathogen (Terry-Lewandowski and Stimart 1983). The Crystal and Star series zinnias are highly resistant or immune to bacterial leaf spots. The Profusion series zinnias are moderately resistant to this disease. Some spotting of the leaves in the lower canopy as well as occasional but unobtrusive leaf death has been seen on Profusion series zinnias in the field (Hagan 2009).

3.3 Ralstonia Wilt (*Ralstonia solanacearum* (Smith 1896) Yabuuchi et al. 1996, comb. nov.)

Geographic occurrence and impact. *Ralstonia solanacearum* (formerly *Pseudomonas solanacearum*) causes a severe and devastating wilt to many economically important crops around the world (Hayward 1991). This disease has been reported on zinnia in Australia, India, and Malaysia (Abdullah 1992; Papdival and Deshpande 1978; Thammakijjawat et al. 2001). Strider et al. (1981) reported that a strain of *R. solanacearum* isolated from geranium was also pathogenic on zinnia. *Ralstonia solanacearum* is a pathogen of quarantine importance.

Symptoms/signs. Infected young plants die rapidly. On older plants, symptoms appear first on the youngest leaves or one-sided wilting and stunting is observed. Finally plants wilt completely and die. The vascular tissues of stem and roots turn brown (Agrios 2005).

Biology and epidemiology. According to host range, *R. solanacearum* strains have been classified into five races (Buddenhagen and Kelman 1964; Hayward 1991). A zinnia strain of *R. solanacearum* originating in Australia has been classified as race 1 (Thammakijjawat et al. 2001). This race attacks all the solanaceous and many non-solanaceous crops and appears as a sudden wilt. The bacteria overwinter in plant debris and spread in soil with water and then enter plants through wounds made in roots by insects, nematodes, or by handling.

Management. There are no chemicals or biological agents to effectively control this bacterium. Infected plants have to be discarded as soon as possible. Pathogen-free propagating plant material is the main way to avoid problems with *Ralstonia.* Soil and equipment should to be disinfested.

3.4 Aster Yellows ("*Candidatus* Phytoplasma Asteris")

Geographic occurrence and impact. This disease has been reported on zinnia in Canada and India (Rao et al. 2012; Singh et al. 2011; Wang and Hiruki 2001).

Symptoms/signs. The pathogen causes general yellowing and dwarfing of the plant, abnormal production of shoots (formation of witches' brooms), and malformation of organs (greening or sterility of flowers, shortening of the internodes). The disease reduces yield and may lead to more or less rapid dieback, decline, and death of the plant.

Biology and epidemiology. Taxonomically, phytoplasmas are member of the class *Mollicutes* and are currently classified within the provisional genus "*Candidatus* Phytoplasma" based primarily on 16S rDNA sequence analysis (Marcone 2014). The phytoplasmas detected in zinnia have been placed in the subgroup II "*Candidatus* Phytoplasma asteris" (16SrI-B) (Rao et al. 2012; Wang and Hiruki 2001). Phytoplasmas are prokaryotic organisms that have no cell walls and can rarely be grown on artificial nutrient media. Most plant mollicutes are vectored by leafhoppers (Cicadellidae) but some by planthoppers (Fulgoroidea). Phytoplasmas are generally present in the sap of a small number of sieve tubes. Insects become vectors when feeding on leaves and stems of infected plants. The phytoplasma then multiplies and infects various body organs of insect vectors, a process that can take 1–3 weeks. When infected leafhoppers feed on healthy plants, the phytoplasmas in their salivary glands are injected into the plant phloem and disease symptoms develop in 10–40 day. A leafhopper carrying the aster yellows phytoplasma can continue infecting plants for the rest of its life. Outbreaks of aster yellows may be expected in cool and wet summers, which favor both leafhoppers and phytoplasma development (Agrios 2005).

Management. There are currently few effective control strategies for aster yellows. Infected plants should to be promptly removed and destroyed. Insect vectors should to be monitored and controlled systematically, because they are essential for

dispersal of the pathogen and long-term survival in nature. Management of aster yellows includes chemical and biological control of leafhoppers, repellence and disorientation of insects in the field, and avoidance of plant production near infected crops or alternate hosts.

4 Viral Diseases

4.1 Bromoviridae

Geographic occurrence and impact. This family contains five genera of viruses: *Alfamovirus, Bromovirus*, *Cucumovirus*, *Ilarvirus,* and *Oleavirus*. Several members of these genera are distributed worldwide. Four viruses from the Bromoviridae have been reported in zinnia: *Alfalfa mosaic virus* (AMV, genus *Alfamovirus*) in Israel, *Cucumber mosaic virus* (CMV, genus: *Cucumovirus*) in India, Iran, and the USA, *Tomato aspermy virus* (TAV, genus *Cucumovirus*), and *Asparagus virus 2* (AV2, genus *Ilarvirus*) in Japan (Fujisawa et al. 1983; Kameya-Iwaki et al. 1996; Nitzany and Cohen 1960; Price 1935; Raj et al. 1997; Shahmohammadi et al. 2015). According to Brunt et al. (1996–), zinnia is also susceptible to the following viruses from this family: *Peanut stunt virus* from genus *Cucumovirus* and *Elm mottle virus*, *Humulus japonicus virus*, *Plum American line pattern virus*, *Prune dwarf virus*, *Prunus necrotic ringspot virus*, and *Tobacco streak virus* from genus *Ilarvirus*.

Symptoms/signs. All viruses detected in zinnia produce similar symptoms: leaf mosaic and distortion, stunting, and flowers malformation (Fig. 6).

Biology and epidemiology. Aphids (Hemiptera: Aphididae) and thrips (Thysanoptera: Thripidae) are main vectors of Bromoviridae. *Aphis gossypii, A. craccivora, A. fabae,* and *Myzus persicae* are responsible for CMV transmission in zinnia (Nitzany and Cohen 1960). All cucumoviruses are transmitted by aphids in



Fig. 6 CMV symptoms (R.J. McGovern © 2017. All Rights Reserved.)

noncirculative (nonpersistent) manner. They may be also transmitted in some species by seeds or mechanically by handling of the plants in the field or greenhouse (e.g., CMV). Seed transmission by zinnia seeds has been reported only for *Asparagus virus 2* (Fujisawa et al. 1983).

Management. Controlling insect vectors, avoiding growing near infected crops, and removing weeds that serve as hosts help to control viral diseases. Vector populations may be reduced by chemical and biological methods and various physical barriers. Use of reflective mulches disorients the vectors and protects the plants.

4.2 Bunyaviridae

Geographic occurrence and impact. *Tospoviruses* make up one genus of the viruses within this family, including several economically important species with an extremely wide host range. The main member of this genus – *Tomato spotted wilt virus* (TSWV) – has been detected in zinnia in Bulgaria and Mexico (Bakardjieva et al. 1998; Morales-Díaz et al. 2008).

Symptoms/signs. Typical symptoms on zinnia are chlorotic rings and spots on the leaves, wilting of the leaves, and plant death.

Biology and epidemiology. Tospoviruses are transmitted by thrips (e.g., *Frankliniella occidentalis*, *F. fusca*, *F. schultzei*, *Thrips tabaci*, *T. palmi*, etc.). The virus is acquired from infected plants by thrips larvae after at least 30 min of feeding. The thrips transmits the virus to healthy plants for the rest of its life.

Management. Refer to the Bromoviridae management section above and introductory (▶ Chap. 4, "Insect Management for Disease Control in Florists' Crops").

4.3 Caulimoviridae

Geographic occurrence and impact. *Dahlia mosaic virus* DMV belonging to the genus *Caulimovirus* has been reported on zinnia in the USA (Kitajima et al. 1969).

Symptoms/signs. The symptoms range from none to occasional chlorotic spots to severe mosaic and stunting.

Biology and epidemiology. DMV can be transmitted by 16 different aphid (Hemiptera: Aphididae) species, mechanical inoculation, and through the seed (not recorded for zinnia).

Management. Refer to the Bromoviridae management section above and introductory (▶ Chap. 4, "Insect Management for Disease Control in Florists' Crops").

4.4 Closteroviridae

Geographic occurrence and impact. Two viruses belonging to genus *Crinivirus* have been reported in zinnia in Taiwan: *Tomato chlorosis virus* (ToCV) and *Tomato infectious chlorosis virus* (TICV) (Tsai et al. 2004). According to Brunt et al. (1996), zinnia is also susceptible to two other viruses from this genus: *Beet pseudoyellows virus* and *Lettuce infectious yellows virus*.

Symptoms/signs. Pronounced yellowing symptoms on the lower leaves of plants, similar to those caused by nitrogen deficiency; the brittleness of the discolored leaves; and occasional upward leaf rolling were observed on infected zinnia plants.

Biology and epidemiology. Criniviruses are confined to the phloem and phloem parenchyma cells. TICV is transmitted exclusively by the whitefly *Trialeurodes vaporariorum*, whereas ToCV is vectored by *T. vaporariorum*, and another whitefly, *Bemisia tabaci*.

Management. Refer to the Bromoviridae management section above and introductory (▶ Chap. 4, "Insect Management for Disease Control in Florists' Crops").

4.5 Comoviridae

Geographic occurrence and impact. Two nepoviruses have been reported on zinnia: *Tobacco ringspot virus* (TRSV) in Japan and the USA and *Tomato black ring virus* (TBRV) in Kenya (Iizuka 1973; Kaiser et al. 1978; Moore and McGuire 1968). According to Brunt et al. (1996–), zinnia is also susceptible to the following viruses from this family: *Cowpea mosaic virus* from genus *Comovirus* and *Arabis mosaic virus*, *Artichoke vein banding virus**, *Caraway latent virus**, *Cherry leaf roll virus*, *Dogwood mosaic virus**, *Peach enation virus**, *Croton yellow vein mosaic virus,* and *Soybean crinkle leaf virus* from genus *Nepovirus.* (*Species not recorded currently in the ICTV Master Species List).

Symptoms/signs. Common symptoms of plant infection by these viruses are mosaic and ring spot, sometimes accompanied by systemic necrosis.

Biology and epidemiology. *Tobacco ringspot virus* is transmitted by the nematode *Xiphinema americanum* and also nonspecifically by insects and mites such as: *Aphis gossypii, Myzus persicae, Melanopus* sp. *Epitrix hirtipennis, Thrips tabaci*, and *Tetranychus* sp. Iizuka (1973) reported 5% transmission of TRSV by zinnia seeds. This virus causes systemic infection in susceptible cultivars, moving from infected leaves to the tips of stems and into roots. Movement from roots to leaves is uncommon (Moore and McGuire 1968). Kaiser et al. (1978) detected seed transmission of TBRV in 1 seedling infected out of 4 tested.

Management. Refer to the Bromoviridae management section above and introductory (▶ Chap. 4, "Insect Management for Disease Control in Florists' Crops").

4.6 Geminiviridae

Geographic occurrence and impact. These viruses are responsible for a significant amount of crop damage worldwide. Four species in genus *Begomovirus* were detected in zinnia: *Ageratum enation virus* (AEV) in India, *Ageratum yellow vein China virus* (AYVCNV) and *Alternanthera yellow vein virus* (AlYVV) in Vietnam, and *Tomato yellow leaf curl virus* (TYLCV) in China. Two other species *Zinnia leaf curl virus* (ZLCV) identified in India and *Zinnia mosaic virus* recorded in India and Spain are not recorded currently in ICTV Master Species List and their taxonomic position is unsure (Ha et al. 2008; Huertos 1953; Jabri et al. 1985; Kumar et al. 2010; Li et al. 2013, 2014); Maritan et al. 2004; Panday and Tiwari 2012; Verma and Singh 1973).

Zinnia plants are also susceptible to the following viruses from this family: *Beet curly top virus* belonging to genus *Becurtovirus* and two species from genus *Begomovirus*: *Croton yellow vein mosaic virus and Soybean crinkle leaf virus* (Brunt et al. 1996–).

Symptoms/signs. Symptoms caused by AEV, AYVCNV, TYLCV, and ZLCV have been described as leaf curling, yellow mosaic, and stunting (Figs. 7 and 8). Zinnia mosaic symptoms have been characterized as a strong mosaic, stunting, decrease of the number of flowers, and color breaking.

Biology and epidemiology. Begomoviruses are transmitted circulatively and nonpropagatively by the whitefly *Bemisia tabaci*. Once the virus is acquired, the whitefly remains viruliferous for life.

Management. Refer to the Bromoviridae management section above and introductory ▶ Chap. 4, "Insect Management for Disease Control in Florists' Crops").

4.7 Potyviridae

Geographic occurrence and impact. Potyviruses include many of the viruses causing some of the most severe and economically important plant diseases. Five species belonging to this genus have been identified in zinnia: *Bidens mottle virus* (BiMoV) and *Sunflower mosaic virus* (SuMV) in the USA, *Sunflower chlorotic mottle virus* (SuCMoV) in Brazil, and *Vanilla distortion mosaic virus* (VDMV) and *Zinnia mild mottle virus** in India (Balaji et al. 2014; Gulya et al. 2002; Logan et al. 1984; Maritan et al. 2004; Padma et al. 1972, 1974).



Fig. 7 *Ageratum enation virus* symptoms (Kumar et al. 2010)



Fig. 8 *Tomato yellow leaf curl virus* symptoms (Li et al. 2014)

According to Brunt et al. (1996–), zinnia is also susceptible to many other viruses from this family: *Sweet potato mild mottle virus* from genus *Ipomovirus* and *Bean yellow mosaic virus*, *Beet mosaic virus*, *Bidens mosaic virus*, *Carrot thin leaf virus*, *Celery mosaic virus*, *Clover yellow vein virus*, *Lettuce mosaic virus*, *Nasturtium*

*mosaic virus**, *Patchouli mosaic virus*, *Pea seed-borne mosaic virus*, *Pepper veinal mottle virus*, *Plum pox virus*, *Primula mosaic virus*, *Tobacco etch virus*, *Tropaeolum 2 virus*, *Turnip mosaic virus*, *Watermelon mosaic 2 virus*, and *Wisteria vein mosaic virus* belonging to genus *Potyvirus.* (*Species not recorded currently in ICTV Master Species List).

Symptoms/signs. Diseases caused by potyviruses appear primarily as mosaics, mottling, chlorotic rings or color breaking on foliage, flowers, and stems. Many of these viruses may cause severe stunting of young plants, stem malformations, and necroses of various tissues.

Biology and epidemiology. Potyviruses are spread by aphids (Hemiptera: Aphididae) in a noncirculative (nonpersistent) manner and several are transmitted through seeds (not recorded for zinnia). These viruses overseason in perennial cultivated and weed hosts.

Management. Refer to the Bromoviridae management section above and introductory ▶ Chap. 4, "Insect Management for Disease Control in Florists' Crops").

4.8 Other Viruses

According to the VIDE Database (Brunt et al. 1996–), zinnia is also susceptible to the following viruses:

Family Flexiviridae, genus *Carlavirus:* *Artichoke latent S virus**, *Butterbur mosaic virus**, *Cassia mild mosaic virus**, *Pea streak virus**, *Poplar mosaic virus*; **genus *Potexvirus:*** *Asparagus 3 virus*, *Cymbidium mosaic virus*, *Foxtail mosaic virus*, *Plantain X virus*

Family Luteoviridae, genus *Luteovirus:* *Beet mild yellowing virus*, *Beet western yellows virus*, *Subterranean clover red leaf virus*

Family Rhabdoviridae, genus *Nucleorhabdovirus:* *Sonchus yellow net virus*

Family Secoviridae: *Strawberry latent ringspot virus*

Family Tombusviridae, genus *Carmovirus*: *Cucumber leaf spot virus*, *Galinsoga mosaic virus*; **genus *Dianthovirus:*** *Carnation ringspot virus*; **genus *Necrovirus*:** *Lisianthus necrosis virus**, *Tobacco necrosis virus*; **genus *Tombusvirus:*** *Cymbidium ringspot virus*, *Pepper Moroccan virus*, *Tomato bushy stunt virus*

Family Virgaviridae, genus *Tobamovirus:* *Maracuja mosaic virus*, *Odontoglossum ringspot virus*, *Ribgrass mosaic virus*

Genus *Sobemovirus:* *Lucerne transient streak virus*

Genus *Ourmiavirus:* *Ourmia melon virus*

(*Species not recorded currently in ICTV Master Species List).

5 Nematode Diseases

5.1 Angular Spots on the Leaves (*Aphelenchoides* spp.)

Geographic occurrence and impact. Foliar nematodes are widespread pathogens of ornamentals grown in greenhouses, nurseries, and in the field. There are three species of economic importance: *A. besseyi*, *A. fragariae,* and *A. ritzemabosi. Aphelenchoides besseyi* prefers warmer climates, while *A. fragariae* and *A. ritzemabosi* are often found in temperate zones (Kohl 2011). All three species can cause angular spots on *Z. elegans* leaves; however, *A. ritzemabosi* has been the most prevalent. *Aphelenchoides* spp. have been reported on zinnias in India, New Zealand, Poland, and the USA (Boesewinkel 1977; Crossman and Christie 1936; Gill and Sharma 1979; Gill and Uppal 1979; Gill and Walia 1980; Kohl 2011; Madej et al. 2000).

Symptoms/signs. Infected plants appear stunted and dwarfed. Infected leaves become shrunken, occasionally accompanied by discoloration, with blotches and chlorotic patches turning into brown and white-yellow areas limited by the veins.

Biology and epidemiology. Water is necessary for the movement and dispersal of foliar nematodes; however, spread from the direct contact of an infected leaf with uninfected plant tissue is possible. Adult and fourth-stage juvenile *Aphelenchoides* spp. enter leaf tissue through stomata on the leaf undersurface where feeding and reproduction occurs. The nematodes feed by piercing neighboring cells with their stylets. Eggs are laid within healthy, green sections of the leaf tissues. The endoparasitic feeding results in the disintegration of the spongy parenchyma and palisade cells; however, nematodes also feed ectoparasitically on stems, buds, and flowers. Males are necessary for reproduction. Fertilized females are able to lay eggs even after emergence from months of dormancy in an anhydrobiotic state. Each female lays approximately 32 eggs, which hatch in 4 days. The second stage juveniles reach reproductive maturity in 6–7 days. The life cycle of *A. ritzemabosi* can be completed in 14 days, with 5 days for embryonic development and another 5 days for maturation. Adults and fourth-stage juveniles are able to overwinter in an anhydrobiotic state within desiccated plant tissue (dried leaves, dormant buds, but not in plant roots) and can survive for several months to up to 3 years. Low air temperature and high relative humidity increase nematodes populations in leaves (Kohl 2011).

Management. Foliar sprays of quinalphos and methyl parathion at 0.05% applied five times at 10-day intervals effectively controlled *A. ritzamabosi* on *Z. elegans*, reducing both the symptoms of infestation and the final nematode population (Gill and Walia 1980). However, due to environmental concerns and toxicity, these nematicides are no longer available. Many other nematicides were proposed (e.g., chlorfenapyr); however, the most effective chemical controls will be those that are truly systemic within the plants (Kohl 2011). Integration of chemical methods with

cultural practices such as sanitation, removal of crop debris from the planting area, management of leaf wetness by reducing overhead irrigation, and increasing air flow through a crop are necessary to manage foliar nematodes (LaMondia 1999).

5.2 Root Knot (*Meloidogyne* spp.)

Geographic occurrence and impact. Root knot nematodes have been reported on zinnias in Pakistan, Taiwan, and the USA (Jabri et al. 1985; Mc Sorley and Frederick 1994; Tsay et al. 2004).

Symptoms. Infected plants are stunted and poorly growing with yellowing leaves. Infected root systems show characteristic knots or galls.

Biology and epidemiology. *Zinnia elegans* exhibited some galling in response to *Meloidogyne incognita,* but incidence of galling increased when the plants were infected with *Zinnia mosaic virus* (Jabri et al. 1985). Mc Sorley and Frederick (1994) found that *Z. elegans* cv. Scarlet was nearly free of galling from *M. incognita* and *M. arenaria* but was susceptible to *M. javanica* (3,400 eggs per plant). Tsay et al. (2004) also observed that some cultivars of *Z. elegans* and *Z. hageana* were highly and moderately resistant to *M. incognita*, while *Z. angustifolia* was more susceptible.

The life cycles of *Meloidogyne* spp. differ slightly (De Guiran and Ritter 1979). The life cycle of M. *incognita* can be completed in 37 days. Second-stage juveniles penetrate root tips, occasionally invading roots in the zone of root elongation. Invasion of nematodes results in development of giant cells in the meristematic, cortical, and xylem tissues of the root and galling of roots occurs. Third- and fourth-stage juveniles and young females occur after about 6–8 and 15 days, respectively. Adult females are observed after 20 days and egg laying begins after 25 days. *Meloidogyne* spp. are mostly spread by water and through equipments contaminated with infested soil.

Management. Control of root knot nematodes is difficult and options are limited, especially in field production. Equipment should to be frequently cleaned to prevent dissemination of the nematodes from field to field. In greenhouses soil, disinfestation may be obtained by steam heat treatment, solarization, fumigation, or chemical drenches.

- ***Biocontrol*** – The biological control agent *Paecilomyces lilacinus* strain 251 has been successfully applied in several crops to control *M. incognita* (Anastasiadis et al. 2008; Hashem and Abo-Elyousr 2011; Kiewnick and Sikora 2006).
- ***Resistance*** – There have been several reports about the resistance of various zinnia cultivars against root knot nematodes (Mc Sorley and Frederick 1994; Tsay et al. 2004).

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Part III

Florists' Crops Diseases: Potted Flowers

Diseases of Azalea

29

Robert G. Linderman

Abstract

Florist azaleas (*Rhododendron obtusum* (Lindl.) Planch) are grown worldwide in greenhouses as a potted flower plant. They belong to the evergreen group but vary in their cold hardiness, so they must be protected against cold winter conditions. Many cultivars have been developed based largely on their horticultural traits, especially flower size and color. Some cultivars are prone to develop witches' broom, thought to be a genetic disorder. However, most florist azaleas are susceptible to a number of diseases, but little attention has been paid to their resistance/susceptibility through breeding. Of concern to growers are diseases caused by species of *Cylindrocladium*, *Pythium*, *Phytophthora*, *Rhizoctonia*, and occasionally *Exobasidium.* Many of the diseases of florist azaleas, such as infections caused by *Cylindrocladium*, occur during the propagation phase of rooting cuttings, with surviving, infected-but-symptomless plants later succumbing to the disease.

Keywords

Phytophthora spp. • *Pythium* spp. • *Cylindrocladium* spp. • *Exobasidium* spp. • *Rhizoctonia* spp. • Witches' Broom

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R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_29

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1 Introduction

Evergreen azaleas originated in Japan and are classified in the genus *Rhododendron*, subgenus *Tsutsutsi*. These grow indoors and outdoors. Most azaleas bloom in spring, although evergreen florist's azaleas can be forced into bloom under greenhouse conditions for gift giving at any time. Evergreen azaleas vary in cold hardiness with the particular cultivar, but the group spans US Department of Agriculture hardiness zones 5 through 9.

A significant component of the florist industry involves the sale of potted evergreen azaleas (*Rhododendron obtusum* (Lindl.) Planch). Plants are grown in production greenhouses for protection against winter cold, since they vary in cold hardiness. Accordingly, they can be "forced" into flower any time by manipulating the environmental conditions to set flower buds that will bloom predictably to match sales for special occasions. Many cultivars have been selected, mainly for their predictable and extraordinary flowering, with variation in flower size and color. Cultivar development has paid little attention to disease susceptibility/resistance. Some diseases, such as Cylindrocladium leaf spot, blight, and/or root rot, have caused tremendous losses in the nursery industry over the years. Other diseases, common to many other greenhouse and nursery crops, include root rots caused by species of *Pythium*, *Phytophthora*, and *Rhizoctonia*. Some foliage and flower diseases, such as leaf and flower gall, caused by species of *Exobasidium* occur mainly on landscape plants and rarely on florist azaleas, mainly because it is readily controlled by judicious removal of galls and application of chemical sprays.

Most of the more serious diseases are caused by soilborne pathogens that infect newly rooted cuttings. Surviving plants are infected but remain symptomless until some later time when poor growth, dieback, and mortality occur. Soilborne root pathogens are much more challenging to manage because of the multiple ways they can invade the greenhouse cultural system and the limitations on effective management. Nonetheless, numerous methods have been identified to block entry, largely by eliminating the pathogens from the sources. For example, sanitation of containers with heat, treatment of irrigation water, using pathogen-free growth media, chemically sanitizing cutting propagation material, and maintaining strict sanitation practices within the greenhouse environment can be effective measures for preventing entry into the greenhouse. Some pathogens like *Cylindrocladium* spp. are especially troublesome because they can disperse spores (inoculum) via an aerial phase within the greenhouse environment.

2 Fungal and Fungus-Like Diseases

2.1 Phytophthora Root Rot, Dieback, and Wilt (*Phytophthora* spp.)

Geographic occurrence and impact. *Phytophthora* spp. are known to infect azaleas and rhododendrons worldwide. These fungus-like organisms (Phylum Oomycota and genetically related to the brown algae) infect many other plants within the Ericaceae family as well (Jones and Benson 2001; Linderman and Benson 2014). Interestingly, some of those plants occur as wild species, but the diseases do not occur in natural settings, even when the pathogen is present, for unknown reasons. They cause root rot, crown canker, and branch dieback, depending on the species. Some species can cause all of those symptoms, while others are confined to the roots and crown. Most of the Phytophthora diseases occur in landscapes or in nursery field plantings, in ground or in containers. When the diseases occur in the nurseries, significant economic losses result. Beyond the nursery, after shipment and sale, further disease development, even mortality, may occur in the hands of the consumer. Those occurrences are frequently a result of earlier undetected infections or contamination that develops after time and/or a change in environmental and soil conditions. Sometimes, infected liner plants appear healthy and are shipped to other wholesale nurseries for growing on, and with further development of the infections, symptoms appear.

Symptoms/signs. Root infections may develop at any time in the life of florist azaleas, from cutting propagation to field and landscape plantings. Most frequently involved are *P. cinnamomi* Rands and *P. nicotianae* Breda de Haan (syn. *P. parasitica* Dastur), but in some areas, other species such as *P. citricola* Sawada are recovered. Infected roots are brown and become necrotic. When enough roots are infected, the root system becomes impaired, and as a result leaves may become chlorotic and then necrotic but rarely wilt. Those leaves generally drop off the plant, causing partial defoliation (Fig. 1). Leaves on infected plants, as well as the plants themselves, are also often smaller than with healthy plants. With some *Phytophthora* species, root infections progress to the lower stem tissue where lethal crown cankers occur. These cankers are revealed by removal of the bark, exposing the internal wood which appears brown.

Biology and epidemiology. Root, crown, stem, and foliage infections caused by a number of species of *Phytophthora* can occur on florist azaleas when sufficient inoculum is present and environmental conditions are conducive. Those infections occur as a result of inoculum being disseminated by air movement, or by water splash of spores from the soil to aboveground tissues landing on susceptible sites. Free water is required for spore germination and infection. The source of inoculum (zoospores, sporangia, chlamydospores, or oospores, depending on the species) in greenhouse culture of florist azaleas is often unknown. Temperature conditions in the



Fig. 1 Mortality of azalea (*left*) caused by *Phytophthora citricola* (R.G. Linderman © 2017. All Rights Reserved.)

greenhouse systems are generally in the right range for infections by *Phytophthora* spp., as with *Pythium* spp. infections. So the reduction of infections by environmental conditions that might occur outdoors does not apply for florist azaleas. Temperature is modified by heating and cooling to enhance plant growth. When flower bud setting is involved, temperatures are usually lowered. This allows plants destined for the florist trade to be programmed for forcing in time for special holiday sales.

The source of *Phytophthora* spp. inoculum varies, depending a lot on the practices of the nursery. For soilborne, root-infecting pathogens, the medium, recycled containers, irrigation water, and plant propagules used for cutting propagation could be sources. Strict sanitation practices within the greenhouse, as well as the propagation area, must be maintained. Soilless growth media are used in growing florist azaleas, so they are least likely to be a source. Use of well or municipal water is also not likely to be a source. Likely sources are reused containers, recycled irrigation water, and source of propagation cuttings.

Management. The primary management strategy for *Phytophthora*-caused diseases of florist azaleas is prevention. Eliminating the pathogen from the major sources of irrigation water, the growth medium, the containers, and even the cuttings can go a long way to preventing these diseases. Thereafter, application of preventative chemical fungicides provides some protection against invasion from a number of sources, including greenhouse workers. The chemicals used for *Phytophthora* control are the same as for *Pythium* control, and they generally target only those genera and some other fungus-like pathogens, but not fungal pathogens. There are preventative chemicals such as products containing etridiazole, mefenoxam, cyazofamid, fenamidone, and phosphonates. The insensitivity of some species of *Pythium* to mefenoxam has also been reported for *Phytophthora* spp. However, there

can be variation in effectiveness of specific chemicals to different *Phytophthora* spp. (Linderman and Davis 2008a). Rotation or combinations with other chemicals should be practiced, as with management of *Pythium* spp.

In addition to chemical agents, there are some biological control agents that target certain species of *Phytophthora* and *Pythium*. Species of *Trichoderma* and *Streptomyces* have been shown to have that activity but usually have not been tested on a range of pathogen species.

Suppressive soils that prevent or limit some root diseases, including those caused by species of *Phytophthora*, have been reported. Often suppression is created by incorporating mixtures of microbes with great diversity into the growth medium. Within that microbial population are members with specific capacity to block the development of specific diseases. Some have the capacity to block sporangium development, and therefore reduce zoospore release (Linderman et al. 1983). Some are able to lyse the pathogen mycelium, while others produce other antibiotic compounds that inhibit growth of the pathogen in soil. With production of florist azalea, however, such practices generally are not used.

2.2 Pythium Root Rot (*Pythium* spp.)

Geographic occurrence and impact. Roots of many plant species can be infected by species of *Pythium*. Most often *Pythium* spp. are known as seedling damping-off pathogens, but they are root pathogens of many woody plants, including florist azaleas (Ivors 2014). They do not cause immediate death of plants but cause root rot that can debilitate plants to various degrees, depending on the time and extent of infection of the root system. Young rooted azalea cuttings can be killed if enough of the root system is involved. That will depend on when the pathogen begins to infect, relative to the rooting process. If infection occurs after a substantial number of roots have formed, the infections might not be detected, and the plants become infected-but-symptomless until the percentage of infected roots increases. A number of species of *Pythium* can be involved in azalea root rot. Some may be more pathogenic than others (Weiland et al. 2013). *Pythium* spp. might also be secondary invaders to infections by species of *Phytophthora*. The net result and impact of Pythium root infections, therefore, is difficult to determine. If growing conditions in the greenhouse are favorable, *Pythium* infections might go unnoticed until some other stress occurs on the plants. Then the combination of Pythium root rot and stress causes the plants to show symptoms on the foliage, such as leaf chlorosis, small leaves, even dieback of young branches. Examination of the roots and detection of root rot suggest the role that *Pythium* played in the syndrome. Such plants, however, do not make it into the florist trade because they are discarded.

Symptoms/signs. A range of symptoms of Pythium root rot can occur, including failure of cuttings to root, rooting followed by root rot, young plant mortality, stunting of plants at later growth stages, chlorosis of foliage, wilting under water deficiency, loss of vigor, necrotic larger roots, and leaf drop (Linderman and Benson



Fig. 2 Symptoms of Pythium root rot of azalea: *Left photo* showing greater root browning of plant on *left. Right photo* showing aboveground symptoms of smaller plant, smaller leaves, and off-colored foliage, compared to plant on the-*left* (R.G. Linderman © 2017. All Rights Reserved.)

2014) (Fig. 2). Examination of the root ball of affected plants, compared to nonaffected healthy plants, usually shows a greatly reduced size. Often it is difficult to distinguish many of the aboveground symptoms from a nutritional disorder. Examining the root ball is critical. Over-watering or drought episodes may hasten the development of aboveground symptoms, but plants without Pythium root rot generally show no or fewer symptoms.

Biology and epidemiology. Identifying the species of *Pythium* involved in root rot of azalea is extremely difficult, even for the specialist. Ten species have been identified on rhododendrons and azaleas, but those affecting florist azaleas have not. It is possible, even likely, that more than one species can be involved. Further, some species/strains can be more pathogenic than others (Weiland et al. 2013).

There can be many sources of inoculum of *Pythium* species that can invade the florist azalea production system: (1) Used and recycled containers have been shown to harbor *Pythium* species, even after rigorous washing. Baiting residual contaminated medium from propagation flats or larger containers reveals that *Pythium* spp. are present. It generally is impossible to predict just how pathogenic each might be without inoculation experiments. The variability of species or strains within a crop has been demonstrated, however (Weiland et al. 2013). (2) Contaminated media is another potential source of inoculum leading to Pythium root rot. Most growers rely on the soilless medium they buy or mix themselves being pathogen-free. However, *Pythium* has been found in some peat mosses, albeit infrequently. (3) Irrigation water is a likely source of *Pythium* where water is being recycled in the summer months. However, often well water is used in propagation, and it is believed to be pathogen-free. Water in irrigation reservoirs should be treated in order to prevent inoculum from being delivered into production pots. Generally chlorine compounds, such as hypochlorite or chlorine dioxide, or even chlorine gas have been used for that purpose. (4) Insect vectors, such as fungus gnats, have been suggested to carry propagules of *Pythium* from an infected pot to healthy pots. However, there is some

degree of controversy in this regard, in that some studies show that the pathogen does not carry over from the larval to the adult stage that flies. However, there is no doubt that the larvae transmit the pathogen from root to root within a pot.

Pythium root rot often occurs in combination with some environmental stress, such as high soluble salts in the medium, over-watering, under-watering (drought), or poor drainage. Water saturation of the medium can allow the pathogen to produce sporangia and then zoospores spread from root to root. Those conditions also stress plants in a physiological manner that makes them more susceptible, even for cultivars that generally are thought to be somewhat resistant.

Having covered all of the primary sources of *Pythium* spp. of potting medium, used containers, plant propagules, or irrigation water, it is still essential to prevent the introduction of *Pythium* thereafter. *Pythium* spp. can be lurking around, just waiting for some lack of sanitation. So it is important to disinfect all bench surfaces, potting benches, tools, and equipments that will contact the potting mix.

Management. Preventing Pythium root rot should be the primary management strategy. Eliminating the pathogen from the major sources of irrigation water, the growth medium, the containers, and even the cuttings can go a long way to managing this disease. Thereafter, application of preventative chemical fungicides provides some protection against invasion from a number of sources, including greenhouse workers. The chemicals used for *Pythium* control are the same as for *Phytophthora* control, and they generally target only those genera and some other fungus-like microorganisms, but not fungal pathogens. They are preventative chemicals, such as products containing etridiazole, mefenoxam, cyazofamid, fenamidone, and phosphonates. However, some species of *Pythium* are not sensitive to mefenoxam (Aegerter et al. 2002), so rotation or combinations with other chemicals should be practiced. In addition to chemical agents, there are some biological control agents that target *Pythium* spp., such as species of *Trichoderma* and *Streptomyces*.

There are examples of suppressive soils that prevent or limit some root diseases, often created by incorporating organic materials, such as composts, that contain mixtures of microbes with great diversity. Within that microbial population are members with specific capacity to block the development of specific diseases. Some have the capacity to block sporangium development and therefore reduce zoospore release. Some are able to lyse the pathogen mycelium, while others produce other antibiotic compounds that inhibit growth of the pathogen in soil.

2.3 Cylindrocladium Blight and Wilt (*Cylindrocladium scoparium* Morgan)

Geographic occurrence and impact. A serious disease of evergreen, florist azaleas occurred in the USA and in European countries in the mid-1950s; within 10 years it appeared in major proportions in many parts of the USA and other countries. The cause of the disease was *Cylindrocladium scoparium*, a fungus previously known to attack primarily conifer and other tree species in seedling

nurseries. Subsequently, the disease occurred on several other major nursery crops. The diseases varied in symptom expression, including root rot, stem lesions, leaf spots and flower blight, and wilt. In recent years to the present, serious *Cylindrocladium* diseases have occurred on azalea and mini rose, as well as other ericaceous plants, primarily in the early stages of cutting propagation. Subsequent sporulation of the pathogen under greenhouse conditions and spread by water splash or air movement of spores results in devastating incidence and severity on many, often thousands, of plants. During that phase of sporulation and spread of the disease, more plants become infected, but remain symptomless, only to wilt and succumb later. Growers have sustained, and are still sustaining, severe economic losses from these diseases, often in the range of hundreds of thousands of dollars per year per grower.

Symptoms/signs. There are multiple symptoms of *Cylindrocladium* infections of florist azaleas, depending on the production stage and environmental conditions (Linderman and Benson 2014). Leaf spot infections can occur if there is airborne inoculum in the greenhouse (Fig. 3). Often those spots go unnoticed because the infected leaf will abscise (due to the production of ethylene) and be lost on the pot surface among other leaves that had fallen for other reasons. Leaf spots are discrete spots that become necrotic as seen from the upper leaf surface. On the lower surface, however, mainly on red or pink cultivars, the veins radiating out from the spot are red. That symptom is absent or less pronounced on white cultivars. Flowers may also be infected by aerial inoculum, forming necrotic flecks that rapidly coalesce (Fig. 4).

Infected fallen leaves become completely colonized by the pathogen in time as they remain moist. Internally, the pathogen forms microsclerotia in the leaf tissue



Fig. 3 Leaf spots on azalea plant caused by *Cylindrocladium scoparium* (*left*); close-up of spot on cutting during propagation (*right*) (R.G. Linderman © 2017. All Rights Reserved.)



Fig. 4 Flower infections on azalea caused by inoculation with *Cylindrocladium scoparium* (R.G. Linderman © 2017. All Rights Reserved.)

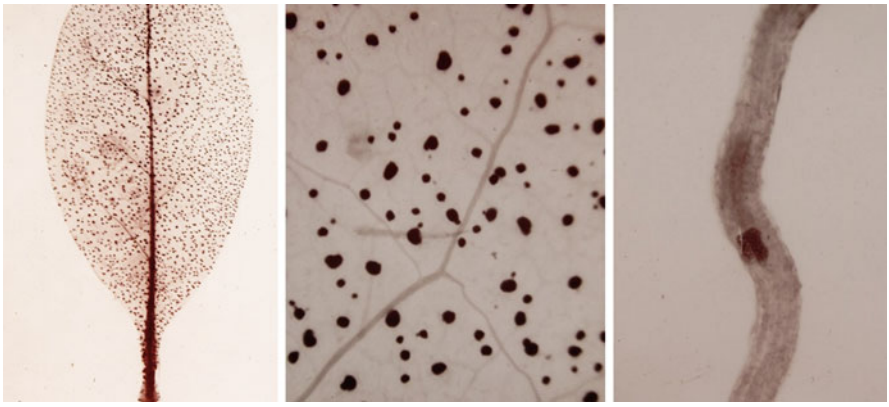


Fig. 5 Microsclerotia of *Cylindrocladium scoparium* formed in infected leaves (*left* and *middle*) or roots (*right*) (R.G. Linderman © 2017. All Rights Reserved.)

that provide it long-term survival. Microsclerotia may also form in infected roots (Fig. 5). Under moist conditions, the sclerotia can generate abundant leaf-surface conidial sporulation that can contribute to the pathogen's dispersal within the greenhouse (Fig. 6). Examination of such leaves, even with a hand lens, will reveal the diagnostic sporulation.

Conidia released from fallen, infected leaves can wash into the medium and initiate root rot on newly formed roots. Infected roots are brown initially and then become necrotic. The extent of roots infected determines when the plant will succumb. Often, infected cuttings will die in pots where four cuttings were stuck

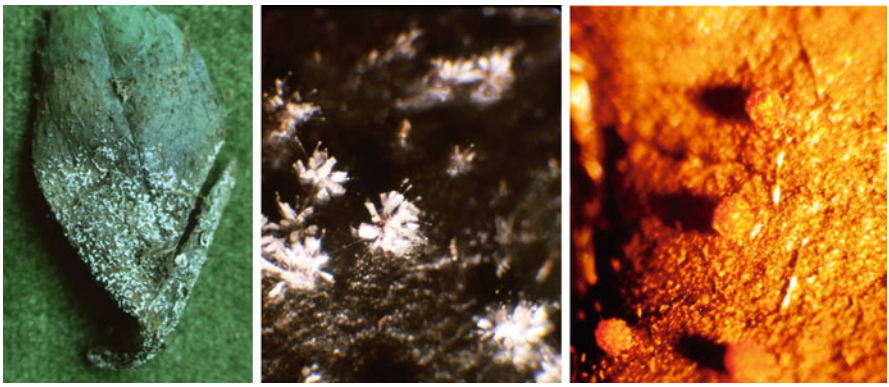


Fig. 6 Photo on the *left* shows sporulation of *Cylindrocladium* spp. on abscised azalea leaf. Photo in the *center* is a close-up of conidial clumps generated from microsclerotia imbedded in infected leaf. Photo on the *right* shows perithecia of *Calonectria kyotensis* (teleomorph) formed on infected azalea leaf (R.G. Linderman © 2017. All Rights Reserved.)



Fig. 7 Wilt symptoms on azalea cuttings (*left*) and older plant (*right*) where one of four cuttings was infected. Note distinctive defoliation from infection in photo on *right* (R.G. Linderman © 2017. All Rights Reserved.)

(Figs. 7 and 8). In many cases, however, the infections do not affect the appearance of the aboveground portions of the plant, mainly because new roots are forming fast enough to maintain the plant in spite of the infected roots. At a later stage, however, mortality may occur. Often, three or four rooted cuttings are transplanted into a single pot for growing on, and only one or two of them die (Fig. 9). That is a sign that the infection began at an earlier stage in propagation. Historically, however, when only one plant was transplanted to a larger pot, that plant eventually wilted due to the



Fig. 8 Root systems of azalea plants from one pot showing effects of root rot caused by *Cylindrocladium* (R.G. Linderman © 2017. All Rights Reserved.)



Fig. 9 Wilting of one azalea plant of four in the pot due to earlier infection by *Cylindrocladium* (*left photo*). Older plant wilting and death due to earlier root infection (*right photo*) (R.G. Linderman © 2017. All Rights Reserved.)

initial root infections on the cuttings. At that time, cutting into the lower stem would usually reveal an internal, brownish canker. That symptom alone, however, would not distinguish *Cylindrocladium* as the cause from cankers caused by *Phytophthora*, for example. Additional diagnostic tests need to be done to confirm Cylindrocladium blight.

Biology and epidemiology. The main causal agent is *Cylindrocladium scoparium* Morgan, but other species with a perfect stage (teleomorph), such as *Calonectria kyotensis* and *Calonecria theae*, were also shown to be pathogenic (Alfieri et al. 1972). Those species with a *Calonectria* stage were shown to eject ascospores aerially when relative humidity declined (Linderman 1974a).

In nearly all cases, cutting and root rot of azaleas caused by species of *Cylindrocladium* begins during cutting propagation and spreads thereafter, depending on the greenhouse production system. It was demonstrated experimentally (Linderman 1973, 1974b) that the pathogen produced microsclerotia in infected leaves that dropped from plants with leaf spot infections. Those infections were often not noticed, and leaf abscission occurred as a result of ethylene production (Axelrood-McCarthy and Linderman 1981). The microsclerotia imbedded in the fallen leaves generated conidiophores on the surface, and conidia then splashed to other cuttings, causing root rot (Linderman 1974a). Some cutting roots became infected, but the plant remained symptomless. In the absence of stress under ideal growing conditions, liner plants moved through the production stages were sold and shipped only to die at some later time.

Management. Aerially dispersed conidia that landed on plants from which cuttings would be taken can be contaminants, not yet causing infections. Therefore, chemical sanitization by dipping cuttings in solutions of fungicides or chlorine compounds, killing the contaminating conidia, is an effective management practice (Linderman, unpublished). Failure to sanitize cuttings has led to disastrous consequences from this disease.

The source of airborne conidia in a greenhouse may be unknown, but infected plants with sporulation at the lower stem could be one source (Linderman, personal observation). Fallen, infected leaves on or below the production benches also can harbor the pathogen and become a source of dispersed conidia. Removal of such debris and flaming the areas under the benches and chemically sanitizing the benches is essential to removing sources of the pathogen inoculum.

Assessment of the presence of *Cylindrocladium* spp. in soil or soilless media might also be needed, especially if the propagation medium is to be reused. Inserting azalea leaves into the medium and incubating for a week was shown to be effective in detecting the pathogen. The leaves were selective in being only infected by *Cylindrocladium* spp. (Linderman 1972). The method was also effective in isolating the pathogen from the stem by inserting a leaf into the cut end. If *Cylindrocladium*, or any other soilborne fungal pathogen, was detected, the medium must be sanitized with heat or a fumigant like Metam sodium prior to reuse (Linderman and Davis 2008b).

Application of fungicides can also provide protection of plants exposed to aerial inoculum. Frequency of application is also critical due to the rate of sporulation of the pathogen as well as development of new, unprotected leaf surface from expansion. Fungicides containing thiophanate methyl have been shown to be effective.

2.4 Leaf and Flower Gall (*Exobasidium* spp.)

Geographic occurrence and impact. Leaf and flower gall disease of azaleas occurs widely throughout the world, mainly on landscape plants, but it can occur on florist azaleas under moist conditions. In the United States, the causal agent is generally considered to be *Exobasidium vaccinii* (Fuckel) Woronin. In China, Japan, and Korea, however, *E. japonicum* Shirai is considered to be the pathogen. *Exobasidium rhododendri* (Fuckel) C. Cramer has been reported on both azalea and rhododendron worldwide. The origin of the disease is not known, but likely first occurred on rhododendrons in their centers of origin. If enough galls appear on a plant, there can be some loss of vigor of the plant. Such severe infections rarely occur in commercial greenhouse production, however. The impact of the disease comes from the time and cost of its management, involving labor and preventative chemical applications.

Symptoms/signs. Leaf gall first appears as light green swellings on the edges and undersides of leaves. Both expanding leaves and flower buds and young, developing shoots are susceptible to infection. The infected tissue expands to form gall-like structures that generally involve the entire leaf (Fig. 10). As the galls mature, they turn white from surface sporulation of the fungal pathogen. The galls can be small or large and bladder shaped. The fungus causes the plant cells to divide and enlarge, making the tissue soft and succulent. Once galls turn white from sporulation, the pathogen can spread very rapidly by water splash or air movement to other plants, initiating new, secondary infections on leaves and even flowers. After sporulation occurs, the leaf galls slowly turn brown, shrink, and become quite hard.

Biology and epidemiology. Spores of the pathogen apparently overwinter in bud scales. As buds break, the spores germinate and infect the young bud tissue as it expands. Humidity level in the greenhouse is a critical factor in the epidemiology of the leaf gall disease. High relative humidity and free water on the leaves and flowers



Fig. 10 Azalea leaf gall caused by *Exobasidium vaccinii*. *Left*: young galls; *Middle*: pathogen sporulation on gall; *Right*: older, necrotic galls (R. Rosetta © 2017. All Rights Reserved.)

is required for spore germination and initiation of new infections, so irrigation scheduling is very important. Overhead watering must be done early in the day so the foliage is not wet through the night. Because of the variation in relative humidity, often the lower leaves, where humidity is higher, are more susceptible than upper leaves where air circulation is better. Once infection has begun, the fungus grows in the intercellular spaces of the surface tissue, producing haustoria that penetrate cells to derive its nutrition. Eventually, often within a few days, the pathogen sporulates on the gall surface, causing it to turn white. Spores produced on a gall can be released and spread to other expanding buds, causing secondary infections. Some of the spores settle into protected sites in forming buds and are in position to initiate new infections.

Management. The main strategy for managing azalea leaf gall is to diligently scout production plants for the first signs of gall development. Small galls must be removed before they turn white with sporulation. Prevention of galls also generally involves application of fungicides as buds break and new foliage appears. Plants should be sprayed before and after removing galls with chemicals such as mancozeb-containing fungicides, copper-containing materials, quaternary ammonium materials, and even oxidizing agents such as hydrogen peroxide. A good sticker may be needed to help retain the applied chemical. Application of any of these materials is strictly preventative, not curative.

2.5 Rhizoctonia Root Rot and Web Blight (*Rhizoctonia* spp.)

Geographic occurrence and impact. Rhizoctonia root rot and subsequent blighted foliage occurs occasionally on florist azaleas in the United States (Copes and Benson 2014). The disease also has been reported in Australia. This disease can occur on many different woody ornamental plants, including rhododendrons. Most often, the disease occurs during cutting propagation where moist, warm conditions prevail. Economic losses result from the loss of affected rooted cuttings, but some infections aren't expressed until later when canopy growth is affected and some dieback/blight occurs.

Symptoms/signs. When Rhizoctonia root rot occurs during propagation of cuttings, the affected cuttings fail to root or the new roots become infected, causing the entire cutting to collapse (Fig. 11). Often the infection site will expand and develop, spreading to adjacent cuttings. Webbing of aerial hyphae occurs, looking like a spider web moving from plant to plant. The pathogen moves both in the rooting medium and from leaf to leaf above the medium surface.

Biology and epidemiology. Some propagators root cuttings under a reemay cover in order to retain heat and to reduce the frequency of misting from above. The high humidity under the reemay allows the fungus to move from one infected cutting to



Fig. 11 Hyphal webbing site of azalea cuttings in propagation caused by *Rhizoctonia solani*. Note discrete spots on leaf in *lower right* (R.G. Linderman © 2017. All Rights Reserved.)

adjacent cuttings, often leading to a patch of infected cuttings. The occurrence of a web-blight pattern where the fungus becomes aerial and infects the lower leaves results in a distinct leaf spot at the infection site. The primary causal agent, *Rhizoctonia solani* J. G. Kuhn, is a nonsporulating Deuteromycete, so how the pathogen invades the propagation system is unknown. The possible sources could be the medium, reused rooting flats, or some cuttings becoming contaminated during cutting collection. The incidence of this disease is very infrequent and spread is by vegetative means. Spread may also occur in particulate material moved by air currents.

Management. The infrequent occurrence of *Rhizoctonia* root rot and cutting death supports the idea that no active management is required, other than removal of affected cuttings at an infection site, including adjacent cuttings that usually show no signs of infection. Often that means removal of the entire affected plug flat. Addressing the possible sources of the pathogen, however, is in order. If propagation flats are being reused, then their sanitization with aerated steam (Linderman and Davis 2008b) is required to kill any carryover of the pathogen from a previous crop, even crops other than azalea. If the rooting medium is suspected as the source, then a decision would be needed as to steam treatment that is generally not a course of action unless other diseases have occurred that would also be curbed by medium pasteurization. Since *R. solani* can colonize organic components of the medium as a saprophyte, rigid sanitation in the propagation area is called for to prevent that from happening. Affected areas of the propagation area should also be treated chemically to avoid spread and carryover. Chemicals that specifically target Rhizoctonia pathogens should be used; broader spectrum chemicals might be used if other causal agents that can cause similar

symptoms are known to occur, such as species of *Pythium* or *Phytophthora*. Since sporulation is not involved in disease initiation or spread, cuttings are less likely to be contaminated, so their sanitation generally is not necessary.

3 Genetic Abnormalities

3.1 Witches' Broom

Geographic occurrence and impact. Some cultivars of florist azaleas have a tendency to develop a cluster of small leaved branches in a tight configuration characterized as a witches' broom. The occurrence of these structures has little impact on the growth of the plant, and plants that exhibit them are generally discarded as being offtype or the affected branch simply pruned off (Linderman 2014).

Symptoms/signs. The witches' broom branches occur generally on one location on the plant, often near the base of the plant. The tight branching and greatly reduced leaf size distinguish it from the rest of the plant (Fig. 12).

Biology and epidemiology. Evergreen azaleas and rhododendrons occasionally exhibit abnormal, miniaturized vegetative growth in the form of a witch's broom. Researchers generally agree that the condition is the result of a genetic change that occurred, and that no biotic causal agent is involved. Therefore, there is no risk of transmission from affected plants to adjacent plants.

Management. Since no biotic agent is involved in the development of witches' broom, no management is required other than to either prune off the broom, or simply discard the plant as being off-type.



Fig. 12 Witches' broom on azalea (R.G. Linderman © 2017. All Rights Reserved.)

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Diseases of Begonia

30

Cristina Rosa and Gary W. Moorman

Abstract

Begonias are susceptible to a wide variety of fungi, bacteria, and viruses, as well as nematodes and abiotic diseases. The systemic nature of some of the pathogens makes it likely that the diseases they cause can be found wherever vegetatively propagated begonias are shipped. Management of these pathogens is paramount for specialty propagators while growers purchasing plants must inspect incoming plants for symptoms and understand the biology of the pathogens involved in order to manage them effectively.

Keywords

Fusarium wilt • Botrytis blight • Powdery mildew • Pythium root rot • Bacterial spot • Viruses • Foliar nematodes • Abiotic diseases

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© Springer International Publishing AG 2018

R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_30

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1 Introduction

Begonias are a very diverse and complex group of plants. The name "Begonia" was first used by Charles Plumier, a Franciscan monk and botanist, in 1703. Linnaeus described all the species known to him in 1753, and by the end of the 1700s, begonias were being cultivated widely in Europe. By the early 1800s, hybrids were being developed. Based on their dominant horticulture characteristics, begonias can be grouped in categories including cane-like, shrub-like, semperflorens, rhizotamous, rex cultorum, tuberous, and trailing-scandent (Thompson and Thompson 1981).

2 Fungal and Fungus-Like Diseases

2.1 Fusarium Wilt (*Fusarium foetens*)

Geographic occurrence and impact. This disease has been found in Europe, Japan, and North America causing wilt and sometimes stem rot (Elmer and Vossbrinck 2004; Schroers et al. 2004; Sekine et al. 2008; Tian et al. 2012; Tschope et al. 2007; Van der Gaag and Raak 2010).

Symptoms/signs. Leaves become dull green and the leaf veins may yellow. The leaves wilt and die. The vascular tissue of infected plants turns brown. A basal stem rot may develop as the disease process proceeds (Fig. 1). Large numbers of pale orange spores develop on the dying tissue, particularly if the tissue is placed on a wet surface in a container and incubated.

Biology and epidemiology. *Fusarium foetens* is related to the *Fusarium oxysporum* complex that contains many form species responsible for vascular wilts of various plants. It is thought that the pathogen was introduced to Europe on plant material brought there for breeding (Schroers et al. 2004), indicating that the pathogen may be spread long distances associated with plant shipments.



Fig. 1 Fusarium wilt on begonia (Wade Elmer © 2017. All Rights Reserved.)

Management. Infected plants should be destroyed. The various types and cultivars of begonia differ in relative susceptibility (Brand and Wienberg 2005; Tian et al. 2012; Tian and Zheng 2012). The biological control agents *Bacillus subtilis*, *Streptomyces griseoviridis*, *Gliocladium catenulatum*, *Streptomyces lydicus*, and *Trichoderma harzianum* have been reported to provide protection of begonias against *Fusarium foetens* in greenhouse experiments (Tian and Zheng 2013). To reduce the presence of the fungus in production systems, chlorine bleach, hydrogen peroxide, and quaternary ammonia sanitizers have been found effective (Elmer 2008).

2.2 Gray Mold (Botrytis Blight; *Botrytis cinerea* Pers.: Fr.)

Geographic occurrence and impact. This disease is very common and occurs wherever plants are grown if the environmental conditions are conducive to its development. The pathogen, *Botrytis cinerea*, can infect almost any plant in a greenhouse including begonias.

Symptoms/signs. Cuttings rot at their base. Tan spots develop on leaves. Established plants rot at the crown or a dry shriveled area develops along a branch (Tompkins 1950). Abundant, dusty, gray masses of spores form on infected tissue if the humidity is high or if infected tissue is placed in a container with moisture.

Biology and epidemiology. Seedlings and all above ground parts of mature plants are susceptible. The fungus can infect intact tissue or through wounds. Infection occurs most readily when the fungus has a food base from which it attacks, such as fading flowers or senescent leaves that have fallen onto healthy leaves. High relative humidity, wetness on the plant, and temperatures between 18 °C and 25 °C greatly favor *Botrytis* infection (Jarvis 1980). The spores are readily spread by air currents (Hausbeck and Pennypacker 1991).

Management. Maintain low humidity within the crop canopy by spacing plants well and venting in a greenhouse setting to improve air circulation. Remove dead and dying flowers and leaves from the area around the plants, from the potting soil surface, and from the plant. Remove this debris from the greenhouse promptly or put it in a closed waste container. Avoid unnecessarily damaging plants. Apply a fungicide preventively but note that the exclusive use of one class of chemical, particularly systemic chemicals, can result in the selection of fungicide resistance to the chemical class. Resistance to benzimidazole fungicides is very widespread in *Botrytis* populations Do not rely on only one chemical class (Elad et al. 1992; Gullino and Garibaldi 1987; Hausbeck and Moorman 1996; Moorman and Lease 1992).

2.3 Powdery Mildew (*Oidium begoniae*, Asexual Stage)

Geographic occurrence and impact. This disease is widespread, probably found wherever begonias are grown, and it significantly damages the aesthetic quality of affected plants. The obligate plant pathogenic organism exhibits dimorphism. That is, the asexual phase of growth looks significantly different from the sexual stage (Bélanger et al. 2002). The asexually reproducing phase of the pathogen is named *Oidium begoniae*. The sexual stage is thought to be a member of the genus *Microsphaera* because the conidia of the asexual stage are not formed in chains (Quinn and Powell 1981), but the structures formed as a result of sexual reproduction (chasmothecia; formerly cleistothecium) are rarely found associated with begonia. It is known that *Sphaerotheca fuliginea* (Powell 1985) and *Golovinomyces cichoracearum* (formerly *Erysiphe cichoracearum*) (Sammons et al. 1982) may also cause powdery mildew in begonia.

Symptoms/signs. White, mealy fungal growth develops on leaves, flowers, and stems (Fig. 2). Tissue beneath the fungus may die.



Fig. 2 Powdery mildew on begonia (The Pennsylvania State University Department of Plant Pathology and Environmental Microbiology © 2017. All Rights Reserved.)

Biology and epidemiology. Disease develops most rapidly when temperatures are 20–21 °C and is greatly inhibited if temperatures are above 28 °C (Quinn and Powell 1982). Unlike essentially all other fungal plant pathogens, wetness has little influence on disease development of this powdery mildew. In fact, it flourishes when leaf surfaces are relatively dry. Severe powdery mildew outbreaks should be anticipated when cool, damp night conditions alternate with warm, dry days.

Management. Examine plants carefully and frequently to detect the onset of disease. Maintain a fungicide program to protect plants. It is known that horticultural oil can kill powdery mildew fungi when it comes in direct contact with the pathogens. Once the oil dries on the plant surface, it has no activity against powdery mildew. That is, it has no residual activity. Applied under the wrong conditions or when the plant is wilting, oils are phytotoxic.

2.4 Pythium Root Rot (*Pythium* sp.)

Geographic occurrence and impact. Several species of *Pythium* have been reported to cause root rot and lower stem rot of begonia around the world including *P. ultimum* (*Globisporangium ultimum*) and *P. splendens* (*Globisporangium splendens*) (Farr et al. 1989). *Phytopythium helicoides* (formerly, *Pythium helicoides*) has also been found on begonias (Yang et al. 2013).

Symptoms/signs. Infected seedlings die. Shiny tan, water-soaked areas develop on the stems and petioles of established plants at or just above the soil line as plants collapse and die (Middleton 1942). Under very high humidity conditions, the pathogen may be seen as a fluffy, white mass near the soil line but this is unusual to see.

Biology and epidemiology. *Pythium* species are often found in field soil, sand taken from streams and rivers, and can be found in pond and lake sediments, and dead roots of previous crops (Ivors and Moorman 2014). Excessively wet potting mixes greatly favor the development of root rot.

Management. Plant in pasteurized, not sterilized, potting media. If the potting mix has been sterilized (killing all living organisms in it) by heating it to too high a temperature or heating it too long, a biological vacuum will have been created. If *Pythium* then contaminates the sterile potting mix, it can cause very severe crop losses, because it has no competition and no natural inhibition by other microbes. Keep hose ends off the ground in order to avoid picking up *Pythium*-contaminated soil and then spraying it onto the crop. Use a well-drained potting mix and do not over-water the plants, particularly if plants are not utilizing a great deal of

water because of prevailing weather conditions. Apply a fungicide or biological control agent at planting for the best protection. If the crop is grown for several months, any chemical or biological control agent will have to be applied repeatedly.

Listed below are additional fungus or fungus-like organisms which have been associated with *Begonia* (Farr et al. 1989). For the most up-to-date listing, search http://nt.ars-grin.gov/fungaldatabases/fungushost/fungushost.cfm:

Aecidium begoniae
Agrobacterium tumefaciens
Alternaria sp.
Alternaria tenuis (*Alternaria alternata*)
Armillaria mellea
Bartalinia begoniae
Botryosphaeria sp.
Botryotinia fuckeliana (*Botrytis cinerea*)
Ceratobasidium sp.
Cercospora begoniae
Cercospora sigesbeckiae
Choanephora cucurbitarum
Cladosporium inconspicuum
Cladosporium sphaerospermum
Coleosporium begoniae
Colletotrichum capsici (*Colletotrichum truncatum*)
Colletotrichum gloeosporioides
Colletotrichum sp.
Corticium solani (*Rhizoctonia solani*)
Corynespora cassiicola
Curvularia inaequalis
Erysiphe begoniae
Erysiphe begoniicola
Erysiphe communis (*Erysiphe pisi* var. *pisi*)
Erysiphe orontii (*Golovinomyces orontii*)
Erysiphe polygoni
Erysiphe polyphaga (*Golovinomyces orontii*)
Fusarium begoniae
Fusarium equiseti
Fusarium roseum
Fusarium solani
Gloeosporium begoniae
Glomerella cingulata (*Colletotrichum gloeosporioides*)
Golovinomyces orontii
Helminthosporium sp.
Lasiodiplodia theobromae

Macrophoma sp.
Macrophomina phaseoli (*Macrophomina phaseolina*)
Marssonina mali
Meliola begoniae
Microsphaera begoniae (*Erysiphe begoniicola*)
Moniliopsis aderholdii (*Rhizoctonia solani*)
Mycena citricolor
Myrothecium roridum
Oidium begoniae (*Golovinomyces orontii*)
Oidium ellipticum
Penicillium bacillisporum (*Talaromyces bacillisporus*)
Pestalotiopsis sp.
Phoma sorghina (*Epicoccum sorghi*)
Phoma sp.
Phomopsis sp.
Phyllachora begoniae
Phyllosticta begoniae
Phyllosticta sp.
Phyllostictina sp.
Phytophthora cactorum
Phytophthora cryptogea
Phytophthora cryptogea f. sp. *begoniae*
Phytophthora nicotianae var. *nicotianae* (*Phytophthora nicotianae*)
Phytophthora niederhauserii
Phytophthora parasitica (*Phytophthora nicotianae*)
Pseudoidium sp.
Pucciniastrum boehmeriae
Pythium aphanidermatum
Pythium debaryanum (*Globisporangium debaryanum*)
Pythium intermedium (*Globisporangium intermedium*)
Pythium irregulare (*Globisporangium irregulare*)
Pythium vexans (*Phytopythium vexans*)
Rhizoctonia solani
Sclerotinia sclerotiorum
Sclerotium rolfsii (*Athelia rolfsii*)
Septonema sp.
Septoria begoniae
Shrungabeeja begoniae
Sphaeropsis begoniicola
Stemphylium sp.
Thanatephorus cucumeris (*Rhizoctonia solani*)
Thielaviopsis basicola
Verticillium albo-atrum
Verticillium dahliae

3 Bacterial and Phytoplasma Diseases

Where possible, the scientific names used are those accepted by the International Society of Plant Pathology Committee on the Taxonomy of Plant Pathogenic Bacteria (Bull et al. 2010).

3.1 Bacterial Spot (Bacterial Blight; *Xanthomonas campestris* pv. *begoniae* (Takimoto) Dye)

Geographic occurrence and impact. This disease, one of the most damaging to begonias, was first described in the 1920s (Powell 1985) (Fig. 3). It causes a leaf spot or blighting of some cultivars but can be systemic in Rieger elatior begonias (Harri et al. 1977). In some cases such as a slight infection, the disease is easily overlooked. Thus, the pathogen can be inadvertently shipped with plants if infection goes undetected.

Symptoms/signs. Water-soaked circular lesions surrounded by yellow halos develop on leaves. In wax begonias (semperflorens types), the spots may be very small (1 mm diameter) (Daughtrey et al. 1995). Plants slowly die one leaf at a time. If infection begins at the leaf margin, wedge-shaped brown dead areas develop with the wide part of the wedge at the margin and the point of the wedge pointing toward the plant stem. When infected tissue is cut, placed in a clear drop of water on a microscope slide, and observed with a bright field microscope with the iris diaphragm mostly closed, bacteria can be seen to stream from the tissue. When Rieger begonias are infected systemically, they wilt and die.

Biology and epidemiology. The bacteria can enter injured roots or leaves and become systemic in some cultivars of Rieger begonias. Eventually wilting and death of the plant occurs. In some other begonias such as Rex, the pathogen remains

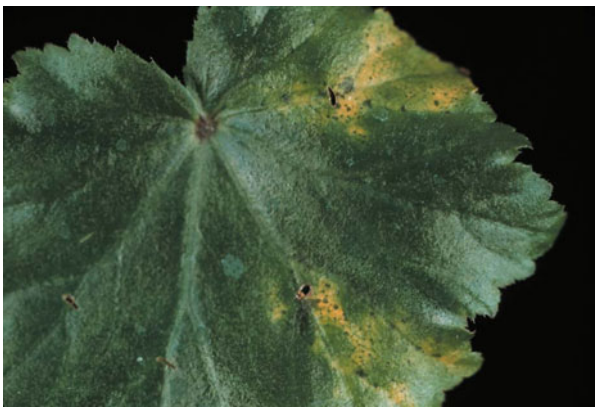


Fig. 3 Bacterial blight on begonia (The Pennsylvania State University Department of Plant Pathology and Environmental Microbiology © 2017. All Rights Reserved.)

localized in leaf spots (Jodon and Nichols 1974). The pathogen can survive for extended periods of time if the leaf debris is dry. The pathogen can be spread from plant to plant on workers' hands, clothing, and tools and has been shown to be disseminated in recycled irrigation water (Atmatjidou et al. 1991).

Management. Purchase plants free of the pathogen. Discard infected plants, particularly Rieger-types which are systemically infected. Remove infected leaves from Rex and tuberous types because they are not systemically infected. Irrigate plants in a manner that keeps water off the foliage. Do not propagate from infected plants. Remove the debris from infected plants from the greenhouse or place it in a closed container. Workers should wash their hands thoroughly periodically when performing tasks on the plants and should disinfect any tools that come in contact with the plants.

Additional Diseases with Bacterial Pathogens

Crown gall (*Rhizobium tumefaciens*, formerly *Agrobacterium tumefaciens*) (Powell 1985)

Bacterial fasciation (*Rhodococcus fascians*) (Powell 1985)

Soft rot (*Pectobacterium carotovorum*, formerly, *Erwinia carotovora*) (Powell 1985)

Additional Diseases with Phytoplasma Pathogens

Phytoplasmas induce symptoms such as phyllody, shoot proliferation, small leaves and flowers, and stunted plant growth. These bacteria are vectored primarily by leafhoppers, planthoppers, and psyllids.

"*Candidatus* Phytoplasma asteris"

A phytoplasma in the 16SrI group was reported to infect begonia (Powell 1985). Shoot proliferation.

A phytoplasma belonging to the 16SrIII group was associated with shoot proliferation in begonia in Brazil in 2006 (Ribeiro et al. 2006).

4 Viral Diseases

Viruses of begonia are associated with mild to severe mosaic and mottling, ringspots, leaf malformation, stem necrosis, chlorosis, and stunting. Generally viruses can be controlled by using clean propagative material, by discarding diseased plants and isolating surrounding plants and controlling vector populations for viruses that are vector transmitted. Clean potting soil needs to be used to control viruses whose vectors are soil inhabitants. The presence of viruses can be diagnosed by use of molecular tests such as reverse transcriptase PCR or immune-based assays such as ELISA or rapid immunostrips, or by indicator hosts.

The EPPO Panel on "Certification of Ornamentals" developed procedures for the production of healthy carnation, pelargonium, lily, narcissus, chrysanthemum, tulip, crocus, iris, begonia, impatiens, rose, freesia, hyacinth, kalanchoe, and petunia.

In the USA, certification programs are managed by the major commercial propagators. Pelargonium and other major flower crops are tested for the presence of the most prevalent viruses either by in house plant pathologists or by testing services contracted with by the propagators. In the USA, phytosanitary certificates are required for intra- and interstate plant movement. Data on pathogens of high consequence, including viruses, are recorded by the National Plant Diagnostic Network.

Where available, information was taken from Adams and Antoniw (2006).

4.1 Clover Yellow Mosaic Potexvirus (ClYMV) Described by Johnson (1942)

Geographic occurrence and impact. It is common in the Western USA and Canada (Agrawal et al. 1962; Pratt 1961). It has occasionally being imported to the UK, but it has not established in the EPPO region.

Symptoms. ClYMV induces color variations and poor vigor.

Biology and epidemiology. ClYMV is in the family *Alphaflexiviridae*. ClYMV is transmitted by infected sap but no vector is known. Reports indicate that it is also seed transmitted (Hampton 1963). Clover yellow mosaic virus is serologically distantly related to *White Clover mosaic virus*.

Management. Use of healthy propagative material and sanitation is recommended.

4.2 Cucumber Mosaic Virus (CMV)

Geographic occurrence and impact. CMV is found worldwide, has an extremely broad host range, and can infect species in more than 100 plant families (Zitter and Murphy 2009).

Symptoms/signs. Symptoms observed on infected begonia are mosaic, leaf deformation and curling, and vein clearing.

Biology and epidemiology. CMV (Jacquemond 2012; Palukaitis and García-Arenal 2003) belongs to the family *Bromoviridae*. CMV is a ss + RNA virus with a tripartite genome, is aphid transmitted in a noncirculative (nonpersistent) manner (Hoggan 1933; Simons 1955; Watson and Roberts 1939), and can be seed transmitted in some plant species (Neergaard 1977).

Management. Control for this virus is particularly difficult, since CMV is a common virus on many plant Families and it can be transmitted by multiple aphid species. Because aphids transmit CMV readily during probing, aphid control is

partially efficacious at reducing the spread of the virus (Zitter and Murphy 2009). Mineral oil has been proposed to delay viral symptoms in different crops (Simons and Zitter 1980).

4.3 Impatiens Necrotic Spot Virus (INSV) and Tomato Spotted Wilt Virus (TSWV)

Geographic occurrence and impact. INSV and TSWV are prevalent in many plant families and species, making their control particularly challenging. INSV can infect around 800 plant species and TSWV more than 1,000.

Symptoms/signs. Symptoms associated with INSV and TSWV are mosaic, mottling, stem necrosis, ringspots on leaves, and leaf deformation (Fig. 4).

Biology and epidemiology. INSV (Law and Moyer 1990) and TSWV (Samuel et al. 1930) are in the Family *Bunyaviridae*. These ssRNA viruses have tripartite genomes, with negative or ambisense orientation. INSV and TSWV are transmitted by thrips in a circulative and propagative manner (Ullman et al. 1993; Wijkamp et al. 1993); thus, they can replicate in their plants as well as in their insect hosts.

Management. One of the best ways to manage these viruses is to control thrips numbers, since thrips need a relatively long acquisition and transmission time, and since the viruses encounter a latent period in the vector prior to becoming transmissible. For instance, in the Netherlands, where INSV and TSWV have been reported on begonia, the incidence of disease has decreased significantly thanks to the implementation of a rigorous thrips management program. The Netherlands has also established a certification program for many ornamental plants, including begonia. To complicate matters, many thrips are insecticide resistant (Brødsgaard 1994; Zhao et al. 1995); thus, care needs to be taken to rotate different classes



Fig. 4 Impatiens necrotic spot virus on begonia (The Pennsylvania State University Department of Plant Pathology and Environmental Microbiology © 2017. All Rights Reserved.)

of insecticides. Weed management is also necessary (Bond et al. 1983; Cho et al. 1986; Kobatake et al. 1984), since weeds can serve as reservoirs for tospoviruses and thrips.

4.4 Tobacco Mosaic Virus (TMV)

Geographic occurrence and impact. TMV can infect a variety of plants in 30 Families worldwide (Shew and Lucas 1991).

Symptoms/signs. Symptoms on begonia are reported to be yellowing, necrosis, mosaic, leaf distortion, and plant stunting.

Biology and epidemiology. TMV belong to the *Virgaviridae* family. The virus has a ss + RNA genome. Symptoms attributed to TMV are often caused by other Tobamoviruses, and many *Tobamovirus* species were once classified as strains of TMV. TMV is occasionally transmitted by chewing insects, but most commonly it is mechanically spread (Harris and Bradley 1973; Lojek and Orlob 1969), in fact TMV virions are extremely stable. TMV can persist in the soil, probably on plant debris, and can infect roots. It can penetrate wounded embryos from infected seed coats (Broadbent 1965). TMV is a special concern in greenhouses, where it can be very hard to eradicate (Broadbent and Fletcher 1963).

Management. Sanitation is the best TMV control method.

4.5 Tobacco Necrosis Necrovirus (TNV) and Carnation Mottle Carmovirus (CarMV)

Geographic occurrence and impact. TNV (Price 1940) can experimentally infect 88 species in 37 plant families and CarMV can infect members in more than 9 families of plants worldwide.

Symptoms/signs. On most hosts TNV causes necrotic lesions and rarely systemic symptoms. TNV can support coinfection with satellite viruses. Carnation mottle virus can infect *Begonia elatior* and *Begonia x cheimantha* where it is associated with symptoms of vein clearing, leaf curling, and flower breaking.

Biology and epidemiology. These viruses are in the Family *Tombusviridae*. Members of this family have a ss + RNA monopartite genome. TNV (described first by Smith and Bald 1935 and more recently by Fraenkel-Conrat 1988) is transmitted by the zoospores of the fungus-like microorganism *Olpidium brassicae* (Kassanis and MacFarlane 1964; Teakle 1962; Teakle and Gold 1963), is not transmitted by seed or by pollen, and can be transmitted mechanically.

CarMV is not known to be transmitted by a vector or by seed, but it is probably transmitted by plant to plant contact and mechanically during cultural practices. The

virus is also stable in water and can be transmitted through irrigation. An unconfirmed report from India indicates that aphids could be vectors of this virus.

Management. To control TNV, care should be taken in using clean irrigation water and potting soil, and in avoiding diseased propagative material. Control of CarMV consists of using certified virus-free material, strict sanitation, and care in handling infected plants.

4.6 Tobacco Ringspot Virus (TRSV) and Arabis Mosaic Nepovirus (ArMV)

Geographic occurrence and impact. ArMV (Smith and Markham 1944) has been detected in 13 States in the USA where it has been the object of quarantine, but it is not widespread or established outside Europe. ArMV has a wide host range. TRSV (Fromme et al. 1927) geographic distribution is mainly in northern USA and China, but it has also been found in Europe and Australia. More than 17 plant families are susceptible to TRSV (Price 1940).

Symptoms/signs. Begonia yellow spot, caused by TRSV, shows symptoms of chlorotic local lesions and yellow mottle patterns, sometimes resembling natural variegation.

Biology and epidemiology. ArMV and TRSV belong to the family *Secoviridae*. Their genomes consist of two linear ss + RNA segments. ARMV is nematode and seed transmitted and can be transmitted by *Cuscuta* sp. Nematodes lose the virus during molting and do not transmit it to their progeny. TRSV is transmitted by nematodes (McGuire 1964), pollen, and seeds as well as by insects (Dunleavy 1957; Komuro and Iwaki 1968; Messieha 1969; Schuster 1963) and mites (Thomas 1969) in a nonspecific manner. TRSV does not replicate in its nematode vector, is lost during molting, and is not transmitted to the nematode progeny.

Management. TRSV is controlled by using clean potting soil, discarding infected propagative material, and by using clean seed.

4.7 Broad Bean Wilt Virus (BBWV) Described by Stubbs (1947)

Geographic occurrence and impact. The virus has been reported worldwide (Lisa and Boccardo 1996). The virus was reported in the USA in New York state, South Carolina and Minnesota in the early 1980s. BBWV was subdivided in two species, BBWV-1 and BBWV-2, in 2000. The two species are serologically distinct, with BBWV-1 more prevalent in Europe, while BBWV-2 is more prevalent in North America, Asia, and Australia. BBWV has a broad host range (Edwardson and Christie 1991).

Symptoms/signs. Symptoms associated with BBWV on begonia are leaf mottling, faint ringspots, color-break in flowers, and generalized stunting. Symptoms tend to be more severe in winter.

Biology and epidemiology. As ArMV and TRSV, BBWV is in the family *Secoviridae*, and its genome consists of two ss + RNA segments. In contrast to ArMV and TRSV, BBWV is aphid transmitted in a noncirculative (nonpersistent) manner (Lisa and Boccardo 1996).

Management. Control of BBWV is difficult because its host range is broad and it is transmitted in a nonpersistent manner; thus, exclusion of aphids is advised, as well as of weeds that serve as virus reservoirs.

4.8 Zucchini Yellow Mosaic Virus (ZYMV) Described by Lisa et al. (1981)

Geographic occurrence and impact. The virus was detected on begonia in Taipei in 2008. As for CMV, another nonpersistently aphid transmitted virus, several plant families are susceptible to ZYMV and the virus is widely distributed, making its control difficult.

Symptoms. Symptoms in begonia consist of faint ringspots on leaves at the early stage of infection; the spots become chlorotic and coalesce during disease progression.

Biology and epidemiology. ZYMV is a potyvirus in the family *Potyviridae*. Its genome consists of a ss + RNA filament and is monopartite. The virus is aphid transmitted in a noncirculative (nonpersistent) manner.

Management. While the use of insecticides is not recommended to control non-persistent viruses and not many experiments have been reported on control of ZYMV in begonia, strategies used to control this virus on other crops would be probably appropriate. These strategies are: the use of resistant material, the use of clean propagative material and adoption of good hygiene practices, the use of mineral oil, and the exclusion of aphids from greenhouses.

5 Nematode Diseases

5.1 Foliar Nematode (*Aphelenchoides fragariae*)

Geographic occurrence and impact. *Aphelenchoides* affects a large number of ornamental plants, including begonia. In some cases, the nematode invades the

vascular tissue and the begonia remains mostly symptomless. Thus, the nematode can be inadvertently shipped from place to place in infected plants. Severe damage can occur in begonias.

Symptoms/signs. Plants may be stunted. In some cases, excessive red pigmentation develops in infected leaves. Bronzed or water-soaked areas develop on leaves of some cultivars. Fibrous-rooted cultivars have small brown leaf spots. Some cultivars exhibit no symptoms despite heavy infection. If leaf or stem tissue is placed in a drop of water in a clear dish and teased into small pieces, numerous colorless nematodes can be seen with some magnification, vigorously moving in a snake-like motion in the water. The nematodes will tend to settle to the bottom of the dish.

Biology and epidemiology. This nematode can enter stomata or wounds, continue to move in the plant, and feed on cells inside the plant (migratory endoparasitic life style). Or the nematode can remain on the surface of the plant, continue to move at will if there is water on the plant tissue, and feed on surface cells (migratory ectoparasitic life style). There is some suggestion that the nematode may have the ability to enter intact leaf surfaces directly (Riedel 1985). Peirson (1974) found that the "Aphrodite Rose" cultivar of Rieger begonia can harbor 12,000 larvae of this nematode per gram of fresh weight of leaves, yet exhibit no symptoms.

Management. Purchase nematode-free plants. Irrigate plants in a manner that keeps water off the foliage so that movement of the nematodes on the leaf surface is inhibited and localized dispersal among closely spaced plants by splashing is avoided. Discard infected plants. A very high value stock plant can be heat-treated to eliminate nematodes (Guba and Gilgut 1938). Some experimentation is required to determine the best temperature and exposure duration for eliminating the nematodes while not killing the plant. An effective hot water dip may range from 1 min at 49 °C (120 °F) to 5 min at 45 °C (115 °F), depending upon the cultivar. A treated plant may be severely damaged, but it will recover over time and be free of the nematode.

Additional Diseases with Nematode Pathogens
Root-knot (*Meloidogyne* spp.) (Powell 1985)

6 Abiotic Diseases

A variety of leaf symptoms develop as a result of nutrient deficiencies in begonia. If yellowing is the predominant symptom and the yellowing is between the veins, iron or magnesium may be lacking. Uniformly yellowing leaves indicates nitrogen or calcium deficiency. Dead tissue at the leaf margin may indicate potassium deficiency, while stunting of an otherwise green plant could be due to lack of phosphorus. A lack of boron causes the foliage to russet and cracks may develop in the leaf petioles and the plants may be very brittle (Nelson et al. 1977).

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Diseases of Coleus

31

Blair R. Harlan and Mary K. Hausbeck

Abstract

Coleus (*Plectranthus scutellarioides*), an herbaceous bedding plant, has been prized by gardeners for its bright colorful foliage since Victorian times and in recent years has seen a resurgence in popularity. At retail greenhouses, coleus can be purchased as flats of mixed cultivars, hanging baskets, or individually potted plants. The most serious pathogen of coleus is downy mildew. Downy mildew on coleus, identified as a *Peronospora* sp. based on morphological characteristics, was first reported in the United States in 2005. Coleus are not considered to be susceptible to the pathogens commonly associated with other bedding plants. However, Rhizoctonia crown and stem blight, Botrytis gray mold, and select viruses have been observed. Management of coleus diseases in the greenhouse and landscape usually centers on the control of downy mildew and includes chemical and cultural control and host resistance.

Keywords

Downy mildew • Botrytis • INSV • Pythium • Rhizoctonia • Scutellarioides • Herbaceous

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R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_32

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1 Introduction

Coleus (*Plectranthus scutellarioides* L. (Codd)) is a species of flowering plant in the family Lamiaceae. Coleus is a popular foliage plant grown in gardens, containers, and interiorscapes throughout the United States. In recent years, coleus has become increasing popular with gardeners who are drawn to its vibrantly colored foliage and variegation patterns. The 2014 wholesale value of coleus in the United States was $14.3 million (Anonymous 2015). Coleus plants are considered to be tropical to subtropical, preferring temperatures ranging between 16 °C and 24 °C (61 °C and 75 °F) (Pedley and Pedley 1974). Temperatures higher than 24 °C will not negatively affect growth, although temperatures lower than 10 °C/50 °F result in decreased vigor. Coleus can survive in most sunlight conditions; however, foliage becomes more vibrant when shaded. Coleus is commercially propagated with vegetative cuttings or by seed. Prior to the emergence of downy mildew as a pathogen, coleus was relatively easy to grow with minimal chemical inputs needed during cultivation. Coleus grown in the United States may be treated with fungicides to prevent and limit downy mildew infection.

2 Fungal and Fungus-Like Diseases

2.1 Botrytis Blight (*Botrytis cinerea*)

Geographic occurrence and impact. This disease has been officially reported in Alaska; however, due to the endemic nature of this pathogen, it is likely much more widely distributed (Anonymous 1960).

Symptoms/signs. Beginning as small water-soaked spots on leaves, stems, or blossoms, the spots coalesce rapidly, affecting large portions of tissue (Hausbeck and Moorman 1996). Leaf blight may be initiated when infected and senescent tissue falls onto healthy leaves. Stem blight typically begins in a broken or cut stem surface and progresses downward, causing a dieback of the entire stem; in severe cases, the blight extends into the base of the plant and kills it. *B. cinerea* readily sporulates on necrotic and diseased tissue, appearing as a fuzzy or powdery gray mold. This sporulation is quite different when compared to downy mildew sporulation, which tends to be sparser and only on the underside of the leaf surface. When sporulation of

B. cinerea is extensive over the plant, a cloud of gray conidia typically can be observed if the plant is physically disturbed.

Biology and epidemiology. Conidia, ascospores, mycelial fragments, and sclerotia all contribute to dispersal of *B. cinerea* (Jarvis 1980). In the majority of cases, conidia serve as the initial inoculum for *B. cinerea* infection, and although conidia can land on plant surfaces and invade immediately, they may remain dormant before germinating (Hausbeck and Moorman 1996). Hyphae may spread the disease when infected senescent plant parts fall onto healthy plant parts. Chlamydospores are thick-walled spores that can remain dormant in crop debris and give rise to new infections when conditions are favorable. A rise and subsequent decrease in relative humidity have been correlated with high counts of atmospheric *B. cinerea* conidia. Once the conidia land on a suitable host, under moist conditions, germ tubes are produced that can penetrate the plant surface leading to infection.

Management

- ***Cultural control*** – Shorten the intervals between scouting events during periods of high relative humidity. Be vigilant in watching for stem infections after pinching or pruning. Watering in the morning so that the foliage can dry rapidly is one way to minimize *Botrytis*. Reducing relative humidity by spacing plants farther apart and providing good air circulation can be helpful (Westcott 1979). Removing leaves with the sporulating pathogen from plants may reduce the amount inoculum for future infections.
- ***Sanitation*** – Sanitation is important and includes removing fading flowers and blighted foliage (Westcott 1979). Plant debris should not be allowed to accumulate on greenhouse benches and floors. This plant debris supports sporulating *B. cinerea* and allows continued release and dispersal of conidia (Hausbeck and Moorman 1996). Keep waste containers that contain old plant material closed and away from greenhouse intake vents.
- ***Fungicides and biocontrols*** – Due to the relative infrequency of Botrytis blight on coleus, fungicide treatments may not be necessary. However, if *Botrytis* is observed on coleus, fungicides can effectively limit the spread of the pathogen. For best results, apply fungicides at the highest label rate. In a trial conducted on geraniums by Hausbeck and Harlan (2010), applications of effective fungicides significantly reduced infection compared to untreated plants. The most effective active treatments included chlorothalonil, fenhexamid, thiophanate-methyl, fludioxonil/cyprodinil, and pyraclostrobin/boscalid. However, resistance to benzimidazole and dicarboximide fungicides has been well documented and should be avoided as standalone treatments (Moorman and Lease 1992).
  The biopesticide polyoxin D zinc salt has been shown to be highly effective in controlling *Botrytis* in greenhouse crops (Hausbeck et al. 2002). Other biopesticides have shown varying degrees of efficacy; *Bacillus subtilis* was effective in limiting *Botrytis* lesions in a trial conducted by Webster and Hausbeck (2004).
- ***Resistance*** – At this time there is no published information on the resistance of coleus to *B. cinerea*.

2.2 Downy Mildew (*Peronospora* sp.)

Geographic occurrence and impact. This disease has been reported in Japan, the USA (Louisiana, New York, Michigan, Florida, Tennessee), the United Kingdom (Daughtrey et al. 2006; Denton et al. 2015; Harlan and Hausbeck 2011; Ito et al. 2015; Palmateer et al. 2007; Rivera et al. 2016), and Germany (Anonymous 2008).

Symptoms/signs. Downy mildew has been observed on both seed-propagated and vegetatively propagated coleus. Symptoms of downy mildew infection include plant stunting, leaf distortion, and leaf abscission (Harlan and Hausbeck 2011). Necrotic lesions may be present on infected leaf tissue. Under high relative humidity, downy mildew-like growth may be observed on the undersides of infected leaves, often prior to any signs of necrosis (Fig. 1). Leaf abscission may occur on older infected leaves, giving the plant a "palm tree" shape. Downy mildew can be difficult to detect in some cases as the pathogen may become latent within an infected plant. These latently infected plants may appear healthy until environmental conditions become favorable for the growth and sporulation of the pathogen (Harlan and Hausbeck 2013).



Fig. 1 Downy mildew signs and symptoms: The most recognizable sign of downy mildew on coleus is sporulation observed on the underside of leaves (*top left*). Infected leaf tissue often displays necrotic lesions (*top left*, *top right*, *lower right*). Symptoms of infection include stunting (*bottom left*) and leaf abscission (*bottom right*) (B.R. Harlan © 2017. All Rights Reserved.)

Biology and epidemiology. The causal agent of downy mildew on coleus has been classified as a *Peronospora* sp., from the group Peronosporaceae. Downy mildew is an obligate pathogen, meaning that the organism must live and reproduce on living plant material. *Peronospora* produces sporangia, which germinate by germ tubes (Shaw 1981). In most cases sporulation occurs on the underside of the leaf surface; however, under favorable environmental conditions, it can be observed on the upper leaf surface and petioles.

The environment plays a crucial role in the development of downy mildew. Studies have concluded that infection and sporulation will occur readily at 15 °C and 20 °C (59 °F and 68 °F). When temperatures reached ≥25 °C/77 °F, infection and sporulation was significantly reduced. Infection and sporulation was not observed on any of the plants incubated at 30 °C/86 °F (Harlan et al. 2012). The prevalence of atmospheric downy mildew sporangia in relation to environmental conditions has been well documented. The release of *Peronospora* sp. sporangia has been correlated to long dew periods and periods of leaf wetness in field-grown ornamentals (Byrne et al. 2005). Greenhouse studies have shown increased levels of coleus downy mildew sporangia in the atmosphere when high relative humidity (>95%) was followed by lower relative humidity. Like other *Peronospora* spp. (Pinckard 1942), sporangial release of coleus downy mildew followed a daily periodicity with sporangial counts highest between 1000 and 1500 h.

Management

- ***Cultural control*** – Coleus should be scouted on a regular basis for signs and symptoms of downy mildew. Infected plants should be kept separate and isolated from healthy plants, as the sporangia can be airborne. Intervals between scouting events should be shortened during periods of high relative humidity. Environmental conditions that may limit downy mildew include keeping relative humidity below 85% and temperatures at or over 25 °C/77 °F. Limiting extended leaf wetness periods by watering in the morning or early afternoon will ensure that the foliage does not remain wet overnight. Increased plant spacing will ensure better ventilation and shorter leaf wetness periods.
- ***Sanitation*** – Removing downy mildew-infected plant material is essential (Harlan and Hausbeck 2011). Although *Peronospora* is an obligate pathogen, needing live plant material to reproduce, the sporangia on abscised leaves may be viable for a short time.
- ***Fungicides and biocontrols*** – If effective fungicides are used, downy mildew on coleus can be adequately controlled (Harlan and Hausbeck 2010). Fungicide active ingredients that have been shown to be highly effective in limiting *Peronospora* infection on coleus include mefenoxam, dimethomorph, fluopicolide, azoxystrobin, fosetyl-al, mandipropamid, fluoxastrobin, and fenamidone (Harlan and Hausbeck 2010). Research on other *Peronospora* species has shown that drench applications of mefenoxam, fluopicolide, and oxathiapiprolin have offered long-term protection from infection. In particular, these applications, when applied to greenhouse plants, have offered protection from downy mildew long after transplanting into the landscape (Harlan and

Hausbeck 2016). Due to the prevalence of the coleus downy mildew pathogen in recent years, preventive fungicide applications are warranted.

Biocontrol products have been tested against *Peronospora* spp. with limited success. An extract of *Reynoutria sachalinensis*, when applied as a preventive spray, has shown some efficacy against coleus downy mildew (Harlan and Hausbeck 2010; Ivors et al. 2011). In a trial conducted by Warfield et al. (2008), spray applications of a phosphorous acid salts product were highly effective.

- ***Resistance*** – With the vast number of different coleus cultivars commercially sold and the varying colors and shapes observed among these cultivars, it is not surprising that a wide range of susceptibility to downy mildew has been observed. Daughtrey et al. (2014) tested 147 cultivars over a multiyear study and rated 32% of the cultivars with low susceptibility, while 27.2% were rated as highly susceptible to downy mildew. In particular, all 11 cultivars in the Wizard series were rated as having medium to high susceptibility, while seven of the nine Fairway series cultivars were rated as having a low or medium susceptibility. When growing highly susceptible cultivars, it is recommended that preventive fungicides be applied prior to disease development.

2.3 Pythium Root Rot (*Pythium* spp.)

Geographic occurrence and impact. This disease has been reported in Florida, Mississippi, California, and Maryland (Alfieri et al. 1984; Anonymous 1960; Parris 1959).

Symptoms/signs. *Pythium* is a water mold that can "nibble" the feeding roots of plants, resulting in wilting and stunted growth. This reduced plant vigor can eventually affect marketability (Garzon et al. 2011). In severe cases, stems of seedlings or recent transplants collapse at the soil line and die (Pscheidt and Ocamb 2016). Plants that survive the initial infection phase are likely to remain stunted. Both cutting- and seed-propagated cultivars of coleus are susceptible. If Pythium root rot is suspected on a plant showing aboveground symptoms, the root system should be inspected. Infected roots will be brown or water-soaked and sparse when compared to a healthy root system (Fig. 2).

Biology and epidemiology. *Pythium* species are soilborne oomycete organisms that prefer wet conditions for infection and reproduction. This preference for wet conditions explains *Pythium*'s characterization as a water mold. Three distinct spore types are produced by *Pythium*: oospores, sporangia, and zoospores. Oospores are thick-walled sexually produced spores that can lay dormant for extended periods. Oospores can withstand long periods of drying, and once environmental conditions are conducive, or a suitable host is presented, they can germinate and infect via fungal-like strands. Sporangia and zoospores are the most common spore type associated with *Pythium* epidemics as they are produced in large numbers.



Fig. 2 Pythium root rot symptoms: Infected roots are *brown*, water soaked, and sparse (M. Daughtrey © 2017. All Rights Reserved.)

Zoospores, in particular, are associated with the spread of the pathogen in ebb-and-flow irrigation systems as this particular spore type is motile in water (Koike and Wilen 2009).

Management

- ***Cultural control*** – Overwatering, creating soil conditions that are overly saturated for extended periods, favors *Pythium* development. In particular, soil moisture conditions of 70% or higher are conducive to infection by *Pythium*. Keep fungus gnats and shore flies under control as they can spread the pathogen (Koike and Wilen 2009).
- ***Sanitation*** – This pathogen can become a greenhouse "resident" that hibernates on dirty plant containers, benches, hoses, and greenhouse walkways (Hausbeck and Harlan 2013). The reuse of flats, pots, and containers is not recommended; however, if they are reused, they should be thoroughly disinfected with a cleaning agent such as quaternary ammonium chloride salts or chlorine bleach. (Refer to ► Chap. 9, "Sanitation for Management of Florists' Crops Diseases" for additional information about disinfectants.) Plants with a suspected *Pythium* infection should be isolated from healthy plants to reduce the further spread of the pathogen.
- ***Fungicides and biocontrols*** – Unfortunately, there are very few effective fungicides registered against Pythium root rot. When attempting to control root rot pathogens with fungicide drenches, it is important to make applications to the soil. Applications of the low risk fungicide mefenoxam can be highly effective in limiting *Pythium* infection. However, resistance of the pathogen to mefenoxam is an issue (Del Castillo Munera and Hausbeck 2016). Etridiazole is another product that is highly effective against *Pythium*. Other products, including fluopicolide, fenamidone, and phosphorous acid salts, have shown promise in managing *Pythium* (Hausbeck and Harlan 2013).

Although using biocontrol against *Pythium* in a greenhouse can be helpful, studies have shown the level of control to be inconsistent. For instance, in a study published by Daughtrey and Tobiasz (2001), four popular biocontrol products were tested against *Pythium*, and all received ratings statistically similar to the untreated control plants. However, Baysal-Gurel and Miller (2013) found applications of biocontrol products effective in limiting Pythium damping-off in the greenhouse.

- ***Resistance*** – At this time there is no published information on the resistance of coleus to *Pythium* spp.

2.4 Rhizoctonia Root and Crown Rot (*Rhizoctonia solani*)

Geographic occurrence and impact. This disease has been reported in Florida, Illinois, New York, and Texas (Alfieri et al. 1984; Anonymous 1960).

Symptoms/signs. *Rhizoctonia* can cause pre- and postemergence damping-off of coleus. Younger plants are more susceptible (Wright 2013). Infected stems will appear blackened and water-soaked (Pscheidt and Ocamb 2016). In severe cases, *Rhizoctonia* can move into the plant canopy, causing spots on leaves and petioles. Root rot symptoms are similar to those caused by *Pythium*, and a diagnostic clinic may be necessary to differentiate with which pathogen you are dealing.

Biology and epidemiology. *Rhizoctonia* is a saprophytic fungal pathogen in the phylum Basidiomycota that is associated with several greenhouse crops throughout the world. *Rhizoctonia* is not host-specific, meaning one isolate can infect several different species of plants (Wright 2013). Unlike many of the other pathogens associated with greenhouse production, spores are not the usual method of dissemination for *Rhizoctonia*. Mycelia, the hairlike fungal strands produced by *Rhizoctonia*, are spread by the movement of infested soil particles, often by workers, which grow and infect nearby hosts. Sclerotia, the resting structure produced by *Rhizoctonia*, can survive for extended periods in soil particles. *Rhizoctonia* can thrive under almost all greenhouse conditions, although warmer, more humid conditions may enhance its spread. Prolonged periods of high humidity can result in the pathogen growing from the soil, up the stem, and into the foliage canopy.

Management

- ***Cultural control*** – One of the primary sources of *Rhizoctonia* inoculum in the greenhouse are infested trays (Gutierrez et al. 2001). The reuse of trays, pots, and other containers is not recommended; however, if they are reused, they should be thoroughly disinfected with a cleaning agent such as quaternary ammonium chloride salts or chlorine bleach.
- ***Sanitation*** – Between crops, clean soil, and organic material from benchtops and floors as this material can harbor the pathogen for extended periods. Because *Rhizoctonia* does not produce spores, one of the more common methods of

transmission is by workers. All pruning tools should be disinfected between crops and foot baths should be installed between greenhouses. Greenhouse carts should also be thoroughly cleaned.

- ***Fungicides and biocontrols*** – Moorman and Lease (1999) conducted a study looking at soil drenches to control Rhizoctonia rot on coleus and found that azoxystrobin and thiophanate-methyl were effective at limiting infection. Other products that have consistently reduced *Rhizoctonia* on ornamental plants include fludioxonil, PCNB, metconazole, and pyraclostrobin. The type of symptoms observed should determine the fungicide application method; drench applications should be used for root and crown rots, while high volume sprays or "sprenches" should be used to control foliar symptoms.
  The biopesticide, polyoxin D zinc salt, is highly effective against *Rhizoctonia* (Hausbeck and Harlan 2015). Using biocontrols in potting mixes may be helpful in managing damping-off when the plants are still young and more susceptible to the pathogen.
- ***Resistance*** – At this time there is no published information on the resistance of coleus to *Rhizoctonia.*

3 Viral Diseases

3.1 Bromoviridae

Geographic occurrence and impact. Viruses in the Bromoviridae are some of the most economically important and widely distributed viruses in the world. One member of this group has been reported in coleus: Cucumber mosaic virus (CMV, genus: *Cucumovirus*). Cucumber mosaic virus has been reported in Louisiana (Holcomb and Valverde 1991).

Symptoms/signs. Symptoms of CMV infection of coleus include ring-spotting, mosaic, and oak-leaf symptoms.

Biology and epidemiology. CMV is spread by more than 80 aphid species (Hemiptera: Aphididae) in a non-circulative (nonpersistent) manner. When these insects feed on CMV-infected plants, the virus moves into the mouthparts of the vector and can spread when the insect moves and feeds on adjacent plants. Although seed transmission has been reported on other crops (Neergaard 1977), it has not been reported or studied on coleus.

Management. There is no cure or chemical control for plant viruses. New shipments of plants should be inspected for symptoms of viruses upon arrival. Vector control is the most important management aspect associated with CMV on coleus. This management plan should include monitoring insect populations and applying chemical controls when needed. Insect populations can be tested for CMV infection.

Stock plants should be tested regularly for CMV, and infected plants should be discarded. Refer to ▶ Chap. 4, "Insect Management for Disease Control in Florists' Crops" for additional virus management information.

3.2 Bunyaviridae

Geographic occurrence and impact. Viruses belonging to the Bunyaviridae include those most commonly associated with ornamental crops. Impatiens necrotic spot virus (INSV, genus: *Tospovirus*) has been reported on coleus in New York (Catlin 2014), Michigan (Byrne 2008), and France (Lambert 2005).

Symptoms/signs. Ring spots are the most typical symptom associated with INSV on coleus (Fig. 3). Other symptoms of INSV include leaf mottle, stem or petiole lesions, irregularly shaped necrotic lesions, or plant stunting (Catlin 2014).

Biology and epidemiology. INSV is spread by the western flower thrips (*Frankliniella occidentalis*) as well as two closely related species *F. fusca* and *F. intonsa*. Unlike viruses in the Bromoviridae family, INSV enters the gut of the vector where it replicates. Thrips must be in their juvenile stage (first and early second larval stages) to acquire the virus, and only immature thrips that acquire these viruses or adults derived from such immatures are vectors.

Management. There is no cure or chemical control for plant viruses. New shipments of plants should be inspected for symptoms of viruses upon arrival. Western



Fig. 3 Symptoms of INSV: ring spots and irregularly shaped necrotic spots (*left*, L. Pundt © 2017. All Rights Reserved.; *right*, M. Daughtrey © 2017. All Rights Reserved.)

flower thrips populations should be closely monitored. In particular, flowers should be inspected, as thrips are likely to reside in such protected areas. If thrips are detected, chemical controls might be necessary. Insect populations can be tested for INSV incidence. Since almost all herbaceous plants associated with horticulture can harbor INSV, greenhouses should be scouted regularly for signs of infection. Any plants showing symptoms of INSV should be disposed of immediately.

3.3 Potyviridae

Geographic occurrence and impact. Potyviruses are fairly common and infect many crops. One member of this group has been recently discovered in coleus: Tobacco etch virus (TEV, genus: *Potyvirus*). TEV has been reported on coleus in Missouri and Minnesota (Lockhart et al. 2010).

Symptoms/signs. Symptoms of TEV are similar to those associated with coleus vein necrosis virus and include foliar and veinal lesions.

Biology and epidemiology. At this time, a number of aphid species are the only known vectors of TEV (Sikora 2004). The virus has a broad host range (>120 species in 19 dicot families) including a number of solanaceous crops such as pepper, tomato, and tobacco.

Management. There is no cure or chemical control for plant viruses. New shipments of plants should be inspected for symptoms of viruses upon arrival. Greenhouses should be scouted regularly for aphids. If aphids are detected, chemical controls might be necessary. Due to the rarity of this virus on coleus, plants showing symptoms of a viral infection should first be tested for INSV and CMV.

3.4 Betaflexiviridae

Geographic occurrence and impact. Of the virus families associated with coleus, viruses in the Betaflexiviridae are the least common and economically important. A new virus was observed on coleus in 2005 and was named coleus vein necrosis virus (CVNV, genus: *Carlavirus*) by Mollov et al. (2007).

Symptoms/signs. Symptoms of CVNV on coleus include abnormal leaf coloration, vein necrosis, and ring patterns (Fig. 4).

Biology and epidemiology. It is unknown how this virus is spread in the environment. In laboratory studies, mechanical viral transmission was successful. However, when aphids (*Myzus persicae*) were tested as a possible vector, transmission of CVNV was not observed (Mollov et al. 2007).



Fig. 4 Symptoms of CVNV: abnormal leaf coloration and ring patterns (M. Daughtrey © 2017. All Rights Reserved.)

Management. There is no cure or chemical control for plant viruses. New shipments of plants should be inspected for symptoms of viruses upon arrival. Due to the rarity of CVNV, if virus symptoms are observed on coleus, testing for INSV and CMV should be completed as they are much more common. At this time, there is not a commercially available test for CVNV.

4 Viroid Diseases

4.1 Pospiviroidae

Geographic occurrence and impact. Viroids are small, single-stranded sections of RNA that do not have protein coats. The coleus blumei viroids (CbVd, genus: *Coleviroid*) are widely distributed and have been classified as six different species, CbVd-1 through CbVd-6. CbVd-1 was first detected in Brazil in 1989 and is the most common species and has been reported worldwide (Fonseca et al. 1989; Tsushima and Sano 2015). CbVds has been reported in Germany, China, India, Indonesia, and Japan (Spieker et al. 1990; Hou et al. 2009; Jiang et al. 2013; Tsushima and Sano 2015).

Symptoms/signs. A wide range of symptoms can be observed on CbVd-infected coleus. In fact, CbVd is often detected on plants that appear to be symptomless and healthy (Fu et al. 2011). Stunting has been identified as a symptom, although yellowing or chlorosis-type symptoms are the most common (Tsushima and Sano 2015). This yellowing can be observed on the leaves in patches, often irregular in shape (Adkar-Purushothama et al. 2013).

Biology and epidemiology. Very little is known on how this pathogen infects and spreads on coleus. Jiang et al. (2014) did show that seed transmission is possible and vegetative cuttings are likely to spread the viroid.

Management. There is no cure or chemical control for plant viroids. Getting seed and cuttings from stock plants that have been tested for CbVd will reduce the spread of the disease.

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Diseases of Gardenia

32

A. J. Palmateer and A. R. Chase

Abstract

Gardenias are a very important crop for both potted flowers for the florist trade and the landscape industries. Gardenias are susceptible to root rot, and the most common and damaging pathogens include *Phytophthora* and *Rhizoctonia*. One of the most devastating diseases affecting gardenias is canker caused by *Diaporthe gardeniae*, which can lead to unsightly cankers and galls on the lower stem causing plant decline and eventual death. Several leaf spot pathogens are reported on gardenia with *Myrothecium* and *Xanthomonas* being among the most common. Disease management practices for gardenia depend on good sanitation, judicious use of pesticides, and cultural practices.

Keywords

Gardenia • *Phytophthora* • *Myrothecium* • *Pythium* • *Rhizoctonia* • Gardenia canker • *Xanthomonas*

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R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_33

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1 Introduction

Gardenia jasminoides is a popular flowering shrub in the Rubiaceae family that can be found growing in tropical and semitropical areas in the landscape worldwide (Fig. 1). It is also grown as a potted flowering crop by the florist industry in temperate climates using protected culture. Patio gardenias are normally not grafted and cuttings are grown in pots. Some cultivars are also grown as standards where the apical meristem is pruned to form a head. Landscape plantings are mostly grafted to the root stock *Gardenia thunbergia* for resistance to root-knot nematodes and *Rhizoctonia* spp.

2 Fungal and Fungus-Like Diseases

2.1 Gardenia Canker (*Diaporthe gardeniae* = *Phomopsis gardeniae*)

Geographic occurrence and impact. This disease has been reported within the continental USA; gardenia canker has been reported in California (Hansen and Barrett 1938), Washington, Nebraska, Kansas, Ohio, Massachusetts, and Florida, and has also been reported in Europe (Italy) (Alfieri 1967) and South America.



Fig. 1 Healthy *Gardenia jasminoides* with showy white blooms (A.J. Palmateer © 2017. All Rights Reserved.)

Symptoms/signs. Initial symptoms of gardenia canker include wilting, yellowing, shriveling, and defoliation of leaves and frequent premature bud drop of unopened flowers. The wilting can affect a portion of the plant or the entire plant where it can appear as a sudden wilt. The cankers are at first small, circular to oblong, swollen brownish lesions on the main stem and branches. Cankers can become as large as 5 cm (2 in.) in diameter and cause partial to complete girdling of stems and branches. Even partial girdling can result in stunting and wilt. The diseased area decays and begins to separate from trunk or branch tissue exposing the wood. In some cases, the periphery of the canker becomes calloused and appears rough and corrugated. The cankers become overgrown with corky callous tissue extending longitudinally in both directions, increasing in size and forming galls. These galls typically increase two to three times in size and produce deep longitudinal cracks. Branches in close proximity to galls begin to lose vigor compared to those further away or originating from unaffected stem tissue. Stem cankers are more common at the base of the plant affecting the crown, but they can occur anywhere on the plant especially where wounded from mechanical injury.

The foliage of older infected plants appears dull eventually turning yellow or drying out and becoming brown before defoliating. Flower buds will abscise before opening. Disease severity and wilting is usually more pronounced under cooler temperatures. During warmer conditions diseased plants may continue to live, but remain stunted and appear unhealthy. Sometimes signs of the imperfect stage of the fungus (*Phomopsis gardeniae*) are present consisting of black fruiting bodies on the lesions (pycnidia) that contain both alpha and beta conidia.

Biology and epidemiology. The fungal pathogen is widely considered to be a wound parasite; however, once infection occurs and the fungus becomes established in host tissue, disease develops and progresses forming mature cankers and subsequent pycnidia. Under conditions of high relative humidity and warm temperatures, an abundance of spores forms within the partially submerged pycnidia and is easily spread by windblown rain and splash dispersion in irrigation water. The fungus is most prevalent on or near cankers and overwinters from one season to the next on the diseased portions of infected plants. The fungus is notorious for entering gardenias through leaf joints at the base of cuttings. Thus, freshly cut leaf bases act as an excellent point of entry for the fungus.

Management

- ***Cultural practices*** – Disease-free stock plants should be used for propagation. Pathogen elimination through sanitation by steam-pasteurizing rooting media, sand, and peat is highly recommended. All cuttings should be taken with sharp blades avoiding rough and jagged edges where spores of the pathogen are more likely to gain entry. All tools contacting plant material should be properly disinfected to minimize contamination and potential spread of the pathogen. Plant injury due to careless cultural practices should be avoided. The productivity of affected plants has been shown to be enhanced or lengthened by piling soil up

around the stem base covering basal cankers to encourage new root formation above the diseased portions of stems.

- ***Fungicides and biological controls*** – Preventative applications of fungicides containing chlorothalonil (FRAC M5), mancozeb (FRAC M3), propiconazole (FRAC 3), or thiophanate methyl (FRAC 1) ensuring excellent coverage of all plant parts may minimize disease outbreaks. In addition, the combination of pyraclostrobin (FRAC 11) and boscalid (FRAC 7) has been effective on similar pathogens on other crops. It is important to rotate fungicides from different classes in order to help prevent the development of resistance. Scientific literature reports good efficacy against Phomopsis on other plant species using biological control products containing *Gliocladium virens*. These products were applied as a soil drench and as a wound treatment.

2.2 Myrothecium Leaf Spot (*Myrothecium roridum*)

Geographic occurrence and impact. This disease was found and described from Pennsylvania in 1941 (Fergus 1957) and has since been found in California (Barrett and Hardman 1947) and Florida.

Symptoms/signs. The leaf spots are mostly circular and vary in size up to 2 cm (~1 in.) in diameter (Fergus 1957). Initial spots appear water soaked and turn brown with age. Often the centers of the lesions fall out giving a shot hole appearance. Under wet conditions, the spots coalesce and large portions of the leaves can rot away. Characteristic greenish-black pillow-shaped sporodochia with white setal margins form on necrotic tissue and can be seen on both the upper and lower leaf surface (Fig. 2).

Biology and epidemiology. Myrothecium leaf spot on gardenia is most common under humid conditions such as those found when attempting to root cuttings.



Fig. 2 *Myrothecium roridum* on gardenia leaves (A.J. Palmateer © 2017. All Rights Reserved.)

Pathogenicity of *M. roridum* isolates is reported to vary greatly with most isolates being wound pathogens on gardenia.

Management

- ***Cultural practices*** – Monitor nutritional programs closely, because over-fertilization has been shown to increase leaf spots and rots caused by *Myrothecium* in some tropical foliage plants. Imbalanced fertilization programs, damage from pesticides, heat or cold injury, and leaf necrosis caused by water stress should be avoided. Wounded or weakened tissue allows *Myrothecium* to form a colony in the canopy, which serves as the focal point for disease spread.
- ***Fungicides and biological control*** – Apply fungicides containing azoxystrobin or pyraclostrobin (FRAC 11), chlorothalonil (FRAC M5), fludioxonil (FRAC 12), iprodione (FRAC 2), or mancozeb (FRAC M3). Good coverage is very important as this fungus can colonize both sides of the leaf surface. No effective biocontrol agents have been reported for this disease.

2.3 Powdery Mildew (*Erysiphe polygoni, Oidium* sp.)

Geographic occurrence and impact. This disease has been reported in Texas and tropical America but is not common.

Symptoms/signs. Powdery mildew of gardenia primarily affects young leaves and shoots where symptoms of infection include leaf yellowing, slight deformation of leaf and bud tissue, and leaf drop. Powdery mildew fungi typically grow on the surface of plants and infect plant cells using a haustorium to absorb nutrients. Signs of infection include a white to gray colored fungal growth that appears like powder and is most often found on the upper leaf surface but may occur on the underside of leaves when conditions are highly favorable.

Biology and epidemiology. Powdery mildew fungi can survive in leaf litter. High relative humidity and moderate to warm temperatures favor outbreaks of powdery mildew. Shade especially during periods of high relative humidity and moderate temperatures are most favorable conditions for powdery mildew.

Management

- ***Cultural practices*** – Proper location of plants in full to partial sun and avoiding heavily shaded areas will help to prevent outbreaks of powdery mildew. Space plants apart to allow for good air movement through the plant canopy. Overhead irrigation may actually reduce the spread of powdery mildew, because the water, unlike with other fungi, actually inhibits spore germination. Closely monitor plants and examine new shoot growth for signs of powdery mildew.
- ***Fungicides and biological controls*** – The use of fungicides may be necessary in production situations when favorable conditions persist. Horticultural oil or plant-based oil such as neem oil can be used to control powdery mildew. Be certain

not to apply oil within 14 days of a sulfur spray or plant injury may occur. Never apply oil when ambient temperatures exceed (30 °C/86°F) or when plants are water-stressed. Sulfur-based products are commonly used for controlling powdery mildew and should be used only as a preventative before signs of powdery mildew appear. Fungicides with efficacy for controlling powdery mildew include thiophanate methyl (FRAC 1); copper (FRAC M1); myclobutanil, propiconazole, triadimefon (FRAC 3); azoxystrobin, pyraclostrobin, trifloxystrobin (FRAC 11); chlorothalonil (FRAC M5); and *Bacillus* spp. (FRAC 44).

2.4 Rhizoctonia Root Rot (*Rhizoctonia solani, Rhizoctonia* sp.)

Geographic occurrence and impact. This disease has been reported wherever gardenia is grown and is considered to be one of the most common and economically important diseases affecting gardenia. Specific reports from the USA (California, Florida, New Jersey), Greece, and Japan have been made.

Symptoms/signs. Common symptoms in cutting beds include damping-off, hypocotyl rot, root rot, stunting, yellowing, and death. On older plants, *Rhizoctonia* causes the roots to turn light to dark brown and macerates the tissue; but the pathogen has been reported to affect all portions of the plant. Light to dark brown irregularly shaped spots form anywhere on the stem and foliage during propagation (Fig. 3) or in the landscape. Mycelium of the fungal pathogen is frequently present and looks like a light brown web colonizing affected tissue giving rise to the common name web blight.

Biology and epidemiology. *Rhizoctonia* can survive in soil and plant debris as mycelium and sclerotia that can be stimulated to germinate by plant exudates. Rhizoctonia stem rot and aerial blight or leaf spot occurs in the warmer months especially during the rainy season when there is an abundance of moisture. Such



Fig. 3 Darkly stained stem cankers on gardenia in propagation due to *Rhizoctonia solani* (A.J. Palmateer © 2017. All Rights Reserved.)

favorable conditions can occur at anytime throughout the year when plants are produced in covered structures that employ overhead irrigation. Under favorable conditions severe outbreaks are common and can occur in as little 7 days.

Management

- ***Cultural practices*** – Prevention is the most important step for managing *Rhizoctonia*. Start with high quality, healthy cuttings, use new or sterilized pots and new potting media. Placing plants on hard surfaces, ground clothes, or benches so that they are not in direct contact with soil is crucial. Due to the quick onset of this disease it is recommended to scout plants weekly for early symptom detection.
- ***Fungicides and biological controls*** – Specific trials on fungicides and biocontrol agents have not been reported on gardenia for *Rhizoctonia*. On other woody ornamental crops, strobilurins (FRAC 11); fludioxonil (FRAC 12); and thiophanate methyl (FRAC 1) are among the most effective fungicides. Additionally, the biocontrol agent *Trichoderma harzianum* T22 can be very effective against root rot but less so against stem rot or cutting rot.

2.5 Phytophthora Root and Stem Rot (*Phytophthora cinnamomi* and *P. nicotianae*)

Geographic occurrence and impact. *Phytophthora cinnamomi* is typically the most common species associated with container and field grown gardenias in the Southeastern USA, but other species have been widely reported to cause root and stem rot of gardenia. *P. nicotianae* has been reported from the USA in North Carolina (Olson and Benson 2011), California, and Florida and also Greece.

Symptoms/signs. Phytophthora usually reduces the overall volume of roots that are present, thus greatly inhibiting the plant's ability to take up water and nutrients for proper growth. The roots of diseased plants appear brittle and brown to reddish brown in color (Fig. 4a). The pathogen usually colonizes the crown often girdling the stem at or just above the soil line. A brown to reddish brown discoloration of the tissues occurs just below the bark layer and may extend up the stem above the soil. Affected tissue is most often wet in appearance. Aboveground symptoms can easily be confused with those caused by a nutritional disorder, overwatering, drought stress, and other abiotic disorders. Slight yellowing of the leaves followed by wilting and possibly plant death (Fig. 4b) are common symptoms associated with *Phytophthora*. Landscape or field grown gardenias may show symptoms of general decline for up to 1 year or more before succumbing to root rot caused by *Phytophthora*.

Biology and epidemiology. *Phytophthora* species are fungus-like water molds or Oomycetes that most often survive as resting structures consisting of chlamydospores, oospores, and mycelia in diseased roots, crowns, and other plant debris. These structures are released into the soil or potting medium from infested crop



Fig. 4 (**a**) Brittle and brown diseased roots due to infection by *Phytophthora nicotianae*. (**b**) Necrotic and wilted leaves on gardenia with stem infection from *Phytophthora nicotianae* (A.J. Palmateer © 2017. All Rights Reserved.)

debris and can be easily spread throughout propagation and container production areas in windblown plant and potting material and by splashing rain and irrigation water. Heaviest losses occur in poorly drained areas where there is standing water. Overwatering in the nursery and landscape are contributing factors to losses from *Phytophthora*.

Management

- ***Cultural practices*** – An integrated approach is necessary for success in controlling Phytophthora root rot. Prevention and sanitation are key, because once symptoms appear it is often too late to adequately control the disease. Providing for proper plant establishment and following good horticultural practices including adequate nutrition and irrigation will help to reduce potential losses from this disease. Recycling irrigation water does not appear to be a significant source of disease in gardenias.
- ***Fungicides and biological controls*** – Fungicides should be used as a preventative measure, and those used for managing Phytophthora include fosetyl-aluminum, phosphorous acid (FRAC 33); fluopicolide (FRAC 43); mefenoxam (FRAC 4); dimethomorph, mandipropamid (FRAC 40); chlorothalonil (FRAC M5); azoxystrobin, pyraclostrobin, fenamidone (FRAC 11); etridiazole (FRAC 14); propamocarb hydrochloride (FRAC 28); cyazofamid (FRAC 21); oxathiapiprolin (FRAC U5); and *Bacillus* spp. (FRAC 44).

2.6 Pythium Root Rot (*Pythium splendens, P. spinosum*)

Geographic occurrence and impact. Pythium root rot is common in most gardenia production areas although losses are usually minimal and damage is not as severe when compared to that caused by *Phytophthora*.

Symptoms/signs. The aboveground symptoms often include yellowing of the oldest leaves first followed by defoliation and in severe cases wilting (Fig. 5a).



Fig. 5 (**a**) Pythium root rot exhibiting severe leaf chlorosis and wilting caused by *P. splendens* (A.J. Palmateer © 2017. All Rights Reserved.). (**b**) Pythium root rot on gardenia showing typical sparse, necrotic, and discolored roots (A.R. Chase © 2017. All Rights Reserved.). (**c**) Outer cortex of roots infected with *Pythium* spp. often deteriorates leaving the light colored central core (A.R. Chase © 2017. All Rights Reserved.)

The plants typically appear unhealthy for an extended period of time and may not respond to fertilization due to *Pythium* attacking the feeder roots and inhibiting the plant's ability to take up water and nutrients. Healthy roots are white whereas diseased roots appear brown and discolored, or may be completely missing (Fig. 5b). The outer cortical tissue can be easily removed when pulling on the roots (Fig. 5c), leaving behind the threadlike core or stele of the water conducting tissue.

Biology and epidemiology. The biology and epidemiology is very similar to *Phytophthora*. Disease development is highly favored by any factor that encourages wet soil conditions, including poor drainage and over watering. Planting gardenias too deep also contributes to the disease.

Management

- ***Cultural practices*** – An integrated approach is necessary for success in controlling Pythium root rot. Closely monitoring irrigation and using a potting medium that allows for adequate drainage or aeration is key. Providing for proper plant

establishment and following good horticultural practices including adequate nutrition and irrigation will help to reduce potential losses from this disease. Recycling irrigation water does not appear to be a significant source of disease in gardenias.

- ***Fungicides and biological controls*** – Fungicides should be used as a preventative measure and those used for managing *Pythium* include fosetyl-aluminum, phosphorous acid (FRAC 33); fluopicolide (FRAC 43); mefenoxam (FRAC 4); dimethomorph, mandipropamid (FRAC 40); chlorothalonil (FRAC M5); azoxystrobin, pyraclostrobin, fenamidone (FRAC 11); etridiazole (FRAC 14); propamocarb (FRAC 28); cyazofamid (FRAC 21); oxathiapiprolin (FRAC U5); and *Bacillus* spp. (FRAC 44).

3 Bacterial and Phytoplasma Diseases

3.1 Bacterial Leaf Spot (*Xanthomonas maculifoliigardeniae*)

Geographic occurrence and impact. This disease was reported for the first time in California in 1941 (Burkholder and Pirone 1941) on greenhouse grown gardenias. It was later described by Ark and Barrett in 1946. It is commonly found in production and the landscape in Florida and California.

Symptoms/signs. Very young tender leaves are the first to develop symptoms which consist of small yellow spots. The yellow spots are angular to circular and gradually increase in size where the center initially appears water soaked. As the leaves age, the center portion of the spots turns reddish brown and necrotic. Lesions are most often surrounded by a wide chlorotic halo (Fig. 6). Spots eventually coalesce and produce larger necrotic areas. Premature leaf drop is common on severely affected plants.

Biology and epidemiology. Conditions providing high relative humidity and temperature, especially in greenhouse production where gardenias are induced to bloom, favor disease development. Symptoms may appear rather suddenly on gardenias growing outdoors during warm and wet periods.

Management

- ***Cultural practices*** – Overhead irrigation should be avoided or timed for periods when leaves dry quickly as bacterial cells are readily dispersed in irrigation water increasing the incidence of diseased plants and plant parts. The disease is most severe during periods of prolonged leaf wetness.
- ***Bactericides and biological controls*** – As a preventative measure apply a bactericide containing copper hydroxide, copper oxychloride, copper sulfate, or other forms (FRAC M1); mancozeb (FRAC M3); or streptomycin sulfate (FRAC 25). The quaternary ammonium (QA) product DDAC also shows good control of



Fig. 6 Xanthomonas leaf spot on gardenia caused by *Xanthomonas maculifoliigardeniae* (A.J. Palmateer © 2017. All Rights Reserved.)

bacterial diseases on other ornamentals. Possible biological control agents include species of *Bacillus* such as *B. amyloliquifaciens* and *B. subtilis* which have both shown good efficacy against related bacterial pathogens on other ornamentals and agronomic crops.

These are less common diseases that are known to affect gardenia:

Botrytis cinerea – California, Florida Hawaii, US Virgin Islands, Puerto Rico, Greece, Saudi Arabia, and China
Capnodium spp. (sooty mold) – California, Georgia, US Gulf Coast States, and Venezuela
Cercospora (and *Pseudocercospora*) – Indonesia, Venezuela, and Florida
Colletotrichum spp. (anthracnose) – Hong Kong, Cina, India, and Florida
Pestalotia (and *Pestalotiopsis*) – Alabama, Florida, Cuba, Japan, and China
Phyllosticta gardeniae – Japan, Republic of Georgia, India, and the USA (Michigan, North Carolina, New Jersey, and Texas)
Phytoplasma – China (Sun and Zhao 2012)
Tomato spotted wilt virus – USA

4 Abiotic Disorders

4.1 Iron Deficiency

Geographic occurrence and impact. Gardenias are an acid loving plant, so when soil pH is greater than 6.0 the potential for micronutrient deficiencies increase. The most common nutrient deficiency affecting gardenias is iron, and when soil pH is above 7 the iron may be in a form that is not readily plant-available.

Symptoms/signs. Most often iron deficient gardenias are stunted and have pale green to yellow leaves. Symptoms are most obvious in younger leaves which turn completely yellow except for dark green veins. Older leaves may only turn yellow along the edges (Fig. 7).

Management

- ***Cultural practices*** – Anything that can be done to acidify (lower) the pH of the soil will help. Some relevant soil amendments include aluminum sulfate, iron sulfate, or wettable sulfur. Gardenias growing in soils with a high buffering capacity such as calcareous soils require foliar applications of iron chelate or ferrous sulfate ($FeSO_4.2H_20$) can be very effective for correcting iron deficiency in gardenias. Allow the soil to remain evenly moist but not completed saturated.

4.2 Bud Drop

Geographic occurrence and impact. Bud drop is a prevailing issue with gardenias and is especially common after a change in location or growing conditions. It can occur in both container and landscape gardenias.



Fig. 7 Iron deficiency shows stunted chlorotic leaves when plants are subjected to high pH (A.J. Palmateer © 2017. All Rights Reserved.)



Fig. 8 Irregular watering or temperature fluctuations causes premature bud abortion and drop (A.R. Chase © 2017. All Rights Reserved.)

Symptoms/signs. When gardenias are under stress unopened flower buds may prematurely drop off from the plant (Fig. 8). Numerous things can cause stress, but some of the most common include insects feeding, diseases including root feeding nematodes, inadequate fertilization, irrigation and light, and temperature fluctuations such as unusually cold or hot conditions.

Management

- ***Cultural practices*** – Anything that can be done to minimize plant stress. Gardenias do not respond well to changes in growing conditions, so avoid moving or relocating plants that are thriving. Monitor irrigation so that the soil remains evenly moist, but not saturated.

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Diseases of Geranium

33

Cristina Rosa and Gary W. Moorman

Abstract

Geraniums are susceptible to a wide variety of fungi, bacteria, and viruses as well as nematodes and abiotic diseases. The systemic nature of some of the pathogens makes it likely that the diseases they cause can be found wherever vegetatively propagated geraniums are shipped. Management of these pathogens is paramount for specialty propagators, while growers purchasing plants must inspect incoming plants for symptoms and understand the biology of the pathogens involved in order to manage them effectively.

Keywords

Botrytis cinerea • *Pythium* Spp. • *Ralstonia solanacearum* • *Xanthomonas campestris* • Virus • Edema • Culture Indexing • Virus Indexing • Certification

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R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_34

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1 Introduction

Species of *Geranium* (described by Linnaeus) are often grown as herbaceous perennials outdoors, while *Pelargonium* (described by L'Héritier) are generally grown indoors for long periods of time or as annuals or perennials outdoors. Emphasis in this chapter is placed on the pathogens and diseases of *Pelargonium graveolens*, *P. peltatum*, and the complex hybrids *Pelargonium* X *domesticum* and *Pelargonium* X *hortorum*. Common names for these *Pelargonium* include florists, garden, bedding, zonal, ivy, horseshoe, rose, and scented and regal geranium, among others depending upon the species. It is thought that *Pelargonium* was brought into cultivation in Europe from plants initially obtained in South Africa in the early 1600s and are well documented as having been grown in many European gardens by the mid-1700s (Laughner 1993). At one time, *Pelargonium* was the mainstay of the bedding plant industry, and the majority of plants were propagated vegetatively. While vegetatively propagated varieties are still grown extensively, many other varieties are grown from seed. Disease susceptibility varies greatly with the variety and the pathogen involved, and some pathogens pose more of a problem in greenhouse production than when plants are grown outdoors. The geographic distribution

of particular pathogens reported in the literature may provide clues to the environmental conditions that favor the disease, but the worldwide trade in millions of *Pelargonium* cuttings greatly increases the potential for any pathogen to be found anywhere *Pelargonium* is grown. Crucial to the production of *Pelargonium* varieties through vegetative propagation is the establishment of stock plants that are free of the plant pathogens that infect systemically or stay closely associated with plant tissues including *Verticillium*, *Xanthomonas campestris* pv. *pelargonii*, *Ralstonia solanacearum*, *Agrobacterium tumefaciens*, *Rhodococcus fascians*, and all the viruses noted here. Culture indexing is used to detect those plant pathogens that readily grow in broth containing required nutrients, and virus indexing employs various methods of testing plant tissues for the presences of specific viruses (De Boer et al. 1996; Huttinga 1996; Oglevee-O'Donovan 1993). Plants free of selected pathogens are then maintained as stock plants from which numerous cuttings are taken. This "culture/virus indexing" procedure has been central in the production of "pathogen-free" *Pelargonium*, but note that they are "free" of selected pathogens. Other pathogens may be present in or on the plants.

2 Fungal and Fungus-like Diseases

For a list of pathogens of this plant in the USA, see Farr, et al. (1989). For the most up-to-date listing, search http://nt.ars-grin.gov/fungaldatabases/fungushost/fungushost.cfm.

2.1 Alternaria Leaf Spot (*Alternaria Sp.*)

Geographic occurrence and impact. This disease occurs when conditions are not well suited to geranium growth, particularly if the temperatures are too high or low or when other factors favor plant senescence. Generally, economic losses are minor (Engelhard 1993; Munnecke 1956).

Symptoms/signs. Water-soaked spots initially formed on the underside of the leaf enlarge to 5–10 mm (1/4–1/2 in.). Leaves may have numerous spots, and some spots may have a yellow halo. Concentric dark rings form in the spots creating a target-like pattern where the fungus has formed dark brown, club-shaped, multi-celled spores on the surface of the leaf (Fig. 1). The spots may merge and occupy most of the leaf. Diagnosis is facilitated if infected leaves are placed in a container with moisture, and the spores are allowed to develop (Engelhard 1993).

Biology and epidemiology. Warm, wet conditions greatly favor infection and development of symptoms particularly on older leaves. The fungus survives on dead leaves on the soil surface, and spores are dispersed by air currents. The disease generally begins on older leaves, particularly if they are senescing, and progresses upward on the plant.



Fig. 1 Alternaria leaf spot (The Pennsylvania State University Department of Plant Pathology and Environmental Microbiology © 2017. All Rights Reserved.)

Management. Irrigate plants in a manner that keeps water off the foliage. Ensure good air circulation among plants so that the humidity within the canopy is kept low. Remove and destroy infected leaves and any crop debris in pots, or on benches, and walkways. Apply a fungicide to protect plants.

2.2 Black Root Rot (*Thielaviopsis basicola (Berk. And Br) Ferris*)

Geographic occurrence and impact. This disease can occur in *Pelargonium* and *Geranium* production (Linderman 1993). In *Pelargonium*, seed-type geraniums vary in susceptibility. While individual growers may sustain serious losses, the occurrence of this disease appears to be very sporadic and rare, particularly with the wide use of *Thielaviopsis*-free, peat-based potting mixes that have replaced field soil-based mixes.

Symptoms/signs. Infected plants may be symptomless or may be stunted and have yellowed older foliage. Older infected roots may be very dark brown, almost black, while infected young roots may be only slightly darkened or may appear healthy unless observed microscopically. *Thielaviopsis* forms very dark brown, thick-walled, cigar-shaped spores in root cells that are readily found through microscopic observation and a thin-walled colorless spore on the surface of plant tissues (Linderman 1993).

Biology/epidemiology. *Thielaviopsis* survives well in soil. If infested soil is brought into contact with plants, infection is likely to occur if environmental conditions and the physiological status of the plant favor disease development. It appears that when plants are under high temperature and low soil moisture stress, disease is likely to develop. Though rare, *Thielaviopsis* has been found in peat-based potting mixes (Graham and Timmer 1991).

Management

- ***Cultural practices*** – Start with healthy, pathogen-free cuttings and seedlings. If the potting mix contains soil, pasteurize it before use. Black root rot is less severe

in soil with a pH of 5–5.5, but growers should exercise caution when lowering the pH outside of the optimal range for plant growth.

- ***Fungicides*** – They are available for suppressing *Thielaviopsis* but should be used routinely for this pathogen only if the operation has a history of crop losses from it. Discard infected plants.

2.3 Cercospora Leaf Spot (*Cercospora Sp.*)

Geographic occurrence and impact. The pathogen is reported to infect *Pelargonium* in North America and the Philippines (Daughtrey et al. 1995) and has been found on *Geranium*.

Symptoms/signs. Initially small (1–5 mm; 1/8–1/4 in.) light green spots enlarge and become gray (Fig. 2). Dark fruiting structures develop within the gray area. As the spots enlarge, they may have a yellow area surrounding them, and the spots may merge. Infected flowers often do not open (Engelhard 1993).

Biology and epidemiology. Little is known of the disease cycle of this pathogen in *Pelargonium*, but it is known that the pathogen is wind disseminated and that infection is greatly favored by wetness of the foliage (Daughtrey et al. 1995).

Management. Irrigate plants in a manner that keeps water off the foliage. Remove infected foliage. Apply a fungicide to protect plants. The chlorothalonil and mancozeb fungicides are effective against Cercospora diseases. Check production guides for your region.

2.4 Gray Mold (Botrytis Blight; *Botrytis cinerea Pers.: Fr.*)

Geographic occurrence and impact. This disease is probably the most common and widespread one in *Pelargonium* production, and the pathogen infects many



Fig. 2 Cercospora leaf spot on Pelargonium (Robert McGovern © 2017. All Rights Reserved.)

additional plants (Daughtrey et al. 1995). In fact, the very high susceptibility of *Pelargonium* results in *Pelargonium* being a significant harbor of *Botrytis* for other greenhouse crops (Hausbeck and Pennypacker 1991a; Hausbeck and Pennypacker 1991b).

Symptoms/signs. Flowers turn dark and fall prematurely. Where infected petals land on leaves, *Botrytis* grows from this nutrient source, and an irregular leaf spot forms on the green leaf (Fig. 3). Cuttings develop a dark brown to black rot (Fig. 4) near the base (also see Pythium root rot, blackleg). Abundant, dusty, gray masses of spores form on infected tissue if the humidity is high or if infected tissue is placed in a container with moisture.

Biology and epidemiology. All aboveground parts of the plant are susceptible, and the fungus can infect intact tissue or through wounds. Seedlings and mature plants are susceptible. Infection occurs most readily when the fungus has a food base from which it attacks, such as fading flowers or senescent leaves. High relative humidity, moisture on the plant, and temperatures between 18 °C and 25 °C (64 °F and 77 °F) greatly favor *Botrytis* infection (Jarvis 1980). The spores are readily spread by air



Fig. 3 *Botrytis* infected petal has fallen on a leaf. *Botrytis* grows from this food base into the healthy leaf (The Pennsylvania State University Department of Plant Pathology and Environmental Microbiology © 2017. All Rights Reserved.)



Fig. 4 *Botrytis*-infected *Pelargonium* cuttings (The Pennsylvania State University Department of Plant Pathology and Environmental Microbiology © 2017. All Rights Reserved.)

currents, and any activity in the greenhouse that changes the relative humidity quickly or disturbs the plants can result in the release of spores (Hausbeck and Pennypacker 1991a, Hausbeck and Pennypacker 1991b).

Management

- ***Cultural practices*** – Heat and ventilate plants in a greenhouse setting, and space plants to ensure good air circulation and low humidity within the crop canopy. It is best to not crowd plants and to not hang plants above *Pelargonium* if those plants tend to drop faded flowers or if irrigating the pots above will result in excess irrigation water dripping onto the geraniums. Avoid unnecessarily damaging plants. Do not leave large stubs of tissue on the stock plant when taking cuttings. Remove and destroy fading flowers and leaves from the plant, the surface of the potting soil, from benches and greenhouse floors. Remove this material promptly from the greenhouse or place it in a closed container.
- ***Fungicides*** – Apply a fungicide. Exclusive use of one class of chemical, particularly systemic chemicals, can result in the selection of resistance to the chemical class. Resistance to benzimidazole fungicides is very widespread in *Botrytis* populations. Do not rely on only one chemical class (Elad et al. 1992; Gullino and Garibaldi 1987; Hausbeck and Moorman 1996; Moorman and Lease 1992).

2.5 Pythium Root Rot and Blackleg (*Pythium Sp.*)

Geographic occurrence and impact. Many species of *Pythium* are known to be pathogens of *Pelargonium*. *P. aphanidermatum*, *P. cryptoirregulare*, *P. irregulare*, *P. myriotylum*, and *P. ultimum* are recovered the most frequently, but *P. complectens* (*Phytopythium vexans*), *P. debaryanum* (*Globisporangium debaryanum*), *P. deliense*, *P. mamillatum* (*Globisporangium mamillatum*), *P. splendens* (*Globisporangium splendens*), and *P vexans* (*Phytopythium vexans*) have reported. These species can be found worldwide, but a particular species may be more important in a given region than other species. Pythium root rot and blackleg (rot of the base of cuttings) can cause extensive crop losses.

Symptoms/signs. Blackleg, the coal black rot of cuttings, first develops as brown water-soaked rot at the cut end of the tissue. The rot proceeds up the stem and kills the cutting rapidly. Seedling geraniums yellow and collapse when root rot occurs (Fig. 5). In the case of root rot of established plants, root tips appear translucent and water-soaked. The outer layers of root tissue strip off when plants are pulled from soil leaving the central core of vascular tissue bare. Usually, spherical spores can be found in root cells when examined microscopically. If *Pythium* is suspected but no spherical spores are observed in the tissue, plate the tissue on plain water agar, and *Pythium* usually grows out within 24 h. There are simple kits available commercially that can be used easily to detect the presence of *Pythium* sp. These immunoassay kits provide a result in less than 30 min.



Fig. 5 Pythium root and blackleg (Robert McGovern © 2017. All Rights Reserved.)

Biology/epidemiology. *Pythium* species are often found in field soil, sand taken from streams and rivers and can be found in pond and lake sediments, and dead roots of previous crops (Ivors and Moorman 2014). While some species are a problem in hydroponic systems, species of *Pythium* present in the clear water (not the sediment) of ebb and flood irrigation systems may not all be serious plant pathogens (Lanze 2015). It is important to know what species is present. *Pelargonium* most susceptible are those being grown at high fertilizer levels (Gladstone and Moorman 1989, Gladstone and Moorman 1990).

Management

- ***Cultural practices*** – Pythium root rot is difficult to control once rot has begun. Every effort should be directed toward preventing the disease before it begins by using a heat-pasteurized potting mix. The entire pile must be heated to 75°–80 °C (165°–180 °F) and held at that temperature for 30 min. Potting mixes heated for very long times or temperatures higher than 80 °C will result in the death of most beneficial organisms in the soil and create a "biological vacuum" that is readily filled by *Pythium* if the mix becomes contaminated in some way. If the commercial mix is purchased in sealed bags or bales, try to keep the covering on until used in order to prevent contamination. *Pythium* is easily introduced into pasteurized soil or soilless mixes by using dirty tools, dirty pots or flats, and dirty loading equipment, by walking on or allowing pets to walk on the mixes, and by dumping the mixes on benches or potting shed floors that have not been thoroughly cleaned and disinfected. Discard infected cuttings since affected rooted cuttings later develop root rot. Keep hose ends off the ground to prevent picking up contaminated soil. If pond or stream water is used for irrigation, be certain the intake pipe is well above the bottom so that sediment is not drawn in. If a known plant pathogenic species is found in the water, treatment may be required (refer to ► Chap. 9, "Sanitation for

Management of Florists' Crops Diseases," and pertinent chapters in Hong et al. (2014)). For example, slow sand filtration has been shown to be an effective, simple, and inexpensive method for removing *Pythium* from water. Heat, ultraviolet light, ozone, and chlorination can also be effective. The best of these methods is the one that can be used consistently and efficiently in an operation. Disinfect all bench surfaces, potting benches, tools, and equipment that will contact the potting mix. Periodically, thoroughly clean and disinfect ebb and flood reservoirs, benches, and flood and drain floors.

- ***Fungicides and biocontrols*** – In a greenhouse operation with a history of Pythium root rot, apply a fungicide or a biological control agent as early in the cropping cycle as possible. Biological agents are generally applied to the potting mix before, during, or immediately after transplanting. However, read the label of the product to obtain information on exactly when the agent should be applied. In some cases, the agent must be in the potting mix for several days before plants are put in the mix in order to avoid phytotoxicity. Some biological control agents can be applied to plants in plug trays before transplanting. If chemical fungicides are also to be used, a general guideline is to not apply any chemical pesticides to the potting mix 10 d before and 10 d after applying the biological control agent. Biological control agents and fungicides may have to be applied more than once in order to maintain adequate protection for several weeks. They do not cure a plant once the plant is infected. Populations of some *Pythium* species have resistance to fungicides (Moorman and Kim 2004; Moorman et al. 2002).

2.6 Rhizoctonia Root and Crown Rot (*Thanatephorus cucumeris* (*Rhizoctonia solani* Kuhn) *Rhizoctonia Sp.*)

Geographic occurrence and impact. Although the pathogen is well known worldwide, this disease is not widespread in *Pelargonium* production, particularly with the use of soilless potting mixes.

Symptoms and signs. A brown, dry rot develops at the base of cuttings (Manning et al. 1973) or seedlings are killed (Powell 1993). When infected tissue is incubated on a moist surface in a closed container, threads of fungal hyphae grow from the infected tissue directly down to the moist surface in 24 h.

Biology and epidemiology. Little is known of this disease. It is reported that drought and high soluble salts renders plants more susceptible to the pathogen (Powell 1993). This species infects a wide range of host plants.

Management. Every effort should be directed toward preventing the disease before it begins by using a heat-pasteurized potting mix. The entire pile must be heated to 75°–80 °C (165°–180 °F) and held at that temperature for 30 min. If this pathogen

has been a problem before in production, the application of a fungicide is recommended in order to protect plants not yet infected.

2.7 Rust (*Puccinia pelargonii-zonalis Doidge, Puccinia granularis, Puccinia morrisoni*)

Geographic occurrence and impact. The pathogen was first found in South Africa (Doidge 1926) but now can be found sporadically wherever *Pelargonium* is grown. However, because it is readily recognized and prevented, it is generally not a major problem.

Symptoms/signs. Chlorotic specks on the upper leaf surface appear directly opposite pustules of rust-colored spores on the underside of leaf. Spores erupt in concentric rings forming a "target" spot (Fig. 6).

Biology/epidemiology. This rust requires only one host (autoecious; there is no alternate host plant), and it produces only two different types of spores: brown single-celled urediniospores and pale brown, two-celled teliospores. When temperatures are between 16 °C and 21 °C (61 °F and 70 °F) and there is water on the leaves, the fungus can continuously produce urediniospores that germinate and infect plants through the stomata (Harwood and Raabe 1979). The urediniospores can be spread by wind or splashing or on workers' hands and clothing. Apparently, teliospores form rarely.

Management. Purchase rust-free cuttings. Irrigate plants in a manner that keeps water off the foliage. Discard unwanted geraniums at season's end unless they are to be kept under observation and treated with fungicides if necessary. When infected plants are found, discard them and treat the remaining plants with a fungicide.



Fig. 6 Geranium rust on *Pelargonium* (The Pennsylvania State University Department of Plant Pathology and Environmental Microbiology © 2017. All Rights Reserved.)

2.8 Sclerotinia Crown Rot or Cottony Stem Rot (*Sclerotinia sclerotiorum (Lib.) de Bary*)

Geographic occurrence and impact. This pathogen infects many different bedding plants in the seeding stage, including *Pelargonium*, where growing conditions are hot and humid. It is generally not a problem in greenhouses in temperate regions.

Symptoms/signs. White, cottony fungal growth develops quickly near the soil line. As the mycelium ages, black sclerotia (up to 2–3 mm in size) develop on the infected tissue.

Biology and epidemiology. The pathogen is carried in soil or on infected plants. It survives as sclerotia in crop debris and in soil.

Management. If soil is a component of the potting mix, the potting mix should be heat pasteurized first. Benches, potting areas, and equipment used to move potting mix should be cleaned and disinfected periodically. Crop debris should be collected and removed from the growing area, particularly between cropping cycles. Infected plants should be discarded (Strider 1985).

2.9 Verticillium Wilt (*Verticillium albo-atrum* Reinke and Berthe or *V. dahliae Kleb.*)

Geographic occurrence and impact. This is a problem primarily where plants are grown outdoors exposed to soil. But if infected plants or infested soil are shipped from place to place, this is an avenue for long distance spread to wherever *Pelargonium* is grown.

Symptoms/signs. Middle and upper leaves yellow, collapse, dry, and fall. Vascular tissue of affected stems is browned or blackened. Symptoms are readily confused with those of bacterial blight. Sometimes, infected plants are severely stunted before any other symptoms are exhibited (Strider 1985).

Biology and epidemiology. The pathogen survives well in soil and in the tissues of many species of infected plants.

Management. Purchase culture-indexed cuttings. Use pasteurized potting mixes. Destroy infected plants.

Fungus or fungus-like organisms reported to be associated with Pelargonium [see Farr et al. (1989) and Farr, and Rossman Fungal Databases, Systematic Mycology and Microbiology Laboratory, ARS, USDA. Retrieved from http://nt.ars-grin.gov/fungaldatabases/]:

Aecidium pelargonii.
Alternaria alternata.
Alternaria pelargonii.
Alternaria tenuis (*Alternaria alternata*).
Alternaria tenuissima.
Armillaria fuscipes.
Armillaria heimii.
Armillaria mellea.
Ascochyta sp.
Aspergillus fischeri.
Aspergillus fischerianus (*Aspergillus fischeri*).
Bipolaris maydis.
Botryosphaeria berengeriana (*Botryosphaeria dothidea*).
Botryosporium pulchrum.
Botryotinia fuckeliana (*Botrytis cinerea*).
Botrytis pelargonii.
Calonectria morganii.
Cladosporium fumago (*Fumago salicina*).
Cladosporium sphaerospermum.
Coleroa circinans.
Colletotrichum gloeosporioides.
Coniella australiensis.
Coniothyrium trabutii.
Cryptovalsa ampelina.
Cylindrocarpon olidum (*Thelonectria olida*).
Cylindrocladiella camelliae.
Cylindrocladiella parva.
Cylindrocladium scoparium.
Cyphella pelargonii.
Diaporthe medusae (*Diaporthe rudis*).
Diaporthe rudis.
Discohainesia oenotherae (*Pilidium lythri*).
Drechslera setariae (*Bipolaris setariae*).
Erysiphe communis (*Erysiphe pisi* var. *pisi*)
Fibroidium pelargonii.
Fusarium oxysporum.
Fusarium pelargonii.
Fusarium semitectum (*Fusarium incarnatum*).
Gloeosporium pelargonii.
Glomerella cingulata (*Colletotrichum gloeosporioides*).
Helicobasidium purpureum.
Leptosphaeria elaoudi.
Leptosphaeria pelargonii.
Macrophomina phaseoli (*Macrophomina phaseolina*).
Macrosporium geraniaceae.
Macrosporium pelargonii.

Oidium sp.
Patellaria atrata (*Lecanidion atratum*).
Peroneutypa heteracantha.
Pestalotia pelargonii.
Pestalotia versicolor (*Pestalotiopsis versicolor*).
Pestalotiopsis sp.
Phaeosolenia pelargonii.
Phyllosticta geraniicola.
Physalospora geranii.
Phytophthora cactorum.
Phytophthora drechsleri.
Phytophthora palmivora var. *palmivora* (*Phytophthora palmivora*)
Phytophthora x *pelgrandis.*
Pilidium concavum (*Pilidium lythri*).
Pleosphaerulina sp.
Pleospora herbarum.
Pleospora phaeocomoides.
Rhizoctonia solani.
Schizophyllum commune.
Sclerotiopsis sp.
Sclerotium sp.
Septoria canberrica.
Septoria geranii.
Septoria pelargonii.
Sphaeropsis sp.
Sphaerotheca fugax (*Podosphaera fugax*).
Sphaerulina pelargonii.
Sporotrichum epiphyllum.
Stemphylium solani.
Tuberculina pelargonii.
Uredo pelargonii.

3 Bacterial and Phytoplasma Diseases

Where possible, the scientific names used are those accepted by the International Society of Plant Pathology Committee on the Taxonomy of Plant Pathogenic Bacteria (Bull et al. 2010).

3.1 Bacterial Blight (*Xanthomonas hortorum* pv. *pelargonii*; Formerly, *Xanthomonas campestris* pv. *pelargonii*)

Geographic occurrence and impact. At one time, this was the most serious disease of *Pelargonium* worldwide. The pathogen is spread in *Pelargonium* production and throughout the world wherever infected stock plants or cuttings are shipped.

Losses can approach 100% in some cultivars. Since first reported in the early 1900s, this disease has increased or decreased depending upon the vigilance of growers, diligence in eliminating infected stock plants, and acceptance of growing plants from culture-indexed cuttings. Over the decades of *Pelargonium* production, outbreaks of this disease have severely damaged the reputations of specialty propagators. Seedling geraniums are susceptible to the pathogen but are free of the disease unless grown in close proximity to vegetatively propagated *Pelargonium*. Although perennial *Geranium* is not severely affected; they can be an ongoing source of the pathogen for *Pelargonium* (Nameth et al. 1999).

Symptoms/signs. (Figs. 7, 8, and 9). Small spots [less than 6.4 mm (1/4 in.)] develop on the underside of the leaf and become sunken and well defined. The leaf wilts and dies as the bacteria spread through water-conducting vessels of veins and petioles. V-shaped areas form with the wide part of V on the leaf margin and point of the V on veins. These symptoms are seen on both *Pelargonium* and *Geranium*. On *Pelargonium*, the leaf spotting may occur late in disease development. Prior to leaf spotting, lower leaves may wilt at the margins, while the blade and petiole remain turgid. As wilt progresses, the entire leaf collapses. The vascular tissue of the main stem on the side of the affected portion of the plant is discolored gray to brown. Lower leaves die and fall. Note that although many of these symptoms can be confused with those caused by *Ralstonia* (bacterial or southern wilt), *Ralstonia* does not cause leaf spots in *Pelargonium*. There are simple, rapid test immunological kits commercially available for testing plants for both *Xanthomonas hortorum* pv. *pelargonii* and for *Ralstonia solanacearum*, and there are molecular methods for identifying the presence of *Xanthomonas hortorum* pv. *pelargonii* in plant tissues (Sulzinski et al. 1996; Sulzinski et al. 1997). Note that other species are known to cause leaf spots on *Pelargonium* but that they are relatively minor problems (Rockey et al. 2015).



Fig. 7 Bacterial blight leaf spot (*Xanthomonas*) on *Geranium* (The Pennsylvania State University Department of Plant Pathology and Environmental Microbiology © 2017. All Rights Reserved.)



Fig. 8 Bacterial blight on ivy geranium (*Pelargonium peltatum*) (The Pennsylvania State University Department of Plant Pathology and Environmental Microbiology © 2017. All Rights Reserved.)



Fig. 9 Bacterial blight – vascular discoloration (*Xanthomonas*) in *Pelargonium* (The Pennsylvania State University Department of Plant Pathology and Environmental Microbiology © 2017. All Rights Reserved.)

Biology and epidemiology. The pathogen survives in infected plants and therefore is primarily a problem in *Pelargonium* propagated vegetatively. Disease develops most rapidly when temperatures are between 20 °C and 30 °C (68 °F and 86 °F). When the pathogen is splash dispersed and enters leaves through hydathodes, bacteria enter the vascular tissue and move throughout the plant. The only tissues not infected are the meristem tips where the vascular tissue is not fully differentiated. Viable bacteria survive on moist plant surfaces and within infected tissue and in the vascular tissue. At temperatures between 10 °C and 15 °C (50 °F and 50 °F), bacterial activity is greatly suppressed, and infected plants may remain symptomless. This can lead growers to believe that stock plants are not infected. However, as temperatures rise, symptoms become apparent. In the meantime, if cuttings are taken from infected stock plants, the cuttings may root but will eventually exhibit symptoms. In addition to splash dispersal, the bacteria can be spread on workers' hands and on knives used for taking cuttings (Munnecke 1954).

Management. Purchase culture-indexed cuttings or grow plants from seed. Immediately discard infected plants after a positive diagnosis is made. Irrigate plants in a manner that keeps water off the foliage. Discard all unwanted geraniums at season's end if they will not be examined periodically for symptoms. If cuttings are taken with knives, the knives should be washed and thoroughly disinfested after use on one plant and before use on another plant. If branches to be used for cuttings are broken off plants by hand, workers should wash their hands frequently or wear disposable gloves and disinfect them after use on one plant and before use on another plant.

3.2 Bacterial Fasciation (*Rhodococcus fascians*, Formerly Known as *Corynebacterium fascians*, *Bacterium fascians*, and *Phytomonas fascians*)

Geographic occurrence and impact. Although the bacteria can persist in infested soil, the probable main source of the pathogen in *Pelargonium* production is through the propagation from infected stock plants. For that reason the disease may appear anywhere Pelargonium is produced vegetatively propagation is grown (Strider 1985).

Symptoms/signs. Short, thick, fleshy, leafy galls formed at base of main stems at or below soil level are pale green or green-yellow (Fig. 10). The rest of the plant appears healthy.

Biology and epidemiology. *Rhodococcus fascians* survives associated with live plants or in soil. Many plants in addition to *Pelargonium* are susceptible to this pathogen. It is believed that *Rhodococcus fascians* does not require a wound to enter the plant. The pathogen is very difficult to grow in culture from infected plant tissues.

Management. Purchase culture-indexed plants. Discard infected stock plants and cuttings and infested media.



Fig. 10 Bacterial fasciation on *Pelargonium* (The Pennsylvania State University Department of Plant Pathology and Environmental Microbiology © 2017. All Rights Reserved.)

3.3 Bacterial Wilt or Southern Wilt (*Ralstonia solanacearum*, Formerly Known as *Burkholderia solanacearum*, and *Pseudomonas solanacearum*)

Geographic occurrence and impact. *Ralstonia solanacearum* attack almost 200 plant species in 33 different plant families. The common name for the diseases this organism causes varies with the host that is attacked and geographic location. It is sometimes called southern wilt (in the northern hemisphere) or bacterial wilt. This bacterium is noted for diseases caused outdoors in land areas bounded by 45°N and 45°S latitudes where rainfall averages above 100 cm/year (39 in./year), the average growing season exceeds 6 months, the average winter temperatures are not below 10 °C (50 °F), the average summer temperatures are not below 21 °C (70 °F), and the average yearly temperature does not exceed 23 °C (72 °F) (Lucas 1975).

Symptoms/signs. Lower leaves wilt, yellow, and fall. Vascular tissue of affected stems turns brown or black; roots may be discolored and rotted (Figs. 11 and 12). Note that although these symptoms can be confused with those caused by *Xanthomonas* (bacterial blight), *Ralstonia* does not cause leaf spots in *Pelargonium*. Slimy, sticky ooze forms tan-white to brownish beads where the vascular tissue is cut. When an infected stem is cut across and the cut ends held together for a few seconds, a thin thread of ooze can be seen as the cut ends are slowly separated. If one of the cut ends is suspended in a clear container of clean water, bacterial ooze will form a thread in the water. Back lighting of the container helps reveal the ooze.

Biology and epidemiology. Although the primary location of survival in the environment is in crop and weed hosts, the bacteria can also survive in soil. They can be readily spread through the movement of contaminated soil and infected vegetatively propagated plants, in contaminated irrigation water, and on the surfaces of tools (cutting knives) and equipment used to work with the plants, and on soiled



Fig. 11 Bacterial wilt (Ralstonia) (Robert McGovern © 2017. All Rights Reserved.)



Fig. 12 Bacterial wilt (Ralstonia) (Robert McGovern © 2017. All Rights Reserved.)

clothing. Populations within this genus and species can be divided into races and biovars based on differing host ranges, biochemical properties, susceptibility to bacteria-infecting viruses (phages), and serological reactions. Race 1 is endemic to North America where it attacks many floricultural and vegetable bedding plant crops including *Pelargonium*, *Catharanthus*, *Impatiens*, *Ageratum*, *Chrysanthemum*, *Gerbera*, *Tagetes*, *Zinnia*, *Salvia*, *Capsicum*, *Lycopersicon*, *Nicotiana*, *Petunia*, *Solanum melongena* (eggplant), *Tropaeolum* (nasturtium), and *Verbena*. Race 3 is tropical in distribution and does not occur naturally in North America. Race 3 biovar 2 infects potato (Williamson et al. 2002) and other hosts including *Pelargonium*, tomato, peppers, eggplant, bean, and beet. Weed hosts include black nightshade, climbing nightshade, horsenettle, Jimson weed, purslane, mustards, lamb's-quarters, and bitter gourd. The bacteria can infect through roots and through any fresh wounds. The bacterium can be difficult to work with in the laboratory because it quickly loses pathogenicity and viability in artificial culture. Race 3 biovar 2 is considered a quarantinable pathogen.

Management. Growing and propagating from pathogen-free plant material is the main way to avoid problems with *Ralstonia*, regardless of the race and biovar involved. It is imperative that propagators use pathogen-free potting soil or other media, establish stock plants that are tested and known to be free of the bacteria, train workers handling the stock plants in methods and procedures that prevent the pathogen from contaminating the potting soil or coming in contact with the stock plants, and then maintaining this system throughout the propagation phase of crop production. Do not bring ground-planted geraniums into the production area or propagate from them. Destroy infected plants. There are no chemicals or biological agents that adequately control *Ralstonia*. Infected plants MUST be discarded as soon as possible. The purchaser of cuttings or prefinished plants should isolate all new, incoming plants as if the health of the plants were unknown, even if the plants have been certified as healthy. New plants must not be commingled or dispersed among

other plants in the greenhouse from other sources. This procedure is crucial because keeping plants originating from one source together limits the area that may need to be quarantined, sanitized, or isolated should *Ralstonia solanacearum* be found. If the pathogen is found in plants being irrigated using a recycling water system (such as ebb and flood), all susceptible plants in the system are at risk to infection. New, incoming plants should not be put into such a system being used for any other plants until their health is verified, and it is known that they do not harbor the pathogen.

3.4 Crown Gall (*Rhizobium tumefaciens*, Formerly *Agrobacterium tumefaciens*)

Geographic occurrence and impact. The bacteria can persist in infested soil, but the probable main source of the pathogen in *Pelargonium* is through the propagation of new plants from infected stock plants. Although this disease may appear anywhere *Pelargonium* are produced by vegetative propagation, it is usually recognized by the propagator and infected stock plants, and cuttings are discarded. Thus, the disease is very seldom a problem except where a local grower maintains the pathogen in a favorite stock plant (Strider 1985). An understanding of this pathogen's biology and its disease cycle has significantly contributed to modern agriculture in the area of biotechnology and genetic engineering (Nester et al. 2005).

Symptoms/signs. Spherical white- to light tan-colored galls develop near the soil line on the roots and stems of infected plants.

Biology and epidemiology. *Rhizobium tumefaciens* survives in infested soil and in infected plants. The pathogen is spread through the movement of these materials from place to place. Wounding of plants through taking cuttings allows entry of the pathogen.

Management. Growing and propagating from pathogen-free stock plants is the main way to avoid crown gall. Propagators must use pathogen-free potting soil or other media, establish stock plants that are known to be free of the bacteria, and destroy infected plants. Ground-planted geraniums should not be brought into the production area nor should cuttings be taken from them.

3.5 Pseudomonas Leaf Spot (Bacterial Leaf Spot; *Pseudomonas cichorii*)

Geographic occurrence and impact. This disease was first described on *Pelargonium* X *hortorum* being grown in Florida, USA, under warm, very humid conditions (Engelhard et al. 1983). In greenhouses or outdoors, the disease can be very damaging particularly if plants are irrigated in a manner that puts water directly on the foliage. Long distance spread of the pathogen occurs when infected plants are shipped from place to place (Daughtrey et al. 1995). The pathogen can infect many different plants in addition to *Pelargonium*.



Fig. 13 Pseudomonas leaf spot on *Geranium* (The Pennsylvania State University Department of Plant Pathology and Environmental Microbiology © 2017. All Rights Reserved.)

Symptoms. Water-soaked spots, 5–10 mm (1/4–1/2 in.) in diameter, form on leaves (Fig. 13). Spots become dark brown to black and irregularly shaped. A yellow halo may surround each spot (Engelhard et al. 1983). Under ideal conditions, spots merge, and the entire leaf may be affected. Infected flower buds and sometime the entire inflorescence are blackened.

Biology and epidemiology. Spotting severity increases with temperature between 16 °C and 28 °C (61 °F and 82 °F) but is greatly inhibited at temperatures above 28 °C. High relative humidity greatly favors infection and spot enlargement (Jones et al. 1984). Sprinkler irrigation and rain splash the pathogen among leaves and flowers and provide the water on the plant tissues required by the bacteria for infection.

Management. Purchase pathogen-free cuttings. It is of utmost importance to water plants in a manner that keeps leaf surfaces dry and to maintain low relative humidity among the plants. Discard infected plants and treat the remaining plants with a bactericide.

3.6 "*Candidatus* Phytoplasma Asteris"

Yellows diseases caused by phytoplasma can occur on *Pelargonium*. For instance, *Candidatus* phytoplasma asteris (Group 16SRI) was reported to infect geranium in Pakistan for the first time by Fahmeed et al. (2009).

4 Viral Diseases

The EPPO Panel on "Certification of Ornamentals" developed procedures for the production of healthy carnation, pelargonium, lily, narcissus, chrysanthemum, tulip, crocus, iris, begonia, impatiens, rose, freesia, hyacinth, kalanchoe, and petunia. A

combination of thermotherapy, meristem tip culture and virus indexing can be used in geranium to eliminate viruses (Horst et al. 1977; Horst and Klopmeyer 1993).

In the USA, certification programs are managed by the major commercial propagators. Pelargonium and other major flower crops are tested for the presence of the most prevalent viruses either by in-house plant pathologists or by companies that provide testing services. In the USA, phytosanitary certificates are required for intra- and interstate plans movement. Data on pathogens of high consequence, including viruses, are recorded in the USA by the National Plant Diagnostic Network (https://www.npdn.org/home).

Where available, references for viruses are from Adams and Antoniw (2006).

4.1 *Arabis Mosaic Virus* (ArMV), *Tobacco Ringspot Virus* (TRSV), *Tomato Ringspot Virus* (ToRSV), and *Artichoke Italian Latent Virus* (AILV)

Geographic occurrence and impact. ArMV (Smith and Markham 1944) has been detected in 13 states in the USA where it has been the object of quarantine, but it is not widespread or established outside Europe. ArMV has a wide host range. TRSV (Fromme et al. 1927) geographic distribution is mainly in northern USA and China, but it has been found in Europe and Australia. More than 17 plant families are susceptible to the virus (Price 1940). ToRSV (Price 1936) is originally from the west coast of the USA (Frazier et al. 1961), and it is probably distributed to ornamental plants in many parts of the world. It can infect plants in 35 families. AILV (Majorana and Rana 1970; Vovlas et al. 1971) is found in Southern Italy and Bulgaria. AILV can infect a number of woody and herbaceous plants, including weeds.

Symptoms/signs. ArMV is associated with mosaic symptoms in pelargonium. Pelargonium ringspot is a disease associated with both TRSV and ToRSV, but little evidence is found to confirm if the disease is the result of double or single infections. In fact, since the two viruses can infect the same hosts in the same geographic area, they have been often confused. ToRSV has also been associated with ringspots and mosaic symptoms in pelargonium. AILV in *Pelargonium zonale* produces severe leaf malformations and reduction in size, elongation of petioles, and stunted plant growth (Vovlas 1974).

Biology and epidemiology. These three viruses are *Nepoviruses* in the family Secoviridae. Their genomes consist of two linear ss+RNA segments. ArMV is nematode transmitted (Fritzsche and Schmidt 1963; Harrison and Cadman 1959; Jha and Posnette 1959) and seed transmitted (Lister and Murant 1967, Murant and Lister 1967) and can be transmitted by dodder (*Cuscuta* sp.). Nematodes lose the virus during molting and do not transmit it to their progeny.

TRSV is transmitted by the nematode *Xiphinema americanum* (McGuire 1964), by pollen and seeds, as well as by many insects (Dunleavy 1957; Komuro and Iwaki 1968; Messieha 1969; Schuster 1963) and mites (Thomas 1969) in a nonspecific

manner. TRSV does not replicate in its nematode vector, is lost during molting, and is not transmitted to the nematode progeny. TRSV and ToRSV are not serologically related, and serological tests can be used to confirm their identification. ToRSV is transmitted by the nematode *Xiphinema americanum* (Téliz et al. 1966), but seed transmission has also been reported in some plant species. Small ssRNA satellite viruses have been associated with some strains of ToRSV, but scant information is available on the presence of these satellite viruses in pelargonium. AILV is transmitted by the nematode *Longidorus apulus* (Rana and Roca 1976), and it is not serologically related to any other *Nepovirus*.

Management. Management of these viruses involves the use of virus-free seed and other propagative material, nematode avoidance through soil and water treatment where necessary, and elimination of weedy hosts.

4.2 *Beet Curly Top Virus* (BCTV)

Geographic occurrence and impact. The virus originated from the Eastern Mediterranean basin and is common in the Western part of the USA from Mexico to Canada (Bennett and Tanrisever 1957). Today it is present also in Africa, Asia, and Central and South America. BCTV has a broad host range and can infect plant species in 44 families (Bennett 1971).

Symptoms/signs. The disease caused by BCTV on pelargonium, called leaf cupping, is very severe, with pronounced tissue yellowing, leaf curling, and distortion.

Biology and epidemiology. BCTV is a member of the family Geminiviridae with an ss+DNA genome contained in geminate particles. Two species of leafhoppers from the arid and semi-arid regions, *Circulifer tenellus* described by Ball (1909) and *C. opacipennis* (Kheyri and Alimoradi 1969; Stahl and Carsner 1923), are vectors of the virus, and the virus is phloem limited and does not multiply in its insect vectors. The virus is also not transmitted through the vector eggs and requires a latent period before transmission to plants. The virus is present in the seed but cannot invade the embryo.

Management. Control of this virus consists of controlling the insect vector, excluding infected plants, and using clean propagative material. Usually plants infected with this virus do not survive and thus are not found commercially.

4.3 *Impatiens Necrotic Spot Virus* (INSV) *and Tomato Spotted Wilt Virus* (TSWV)

Geographic occurrence and impact. INSV (Law and Moyer 1990) and TSWV (Samuel et al. 1930) together with their thrips vectors are prevalent worldwide in

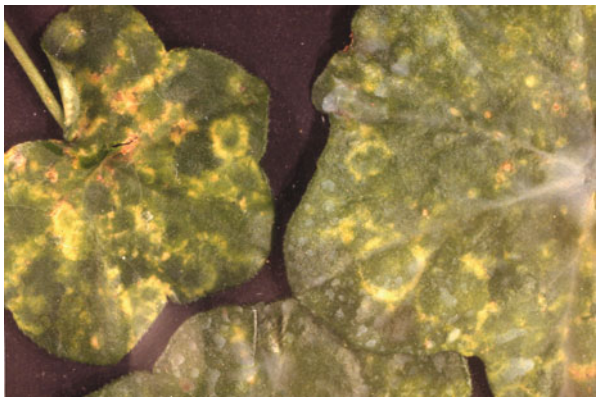


Fig. 14 Symptoms of INSV (Jay Pscheidt © 2017. All Rights Reserved.)

many plant families and in hundreds plant species, making their control particularly challenging.

Symptoms/signs. Symptoms associated with INSV and TSWV are mosaic, mottling, stem and leaf necrosis, ringspots on leaves, leaf deformation, and stunted growth (Fig. 14). Symptomatology is driven by plant phenology and environmental conditions.

Biology and epidemiology. INSV and TSWV are in the family Bunyaviridae. These ssRNA viruses have tripartite genomes, with negative or ambisense orientation. INSV and TSWV are transmitted by thrips in a propagative (persistent manner) (Ullman et al. 1993; Wijkamp et al. 1993); thus, they can replicate in their plants as well as in their insect hosts.

Management. One of the best ways to manage these viruses is to exclude thrips from greenhouses and to reduce their numbers, since thrips need a relatively long acquisition and transmission time, and since the viruses encounter a latent period in the vector prior to becoming transmissible. Unfortunately, thrips are now resistant to many insecticides (Brødsgaard 1994; Zhao et al. 1995). Weed management is also necessary (Bond et al. 1983; Cho et al. 1986; Kobatake et al. 1984). (For additional information, refer to ▶ Chap. 4, "Insect Management for Disease Control in Florists' Crops").

4.4 *Pelargonium Zonate Spot Virus* (PZSV), *Cucumber Mosaic Virus* (CMV)

Geographic occurrence and impact. PZSV is geographically restricted to Southern Italy. The virus can infect tomato and artichoke and is common in weeds. CMV is found worldwide, has an extremely broad host range, and can infect species in more than 100 plant families (Zitter and Murphy 2009).

Symptoms/signs. Symptoms induced by PZSV are leaf malformations, puckering, and zonate yellow bands (Martelli and Cirulli 1969; Quacquarelli and Gallitelli 1979). CMV in geranium causes flower breaking disease.

Biology and epidemiology. PZSV and CMV (described by Doolittle 1916; Jagger 1916, and more recently by Jacquemond 2012; Palukaitis and Garcia-Arenal 2003) have tripartite ss+RNA genomes. Both viruses are in the family Bromoviridae. PZSV belong to the *Anulavirus* genus (Gallitelli 1982) and CMV to the *Cucumovirus* genus (Wildy 1971). PZSV is seed and pollen transmitted but apparently not vector-transmitted. Thrips can serve as carriers of the infected pollen from host to host. CMV is aphid transmitted (Kennedy et al. 1962) in a non-propagative (nonpersistent) manner (Hoggan 1933; Simons 1955; Watson and Roberts 1939), and it can be seed transmitted in some plant species (Neergaard 1977).

Management. PZSV can be controlled using clean propagative material. Control for CMV is particularly difficult, since the virus is common on many plant families, and it can be transmitted by multiple aphid species. Because aphids transmit CMV readily during probing, aphid control is particularly important for reducing the spread of the virus. In open field, the use of mineral oil to discourage aphid feeding has been used especially in Europe, but thus far its use in a greenhouse setting has been limited.

4.5 *Tobacco Mosaic Virus* (TMV) and *Tobacco Rattle Virus* (TRV)

Geographic occurrence and impact. These viruses can infect a variety of plants in 30 families for TMV (Shew and Lucas 1991) and more than 50 (Horváth 1978; Noordam 1956; Schmelzer 1957; Uschdraweit and Valentin 1956**)** for TRV in Europe, Japan, New Zealand, and North America.

Symptoms/signs. Symptoms attributed to TMV (Siegel and Wildman 1954) are often caused by other tobamoviruses, and many tobamovirus species were once classified as strains of TMV. Symptoms on pelargonium are reported to be mosaic and small light colored or brown lesions on leaves.

Biology and epidemiology. TMV and TRV belong to the Virgaviridae family. These viruses have an ss+RNA genome. TMV is occasionally transmitted by chewing insects, but most commonly, it is mechanically spread (Harris and Bradley 1973; Lojek and Orlob 1969); in fact TMV virions are extremely stable. TMV can persist in the soil, probably on plant debris, and can infect roots. It can penetrate wounded embryos from the infected seed coat (Broadbent 1965). TMV is a special concern in greenhouses, where it can be very hard to eradicate (Broadbent and Fletcher 1963). TRV is transmitted by nematodes, *Paratrichodorus* spp. and *Trichodorus* spp., (Taylor and Brown 1997), and pelargonium can become infected with this virus if grown in soil infested with viruliferous nematodes. TRV is also transmitted mechanically and by seed in certain hosts.

Management. Sanitation is the best TMV and TRV control method. Sanitation practices include the use of clean tools and pots, disinfected soil, virus-free seeds, and prompt removal of infected plants. Control of nematodes is essential to limit TRV spread; thus, the use of clean irrigation water and sanitized soil is recommended.

4.6 *Tomato Bushy Stunt Virus* (TBSV), *Tobacco Necrosis Virus* (TNV), *Moroccan Pepper Virus* (MPV), and *Pelargonium Leaf Curl Virus* (PLCV)

Geographic occurrence and impact. TBSV has a restricted natural host range and causes damage primarily on agricultural crops, while it can experimentally infect more than 120 plant species. It is geographically distributed in Europe, the Americas, and North Africa. TNV has a natural broad host range (Price 1940) that include tobacco, cucumber, melon, European strawberry, citrus, apple, and pear. It can experimentally infect more than 37 plant families, and it is distributed worldwide. PLCV (Hollings and Stone 1965) is common in the USA and Mediterranean region. It was detected in India in 2013 (Kumar et al. 2013). Susceptible host species are *Pelargonium zonale*, common bean, *Datura*, and *Chenopodium*. Some of these species are considered experimental hosts. MPV is present in Morocco, Iran, USA, Europe, and Central Asia (Wintermantel and Hladky 2013). Its host range includes tomato, lettuce, pepper, pelargonium, jimson weed, and lisianthus.

Symptoms/signs. These viruses induce *Pelargonium* leaf curl or crinkle, and *Pelargonium* necrotic spot, with stunted growth, leaf deformation and splitting, extensive spots first hyaline and then necrotic, and yellowing and necrosis of leaves. Symptoms are strongly influenced by temperature.

In pelargonium, TNV can cause symptomless infection that turns symptomatic under different light and temperature conditions. PLCV is the causal agent of leaf curl disease. The disease symptoms are leaf curl and yellow stellate spots that are more pronounced in winter and at lower temperatures and diminish in summer at higher temperatures.

Biology and epidemiology. These are monopartite ss+RNA viruses and are members of the *Tombusvirus* genus in the family Tombusviridae. TBSV (Ainsworth 1936, Bawden and Pirie 1938 Smith 1935) is soilborne and is transmitted by infected seed and soil. Defective interfering RNAs (DI-RNAs) and satellite RNAs have been associated with TBSV in its naturally infected plants (Galetzka et al. 2000, Gallitelli and Hull 1985), but were not investigated in pelargonium. TNV (Babos and Kassanis 1963; Bawden 1941; Smith and Bald 1935) is a soilborne virus and is transmitted by zoospores of the chytrid fungus *Olpidium brassicae* (Kassanis and MacFarlane 1964, Teakle 1962, Teakle and Gold 1963). TNV can support the multiplication of satellite viruses in some plants and serologically can be divided into groups, but group serology does not coincide with symptomatology. PLCV is

serologically related to TBSV. MPV (Makkouk et al. 1981) is distantly serologically related to TBSV. PLCV and MPV were described to be associated with soil infested by *Olpidium* spp. in Iran in 2011 (Rasoulpour and Izadpanah 2011). PLPV is an unassigned species in the *Tombusviridae*.

Management. Virus control requires the use of clean propagative material and soil, avoiding the spread of the vectoring zoospores by removing infested soil and water, and good sanitation practices. Research on virus-resistant varieties is needed.

4.7 *Pelargonium Flower Break Virus* (PFBV)

Geographic occurrence and impact. Virus described by Stone and Hollings (1973)**.** It infects six plant families and has limited host range. The virus was reported to be the most common viral pathogen of *Pelargonium* sp. in Western Europe (Krczal et al. 1995).

Symptoms/signs. It can infect *Pelargonium domesticatum* and can cause flower-breaking in several cultivars, but it remains usually symptomless on *P. peltatum* and *P. zonale*. Other symptoms can include flower streaking, stunting, and chlorotic spotting (Fig. 15). Seedlings of pelargonium-type "Nittany Lion" appear to be immune to PFBV (Kemp 1969).

Biology and epidemiology. PFBV is an ss+RNA virus belonging to the genus *Carmovirus* in the family Tombusviridae. PFBV is transmitted mechanically and by vegetative propagation, but does not seem to be transmitted by seed. Transmission of the virus in recirculating irrigation systems and by thrips via pollen has been demonstrated (Krczal et al. 1995). The virus is not serologically related to other viruses.



Fig. 15 Symptoms of PFBV (Robert Wick © 2017. All Rights Reserved.)

Management. Use of clean propagative material and discarding of infected plants is recommended. Hollings and Stone (1974) reported that *Pelargonium* can be freed from PFBV by thermotherapy, but plant survival rate was low.

Other viruses associated with Pelargonium:

Tombusviridae family:

Pelargonium chlorotic ring pattern virus.
Pelargonium leaf curl virus.
Pelargonium line pattern virus.
Pelargonium necrotic spot virus.

Pelargonium vein banding virus is a virus related to viruses in the *Badnavirus* genus, not yet approved as a species.

Pelargonium vein clearing is in the genus *Nucleorhabdovirus*. It has a monopartite ss-RNA genome and replicates in the plant cell nucleus.

Other viruses:

Cherry leaf roll virus.
Lilac chlorotic leaf spot virus.
Tomato black ring virus.

5 Nematode Diseases

Nematodes, including dagger nematode (*Xiphinema* sp.), foliar nematode (*Aphelenchoides* sp.), and root-knot nematode (*Meloidogyne* sp.), have been recorded to occur on Pelargonium but do not appear to be a widespread problem (Strider 1985).

6 Abiotic Diseases

A myriad of factors including air pollutants, misuse of plant growth regulators, and individual cultivar responses to environmental conditions can result in symptoms on *Pelargonium* (Freeman 1993).

6.1 Edema

Geographic occurrence and impact. This problem can occur wherever *Pelargonium* is grown. It is most damaging to ivy geranium (*Pelargonium peltatum*). (Balge et al. 1969; Digat and Albouy 1976)

Symptoms/signs. Small water-soaked pimples or blisters form on the underside of lower leaves (Fig. 16). The blisters become corky and brown as they enlarge. Severely affected leaves fall.



Fig. 16 Edema on ivy geranium (*Pelargonium peltatum*) (The Pennsylvania State University Department of Plant Pathology and Environmental Microbiology © 2017. All Rights Reserved.)

Biology and epidemiology. Edema development is favored when the weather is cool, cloudy, and humid while the soil is wet, particularly if mites are feeding on the plants. Although over the decades scientists have referred to this as a physiological disorder and have not linked it to a biotic pathogen, it is interesting to note that symptoms can be induced in otherwise healthy plants by injecting them with sap taken from a plant with edema (Digat and Albouy 1976).

Management. Spacing plants to provide good air circulation, using a well-drained potting mix, not overwatering during cool, cloudy weather, and mite suppression, are reported to help in preventing the development of edema.

6.2 Heat Stress

Geographic occurrence and impact. This problem can occur wherever *Pelargonium* is grown. Sensitivity to high temperatures varies greatly among cultivars (Strider 1985).

Symptoms/signs. Upper leaves and stems completely lose chlorophyll. Sometimes, only the center of the leaf is bleached. Usually only a few leaves are affected, but in severe cases, most of the leaves and stems may appear bleached.

Biology and Epidemiology. *Pelargonium* exposed to temperatures above about 28 °C/82 °F for more than 12 h develop symptoms. While some cultivars exhibit severe symptoms, others in the same greenhouse may be completely free of symptoms.

Management. Monitor temperatures in the area where cultivars known to be heat sensitive are grown. If it appears that temperatures could persist above 28 °C for more than 12 h, mist the sensitive cultivars so that evaporative cooling occurs.

However, many of the pathogens detailed elsewhere in this section are favored when moisture is on the foliage. Apply only enough moisture to slightly dampen the leaf surface and then evaporate completely in minutes. Do not mist to the extent that water begins to drip off the foliage.

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Diseases of Holiday Cacti: *Schlumbergera* and *Hatiora*

34

Robert L. Wick

Abstract

The most important diseases of *Schlumbergera truncata*, the Thanksgiving cactus, are Fusarium basal stem rot, Phytophthora root and stem rot, Bipolaris blight, and bacterial soft rot. *Impatiens necrotic spot virus* (INSV) and *Tomato spotted wilt virus* (TSWV) are a problem because infected plants cannot be sold. *Hatiora gaertneri* (Regel) Barthlott, the Easter cactus is relatively free of important diseases except for Fusarium stem rot which is very destructive to *Hatiora*.

Keywords

Fusarium oxysporum • *Bipolaris cactivora* • *Phytophthora nicotianae* • *Pectobacterium carotovora* • *Pythium* • INSV • TSWV

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R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_48

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1 Introduction

Schlumbergera truncata (Haworth) Moran, Thanksgiving cactus (also called Christmas cactus), is the most common *Schlumbergera* species under cultivation (Boyle 2007). The interspecific hybrid *S.* x *buckleyi* (Buckley) Tjaden (= *S. russelliana* x *S. truncata*) blooms a month later and is commonly called the Christmas cactus (Boyle 2007; Daughtrey et al. 1995). *Schlumbergera truncata* was formerly *Zygocactus truncatus* (Haw.) K. Schum. Members of the Cactaceae are native to Brazil; they are widely cultivated as a flowering potted plant along with *Hatiora gaertneri* (Regel) Barthlott, the Easter cactus. *Hatiora gaertneri* was previously known as *Rhipsalidopsis gaertneri* (Regel) Moran.

2 Fungal and Fungus-Like Diseases

2.1 Basal Stem and Root Rot [(*Fusarium oxysporum* Schlecht); (*Fusarium oxysporum* f. sp. *opuntiarum*)]

Geographic occurrence and impact. Fusarium basal stem and root rot of *S. truncata* was first reported in a proceedings in 1975 (Miller 1975) and formally published in 1980 (Moorman and Klemmer 1980). It has been reported in the USA, Argentina (Petrone et al. 2007), Italy (Lops et al. 2013), the Netherlands (Baayen et al. 2000), and Germany (Baayen et al. 2000; Gerlach 1972). Inoculation trials with *F. oxysporum* isolated from *S. truncata* failed to cause disease on *H. gaertneri* (Chase 1982). However subsequent research showed that an *F. oxysporum* isolated from *S. truncata* caused stem and root rot on several cultivars of *H. gaertneri* similar in extent to several cultivars of *S. truncata* (Mitchell 1987). Nevertheless there is a paucity of information regarding the occurrence of *F. oxysporum* root and stem rot on *H. gaertneri*. *Fusarium oxysporum* f. sp. *opuntiarum* was described from *Schlumbergera* in Germany in 1972. Subsequent reports of *F. oxysporum* on cactus did not include the f. sp. *opuntiarum* epithet; however, it is not known if *F. oxysporum* cited in the USA and Argentina is different or the same as *F. oxysporum* f. sp. *opuntiarum* reported in Europe. Fusarium basal stem and root rot is one of the most destructive diseases of *Schlumbergera*.

Symptoms/signs. The disease can manifest itself as a root rot, basal stem rot, and cladophyll lesion. Basal rot can occur on wounded or unwounded stems and is at first water soaked in appearance with reddish-brown margins. As the lesion ages, the tissue may dry to a papery tan (Moorman and Klemmer 1980). Lesions on wounded inoculated cladophylls of *S. truncata* can become up to 50 mm in diameter in 10 days (Chase and Yuen 1993). Lesions on inoculated *H. gaertneri* cladophylls ranged from about 5 to 20 mm in diameter.

Biology and epidemiology. *F. oxysporum* is the most widely dispersed of *Fusarium* spp. and occurs throughout the world in many types of soils, mostly as a soil saprophyte (Domsch et al. 1980; Leslie and Summerell 2006). A true soil-borne fungus survives in the soil by producing resting structures, chlamydospores, and active growth. Since *Hatiora and Schlumbergera* are vegetatively propagated, the widespread occurrence of *F. oxysporum* on cactus is probably due to movement of infected plant material and infested soil. *Fusarium oxysporum* is best known for the destructive nature of the vascular wilt diseases it causes on a wide variety of crops (Agrios 2005); more than 100 *forma speciales* and races have been described, most of which have a very narrow host range, often restricted to a single plant species or cultivar. While the descriptions of *F. oxysporum* disease of cactus often include wilt, wilt is apparently due to root rot and basal stem canker as opposed to vascular wilt. No one has demonstrated that *F. oxysporum* is a vascular pathogen of *Schlumbergera*.

Management

- ***Cultural Practices*** **–** New plants coming into the greenhouse should be carefully inspected for root rot, basal canker, and lesions on cladophylls. Diseased plants and plant debris should be removed from the presence of healthy plants. When growing plants in soil-less media, take care not to introduce field soil by hands, tools, and hose ends dropped on the floor. If the growing medium contains field soil, it must be pasteurized. Air drying of excised cladophylls up to 72 h before propagation did not prevent *Fusarium* from infecting (Mitchell 1987). With other *Fusarium* diseases, the application of nitrate as opposed to ammonium forms of nitrogen can suppress *Fusarium*. In addition, acid soils <6.0 can increase *Fusarium* disease severity so properly limed potting mixes may reduce disease severity.
- ***Resistance*** **–** *Schlumbergera truncata* cvs. Gold Charm, Christmas Flame, and Cambridge were found to be very susceptible to *Fusarium* (Chase and Yuen 1993). In a separate trial, cvs. Gold Charm and Norris were found to be the most susceptible (Mitchell 1987). Significant resistance to *Fusarium* was not found in either of the abovementioned trials.
- ***Chemical Control*** **–** Chlorothalonil dips and drenches provided good control of Fusarium basal rot but inhibited root growth. High rates of a combination of mancozeb and thiophanate-methyl were phytotoxic, but benomyl provided complete control (Mitchell 1987).

2.2 Bipolaris Blight *Bipolaris cactivora* (Petr.) Alcorn.; Previously *Drechslera cactivora* (Petr.) M. B. Ellis and *Helminthosporium cactivorum* Petr.

Bipolaris blight is also known as top rot, stem rot, and shattering

Geographic occurrence and impact. It has been reported in the USA and Europe (Daughtrey et al. 1995), probably more widely distributed. *Schlumbergera* is only moderately susceptible to this disease; younger plants are more susceptible than older ones. Most cultivars of *Hatiora gaertneri* are very susceptible to *Bipolaris cactivora*.

Symptoms/signs. *Bipolaris cactivora* causes roughly circular dark sunken lesions on cladophylls (Figs. 1 and 2). Cladophylls become soft rotted and abscise from the plant. A dark-brown, felty growth of spores may develop on the surface of the infected tissue.



Fig. 1 Bipolaris blight (R.L. Wick © 2017. All Rights Reserved.)



Fig. 2 Bipolaris blight (R.L. Wick © 2017. All Rights Reserved.)

Table 1 Disease reactions of *Hatiora* species and cultivars to an isolate of *Bipolaris cactivora* (T.H. Boyle and R.L. Wick, unpublished data)

Disease reaction	Entry
Highly susceptible	Annika, Cassiopeia, Crimson Giant, Crystal Lakes 32, Evita, Flash, Leo's Pink, Pink Perfection, Rainbow, Rood, Shocking Pink, Sutter's Gold, Thor-Anne, *H. rosea* MD861
Moderately susceptible	Andre
Resistant	Hatherton Star, *H. salicornioides* Logee

Biology and epidemiology. In 1955, approximately 60 species of cacti were known to be susceptible to "top rot" by *Bipolaris cactivora* (Durbin et al. 1955). Natural infection of *Schlumbergera truncata* by *B. cactivora* was first reported in California, USA (Raabe 1989). Prior to that, inoculations of *Schlumbergera* with *B. cactivora* isolates from *Hatiora* were largely unsuccessful (Chase 1982). Abundant conidia are produced on the surface of the lesions where they may be dislodged by wind or water. Wounding is not necessary for infection, and infection can take place on any of the cladophylls including those below the ground.

Management

- ***Cultural Practices*** – Infected plants should be discarded. Keep relative humidity low and reduce plant wetness duration. A novel approach to controlling this disease on other cacti utilized heat-inactivated conidial suspension of *B. cactivora*. Conidial suspensions were autoclaved, and when applied 2 days prior to inoculation with the pathogen, plants had no or minimal disease development (Choi et al. 2010). The treatment greatly increased wound periderm formation at the site of inoculation.
- ***Resistance*** – At the University of Massachusetts, a diverse array of 19 *Hatiora* genotypes were challenged by *B. cactivora* in growth chambers and in the greenhouse. In all three experiments, *Hatiora* x *graeseri* 'Hatherton Star' exhibited excellent resistance. *H. salicornioides* 'Logee' was also resistant. Resistance was displayed as small lesions with little or no sporulation; susceptible plants had large lesions with abundant spores (Boyle 2007 and Table 1).
- ***Chemical Control*** – Chlorothalonil has been used to control this disease (Daughtrey et al. 1995).

2.3 Pythium Root and Stem Rot [*Pythium aphanidermatum* (Edson) Fitzp. *Pythium irregulare* Buisman, and *Pythium cryptoirregulare* Garzon, Yanez, and Moorman]

Geographic occurrence and impact. The three *Pythium* species listed above have been reported on *Schlumbergera* (Knauss 1975; Garzon et al. 2007; Lee et al. 2010), and Pythium root and stem rot has been reported in Denmark and Florida, USA;

however, it is presumed that many *Pythium* spp. are capable of causing root and stem rot, and the diseases occur worldwide. *Pythium* is one of the most common causes of root rot in greenhouses (Daughtrey et al. 1995).

Symptoms/signs. *Pythium* spp. cause feeder roots to become brown and soft rotted. Usually the cortex easily separates from the vascular system. Root rot causes wilting of the plant. Occasionally a crown rot occurs and *Pythium* spp. can move up the stem for some distance.

Biology and epidemiology. *Pythium* is a widespread and common genus of the Oomycota (water molds) (Daughtrey et al. 1995); nearly every handful of soil contains one or several species. *Pythium* spp. are not usually an aggressive plant pathogen unless plants are growing in a soil-less medium, soluble salts are high and moisture abundant. There are more than 200 species of *Pythium*; they generally have a wide host range and vary in their pathogenicity and distribution. *Pythium* species are not strong soil competitors but can survive in soil for long periods of time via oospores. When soil moisture is abundant, root exudates cause oospores to germinate and produce a sporangium where 10–30 zoospores develop. The zoospores find roots by chemotaxis and they have flagella that propel them through the water. Once on the root surface, they infect, colonize, and kill the root cells and produce oospores and sporangia. Young roots and seedlings are particularly susceptible to infection. Fungus gnats can also spread the pathogen.

Management

- ***Cultural Practices*** – Remove diseased plants from the greenhouse being careful not to spill the soil from the pots. When growing plants in soil-less media, take care not to introduce field soil by hands, tools, and hose ends dropped on the floor. Use a well-drained growing medium and avoid overwatering and overfertilization.
- ***Chemical Control*** – Many modern fungicides have single-site mode of action and readily select for resistance. To avoid resistance development, rotate between different Fungicide Resistance Action Committee groups (FRAC Code List 2015); do not use one fungicide repeatedly. Note that some isolates of *Pythium* and *Phytophthora* may already be resistant to some of these fungicides. Many greenhouse isolates of *Pythium* in the USA have developed resistance to mefenoxam. The fungicides listed here are followed by the FRAC code in parentheses followed by the risk of selecting for resistance. Mefenoxam (4) high, etridiazole (14) low to medium, azoxystrobin (11) high, fluopicolide (43) unknown, fosetyl-Al (33) low, fenamidone (11) high, and Cyazofamid (21) medium to high.

2.4 Phytophthora Root and Stem Rot *Phytophthora nicotianae* Breda de Haan; Formerly *Phytophthora parasitica* Dastur.

Geographic occurrence and impact. *Phytophthora nicotianae* causes many important plant diseases worldwide. *Phytophthora nicotianae* is very destructive



Fig. 3 Phytophthora root and stem rot (R.L. Wick © 2017. All Rights Reserved.)

to *Schlumbergera truncata* (Alfieri and Miller 1971). It is likely that other species of *Phytophthora* can cause root and stem rot of *Schlumbergera*.

Symptoms/signs. Plants collapse and may show brown lesions at the base of the plant (Fig. 3).

Biology and epidemiology. *Phytophthora nicotianae* was considered more or less synonymous with *Phytophthora parasitica* following the combining of the two taxa by Waterhouse in 1963, although the use of *P. parasitica* rather than *P. nicotianae* became preferred over time. Indeed from 1970 to 1994, citations listed *P. parasitica* much more commonly than those listing *P. nicotianae* (Erin and Ribeiro 1996). Currently *P. nicotianae* is the accepted name. *Phytophthora nicotianae* has the widest host range of any *Phytophthora* species and is found all over the world. *Phytophthora* is not as common in greenhouses as *Pythium*, but it is much more aggressive than *Pythium*. There are more than 80 species of *Phytophthora* and most have wide host ranges. Like *Pythium*, *Phytophthora* is an Oomycete and is most active when water is plentiful in the growing medium. Dispersal of inoculum is dependent on movement of contaminated water, soil, or plant material. Nearly all species produce oospores but some need opposite mating types to do so, and some produce resting structures called chlamydospores. *Phytophthora* is a soil-borne organism and can survive for many years in the absence of a host. Oospores are stimulated to germinate when root exudates are present, and germination often results in the formation of sporangia with swimming zoospores. Temperatures of 28° C/82° F C and above favor disease development (Daughtrey et al. 1995).

Management

- ***Cultural Practices*** – Remove diseased plants from the greenhouse being careful not to spill the soil from the pots. When growing plants in soil-less media, take care not to introduce field soil by hands, tools, and hose ends dropped on the floor. Use a well-drained growing medium and avoid overwatering and overfertilization.

- ***Chemical Control*** – Many modern fungicides have single-site mode of action and readily select for resistance. To avoid resistance development, rotate between different FRAC groups (FRAC Code List 2015); do not use one fungicide repeatedly. Note that some isolates of *Pythium* and *Phytophthora* may already be resistant to some of these fungicides. Many greenhouse isolates of *Pythium* in the USA have developed resistance to mefenoxam. The fungicides listed here are followed by the FRAC code in parentheses followed by the risk of selecting for resistance. Cyazofamid (21) medium to high, fenamidone (11) high, mefenoxam (4) high, phosphorous acid salt (no FRAC Code) low, and pyraclostrobin (11) high.

2.5 Other Minor Diseases Caused by Fungi

Note, these diseases may be minor in importance or, they are rare in their occurrence.

Geographic occurrence and impact. The following fungi are listed in various indices and reports as occurring on *Schlumbergera truncata*: *Alternaria* sp., anthracnoses (*Colletotrichum* spp.), *Armillaria* root rot, *Cercospora* leaf spot, *Dichotomophthora* sp. tip blight, *Fusarium spp.* basal stem rot, leaf spot, Myrothecium leaf spot and crown rot, and *Phomopsis* sp. leaf spot (Daughtrey et al. 1995; Wick and Dicklow 1999).

3 Bacterial Diseases

3.1 Bacterial Soft Rot [*Pectobacterium carotovorum* subsp. *carotovorum* (Jones) Hauben et al. emend. Gardan et al.)]. Formerly *Erwinia carotovora* subsp. *carotovora*

Geographic occurrence and impact. *Pectobacterium* has a worldwide distribution and a wide host range. Bacterial soft rot is not a major problem, but it can be destructive and persistent if not controlled completely.

Symptoms/signs. Soft rot generally starts at the soil line where it causes a soft mushy rot.

Biology and epidemiology. *Pectobacterium* produces pectic enzymes which are responsible for soft rot. The bacterium can survive in soil and may contact plants from dirty hands and tools, splashing water, insects, or hose ends that have contacted the ground. *Pectobacterium* may also come in on cuttings from infected mother plants. Bacteria need wounds or natural openings to gain entry into the host; cut cladophylls that are rooting would be particularly susceptible. In general, plants

propagated vegetatively are very vulnerable to infection by soft rot bacteria, especially at high temperatures which are often provided by heating mats to encourage rooting. *Pectobacterium carotovorum* subsp. *carotovorum* has an optimum temperature for growth of 28–30 ° C/82–86° F but can grow up to 37–42 ° C/98–108° F (Daughtrey et al. 1995).

Management

- ***Cultural Practices*** – Before propagation, the cutting bench area should be clean and disinfested. Plants or rooting cladophylls with evidence of soft rot should be discarded. If soft rotting occurs in the propagation area, discard all plant material, and thoroughly disinfest all surfaces with a quaternary ammonium compound or similar product (Daughtrey et al. 1995).
- ***Chemical Control*** – Various formulations of copper can afford some protection against bacteria but are not likely to prevent basal rots that initiate below the surface of the growing medium.

4 Virus Diseases

4.1 Tomato Spotted Wilt Virus (TSWV) and Impatiens Necrotic Spot Virus (INSV)

Geographic occurrence and impact. TSWV and INSV have become distributed worldwide and are known to occur in the USA, South America, Canada, Europe, India, China, and Japan. Reports on *Schlumbergera* include the USA, Canada, Finland, and Europe, but these viruses are probably more widespread. While not destructive on *Schlumbergera*, TSWV and INSV are of economic importance as infected plants would be discarded.

Symptoms/signs. TSWV/INSV cause a variety of symptoms on *Schlumbergera* including sunken chlorotic lesions, dark-green spots, chlorosis, ring spots, and necrosis (Figs. 4 and 5). Infected plants can be non-symptomatic as well (Hausbeck and Fildow 1991).

Biology and epidemiology. TSWV/INSV are *Tospoviruses* in the family Bunyaviridae. Prior to 1990, TSWV was considered to have two strains, TSWV I (impatiens strain) and TSWV L (lettuce strain). In 1990, INSV was described as distinct from TSWV; however, prior to this time, plants showing symptoms of TSWV may in fact have been infected by INSV. At least in the USA, ornamental plants are far more likely to be infected by INSV than TSWV, and the symptoms can be similar (Daughtrey et al. 1995). Antisera are available to distinguish the two viruses. Tospoviruses are vectored exclusively by at least seven species of thrips (Thysanoptera: Thripidae). Only the first and early second



Fig. 4 INSV (R.L. Wick © 2017. All Rights Reserved.)



Fig. 5 INSV (R.L. Wick © 2017. All Rights Reserved.)

instar larvae can acquire the virus, and both the larvae and adults can transmit the virus. The virus/thrip relationship is persistent and INSW and TSWV can replicate in the vector.

Management

- ***Cultural Practices*** – Inspect plants for viruses before introducing them into the greenhouse. *Schlumbergera* can be asymptomatic carriers of the virus so it would be wise to have random testing carried out on newly acquired plants. Control weeds on the greenhouse floor and around the greenhouse. Monitor for thrips

with yellow sticky cards. Screening can be used to cover greenhouse vents, but the material should have apertures of 135 microns or less; this will exclude thrips but can greatly reduce airflow into the greenhouse. Avoid carrying over crop material from season to season.

- ***Chemical Control*** – Insecticides should be used to control thrips; however, many thrips have developed resistance to one or more insecticides.

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Diseases of *Hydrangea*

35

Yonghao Li, Margaret T. Mmbaga, Boru Zhou, Jacqueline Joshua, Emily Rotich, and Lipi Parikh

Abstract

Hydrangea, a native to Asia and America, is a popular ornamental plant due to its interesting large, showy flowers with different colors and inflorescence forms. Many hydrangea species are often used to design landscapes and gardens. In greenhouses, nurseries, and landscapes, hydrangea production and values are affected by fungal, bacterial, and viral diseases. In this chapter, the biology of pathogens, development of diseases, and strategies for their control of common diseases of hydrangea were described.

Keywords

Hydrangea • Powdery mildew • Leaf spot • Rust • Anthracnose • Bacterial • Virus

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© Springer International Publishing AG (outside the USA) 2018

R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_36

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1 Introduction

Hydrangeas are among the most loved flowering shrubs and vines in landscapes. Many species in the genus *Hydrangea* vary in shrub size, texture, flower shape, and color, and their aesthetic values include popular floral arrangements and crafts (Mmbaga et al. 2008). The most common hydrangea is the bigleaf/French/garden hydrangea (*H. macrophylla* (Thunb.) Ser.) that thrives in hardiness US zones 6–9 (Dirr 2004). Although more than 700 cultivars have been described in *H. macrophylla*, only about 25% of these are available in the US trade (Dirr 2004; Van Gelderen and Van Gelderen 2004). Several other common species in this genus are climbing hydrangea (*H. anomala*), oakleaf hydrangea (*H. quercifolia*), smooth hydrangea (*H. arborescens*), and Pee Gee hydrangea (*H. paniculata*) (Dirr 2004).

2 Fungal Diseases

2.1 Powdery Mildew (*Golovinomyces orontii* DC)

Geographical occurrence and impact. Powdery mildew of hydrangea has been reported in the Unites States and Korea (Park et al. 2012). The disease is a common problem in bigleaf hydrangea (*H. macrophylla*) in greenhouses, nurseries, and landscapes when environment conditions are warm and humid. The disease also affects *H. serrata* as well as smooth (*H. arborescens*), bigleaf (*H. macrophylla*), and panicle (*H. paniculata*) hydrangeas (Hagan and Mullen 2001).

Symptoms/signs. Early signs of powdery mildew on hydrangea consist of circular light gray patches composed of mycelia and spores on both sides of the leaf surface.

These pustules of fungal tissue eventually turn into yellow or purple blotches on the leaf (Fig. 1). As the disease progresses, disfiguring white mycelium develops on leaf surfaces along with extensive chlorosis or yellowing of the leaves (Fig. 2). Premature defoliation may occur on severely infected plants. Growth of the plant may be impacted with the reduction in active leaf area and shoot elongation directly reducing the production of photosynthesis (Hagan et al. 2004; Sinclair and Lyon 2005). Powdery mildew is rarely fatal to plants in landscape settings. Its major damage is the reduction in aesthetic quality and plant vigor. In addition economic values of infected plants are lowered and may even render plants unmarketable.

Biology and epidemiology. The bigleaf hydrangea powdery mildew is caused by *G. orontii* (formerly *Erysiphe polygoni* DC.) and *E. poeltii* (Pscheidt 2014). Powdery mildew caused by *Oidium hortensiae* on *H. macrophylla* was first reported in Korea in 2012 (Park et al. 2012). The pathogen is an obligate parasitic fungus that occupies the leaf surface of the plant (Li et al. 2009a). The disease is favored by dry conditions, high humidity, and warm day/cool night temperatures. The pathogen obtains its nutrition from epidermal cells that are parasitized by root-like structures called haustoria (Li et al. 2008). The fungus overwinters as hyphae or spores in buds of previously infected plants in the landscape and living plants in greenhouses.



Fig. 1 The early symptom of powdery mildew of hydrangea with *white* mildew and *brown* patches (Yonghao Li © 2017. All Rights Reserved.)



Fig. 2 Symptoms of hydrangea powdery mildew with infected leaves covered by heavy *white* mildew (Yonghao Li © 2017. All Rights Reserved.)

Management

- ***Cultural practices*** – Propagation should be done using cuttings from healthy stock plants. Efforts to reduce the relative humidity in the greenhouse and increasing plant spacing to improve air circulation will suppress powdery mildew. Placing plants in sunny locations and avoiding overhead sprinkler irrigation will also help to reduce the incidence of disease. Growers should remove infected plants as well as remove debris from previous crops as soon as possible to reduce the amount of inoculum and infection source.
- ***Chemical*** – Fungicides labeled for use against powdery mildew on hydrangea are available. Applications should be initiated when initial symptoms of the disease are observed or conditions are favorable for disease development. Fungicide applications should be repeated at 7–14-day intervals or by following label recommendations to provide effective control. Some labeled fungicides reported to control powdery mildew on hydrangea include azoxystrobin, fenarimol, paraffinic oil, and thiophanate-methyl (Hagan et al. 2004; Hagan and Mullen 2001). Fungicide rotations should be practiced to minimize the development of fungicide resistance. Rotations should include fungicides from different fungicide groups (FRAC groups) that operate using different modes of action.
- ***Biorationals*** – Applications of biorational products are also effective in controlling powdery mildew (Mmbaga and Sauvé 2004). However products have to be applied preventatively when initial symptoms of powdery mildew are observed. Thorough coverage of the tops and undersides of leaves is recommended. Sodium bicarbonate (baking soda) marketed as Armicarb™ (potassium bicarbonate (MilStop)) and fatty acids from neem seed oil marketed as Triact 70® are labeled for powdery mildew control (Hagan et al. 2005; Horst et al. 1992; Mmbaga and Oliver 2007).
- ***Biocontrols – Bacillus subtilis*** strain QST 713 (Hagan et al. 2004) is labeled as a biological control agent against powdery mildew in hydrangea.
- ***Resistance*** – The best way to manage powdery mildew is by growing disease-resistant selections. Previous reports indicate that cultivars belonging to *H. macrophylla* ssp. *serrata* are more resistant than *H. macrophylla* ssp. *macrophylla* cultivars (Dirr 2004). Out of 90 *H. macrophylla* cultivars evaluated for resistance to powdery mildew over a 3-year period, three cultivars, "Amagi Amacha," "Shirofuji," and "Veitchii," were among the most powdery mildew-resistant cultivars each year (Windham et al. 2011). Other cultivars, namely, "Diadem," "Komachi," and "Omacha," were highly resistant in 2006 and 2008, but only moderately resistant in 2007 (Windham et al. 2011). Bigleaf hydrangea "Veitchii" also showed higher resistance to powdery mildew in vitro tests (Li et al. 2009b).

2.2 Cercospora Leaf Spot (*Cercospora hydrangea* L.)

Geographic occurrence and impact. This disease generally affects bigleaf and smooth types of hydrangea as well as oakleaf hydrangea in both landscape and



Fig. 3 Small circular *brown* necrotic lesions with ash-colored centers on a hydrangea leaf infected by *Cercospora* sp. (Margarete Mmbaga © 2017. All Rights Reserved.)

nursery settings (Vann 2010; Mmbaga et al. 2012, 2015). The disease is particularly important in warmer regions of southeastern United States including Arkansas, Alabama, Georgia, and Tennessee (Vann 2010; Hagan and Mullen 2001). This leafspot disease hardly ever kills the plant, but it can cause significant premature defoliation and reduce the plant vigor and flower bud set (Vann 2010; Hagan and Mullen 2001).

Symptoms/signs. Leaf spot symptoms differ based on the type of hydrangea infected. Older leaves at the bottom of the plant usually exhibit the first visible symptoms and then spread upward toward the top of the plant (Vann 2010; Hagan and Mullen 2001). Early spots on the leaves are purple and small with a circular shape (Fig. 3). This is followed by enlargement of the spot that turns angular or irregular in shape and acquires a tan or gray center enclosed by a purple or brown border commonly referred to as frogeye leaf spot pattern in bigleaf hydrangea (Vann 2010; Hagan and Mullen 2001). Alternatively, leaf spots on oakleaf hydrangea are angular shaped and dark brown to purple and may become yellow green and defoliate. Leaf spotting often commences in the midsummer and becomes visible by early fall.

Biology and epidemiology. The primary source of inoculum is the conidia of *C. hydrangeae* that overwinter in the leaf debris. The spores are disseminated to healthy lower foliage via splashing rain or overhead irrigation as well as wind (Hagan and Mullen 2001; Vann 2010). Recurring summer rain showers can significantly accelerate the rate of disease spread, level of spotting, and defoliation. On the other hand, suppression of the disease development and spread can be prolonged by drought periods and prolonged exposure to full sun (Hagan and Mullen 2001; Li et al. 2008; Mmbaga et al. 2012).

Management

- ***Cultural*** – Application of sufficient nitrogen will help ensure good growth of the plants (Hagan and Mullen 2001). Drip irrigation or using soaker hoses and wide plant spacing that allow air movement and reduce leaf wetness have been reported to reduce Cercospora leaf spot incidences in landscape plantings. Proper site

selection is imperative in order to avoid known problematic areas. Choice of site by a grower may not always be feasible, but the use of well-drained areas and partial shade with direct exposure to morning sunlight promotes better growth of the hydrangea and reduces foliage disease incidence (Li et al. 2008). A good sanitation strategy requires the removal and destruction diseased leaf debris that act as the source of infection for new plants. This will help to minimize infection level and disease severity.

- ***Chemical*** – Using protective fungicides is advocated for high-value landscape plants that annually show noticeable damages. The fungicide application should commence when first leaf spot symptoms are observed and continue as needed. Products containing chlorothalonil, myclobutanil, or thiophanate-methyl are highly effective if applied at the onset of the disease. The use of protective chlorothalonil or mancozeb programs was found to significantly reduce Cercospora leaf spot disease severity in bigleaf hydrangea (Morrison 1980). Since protection by these products is not guaranteed for new growth that is not covered by the fungicide, multiple fungicide applications at 10–14-day intervals are necessary to ensure continuous leaf protection and good disease control.
- ***Resistant varieties*** – Selection of resistant and disease-free cultivars for new planting is important to prevent the introduction of the disease to new areas. Mmbaga et al. (2012) reported ten cultivars of *H. macrophylla*, "Ami Pasquier," "Ayesha," "Blue Bird," "Forever Pink," "Fuji Waterfall" ("Fujinotaki"), "Miyama-yae-Murasaki," "Seafoam," "Taube," "Tricolor," and "Veitchii," for expressing resistance or moderate resistance to multiple pathogens including Cercospora leaf spot in full sun, full shade, and partial shade environments (Li et al. 2008; Mmbaga et al. 2012).

2.3 Botrytis Blight (*Botrytis cinerea* Pers.: Fr.)

Geographic occurrence and impact. Botrytis blight, also called gray mold, is a serious disease problem in the production and postharvest of floral crops. *B. cinerea* has a wide host range including many herbaceous annual and perennial plants (Plant Disease Diagnostic Clinic 2015). The fungus is considered an opportunistic pathogen that infects weakened or injured leaves and flowers especially in cloudy, humid, and rainy weather conditions. All species of hydrangea are susceptible to this disease, but bigleaf hydrangea is most susceptible (Hagan and Mullen 2001).

Symptoms/signs. When flowers are infected, the initial symptoms are water-soaked spots on petals, which develop reddish-brown irregular blotches that may, in time, cover the whole flower later. Affected flowers quickly turn brown and wither. The fuzzy gray growth (spores and conidiophores of the fungal pathogen), a characteristic symptom of Botrytis blight, is usually noticed on diseased flowers when environment conditions are wet or highly humid (Hagan and Mullen 2001). Leaves can be infected and show irregular brown necrotic lesions on leaves when diseased petals or other infected debris are fallen on them (Fig. 4).



Fig. 4 Irregular necrotic lesions on hydrangea leaves affected by Botrytis blight (Margarete Mmbaga © 2017. All Rights Reserved.)

Biology and epidemiology. *B. cinerea* can survive in plant debris as a saprophyte or as a pathogen on a broad range of host plants (Daughtrey et al. 2000). The fungus also overwinters as black resting structures (sclerotia) on dead plant tissues or in soil (Hagan and Mullen 2001; Plant Disease Diagnostic Clinic 2015). The pathogen can be transmitted by wind, water splash, insects, and human activities from affected plants. Spore germination needs high relative humidity or water films on plant surfaces. A 5- to 8-h wet period is required for infection of *B. cinerea* (Jewett and Jarvis 2001). Germinated spores directly penetrate healthy blossoms and leaves and also enter through natural openings and wounds. The favorable conditions for the disease development are unbalanced nutrition, low light intensity, cool temperature, and high humidity, especially in the condition of several consecutive days of cloudy, humid, rainy weather (Sinclair and Lyon 2005). The optimal temperature for the onset of Botrytis blight in the greenhouse is approximately 15 °C. All species of hydrangea are susceptible to this disease, but damage is noted most often on the bigleaf hydrangea (Hagan and Mullen 2001).

Management

- ***Cultural practices*** – Venting and concurrently heating greenhouses at dusk can expunge humidity. Avoiding overhead irrigation will help to keep leaf canopies dry and reduce relative humidity which can suppress the disease (Daughtrey et al. 2000). Increasing plant spacing and pruning weakened leaves from the lower part of plants to improve air circulation and encourage rapid evaporation of moist from plants and soil surfaces, avoiding wounds of leaves or shoots which can promote further spread of the disease. In addition, watering plants in the morning to allow rapid evaporation of excess moisture from plant surfaces is recommended.
- ***Sanitation*** – Proper sanitation is crucial in controlling this disease. But, sanitation should not be conducted when plants are wet with dew or rain because it will spread fungal spores into the air. Removing infected leaves and damaged flower

heads from plants and destroying them to reduce the inoculum in greenhouses and landscapes. In the greenhouse, clearing of benches and beds should be done before bringing in fresh plant materials of next crops. This can be followed by treatment of wooden surfaces in propagation and production areas with 2% disinfectant (Hagan and Mullen 2001).

- ***Fungicide*** – Protection of plants against infection can be easily managed using fungicides. The first fungicide application should be made in the spring when continuously cool and wet weather is predicted or where Botrytis blight has been a problem in the previous year. Choice of fungicides is based on the site and type of plant(s) to be treated. Some effective fungicides recommended include iprodione, mancozeb, and thiophanate-methyl (Hagan and Mullen 2001). To prevent fungicide resistance development, always alternate fungicide applications between chemicals with different modes of actions. Resistance to thiophanate-methyl and iprodione has been reported in Botrytis populations.
- ***Biological*** – Some biofungicides, such as Mycostop (*Streptomyces griseoviridis* Strain K61), actinovate (*Streptomyces lydicus*), are effective to control Botrytis blight.
- ***Resistant varieties*** – The use of resistant cultivars is the most effective; however, no major resistance gene to *Botrytis* has been identified in hydrangeas.

2.4 Rust (*Pucciniastrum hydrangeae* (B. & C.) Arth.)

Geographic occurrence and impact. In nurseries, gardens, and landscapes, hydrangea rust is most commonly found on smooth hydrangea, a native shrub in the eastern North America. The pathogen can also affect hemlock, but it is more commonly found on hydrangeas. The disease is reported from New York to Illinois, Arkansas, Tennessee, and Georgia (Sinclair and Lyon 2005; Li et al. 2010).

Symptoms/signs. On hydrangea leaves, symptoms initiate as small yellow or orange spots, which then develops to necrotic angular lesions because fungal growth is normally restricted by major veins (Fig. 5). An orange dust (urediniospores) forms on the lower surface of infected leaves when the fungal fruiting bodies (uredinia) are mature (Fig. 6). As the disease develops, symptoms are noticeable on the whole plant (Fig. 7).

Biology and epidemiology. To complete its life cycle, the fungus, *P. hydrangea*, needs two genetically distinctive hosts, hemlock and hydrangea, which includes *Tsuga canadensis* (eastern hemlock), *T. caroliniana* (Carolina hemlock), *H. arborescens* (smooth hydrangea), and *H. paniculata* (panicle hydrangea). Urediniospores formed on hydrangea leaves repeat several infection cycles on hydrangeas during a season. Flat, reddish-brown telia develop within the epidermal cells of both sides of leaf surfaces of hydrangea in late summer and fall. The teliospores germinate and produce basidiospores that infect hemlock needles in the spring (Sinclair and Lyon 2005). In late spring to early summer, cylindric aecia with



Fig. 5 Yellow spots on a smooth hydrangea leaf infected with hydrangea rust (Yonghao Li © 2017. All Rights Reserved.)



Fig. 6 Yellow to orange pustules on the lower surface of a hydrangea leaf infected with hydrangea rust (Yonghao Li © 2017. All Rights Reserved.)



Fig. 7 A hydrangea plant with rust-infected leaves (Yonghao Li © 2017. All Rights Reserved.)

light cream color are formed on the lower surface of hemlock needles. When aecia are mature, orange-yellow aeciospores are released and infect hydrangea leaves in the spring.

Management

- ***Cultural practices*** – Reduction of relative humidity and the duration of plant tissue wetness are critical for the disease control.
- ***Fungicide*** – Chlorothalonil 720 SC and Daconil Weatherstik fungicides are labeled for hydrangea rust. The first fungicide application should be made in the spring to protect leaves. Fungicide doses and replications need to follow the fungicide labels.
- ***Resistance*** – Significant differences in resistance to hydrangea rust were reported between cultivars of smooth hydrangeas (Li et al. 2010). Among seven tested varieties, "Frosty" was highly resistant, "Green Dragon" was highly susceptible, and the other five varieties, "Ryan Gainey," "Pink Pincushion," "White Dome," "Mayes Starbust," and "Annabelle," were intermediate (Li et al. 2010).

2.5 Anthracnose (*Colletotrichum gloeosporioides* (Penz.) Penz. and Sacc.)

Geographic occurrence and impact. Anthracnose of hydrangea is caused by *C. gloeosporioides* and found periodically in landscape and field plantings of bigleaf hydrangea. This disease affects both leaves and blossoms of hydrangea plants.

Symptoms/signs. Initially, circular or slightly irregular brown spots are formed on hydrangea leaves with the center of the spots measuring up to 2.5 cm or more in diameter and turning light brown to tan in color (Hagan and Mullen 2001). This is followed by alternating dark and slightly lighter rings of dead tissue forming a bull's eye or target-spot appearance (Fig. 8). Large, dark brown, irregular blotches may extend across the flower petals and leaves when environmental conditions are favorable for the disease development.



Fig. 8 Circular to irregular-shaped brown spots with concentric rings and ash-gray center on hydrangea leaves infected by *Colletotrichum gloeosporioides* (Margarete Mmbaga © 2017. All Rights Reserved.)

Biology and epidemiology. *C. gloeosporioides* has a very broad host range in woody shrubs and trees. The pathogen overwinters in diseased leaf debris of hydrangea and other plant debris. The disease is favored by hot, wet weather conditions. Masses of spores ooze from fruiting bodies (acervuli) embedded in leaf debris. In the presence of continuous leaf wetness, spores spread to other leaves and flower petals by splashing water (Hagan and Mullen 2001). The pathogen is able to quickly penetrate and colonize the host tissues at warm temperatures of 24–32 °C. The rate of disease infection and symptom appearance are promoted by prolonged periods of heavy fog, frequent showers, and dew. Hydrangeas that are extensively fertilized may be more sensitive to infection by *C. gloeosporioides* (Hagan and Mullen 2001).

Management

- ***Cultural*** – The use of disease-/symptom-free plant cuttings can significantly minimize the chances of spreading the disease. Removing and destroying diseased leaf debris or blighted blooms eliminate and reduce the inoculum source.
- ***Chemicals*** – The use of protective fungicide treatments is a viable option to protect susceptible hydrangea from anthracnose. The application should be done at 10–14-day intervals during the summer for good efficacy. Some of the recommended fungicides include chlorothalonil and thiophanate-methyl, and they should be administered as per manufacturer's instructions (Hagan and Mullen 2001).

2.6 Corynespora Leaf Spot (*Corynespora cassiicola* (Berk. & M.A. *Curtis*) C.T. Wei)

Geographic occurrence and impact. This disease infects leaves, stems, and flower petals and causes irregularly shaped brown necrotic lesions. The disease may spread rapidly during hot and wet weather and has the potential to kill small plants and severely reduce the aesthetic value of the plants.

Symptoms/signs. Symptoms of this disease are characterized as various sized brown spots on hydrangea leaves (Fig. 9) that are similar to those of Cercospora leaf spot. Like Cercospora leaf spot, Corynespora leaf spot is favored by the similar environments and is often isolated as part of a disease complex (Hagan et al. 2004; Zaher et al. 2005; Smith and Schlub 2007).

Biology and epidemiology. Although *C. cassiicola* has been reported on over 300 plant species, a high degree of host specificity was reported in the fungal populations (Pereira et al. 2003). The pathogen survives in infested plant debris and on stems of previously infected plants.

Management

- ***Cultural*** – Removal and destruction of diseased leaf debris that act as the source of infection for new plants are important. Avoid overhead irrigation and plant crowding.



Fig. 9 Various sized brown spots on hydrangea leaves infected with *Corynespora cassiicola* (Margarete Mmbaga © 2017. All Rights Reserved.)

- ***Chemicals*** – Both Cercospora and Corynespora leaf spot diseases tend to have annual outbreaks when the pathogens have become established in an area (Hagan and Mullen 2001; Hagan et al. 2004; Zaher et al. 2005; Smith and Schlub 2007; Mmbaga et al. 2012). Thus, protective fungicide applications are advocated for high-value landscape plants that annually show noticeable damages. The fungicide application should commence when first leaf spot symptoms are observed and continue as needed.
- ***Resistant varieties*** – Selection of resistant and disease-free cultivars for new planting is important to prevent the introduction of the disease to new areas. Cultivars "Ami Pasquier," "Ayesha," "Blue Bird," "Forever Pink," "Fuji Waterfall" ("Fujinotaki"), "Miyama-yae-Murasaki," "Seafoam," "Taube," "Tricolor," and "Veitchii" were rated resistant or moderate resistant to multiple pathogens including Corynespora leaf spot in full sun (Mmbaga et al. 2012).

2.7 Phoma Leaf Spot (*Phoma* spp.)

Geographic occurrence and impact. This disease can occasionally cause leaf spots in hydrangea. *Phoma exigua* has been reported to cause leafspot disease in hydrangea in different parts of the United States and central Italy (Garibaldi et al. 2006; Mmbaga et al. 2009).

Symptoms/signs. Phoma leaf spot is characterized by small necrotic spots surrounded by chlorotic haloes on the upper side of infected leaves (Garibaldi et al. 2006). The color of the leaf spots may differ slightly based on cultivars as observed by Mmbaga et al. (2010) where *H. macrophylla* cultivar "Lady in Red" developed small reddish-brown lesions and cultivar "Seafoam" had large lighter brown lesions during pathogenicity tests with *P. exigua* (Fig. 10).

Biology and epidemiology. Phoma leaf spot is favored by relatively low temperatures (24 ± 3 °C) for symptom development (Mmbaga et al. 2010). Severely infected leaves become chlorotic and abscised leading to premature defoliation.



Fig. 10 Brown spots on a hydrangea leaf infected by *Phoma exigua* (Margarete Mmbaga © 2017. All Rights Reserved.)



Fig. 11 Concentric ring spots on a bigleaf hydrangea leaf infected by *Myrothecium roridum* (Margarete Mmbaga © 2017. All Rights Reserved.)

Infected plants rarely die, but the presence of lesions on mature plants decreases aesthetic quality and subsequent market values.

Management

- ***Cultural practices*** – Sanitation and removal of diseased leaf debris help to eliminate source of infection.

2.8 Myrothecium Leaf Spot and Blight (*Myrothecium roridum* Tode Ex Fr.)

Geographic occurrence and impact. The disease was first reported in Tennessee in 2010 (Mmbaga et al. 2010; Mmbaga 2010).

Symptoms/signs. In *H. macrophylla*, brown necrotic lesions with concentric rings and ash-colored centers (Fig. 11) characterized this disease and are similar to symptoms from *M. roridum* in other ornamental plants such as salvia, gardenia,

begonia, and New Guinea impatiens (Mmbaga et al. 2010; Mangandi et al. 2007). The pathogen causes blighting of the stems, mostly beginning at the soil line, tan concentric leaf spots, and yellowing and wilting of foliage.

Biology and epidemiology. *M. roridum* resides mostly in the soil and has a broad host range consisting of several vegetable, agronomic, and ornamental crops (Ponappa 1970). Infected plant materials and infested soil disseminate spores of *M. roridum*, and fungal spores may remain viable for 2–3 months in field soil at 20 °C (Murakami et al. 2000).

Management

- ***Cultural practices*** – Some of the management strategies comprise of soil disinfestation using fumigants or steam and soil solarization to kill the pathogen in the soil. Preventative measures focus on sanitation practices to reduce disease incidence.
- ***Host resistance*** – When available, disease resistance is the best method for controlling hydrangea leaf spot diseases. Out of 88 cultivars of *H. macrophylla* exposed to natural airborne leaf spot pathogens in McMinnville, Tennessee, USA, ten cultivars, "Ami Pasquier," "Ayesha," "Blue Bird," "Forever Pink," "Fuji Waterfall" ("Fujinotaki"), "Miyama-yae-Murasaki," "Seafoam," "Taube," "Tricolor," and "Veitchii," were rated resistant or moderately resistant in full shade, full sun, and partial shade environments (Mmbaga et al. 2012). These cultivars may be considered to have multiple resistance to the pathogens including *Cercospora* spp., *C. cassicola*, *C. gloeosporioides*, *M. roridum*, and *P. exigua* (Mmbaga et al. 2012, 2015).

2.9 Alternaria Leaf Spot (*Alternaria* spp.)

Geographic occurrence and impact. Alternaria leaf spot of hydrangea can be caused by several *Alternaria* species that has been reported in Italy and the United States (Daughtrey 1995; Garibaldi et al. 2006, 2008). Infected plants rarely die, but the presence of lesions reduces the aesthetic quality and subsequently the commercial value.

Symptoms/signs. Initial symptoms of Alternaria leaf spot are small brown spots with yellow halos that appear on the upper surface of the infected leaves. And then, lesions become dark brown or black and often coalesced into large necrotic areas. Symptoms are commonly observed on the leaf margins and near the petioles. Severely affected plants are defoliated (Garibaldi et al. 2006, 2008).

Biology and epidemiology. Two species, *A. alternata* and *A. compacta,* have been identified on bigleaf hydrangea and climbing hydrangea, respectively, in Italy (Garibaldi et al. 2006, 2008). The pathogens of Alternaria leaf spot can survive as mycelium or spores in plant debris. In moist and wet conditions, spores of the

pathogen are spread by wind, rain, or irrigation, which causes disease epidemics in landscapes and nurseries. Consequently, periods of moist conditions from rain and overhead irrigation are favorable for the disease development.

Management

- ***Cultural practices*** – Spacing plants to minimize dense canopy formation and improve air circulation. Avoiding overhead irrigation, watering plants in the morning to allow water on leaf surfaces to be evaporated quickly. Appropriately heating and ventilating greenhouses to reduce humidity.
- ***Sanitation*** – Removing infected leaves from infected plants and destroying them or putting them in trash.
- ***Fungicides*** – Fungicide application is a part of integrated disease management programs of the disease. Since most fungicides are preventative, the first application should be started when the disease is detected in nurseries or landscapes. The interval of applications needs to follow label recommendations.

3 Bacterial Diseases

3.1 Bacterial Leaf Spot (*Xanthomonas campestris*)

Geographic occurrence and impact. Bacterial leaf spot caused by a pathovar of *X. campestris* is a common problem on oakleaf hydrangea, smooth hydrangea, and bigleaf hydrangea. The disease has been reported in the United States (Uddin et al. 1996).

Symptoms/signs. On oakleaf hydrangea, the early symptoms of the disease initiate as water-soaked spots on the lower surface of leaves and then develop to angular purple lesions because lesions are usually restricted by leaf veins (Fig. 12). Normally, the disease starts on the lower leaves and spreads upward on the plant. On bigleaf hydrangea and smooth hydrangea cultivars, symptoms are small purple



Fig. 12 Angular *brown* lesions of bacterial leaf spot on an oakleaf hydrangea leaf (Yonghao Li © 2017. All Rights Reserved.)



Fig. 13 Angular brown lesions of bacterial leaf spot on a bigleaf hydrangea leaf (Yonghao Li © 2017. All Rights Reserved.)

angular spots (Fig. 13). At the late stage of lesion development, the necrosis develops in the center of lesions which dries, turns tan to brown, and can frequently fall out giving a shot-hole appearance.

Biology and epidemiology. Humid and moderately wet conditions are favorable for the disease epidemic in greenhouses, nurseries, and landscapes. Infection begins on the lower leaves and then spreads to upper leaves and adjacent plants as the pathogen is transported by splashing water. Small water-soaked spots develop on leaves within 3–5 days after inoculation, with typical purple spots developing within 7 days (Uddin et al. 1996).

Management

- ***Cultural practices*** – Avoid overhead irrigation to prevent the spread of the inoculum through splashing water. Space plants adequately to improve air circulation. Water plants in the morning and avoid handling plant when they are wet.
- ***Sanitation*** – Promptly remove and discard severely infected leaves during the growing season. At the end of a season, remove and destroy fallen leaves.
- ***Fungicide and biological control*** – Kaolin clay (Surround) and neem oil (Neem Gold) were reported to reduce disease severities of bacterial leaf spot in the field experiments (Mmbaga and Oliver 2007). Copper-containing fungicides may provide some control when applied in late spring, but will not be effective in plants that are grown under conditions favoring the disease.

4 Viral Diseases

4.1 Hydrangea Mosaic Virus (HdMV)

The typical symptom of HdMV on bigleaf hydrangea is chlorotic mosaic on leaves. The virus can be transmitted mechanically by grafting, by leaf contact, or by tools, but not by aphids or through seed. Control of HdMV includes using virus-free transplants and making cuttings from healthy plants (Thomas 1983).

4.2 Hydrangea Ringspot Virus (HRSV)

The early symptoms of HRSV on hydrangea leaves are brown spots or rings, and then plants show leaf distortion, leaf roll, and stunted growth. The virus can be transmitted mechanically through saps, but aphids or seed does not transmit HRSV (Daughtrey et al. 1995; Williams-Woodward and Daughtrey 2001). Cultural control of HRSV includes buying clean stock and sanitizing pruning tools.

4.3 Tomato Ringspot Virus (ToRSV)

ToRSV has a wide host range including woody and herbaceous plants. On hydrangea, the virus causes leaf distortion and chlorosis and stunted plant growth. The virus can be transmitted by nematodes, but not by pruning tools. Control strategies for ToRSV are weed management and the use of nematode-free soil mix for container-grown plants (Williams-Woodward and Daughtrey 2001; Brierley 1954; Daughtrey et al. 1995).

4.4 Tobacco Ringspot Virus (TRSV)

TRSV is widespread in woody and herbaceous plants in North America and also occurs in Europe, Asia, and Australia. Various symptoms can be found on infected plants, which include leaf distortion, leaf chlorosis, and stunted plant growth. Nematodes in the genus *Xiphinema* are major vectors of the virus (Williams-Woodward and Daughtrey 2001). Strategies for TRSV control include planting clean stocks in containers in soilless media and controlling weeds that may be reservoirs the virus and nematode vectors.

4.5 Tomato Spotted Wilt Virus (TSWV)

TSWV infects over 1,000 plant species. The virus is transmitted by thrips. Prevention is the first step of the disease control, which can be achieved by purchasing virus-free plant materials. In greenhouses, control of TSWV can be achieved by destroying infected plants and thrip management (Williams-Woodward and Daughtrey 2001).

4.6 Cherry Leaf Roll Virus (CLRV)

CLRV is widespread in Eurasia and North America and also occurs in Australia and New Zealand. Symptoms of CLRV on hydrangea are leaf deformation, chlorosis, and necrotic spots. This virus can be transmitted through seed and pollens and also by grafting (Veerakone et al. 2012). Since CLRV is a relatively new disease on hydrangea and no effective control methods have been reported, using clean and

virus-free plant materials is effective to prevent the introduction of the virus to nurseries and landscapes.

4.7 Alfalfa Mosaic Virus (AMV)

AMV has a wider host range including 150 plant species and is widely distributed in the world (Jaspars and Bos 1980). This virus was first reported on *Hydrangea* spp. in British Columbia (Chiko et al. 1986). In the United States, occurrence of AMV in hydrangea was first reported in 2013 (Lockhart et al. 2013). A typical symptom of AMV on hydrangea is yellow leaf blotching. AMV can be transmitted by various aphid species. Control of AMV is achieved by weed management and aphid control (Daughtrey et al. 1995).

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Diseases of *Kalanchoe*

36

Robert L. Wick

Abstract

Kalanchoe blossfeldiana.

Keywords

Erysiphe sedi • *Golovinomyces orontii* • *Pseudoidium kalanchoes* • *Aecidium kalanchoe* • *Stemphylium solani* • *Stemphylium xanthosomatis* • *Stemphylium bolickii* • *Dickeya chrysanthemi* • *Pectobacterium carotovorum* • *Phytophthora niederhauserii* • *Phytophthora cactorum* • *Phytophthora nicotianae* • *Pythium*

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R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_37

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1 Introduction

The florists' *Kalanchoe*, *Kalanchoe blossfeldiana* Poelln., is in the family Crassulaceae. A succulent perennial herb from Madagascar, it was first introduced to the floriculture trade in Germany in the 1930s. It is now widely propagated in Europe and North America and sold as a potted house plant. The genus also serves as the common name, but it also has many other common names.

2 Fungal and Fungal-like Diseases

2.1 Cercospora Leaf Spot (*Cercospora* sp.).

Geographic occurrence and impact. Cercospora leaf spot was described in 1973 in Mississippi (Spencer 1973). It has been noted in Louisiana and California.

Symptoms/signs. The single published description of *Cercospora* on *Kalanchoe* describes it as a severe purple-brown leaf spot, sunken when young and enlarging up to 3 mm. Older lesions become tan with dark margins and may have chlorotic halos; abscission may occur.

Biology and epidemiology. *Cercospora* spp. are known to survive well on plant debris between crops. They produce spores which are easily disseminated by wind currents. The *Cercospora* sp. from *Kalanchoe* grew best at 28° C/82° F at a pH of 5.5 in a liquid medium.

Management

- ***Cultural Practices*** – Diseased plants and plant debris should be removed from the presence of healthy plants. Once the crop is finished, all crop debris should be removed. Avoid overhead watering and reduce leaf wetness duration as much as possible.

- ***Chemical Control*** – The protective fungicide chlorothalonil and the single-site mode of action triazoles and strobilurins give very good protection against *Cercospora* diseases in field crops.

2.2 Leaf Spot and Scorch (*Stemphylium bolickii Sobers* & C.P. Seym., *S. floridanum* C.I. Hannon & G.F. Weber, *S. solani* G. F. Weber, and *S. xanthosomatis* B. Huguenin)

Geographic occurrence and impact. *Stemphylium bolickii* (originally spelled as bolicki) leaf spot was first described on *Kalanchoe*, *Sedum*, and *Echeveria* in 1963, USA; this was also the first description of the species epithet. *Kalanchoe* was described as a host of *Stemphylium floridanum*, 1965, USA. *Stemphylium solani* was first reported in Taiwan in 2011 and *S. xanthosomatis* in Korea in 2012. *Stemphylium solani* has been reported all over the world on a number of vegetable crops and ornamentals. However, *S. solani* isolated from tomato in Florida failed to infect *Kalanchoe* (Sobers and Seymour 1963). All four *Stemphylium* species are reported to be fairly destructive to *Kalanchoe*.

Symptoms/signs. Leaf spots caused by *Stemphylium bolickii* and *S. floridanum* were compared and described by Sobers (Sobers and Seymour 1963; Sobers 1965). With *S. floridanum*, lesions enlarge rapidly and are circular to irregular, black, depressed, and up to 30 mm in diameter. *S. bolickii* causes lesions that are similar in shape, depressed at first but then become raised and scabby, rarely measuring more than 5 mm. Lesions caused by *S. solani* described in Taiwan measured up to 2 mm in diameter with a chlorotic halo (Shen et al. 2011). *Stemphylium xanthosomatis* is described as causing leaf scorch in Korea (Kwon 2012). Browning of the leaf margins is followed by interveinal chlorosis or darkening; finally leaves become desiccated and develop a velvety black olive growth of conidiophores and conidia. Defoliation also occurs.

Biology and epidemiology. *Stemphylium* survives in plant debris as mycelium and its thick-walled, darkly pigmented spores likely provide protection against UV light and desiccation. Optimum temperature for growth of *S. floridanum* isolated from Kalanchoe was 24–28° C/75–82° F, while optimum temperature for growth of *S. bolickii* was 28–32° C/82–90° F (Sobers 1965). Optimum temperature for growth of *S. solani* from *Kalanchoe* was reported to be 24–28° C (Shen et al. 2011). Conidia are produced on the surface of leaves and spread easily by air currents. Free moisture from dew, fog, rain, or irrigation is necessary for spore germination.

Management

- ***Cultural Practices*** – Diseased plants and plant debris should be removed from the presence of healthy plants. Once the crop is finished, all crop debris should be removed. Avoid overhead watering and reduce leaf wetness duration as much as possible.

- ***Chemical Control*** – Protective fungicides such as chlorothalonil and ethylene bisdithiocarbamates (EBDCs) have been shown to protect plants from infection from some species of *Stemphylium.*

2.3 Phytophthora Root and Stem Rot [*Phytophthora cactorum* (Lebert & Cohn) J. Schrot], *P. nicotianae* (Breda de Haan), and *P. niederhauserii* Z.G. Abad et J. A. Abad

Geographic occurrence and impact. *Phytophthora* causes many important diseases of floriculture crops, vegetables, and trees worldwide. All three of the above species are widely distributed. On *Kalanchoe*, *P. cactorum* was reported in Germany (Farr and Rossman 2015), *P. nicotianae* was reported in Korea (Farr and Rossman 2015), and *P. niederhauserii* was reported in Norway (Abad et al. 2014).

Symptoms/signs. Root rot symptoms are indistinguishable from those caused by *Pythium. Phytophthora* is more likely to cause crown and stem rot than *Pythium* and causes a stem rot of *Kalanchoe*. Infected plants collapse and may have lesions moving up the stem.

Biology and epidemiology. *Phytophthora* is not as common in greenhouses as *Pythium*, but it is much more aggressive. There are more than 80 species of *Phytophthora* and most have wide host ranges, especially the three species reported on *Kalanchoe*. Like *Pythium*, *Phytophthora* is an oomycete and is most active when water is plentiful in the growing medium. Dispersal of inoculum is dependent on movement of contaminated water, soil, or plant material. Nearly all species produce oospores, but some need opposite mating types to do so, and some produce chlamydospores. *Phytophthora* is a soil-borne organism and can survive for many years in the absence of a host. Oospores are stimulated to germinate when root exudates are present, and germination often results in the formation of sporangia with swimming zoospores. Temperatures of 28° C/82° F and above favor *P. nicotianae* disease development (Daughtrey et al. 1995). Optimum temperature for growth of *P. niederhauserii* is 30° C/86° F (Abad et al. 2014). Optimum temperature for *P. cactorum* is 25° C/77° F and the maximum is 31° C/88° F (Erwin and Ribeiro 1991).

Management

- ***Cultural Practices*** – When growing plants in a soil-less medium, take care not to introduce field soil by hands, tools, and hose ends dropped on the floor. Use a well-drained medium and avoid overwatering and overfertilization.
- ***Chemical Control*** – Fungicides specific for *Phytophthora* include mefenoxam, etridiazole, and fluopicolide. See comments listed under chemical control of *Pythium*.

2.4 Powdery Mildews [*Podosphaera fuliginea* (Schltdl.: Fr.) U. Braun & S. Takam.], (*Erysiphe sedi* U. Braun), [*Pseudoidium kalanchoes* (Lustner ex U. Braun) U. Braun & R.T.A. Cook, comb. nov.], *and* [*Golovinomyces orontii* (Castagne) Heluta, Ukrayins'k])

Geographic occurrence and impact. The first report of powdery mildew on *Kalanchoe* was in Germany in 1935 followed by England in 1945 (Wheeler 1978). Powdery mildew of *Kalanchoe* has been reported from the USA, Sweden, Switzerland, England, France, Korea, Germany, Hungary, Norway, Bulgaria, and Venezuela.

Symptoms/signs. Symptoms can be nearly undetectable on *Kalanchoe*, but typically powdery colonies with robust mycelial growth can occur and the entire plant may become covered with mildew. When colonies are not obvious, damage to the epidermis can still be evident as a corky or pitted surface (Fig. 1).

Biology and epidemiology. Powdery mildews are obligate parasites of plants and are unique from other plant pathogenic fungi in that the spores do not need free moisture to germinate and infect, and can be more destructive at a relatively low (50–70%) relative humidity. Germination and infection can be inhibited by free moisture (Agrios 2005). Powdery mildews can survive in the absence of the host via chasmothecia. Many powdery mildews have restricted host ranges. *Podosphaera fuliginea* has been reported on the Plantaginaceae and also on *Kalanchoe* by the

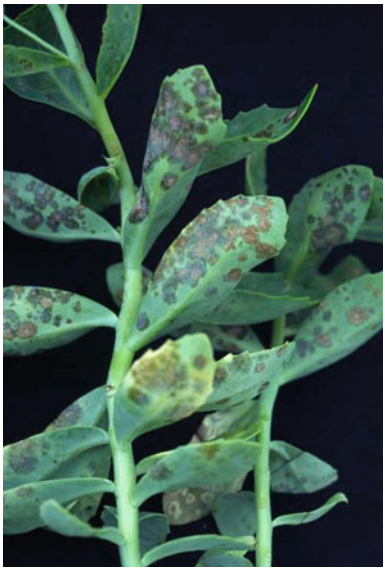


Fig. 1 Powdery mildew of *Kalanchoe blossfeldiana* (L. Pundt © 2017. All Rights Reserved.)

synonyms *Sphaerotheca fuliginea* and *S. humuli* var. *fuliginea* (Braun and Cook). *Pseudoidium kalanchoes* is reported in the Crassulaceae and probably has a wider host range (Braun and Cook 2012). *Golovinomyces orontii* has been reported on about 30 plant families.

Management

- ***Cultural Practices*** – As noted earlier, reduction of humidity is not a practical method to control powder mildews.
- ***Chemical Control*** – Many fungicides will control powdery mildew but nearly all the modern chemistries have a single-site mode of action with some systemic or redistribution potential. Demethylation inhibitors and related chemistry, quinone outside inhibitors, and benzimidazoles all have a single-site mode of action and also select for resistant powdery mildews (Hollomon and Wheeler 2002). Therefore, rotation of fungicides with different modes of action is important.

2.5 Pythium Root and Stem Rot

Geographic occurrence and impact. Pythium root and stem rot have been reported in Denmark and Florida, USA; however, it is presumed to occur worldwide. *Pythium* is one of the most common causes of root rot in greenhouses (Daughtrey et al. 1995)

Symptoms/signs. *Pythium* causes feeder roots to become brown and soft rotted. Usually the cortex easily separates from the vascular system. Root rot causes wilting of the foliage. Occasionally a crown rot occurs, and *Pythium* can move up the stem for some distance.

Biology and epidemiology. *Pythium* is a widespread and common genus of the Oomycota (Daughtrey et al. 1995); nearly every handful of soil contains one or several species. *Pythium* is not usually an aggressive plant pathogen unless plants are growing in a soil-less medium, soluble salts are high, and moisture is abundant. There are more than 200 species of *Pythium*, which generally have a wide host range and vary in their pathogenicity and distribution. *Pythium* species are not strong soil competitors but can survive in soil for long periods of time via oospores. When soil moisture is abundant, root exudates cause oospores to germinate and produce a sporangium where 10–30 zoospores develop in an extra-sporangial vesicle. The zoospores find roots by chemotaxis, and they have flagella that propel them through the water. Once on the root surface, they infect, colonize, and kill the root cells and produce oospores. Young roots and seedlings are particularly susceptible to infection.

Management

- ***Cultural Practices*** – Remove diseased plants from the greenhouse being careful not to spill the soil from the pots. When growing plants in soil-less media,

take care not to introduce field soil by hands, tools, and hose ends dropped on the floor. Use a well-drained growing medium and avoid overwatering and overfertilization.

- ***Chemical Control*** – Many modern fungicides have a single-site mode of action and readily select for resistance. To avoid resistance development, rotate between different FRAC groups (FRAC Code List 2015); do not use one fungicide repeatedly. Note that some isolates of *Pythium* and *Phytophthora* may already be resistant to some of these fungicides. Many greenhouse isolates of *Pythium* in the USA have developed resistance to mefenoxam. The fungicides listed here are followed by the FRAC code in parentheses followed by the risk of selecting for resistance: mefenoxam (4), high; etridiazole (14), low to medium; azoxystrobin (11), high; fluopicolide (43), unknown; fosetyl-al (33), low; fenamidone (11), high; and cyazofamid (21) medium to high.

2.6 Rust (*Aecidium kalanchoe* J. R. Hern., *A. umbilici* Trott.)

Geographic occurrence and impact. *Aecidium kalanchoe* has only been reported in the USA (Idaho) and Colombia. The fungus was reported to cause defoliation in *Kalanchoe blossfeldiana* cvs. Sofie and Colbuco (Hernadez et al. 2004).

Symptoms/signs. Chlorotic lesions composed of spermagonia surrounded by aecia, 1–1.5 cm in diameter form on the leaves (Fig. 2). Defoliation may occur.

Biology and epidemiology. This rust produces only spermagonia with spermatia and aecia with aeciospores; uredinia and telia are unknown. An alternate host is unknown or may not occur.



Fig. 2 Aecia of *Aecidium kalanchoe* on *Kalanchoe blossfeldiana* (J. Hernandez © 2017. All Rights Reserved.)

Management

- ***Cultural Practices*** – Diseased plants and plant debris should be removed from the presence of healthy plants. Reduce humidity and leaf wetness duration.
- ***Chemical Control*** – Ethylene bisdithiocarbamates and chlorothalonil provide broad-spectrum protection against rust. These fungicides have a multisite mode of action and will not select for resistance. Single-site mode of action sterol inhibiting fungicides and strobilurins will provide some curative activity and have longer application intervals.

2.7 Other Minor Diseases Caused by Fungi

Note that these diseases may be minor in importance or they are rare in occurrence. The following fungi are listed in various indices and reports as occurring on *Kalanchoe blossfeldiana*: *Alternaria* sp. Venezuela and West Indies; *Botrytis cinerea*, the USA, Bulgaria, and Korea; *Cylindrocladium* sp., Florida, USA; *Fusarium herbarum*, Germany; *Fusarium oxysporum*, Korea; *Fusarium proliferatum*, Korea; *Glomerella cingulata*, Venezuela, *Lasiodiplodia theobromae*, Venezuela; and *Rhizoctonia solani*, Florida, USA.

3 Bacterial Diseases

3.1 Bacterial Soft Rot and Wilt [*Dickeya chrysanthemi* (Burkholder et al.) Samson et al., *Pectobacterium carotovorum* subsp. *carotovorum* (Jones) Hauben et al. emend. Gardan et al.)]. *Dickeya chrysanthemi* and *Pectobacterium carotovorum* subsp. *carotovorum* formerly *Erwinia chrysanthemi* and *Erwinia carotovora* subsp. *carotovora*, Respectively

Geographic occurrence and impact. Soft rot and wilt have been reported in Italy (Tamietti and Gullino 1988), the Netherlands (Janse and Ruissen 1988), Denmark (Dinesen 1979), and the USA (Engelhard et al. 1986); however, *Dickeya* and *Pectobacterium* have a worldwide distribution and occur on many plants and thus could occur wherever *Kalanchoe* is propagated. Bacterial soft rot is not a major problem, but it can be destructive and persistent if not controlled completely.

Symptoms/signs. Both *Dickeya* and *Pectobacterium* cause soft rot, stunting, and wilt of *Kalanchoe blossfeldiana*. Leaves may become grayish and lose their luster. Browning and soft rot of the pith and basal rot may be evident.

Biology and epidemiology. *Dickeya* and *Pectobacterium* produce pectic enzymes which are responsible for soft rot. These ubiquitous bacteria can survive in soil and may contact plants from dirty hands and tools, splashing water,

insects, or hose ends that have contacted the ground. The bacteria may also come in on cuttings from infected mother plants. Bacteria need wounds or natural openings to gain entry into the host. Plants propagated vegetatively are very vulnerable to infection by soft rot bacteria, especially at high temperatures which are often provided by heating mats to encourage rooting. *Pectobacterium carotovorum* subsp. *carotovorum* has an optimum temperature for growth of 28–30° C/82–86° F but can grow at up to 37–42° C/98–108° F; optimum growth temperature of *Dickeya chrysanthemi* is 34–37° C/93–98° F, and some strains do grow above 40° C/104° F (Daughtrey et al. 1995).

Management

- ***Cultural Practices*** – Do not take cuttings from plants that have evidence of bacterial infections. The cutting bench area should be clean and disinfested. Cuttings should be taken with a sharp knife, and knives should be disinfested frequently. Stick cuttings in growth medium right away or keep plants covered in a disinfested container for a minimum amount of time. If soft rotting occurs in the propagation area, discard all plant materials and thoroughly disinfest all surfaces with a quaternary ammonium compound or similar product (Daughtrey et al. 1995).
- ***Chemical Control*** – Various formulations of copper can afford some protection against bacteria, but are not likely to prevent basal rots and wilts that initiate below the surface of the growing medium.

4 Virus Diseases

4.1 Kalanchoe *Latent Virus* (KLV) (Genus *Carlavirus*; Family Betaflexiviridae)

Geographic occurrence and impact. USA and Denmark but likely to occur wherever *Kalanchoe* is propagated.

Symptoms/signs. This virus is symptomless in *Kalanchoe*.

Biology and epidemiology. Kalanchoe latent virus is a single-stranded RNA *Carlavirus* (Hearon 1982).

Management. Meristem-tip culture can eliminate the virus.

4.2 Kalanchoe Mosaic Virus (KMV) (Genus *Potyvirus*; Family Potyviridae Also Known as Green Island Mosaic)

Geographic occurrence and impact. USA and Denmark but likely to occur wherever *Kalanchoe* is propagated.

Symptoms/signs. Mosaic and "green island" mosaic or a diffuse chlorotic mottle.

Biology and epidemiology. Pinwheel inclusions typical of *Potyvirus* were observed as is the case for other *Potyviruses*. KMV can be transmitted by aphids (Myzus *persicae*) in a non-propagative (nonpersistent) fashion. Symptoms occur 2–3 weeks. after aphid feeding (Husted et al. 1994).

Management. Do not propagate from symptomatic plants. ELISA can be used to detect this virus, and meristem-tip culture can free plants of the virus (Bech and Husted 1996).

4.3 Kalanchoe Top-Spotting Virus (KTSV) (Genus *Badnavirus*; Family Caulimoviridae)

Geographic occurrence and impact. USA and Europe but probably occurs wherever Kalanchoe is propagated.

Symptoms/signs. Newly developing leaves show chlorotic spots which may become depressed as the symptoms fade (Fig. 3).

Biology and epidemiology. Ultrastructural studies showed bacilliform virus particles associated with top-spotting of Kalanchoe (Hearon 1981), and subsequent research confirmed the presence of a *Badnavirus* (Hearon and Locke 1984; Lockhart and Ferji 1988). The virus can be transmitted by vegetative cuttings, grafting, pollen, and seed (Hearon and Locke 1984). The virus can also be vectored by the mealy bug *Planococcus citri* (Brunt et al. 1996). The host range is considered to be very narrow, probably restricted to *Kalanchoe*.

Management. Vegetative propagation is the most likely way this virus is spread. Do not propagate from infected plants. Also, *K. blossfeldiana* can be an



Fig. 3 Top-spotting virus on *Kalanchoe blossfeldiana* (B. Lockhart © 2017. All Rights Reserved.)

asymptomatic carrier. PCR can detect the virus in plants, but unfortunately false positives can occur due to putative integrated pararetroviral elements of KTSV that are non-infective (Yang et al. 2005).

4.4 Tomato Spotted Wilt Virus/Impatiens Necrotic Spot Virus (TSWV/INSV) (Genus *Tospovirus*; Family Bunyaviridae)

Geographic occurrence and impact. The *Tospoviruses* TSWV and INSV have become distributed worldwide; the viruses are known to occur in the USA, South America, Canada, Europe, India, China, and Japan. Reports on *Kalanchoe* include USA, Canada (Tehrani et al. 1990), Finland, and Europe, but it is probably more widespread. TSWV and INSV are not destructive viruses on *Kalanchoe* but they are of economic importance as infected plants would be discarded.

Symptoms/signs. TSWV/INSV can cause distortion of new growth (Fig. 4); ring patterns may or may not occur (Fig. 5).

Biology and epidemiology. TSWV/INSV are *Tospoviruses* in the family Bunyaviridae. Prior to 1990, TSWV was considered to have two strains, TSWV I (impatiens strain), and TSWV L (lettuce strain) (German et al. 1992). In 1990, INSV was described as distinct from TSWV; however, prior to this time, plants showing symptoms of TSWV may in fact have been infected by INSV. At least in the USA, ornamental plants are far more likely to be infected by INSV than TSWV, and the symptoms can be similar (Daughtrey et al. 1997). Antisera are available to distinguish the two viruses. Tospoviruses are vectored exclusively by at least seven species of thrips (Thysanoptera: Thripidae). Only the first and early second stage larvae can acquire the virus, and both the larvae and adults can transmit the virus. The virus/thrip relationship is propagative (persistent) and INSV and TSWV can replicate in the vector.



Fig. 4 Distortion on *Kalanchoe blossfeldiana* foliage by a *Tospovirus* (R.L. Wick © 2017. All Rights Reserved.)



Fig. 5 Ringspots on *Kalanchoe blossfeldiana* foliage by a *Tospovirus* (R.L. Wick © 2017. All Rights Reserved.)

Management

- ***Cultural Practices*** – Inspect plants for viruses before introducing them into the greenhouse. Control weeds on the greenhouse floor and exterior. Monitor for thrips with yellow sticky cards. Screening can be used to cover greenhouse vents, but the material should have apertures of 135 μ or less; this will exclude thrips but can greatly reduce air flow into the greenhouse. Avoid carrying over crop material from season to season.
- ***Chemical Control*** – Insecticides should be used to control thrips; however, many thrips have developed resistance to one or more insecticides. More information can be found in the introductory (▶ Chap. 4, "Insect Management for Disease Control in Florists' Crops").

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Diseases of Poinsettia

37

Margery L. Daughtrey and A. R. Chase

Abstract

Poinsettia, *Euphorbia pulcherrima* Willd. ex. Klotsch, is a major flowering potted plant for winter holidays. Because it is vegetatively propagated and is so widely grown, poinsettia diseases are relatively well known and well studied. Being a woody plant, poinsettias are not prone to tospoviruses, but they are susceptible to a wealth of foliar problems (Botrytis blight, powdery mildew, scab, Alternaria leaf spot, Xanthomonas leaf spot, and anthracnose) as well as many common root and stem diseases (Pythium and Phytophthora root rots, Thielaviopsis root rot) as well as occasional Fusarium or Rhizoctonia stem problems. *Poinsettia mosaic virus* (PMV) has been associated with minor problems, and a phytoplasmal infection has contributed free branching for more attractive plants. Poinsettia diseases may be managed through clean stock production coupled with integrated pest management strategies in greenhouses where the crops are propagated and finished for sale.

Keywords

Poinsettia • *Euphorbia pulcherrima* • IPM • *Botrytis* • Powdery mildew • Scab • *Xanthomonas* • *Pythium* • *Poinsettia mosaic* • Branch-inducing phytoplasma

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© Springer International Publishing AG 2018

R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_39

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1 Introduction

Poinsettia, *Euphorbia pulcherrima* Willd. ex. Klotsch, is a woody plant used as an ornamental, especially as a potted plant for the winter holidays. The poinsettia is native to an area near Taxco, Mexico, where the Aztecs valued it for its red bract

color and made use of it as a dye and for medicines (Ecke et al. 2004). Poinsettias were brought to the United States in 1825 by Joel Roberts Poinsett, the first US ambassador to Mexico. The poinsettias sold from the 1920s to 1960s were largely selections or sports from a single seedling called 'oak leaf' grown in New Jersey in the 1920s, many of them developed by Paul Ecke Sr. in Encinitas, California (Ecke et al. 2004). The plant was developed as an ornamental crop in the United States and Europe in the latter half of the twentieth century, through commercial, university, and federal breeding programs. Their success led to a diversity of bract colors and forms and attractive dark green foliage and branching habit (Ecke et al. 2004). Today the poinsettia is the leading potted flowering plant for indoor use, with a wholesale value of $141 million in 2014 (USDA 2015). Poinsettias are also used as landscape ornamentals in subtropical areas, including the southern United States. Poinsettias for holiday potted plants are produced from cuttings under protective glass or plastic structures in temperate climates during the summer and fall for late fall sales.

Poinsettias are one of the most important flower crops, thus their diseases have received more attention than many. For some of the less common diseases, our knowledge extends only to identification, while for others research has been conducted on poinsettias or other hosts to unravel the secrets of their epidemiology and management (Benson et al. 2001; Strider and Jones 1985). Throughout this chapter, the numeric groupings assigned by the Fungicide Resistance Action Committee (FRAC) will be used to designate mode of action for fungicide active ingredients.

2 Fungal and Fungus-Like Diseases

2.1 Alternaria Leaf Spot [*Alternaria euphorbiicola* E. Simmons and Engelhard, *Alternaria euphorbiae* (Barth) Aragaki and Uchida (comb. nov.)]

Geographic occurrence and impact. Species of *Alternaria* have been reported worldwide on poinsettia (Table 1). The appearance of the disease is inconsistent, however, and its impact on poinsettia production is not high in most greenhouses.

Symptoms/signs. Alternaria leaf spot causes symptoms that are sometimes similar to other foliar diseases of poinsettia, including Phytophthora blight, bacterial canker, and scab. Lesions form on bracts, leaves, petioles, and stems. Purplish-black spots (initially 0.5 mm in diameter) grow to an elliptical shape ($2-4 \times 4-7$ mm); eventually they become irregularly shaped and turn brown, reaching up to 20 mm in diameter (Fig. 1). Chlorotic halos often surround lesions (Fig. 2), with leaf abscission common in severe infections. Lesions on leaf veins lead to distortion. Tan to brown stem lesions (3×8 mm) are elongated and sunken. Infection of cyathia has been reported (Yoshimura et al. 1986).

Biology and epidemiology. In Florida and other subtropical regions that experience significant rainfall, the disease is most severe on poinsettia crops produced in

Table 1 *Alternaria* species reported on poinsettia worldwide (Farr and Rossman 2015)

Alternaria species	Location
Alternaria alternata (syn. *A. tenuis*)	Japan, New Caledonia
Alternaria biproliformis (syn. *A. heveae*)	China
Alternaria euphorbiae	Hawaii
Alternaria euphoribiicola	Florida, Hawaii, Hong Kong
Alternaria obtecta	California, Louisiana
Alternaria pipionipisi, *A. rostellata*	California
A. pseudorostrata	California, United States
*A. protenta (*syn. *A. pulcherrimae)*	Australia
Alternaria sp.	Michigan, Tanzania, Venezuela
Alternaria tenuissima	Hong Kong



Fig. 1 Alternaria spots may occur on bracts as well as leaves (A.R. Chase © 2017. All Rights Reserved.)



Fig. 2 Alternaria leaf spots often show yellow haloes (M. Daughtrey © 2017. All Rights Reserved.)

shade houses or in the landscape. The disease is rarely a problem in greenhouses, where the foliage can be kept dry through careful environmental regulation, especially with the advent of ebb-and-flood or flood-floor production, common for this crop today in many countries.

Management. Cultural practices that eliminate or minimize leaf wetness help limit development of Alternaria leaf spot. Diseased plants and fallen leaf debris should be removed frequently from the growing area.

Thiophanate-methyl should not be used for this disease as it has been shown to increase severity of Alternaria leaf spot on some floricultural crops. Iprodione (FRAC 2), triflumizole (FRAC 3), chlorothalonil (FRAC M5), and trifloxystrobin (FRAC 11) provided about 70% control in Florida trials, but the best control was seen with fludioxonil (FRAC 12), azoxystrobin (FRAC 11), and mancozeb (FRAC M3) (McGovern 1999; McGovern and Wilfret 1998b; McGovern et al. 2001).

Poinsettia cultivars that are relatively resistant to Alternaria leaf spot should be grown. Susceptibility varies among poinsettia cultivars; the only available reports are from cultivars available in the 1980s–1990s. Poinsettia cultivars that are relatively resistant to Alternaria leaf spot should be grown. The most susceptible cultivars included some V-14 cultivars (Glory, White, and Jingle Bells) and Eckespoint C-1 Red. V-10 Amy had intermediate susceptibility. Annette Hegg (Dark Red, Top White, Brilliant Diamond, and Hot Pink) developed only tiny leaf spots with tan centers in resistance trials (Engelhard and Schubert 1985). McGovern and Wilfret (1998a, 1999) reported that the poinsettia cvs. consistently least susceptible to Alternaria leaf spot included Freedom White, Pearl, Petoy, Sonora Pink, Spotlight Dark Red, and Subjibi.

2.2 Amphobotrys Blight [*Amphobotrys ricini* (Buchw.) Hennebert (syn. *Botryotinia ricini* (Godfrey) Whetzel)]

Geographic occurrence and impact. *Amphobotrys* blight and stem rot on poinsettia were first reported from Louisiana and Florida in 1988 and were noted in Bermuda in 1995 (McGovern, personal communication). In 2000, the same pathogen was reported from Florida attacking another species of *Euphorbia* (*E. milii*) causing a flower blight (McMillan et al. 2000). This disease appears to be regional and very rare.

Symptoms/signs. A foliar blight and shoot blight occur; mycelial webbing forms rapidly. Basal stem rot characterized by a soft, watery rot starting at the soil line has also been reported. The rot results in wilting without extensive stem or root rot (Holcomb and Brown 1990). Small, black sclerotia have been found in infected poinsettia stems.

Biology and epidemiology. Several species in the genus *Euphorbia*, as well as *Ricinus communis* (castor bean), are susceptible to this pathogen: *E. supina* Raf. ex Boiss., *E. heterophylla* L., and *E. hirta* L. as well as *E. pulcherrima*.

Management. Shoot tip infections were controlled by used of a thermal fog formulation of chlorothalonil (FRAC M5) but this did not control rot at the stem base (Holcomb and Brown 1990).

2.3 Anthracnose [*Colletotrichum truncatum* (Schwein.) Andrus and W. D. Moore (syn. *C. capsici* (Sydow) E. J. Butler and Bisby)]

Geographic occurrence and impact. Anthracnose on poinsettia caused by *C. capsici* (syn. *C. truncatum*) was reported from Florida and Japan (Sato et al. 2015). Additionally, *C. gloeosporioides* has been reported from Mexico, Florida, and Texas. Anthracnose on poinsettia is not common in the United States.

Symptoms/signs. Leaf and stem spot and blight on poinsettia cultivar Angelika were found in a greenhouse in Japan in 2004. Brown spots 3–5 mm in diameter appeared at first on lower leaves and then enlarged along veins or leaf margins. The spots developed pale brown centers with dark brown borders and dead areas eventually dropped off. Stems became brittle as elongated lesions developed on them. Black acervuli with setae were produced on the lesions under high humidity. Wounded and non-wounded poinsettia plants (cv. Angelika) were inoculated, but symptoms developed only on the wounded plants, 11–21 days after inoculation (Sato et al. 2008).

Management. Utilize environmental control to minimize periods of wetness on leaves and bracts of poinsettias. Avoid wounding foliage or bracts, which might provide entry points for the fungus.

2.4 Botrytis Blight or Gray Mold [*Botrytis cinerea* (syn. *Botryotinia fuckeliana* (de Bary) Whetz)]

Geographic occurrence and impact. *Botrytis cinerea* is a ubiquitous fungus. Botrytis blight occurs wherever poinsettias are grown and causes losses in propagation, finishing, and post-harvest stages.

Symptoms/signs. Botrytis blight is particularly feared as a disease of bracts, but may attack all aboveground portions of poinsettia plants: stems, leaves, bracts, and flowers. Leaf lesions are typically large, light to medium brown, and enlarge further with each period of high humidity, sometimes creating zonate lesions. Infections often start as small light brown lesions at the edge of bracts and grow into large

wedges, having begun as infections at the perimeter of the leaf or bract (Figs. 3 and 4). Stem cankers are usually light brown to tan and may girdle main stems or side branches, causing wilt (Fig. 5). Flower parts may also be infected. The long brown conidiophores topped by clumps of hyaline spores form conspicuous masses on dead plant tissue to give the disease its common name: gray mold.

Biology and epidemiology. There are hundreds of host plants for *Botrytis cinerea*, but poinsettia is one of the most susceptible flower crops. The pathogen is a necrotroph, growing in moribund tissue and secreting cell wall-degrading enzymes in advance of its hyphae. Its ability to infect plants from a morsel of infected tissue is 200 times greater than its ability to cause infection via conidia (Sirjusingh et al. 1996). Wounds in leaves are a common site of infection, especially for cuttings during shipment and propagation.



Fig. 3 *Botrytis* leaf spots often form wedges at the edge of the leaf (M. Daughtrey © 2017. All Rights Reserved.)



Fig. 4 Closeup of *Botrytis* lesion at leaf tip (A.R. Chase © 2017. All Rights Reserved.)



Fig. 5 *Botrytis* stem cankers are tan in color; affected portions of plants will wilt (M. Daughtrey © 2017. All Rights Reserved.)

Disease spread in the greenhouse is largely by dispersal of conidia by air currents (Jarvis 1980). The epidemiology of *Botrytis* populations has been more closely studied in crops other than poinsettia. Conidial germination is best at 20–25 °C/68–77 °F (Blakeman 1980) in the presence of free water (Strider and Jones 1985), although germination has also been noted at extremely high humidity under laboratory conditions. Germination may be delayed for at least 3 weeks (Salinas et al. 1989) when conditions are not sufficiently moist.

Nutrients on the surface of plants are beneficial for infection, which may explain why wounded tissues and flower parts are more likely to be infected than intact leaves (Blakeman 1975). Applications of glucose at 0.1 M and 0.05 M KH_2PO_4 were found to increase germination and infection (Kulek and Floryszak-Wieczorek 2002). Older, more mature leaves are the most susceptible of the intact foliage (Elad and Evensen 1995). Latent infections by *B. cinerea* are one of the main reasons why the disease is so important as a post-harvest problem: symptoms often develop after poinsettias are packed and shipped (Jarvis 1977; Pritchard et al. 1996).

Bract-edge burn in poinsettias, which develops as a result of calcium deficiency at the end of production in certain cultivars, can predispose bracts to *Botrytis* infection that destroys a plant's salability (Barrett et al. 1995).

Inverting the day/night temperature for a poinsettia crop so that night temperatures were warmer than day for the purpose of producing compact plants was shown not to affect poinsettias' susceptibility to Botrytis blight (Pritchard et al. 1996). Higher finishing temperatures, either day or night, did increase blighting and sporulation.

Management. Management of Botrytis blight in greenhouses requires an integrated strategy, as described by Hausbeck and Moorman (1996), comprised of sanitation, environmental control, and appropriate use of fungicides. The cutting wounds made on stock plants and the wounds on the cuttings themselves create critical control points for *Botrytis* blight management. Poinsettia susceptibility has been shown to increase with plant maturity (Pritchard et al. 1996), which increases

the need for careful management just when growers are concerned with shipping. Injury to bracts quickly destroys crop value.

Cultural and environmental controls are extremely important to avoid *Botrytis* losses in poinsettia culture. Organic debris removal from the greenhouse eliminates a source of conidial inoculum, as *B. cinerea* often sporulates profusely when living as a saprophyte on dead plant material. Poinsettias should be grown with adequate nitrogen and light to prevent senescence of lower leaves that could provide a substrate for *Botrytis*. Avoiding the presence of free moisture is largely accomplished by irrigation early in the day and nighttime humidity control. To prevent condensation on plant surfaces, ventilation to remove warm humid air and adequate spacing between plants is necessary. Air movement up between plants is facilitated by a wire-mesh bench, while, in a solid-bench or tray system, horizontal air flow may be employed to provide the necessary air movement (Augsburger and Powell 1986).

When considering nutrient effects on Botrytis blight, it is critical to avoid calcium deficiency. Calcium can reduce the effects of *B. cinerea* on roses (Volpin and Elad 1991). On poinsettias, calcium is an essential nutrient to prevent bract-edge burn, which provides a pathway for the initiation of *Botrytis* lesions on bracts. See ▶ Chap. 3, "Nutritional Disorders of Florists' Crops," for additional information.

Botrytis cinerea is notorious for developing resistance to systemic fungicides. Resistance to benomyl and thiophanate-methyl has been known in greenhouse populations of *B. cinerea* for some time (Maude 1980; Moorman and Lease 1992). Resistance to dicarboximides is known (Pappas 1982; Yourman and Jeffers 1999), and fenhexamid resistance has also been noted in a US greenhouse (Moorman et al. 2012).

A low-residue treatment safe for use on bracts is desired by poinsettia growers, but many fungicides specifically prohibit use on poinsettias after bract development. Treatments with thermal dusts of the fungicide chlorothalonil have been used in the past to protect poinsettias at the end of a crop – but with the negative effect of bract fading noted in some cultivars (Powell 1977). In one study, injury to bracts with a thermal dust treatment of chlorothalonil was found on more cultivars grown with a 24 °C/75 °F night temperature than with at 21 °C/70 °F night temperature (Carlson and Emino 1969). Current labels in the United States indicate that a chlorothalonil thermal dust is to be used on poinsettias prior to bract formation only. The greatest reduction in disease severity was seen with fenhexamid and pyraclostrobin + boscalid treatment in a trial on poinsettias in bract (Leonberger and Beckerman 2009). Prior to bract coloration, a large number of active ingredients are available for use against Botrytis blight on poinsettias in the United States: chlorothalonil (FRAC M5), mancozeb (M3), coppers (FRAC M1), fludioxonil (FRAC 12), cyprodinil + fludioxonil (FRAC 9 + FRAC 12), fenhexamid (FRAC 17), iprodione (FRAC 2), polyoxin D (FRAC 19), pyraclostrobin + boscalid (FRAC 11 + 7), and strobilurins (FRAC 11). Biofungicides labeled in the United States for *Botrytis* management include *Bacillus subtilis* (FRAC NC), *Streptomyces* spp. (FRAC NC), and *Reynoutria sachalinensis* extract (FRAC P). Studies have shown the effectiveness of newer fungicides (Hausbeck and Harlan 2012), including fenhexamid (Benson

and Parker 1999) and strobilurin materials (Benson and Parker 2000a, 2000b), on poinsettia against Botrytis blight.

Genetic control is also relevant for *Botrytis* blight on poinsettias. Cultivars grown in the 1970s varied in susceptibility to the disease (Shanks 1972; Witte and Miller 1976; Manning et al. 1972). Since cultivars grown today are different from those in the 1970s, more recent cultivar comparisons would have more practical value. Elad and Evensen (1995) discussed the various processes associated with senescence that could inform the approach to breeding for resistance to *B. cinerea* in its many hosts. They suggested various techniques for thwarting senescence effects such as increased membrane permeability, including increasing the antioxidant systems and reducing the ethylene response. Physical barriers could also be optimized by developing a thicker, more hydrophobic cuticle, or providing inhibitors of cutinase. Giving poinsettias new defenses against cell wall-degrading enzymes or increasing the wound response rate would be additional strategies. Keeping latent infections quiescent longer and increasing the expression of various chemical defenses are additional goals that would result in improved poinsettia resistance to Botrytis blight.

Learning how to stimulate natural plant resistance mechanisms may provide new avenues of management for *B. cinerea* on poinsettias in the future. Systemic acquired resistance effects were obtained in poinsettia with benzothiadiazole (BTH): treatments at 0.3 mM decreased the susceptibility of 'Coco White' and 'Malibu Red' poinsettia cultivars in a leaf-disc assay (Kulek and Floryszak-Wieczorek 2002).

2.5 Choanephora Wet Rot [*Choanephora cucurbitarum* (Berk. and Rav.) Thaxter]

Geographic occurrence and impact. *Choanephora cucurbitarum* causes a soft, wet rot of poinsettias. The disease usually occurs at the hottest times (August through October) and may affect plants at all stages of production.

Symptoms/signs. Symptoms of Choanephora wet rot resemble those caused by *Rhizopus stolonifer*. A soft, mushy decay can develop on infected leaves and petioles. Infected stems wilt prior to collapsing. Young plants are destroyed under humid conditions, while older plants may develop only a few branches with symptoms. *C. cucurbitarum* produces characteristic whisker-like sporangiophores on diseased tissues (Engelhard 1987).

Biology and epidemiology. *C. cucurbitarum* also causes rot of fruits and vegetables as well as blighting of flowers and, in some cases, immature stems of *Hibiscus rosa-sinensis* and *Petunia* × *hybrida*. Disease develops very rapidly under warm to hot, humid to wet conditions. On bell pepper and swamp *Hibiscus*, the fungus ceases sporulation at temperatures above 14 °C/57 °F.

Management. Minimizing plant stress and spacing to promote air circulation and minimizing leaf wetness have been suggested for management of this disease.

Excellent control of wet rot on poinsettia has been reported with azoxystrobin (FRAC 11), copper pentahydrate (FRAC M1), and chlorothalonil (FRAC M5) (McGovern 1999).

2.6 Corynespora Leaf and Bract Spot [*C. cassiicola* (Berk. and M. A. Curtis) C. T. Wei (syn. *Helminthosporium cassiicola* Berk. and M. A. Curtis)]

Geographic occurrence and impact. Corynespora leaf and bract spot on poinsettia was first reported from Florida (Chase and Simone 1986) and Louisiana (Holcomb and Fuller 1993). The disease has appeared only rarely in the past 20 years.

Symptoms/signs. On poinsettia, large, irregularly shaped, brown to black lesions form on bracts and leaves and strongly resemble those caused by *Botrytis cinerea* infections (Fig. 6). They occur primarily at the tips and margins of leaves and may be as large as 3 cm in diameter. In some cases disease is more prevalent on immature plants.

Biology and epidemiology. Wounding is not usually necessary for infection on poinsettia. *C. cassiicola* is spread by airborne spores. High moisture and humidity are conducive to infection. The fungus has been observed to survive on plant debris for 2 years. Lesions develop within 10 days of infection. Most isolates show no host specialization. A variety of plants are hosts for *C. cassiicola*, including *Aeschynanthus pulcher* (lipstick vine), *Aphelandra squarrosa* (zebra plant), *Catharanthus roseus* (annual vinca), *Hydrangea macrophylla*, *Saintpaulia ionantha* (African violet), and *Salvia splendens* (scarlet sage), as well as vegetable and field crops.

Management. Reduction of humidity and leaf wetness duration are important for preventing infection. Fungicide trials on hydrangeas indicate excellent control with



Fig. 6 *Corynespora* leaf and bract spot on transitional bracts (A.R. Chase © 2017. All Rights Reserved.)

azoxystrobin (FRAC 11), triforine (FRAC 3), copper products (FRAC M1), and a premix of propiconazole and chlorothalonil (FRAC 3 and M5).

2.7 Fusarium Stem Rot [*Fusarium solani* (Mart.) Sacco]

Geographic occurrence and impact. Fusarium stem rot was reported from Wisconsin in 1987 (Heimann and Worf 1987). No additional research and few reports are available: the disease is apparently rare.

Symptoms/signs. Basal cankers, usually about 2 cm above the soil line and 3–6 cm long, are formed (Fig. 7). Lesions noted in Wisconsin were brown to black, somewhat shrunken, and had distinct necrotic margins. All stem tissues except the pith were affected and sometimes the bark tissue appeared shredded. Although cankers became obvious after potting, infected plants developed normally. Stress in the retail environment did result in wilting on retail shelves.

Management. Symptom similarity with Rhizoctonia stem rot may cause misidentification by plant producers. Fungicides for prevention of Rhizoctonia stem rot fortunately could be expected to prevent Fusarium stem rot as well.

2.8 Powdery Mildews: *Leveillula clavata* Nour, *L. taurica* (Lev.) G. Arnaud, *Pseudoidium poinsettiae* (U. Braun, Minnis, and Yañez-Morales), *Phyllactinia poinsettiae*, and *Ovulariopsis erysiphoides*

Geographic occurrence and impact. See Table 2 for reports of occurrence.

The greatest impact of a powdery mildew on poinsettia has been seen from *Pseudoidium poinsettiae* (sometimes referred to as *Oidium poinsettiae*) affecting plants in the United States, Mexico, Puerto Rico, and Europe. The disease was first



Fig. 7 *Fusarium* cankers are brown and form at the soil line (M. Daughtrey © 2017. All Rights Reserved.)

Table 2 Powdery mildew reports on poinsettia from around the world (Farr and Rossman 2015; Siahaan et al. 2015; Daughtrey and Hall 1992; Anonymous 2002)

Powdery mildew species	Location
Leveillula clavata	Canary Islands, Italy, India, Indonesia, Kenya, Tanzania
Leveillula taurica syn. *Oidiopsis taurica*	Canary Islands, Tanzania, India, Indonesia, Kenya
Leveillula sp.	Israel
Oidium sp.	El Salvador, Ethiopia, Guatemala, Mexico, United States
Ovulariopsis erysiphoides	Venezuela
Pseudoidium poinsettiae (syn. *Oidium poinsettiae*), *Erysiphe poinsettiae*	Mexico, Puerto Rico, Denmark, Germany, Sweden, United States
Phyllactinia poinsettiae	Indonesia

seen in the United States in 1990 in Pennsylvania and the Pacific Northwest (Daughtrey and Hall 1992; Kim and Olson 1994); crops in Mexico and Puerto Rico were affected by the disease at about the same time (Daughtrey and Hall 1992). The disease appeared in numerous other states in subsequent years. The mildew seen in the United States has also been seen in the United Kingdom and Europe (Denmark, Germany, and Sweden) starting with the appearance of the *Pseudoidium* in Denmark in 2005 (Anonymous 2002). The impact of this powdery mildew comes not from its effect on plant health, but from the visibility of the fungus: the aesthetic quality of the crop plummets immediately, and infected plants are not saleable. In addition, if poinsettias are sold that have unnoticed infections, the disease will continue to develop in homes and interiorscapes, resulting in obvious white spotting on colored bracts that leads to immense customer dissatisfaction. After the initial few years in which the disease was seen in the poinsettia production industry, when many growers experienced total crop failures, incidence has been sporadic and economic losses have been much less severe. This is due to vigilant scouting and judicious applications of protective fungicide at all levels of production. This powdery mildew continues to be found in a few greenhouses in North America almost every year.

Symptoms/signs. Powdery mildew caused by *Pseudoidium poinsettiae* can be difficult to scout for during the spring and summer, as there are no white colonies then on the upper leaf surface: the only symptoms may be a few round, diffuse, chlorotic spots on the leaves (Fig. 8). Beneath these pale areas, on the abaxial side of the leaf, there are sparse, often indistinct, powdery mildew colonies (Fig. 9). In the fall, when greenhouse temperatures cool, conspicuous white colonies develop on the upper surface of leaves and bracts (Fig. 10). When oval to cylindrical conidia are produced (singly, but forming false chains) from the surface mycelium, the colonies take on a more granulated ("sugar-coated") appearance. If the growth of the powdery mildew is unchecked by fungicide application, colonies will grow until they merge and cover the surface of leaves or bracts.



Fig. 8 Powdery mildew causes chlorotic spots on the upper leaf surface, which mark the location of colonies on the undersurface (M. Daughtrey © 2017. All Rights Reserved.)



Fig. 9 Powdery mildew colonies appear first on the leaf undersurface (M. Daughtrey © 2017. All Rights Reserved.)



Fig. 10 Bracts as well as leaves are susceptible to powdery mildew (M. Daughtrey © 2017. All Rights Reserved.)

Powdery mildew symptoms caused by *Leveillula clavata* (syn. *Oidiopsis taurica*) as described from a 2005 appearance in Italy (Garibaldi et al. 2006) are similar to those in early (summer) infections of poinsettia by *Pseudoidium poinsettiae* in the United States – both fungi cause yellow spots on adaxial leaf surfaces. In other respects the disease caused by *L. clavata* is quite different: bract infections have not been reported with *L. clavata*. Club-shaped conidia are borne singly on conidiophores that emerge through stomates on the abaxial leaf surface, where a rose to reddish mycelium also appears. Chasmothecia with a hemispherical, lenticular or pezizoid shape and simple appendages, containing many asci each with two ascospores, were described on poinsettias in Kenya (Nour 1957). A similar powdery mildew fungus has also been reported on poinsettia from Japan (Horie et al. 2006).

A third genus of powdery mildew, *Phyllactinia poinsettiae* (syn. *Ovulariopsis poinsettiae*), was recently reported from Indonesia (Siahaan et al. 2015). This mildew forms subevanescent colonies on the abaxial side of leaves. Club-shaped conidia with papillate tips are produced singly on conidiophores arising from hyphae.

Biology and epidemiology. Studies of powdery mildew management on poinsettia have largely been confined to *Pseudoidium poinsettiae* (syn. *Erysiphe poinsettiae*). Studies at Michigan State University on this fungus showed conidial germ tubes appearing on poinsettia 'Freedom Red' at 2 h post-inoculation (Celio and Hausbeck 1998); germination reached a peak at 36 h. Appressoria were slightly lobed to lobed. Over half of the germinated conidia had established a globose haustorium within 48 h. Basal cells of conidiophores were arced distinctively. Early steps in colony establishment (percentage of conidial germination, secondary germ tube formation, and haustorium formation) were limited at 30 °C/86 °F as compared to 20 °C/68 °F. The powdery mildew can be maintained on symptomless plants over the summer (Kim et al. 1995a), thus growers should not count on disease eradication through exposure to typical summer greenhouse temperatures. The suppressive effect of high temperatures on powdery mildew of poinsettia is, however, reflected in field observations that the disease does not progress during the summer months in the northeastern United States (Kim et al. 1995a). The optimum for germination of conidia was found to be 25 °C/77 °F and 85% relative humidity (RH) in greenhouse studies (Hausbeck and Kalishek 1994), conditions which are typically available in greenhouses in temperate climates in the fall.

Peak concentrations of conidia in the greenhouse were associated with dips in RH or changes in temperature, which often occurred simultaneously (Shaw and Hausbeck 1995; Byrne et al. 2000). Symptoms were more severe and the conidial concentration was higher (1,221 conidia/m^3 air/h) in one greenhouse with an average RH of 64%, while the conidial concentration in a similar greenhouse with an average RH of 53% was only 317 conidia/m^3 air/h.

Studies in Germany indicated that the powdery mildew from poinsettia (referred to as *Oidium* sp. but presumably the *Pseudoidium* seen in the United States at about the same time) could be transferred to other *Euphorbia* spp. as well *E. exigua*, *E. heterophylla*, *E. helioscopia*, and *E. marginata* (Anonymous 2004). These plants

or wild *E. pulcherrima* could provide a reservoir of inoculum near poinsettia cutting production facilities.

Management

- ***Cultural practices*** – Keeping humidity down (<85%) in the greenhouse and improving air circulation help to slow an epidemic of powdery mildew, but are not sufficient to halt it (Hausbeck and Kalishek 1994). The disease continues to develop after poinsettias leave the greenhouse and enter retail or customer environments. Treatment with elevated temperature (>30 °C/86 °F) has been suggested as a possible technique for specialist propagators to use prior to shipping cuttings (Celio and Hausbeck 1998), but the plant safety and effectiveness of this procedure have not yet been tested.
- ***Sanitation*** – Fortunately *P. poinsettiae* has not been found causing disease on any other hosts in northern temperate climates, so removing all poinsettias from the greenhouse at the end of the growing season is likely to provide complete eradication of inoculum. Inadvertent reintroduction on cuttings the next season is the grower's only concern unless plants are being grown in subtropical areas where poinsettias grow year-round. Removing infected leaves as soon as they are detected is not often a practical solution, but reduces inoculum in the greenhouse and at high greenhouse temperatures could be eradicative early in the growing season (Hausbeck et al. 1994). Early detection of powdery mildew is important, as it alerts the grower to the need for an appropriate fungicide program for that year's crop. Leaf removal must be combined with fungicide treatment to avoid extensive crop loss in the fall, when greenhouse conditions become more conducive to disease development and spread. Chasmothecia have not been found for *P. poinsettiae*, thus there is no persistent inoculum on leaf debris in the greenhouse after plants are sold.
- ***Fungicides and biocontrols*** – Fungicides have been very effective against the *Pseudoidium* on poinsettia, perhaps in part because the fungus was introduced to horticultural productions systems only recently. Both protectant and systemic materials have been very effective when used preventively. Sprays for powdery mildew control need not be applied unless the pathogen is present, so a careful scouting program should be in place in order to detect the disease before it is widespread. Many fungicide labels restrict use on poinsettias to the time before bracts begin to color, so chemical control must be established before the finishing stages. Weekly or biweekly treatments of a wide range of materials including potassium bicarbonate (FRAC NC), copper pentahydrate (FRAC M1), thiophanate-methyl (FRAC 1), trifloxystrobin (FRAC 11), kresoxim-methyl (FRAC 11), polyoxin D (FRAC 19), pyraclostrobin (FRAC 11), iprodione (FRAC 2), and demethylation inhibitor materials (triflumizole, triadimefon, and myclobutanil) (FRAC 3) have provided significant protection (Daughtrey and Macksel 1995; Daughtrey and Tobiasz 1999; Gould and Bergen 1995a, b; Hausbeck and Kusnier 1995a, b, c, 1996; Hausbeck et al. 2002a, b; Daughtrey and Tobiasz 2009). Rotation of active ingredients with different modes of action is essential for powdery mildew management, so it is fortunate that there are many

fungicide options for this purpose during all but the finishing stages of production.

Biological controls are desirable for powdery mildew control in poinsettias, particularly because growers are not comfortable applying fungicides to colored bracts and because many fungicide labels prohibit such use. A program alternating a biological control (*Bacillus subtilis*) with a biorational material (potassium bicarbonate) reduced powdery mildew significantly in an inoculated trial (Daughtrey and Tobiasz 2010).

- ***Resistance*** – There is no known resistance to powdery mildew (*Pseudoidium poinsettiae*) in poinsettia, but the level of susceptibility varies among cultivars. Celio and Hausbeck (1997) tested 11 cultivars for susceptibility, and all developed powdery mildew colonies within 31 days. Three red cultivars tested had significantly more powdery mildew than other cultivars in two trials. Kim et al. (1995b) reported higher susceptibility in leaves and bracts of the cultivar 'Freedom Red' as compared to the older cultivars 'Dark Red Hegg' and 'V-17 Angelika White' in a greenhouse trial. If the most popular red cultivars are prone to powdery mildew, growers may not have the option of growing less susceptible types. Prioritized scouting of the more susceptible red cultivars has been suggested (Celio and Hausbeck 1997) to improve scouting efficiency. In a trial in Germany, *Euphorbia fulgens* and *E. milii* were not infected with the pathogen although four other *Euphorbia* species were shown to be hosts (Anonymous 2004). Plant breeders will ideally expose prospective new lines to *P. poinsettiae* to select for poinsettias with low susceptibility to this disease.

2.9 Pythium Root Rot (*Pythium* spp.)

Geographic occurrence and impact. *Pythium* species are water molds belonging to the Oomycetes and not true fungi. The species reported to occur on poinsettia are *P. aphanidermatum*, *P. cryptoirregulare* (syn. *Globisporangium cryptoirregulare*), *P. debaryanum* (syn. *Globisporangium debaryanum*), *P. irregulare* (syn. *Globisporangium irregulare*), *P. perniciosum*, and *P. ultimum* (Farr and Rossman 2015) as well as *P. myriotylum* and *P. helicoides* (Miyake et al. 2014). Strider and Jones (1985) mention *P. ultimum* as the primary problem in poinsettia production up until that time, but *P. aphanidermatum* has caused the most dramatic losses in recent years (Moorman et al. 2002).

Pythium root rot is widely distributed, and no doubt occurs wherever this crop is grown. Formal reports of Pythium root rot on poinsettia caused by one or more species have come from South America, the United States, Japan, New Zealand, and Brazil (Farr and Rossman 2015). The disease can have imperceptible effects on plant quality or can cause extensive mortality of poinsettias, especially in irrigation systems that allow recirculation of water. The particular *Pythium* species that are important today are different from those that were important in the first half of the twentieth century, largely due to a shift to production systems using soilless media and recirculating subirrigation. Changes in the cultivars grown may also have affected which root diseases are most important, and production of cuttings in new

geographic areas around the world may have led to the introduction of new pathogen species or strains.

Symptoms/signs. There is a soft brown or gray-brown rot of the cortex of the fibrous roots, which contrasts sharply with the white, vigorous growth of a healthy root system (Fig. 11). Severe aboveground symptoms of Pythium root rot include stunting of the whole plant and even wilting and death. Leaves yellow, starting with the lower leaves, and may curl upward as they wilt. In advanced infections, the cortex will slough off the hard xylem core as the plant is pulled from the pot, leaving behind thin white strings of xylem (Fig. 12). Symptoms appear on the roots on the outside of a rooting cube or on roots adjacent to pot sidewalls; they are generally most pronounced in the bottom of the pot. Because *Thielaviopsis basicola* and *Rhizoctonia solani* both cause a drier looking rot, Pythium root rot is most easily confused with Phytophthora or Fusarium root rots. Fungus gnat feeding is often coincident with Pythium root rot, but either the insect or the water mold acting alone can cause serious root injury. See ▶ Chap. 4, "Insect Management for Disease Control in Florists' Crops," for additional information.



Fig. 11 Pythium root rot (*at right*) in contrast to a healthy root system (M. Daughtrey © 2017. All Rights Reserved.)



Fig. 12 Pythium root rot (showing xylem remaining after cortex has sloughed off) (M. Daughtrey © 2017. All Rights Reserved.)

Biology and epidemiology. The biology of the different *Pythium* species varies somewhat, but there are characteristics that they share. They are fungus-like in their appearance, but molecular studies have verified the longtime suspicion that they are members of a unique group, more closely related to algae than to fungi. *Pythium* spp. possess coenocytic hyphae with cell walls composed of cellulose rather than the chitin of a true fungus. In the wet environments that are favorable for infection, most *Pythium* species accomplish asexual reproduction by forming sporangia (in some cases these are merely swollen hyphae). Motile zoospores are released from thin-walled vesicles produced by the sporangium of a *Pythium* sp., rather than being formed within the sporangium in the manner of *Phytophthora*. Often chlamydospores and/or hyphal swellings may be formed by the hyphae as well.

The sexual structures are termed oogonia and antheridia, with these structures sometimes coming from the same individual and sometimes requiring an opposite mating type. The result of fertilization of an oogonium by one or more antheridia results in the formation of an oospore. The thick walls of the oospore make it very resistant to environmental stresses and to other microorganisms, so it is a long-term survival structure. Oospores in organic debris are the main source of contamination in a greenhouse operation. Oospores are sometimes present in the peat moss itself or sometimes linger in recycled pots or trays that have not been thoroughly cleaned and disinfested. Morphological differences were originally used to identify *Pythium* species, but DNA analysis has led to many changes in *Pythium* taxonomy and nomenclature in recent years, including the creation of new genera that were once considered to be within the genus *Pythium* (Uzuhashi et al. 2010).

Because different *Pythium* species have different temperature optima, problems with particular species may appear at different times of year. The most commonly encountered *Pythium* in earlier years, when poinsettias were grown in soil-based media, was *P. ultimum*; root rot from this pathogen was shown to be most severe at 17 °C/62 °F and to cause little effect at temperatures at or above 26 °C/79 °F (Bateman and Dimock 1959). Pythium root rot was at that time seen commonly at the end of the production season, when temperatures were lowered to slow plant development prior to sale.

Today, *P. ultimum* is occasionally encountered, but the most serious *Pythium* pathogen on poinsettia in North America today is *Pythium aphanidermatum*, first seen in 1977 as a pathogen on poinsettia in Canada on plants grown from cuttings propagated in the United States (Moorman et al. 2002; Komorowska-Jedrys et al. 2008). This organism causes disease at higher temperatures than *P. ultimum* (Hendrix and Campbell 1973) and produces prolific numbers of zoospores that make it ideally suited to thrive in recirculating irrigation systems (Sanogo and Moorman 1993). Temperatures are favorable to *P. aphanidermatum* in summer, when most of the vegetative growth of poinsettias takes place. This species was reported causing cutting rot in soilless production systems in Brazil (Palmucci and Grijalba 2007). In addition, Miyake et al. (2014) reported sudden outbreaks of root rot in ebb-and-flood production of poinsettias in Japan, with *P. myriotylum* and *P. helicoides* discovered to be the pathogens. Their subsequent research found that isolates of all three high-temperature-loving *Pythium* species – *P. aphanidermatum*,

P. myriotylum, and *P. helicoides* – gave the greatest incidence and severity of root rot at 35 °C/95 °C when inoculated to poinsettia. Although favored by hot environments, all three were able to cause disease at 20 °C/68 °F at higher inoculum (zoospore) levels.

Bateman (1961) found that overwatering (keeping soil at 70% moisture holding capacity or above) promoted Pythium root rot in 'Barbara Ecke' poinsettias. He also showed that neutral to alkaline soil was favorable to root rot caused by *P. ultimum* (Bateman 1962).

A study by Moorman (1986) showed that growing poinsettias with higher fertility increased Pythium root rot caused by *P. ultimum*. Fertilization rates tested ranged from 100 to 600 μg N/g. Higher soluble salt levels were correlated with increased mortality in poinsettias inoculated with *P. ultimum* early in production in three different soilless mixes as well as in a soil-containing mix.

Management. Identification of the species of *Pythium* present is important to designing a management program. Both *P. irregulare* and *P. cryptoirregulare*, indistinguishable morphologically, occur on poinsettias in greenhouses (Garzon et al. 2007) and are rarely problematic unless cultural controls are mismanaged. *P. aphanidermatum*, *P. helicoides*, and *P. myriotylum* are all relatively aggressive pathogens and are all favored by high temperature. Globalization of the ornamental plant trade is no doubt having an effect on the species and genotypes of *Pythium* and other pathogens that are encountered in greenhouses. An analysis of *P. aphanidermatum* found on poinsettia in Pennsylvania greenhouses noted genotypes from around the world (Lee et al. 2010). Variation in susceptibility to *P. aphanidermatum* among poinsettia cultivars has been noted (Daughtrey and Hyatt 2012); growers experiencing extensive losses may find it worthwhile to trial different cultivars.

Poinsettia growers are generally fearful of Pythium root rot losses and use very careful sanitation procedures and treatments with biological and chemical fungicides to ensure crop safety. Sanitation efforts are directed against the introduction of oospores in field soil, a particular threat in flood-floor operations. Whereas earlier production methods included steam pasteurization to ensure the safety of soil-containing growing media, today's poinsettia growers use soilless growing mixes based largely on sphagnum peat moss. Cuttings are often rooted in artificial foam materials – a practice which eliminates peat moss as a possible source (Graham 1994). Clean growing surfaces and clean irrigation and handling practices using soilless media provide a measure of security that was not available to a poinsettia grower 50 years ago. Sanitation between crops will be especially important in operations with recirculating irrigation systems, which favor dissemination and infection of *Pythium* spp. Miyake et al. (2014) noted that poinsettias were first seen with root rot caused by *P. helicoides* in Japan in ebb-and-flow production following rose and begonia crops that were known hosts of that species. Although monitoring for Pythium root rot is usually a matter of watching for symptoms, bentgrass traps have been used to monitor for populations of high-temperature *Pythium* species in ebb-and-flood systems in Japan. These traps successfully detected the

pathogens 30 days before symptom development was evident from infections of *P. helicoides* in potted miniature roses (Watanabe et al. 2008).

Boehm and Hoitink (1992) found that higher microbial activity in a light-colored, less-decomposed sphagnum peat was associated with short-term suppression of Pythium root rot. Treatments with bioantagonists against *P. ultimum* var. *ultimum* in subirrigated poinsettias were trialed in Canada (Little et al. 2003). Quality was similar when inoculated plants were protected with metalaxyl or with treatments of *Streptomyces griseoviridis* strain K61 or *S. lydicus* strain WYEC 108. Other bioantagonist products including *Trichoderma* spp. and *Bacillus subtilis* active ingredients are also labeled for use against *Pythium* spp. in greenhouses.

There are few fungicides with strong effectiveness against *Pythium*. Trials have shown the relative effectiveness of some of the oomycete fungicides (Parker and Benson 2011, 2012, 2013; Benson and Parker 2001b; Hausbeck and Harlan 2007). Fungicides that have been used for *Pythium* management include metalaxyl/mefenoxam (FRAC 4), the effectiveness of which has been reduced in the greenhouse arena due to the development of resistance in some *Pythium* populations (Moorman et al. 2002). The effectiveness of etridiazole (FRAC 14), sold singly and also in combination with thiophanate-methyl (FRAC 1) for broad-spectrum root rot management, continues: there has not been any resistance to this active ingredient in greenhouse *Pythium* populations. Fosetyl-Al and other phosphorous acid materials (FRAC 33) can reduce *Pythium* losses to some degree. More recently, cyazofamid (FRAC 21) and fluopicolide (FRAC 43) have been introduced for control of *Pythium* root rot in greenhouses. Strobilurins (FRAC 11) give partial but not strong control of *Pythium*.

2.10 Phytophthora Crown and Root Rot (*Phytophthora drechsleri, P. nicotianae, P. cryptogea*)

Geographic occurrence and impact. Three species of *Phytophthora* have been reported from poinsettia: *P. drechsleri* (Korea and the United States) and *P. nicotianae* (United States, Korea, Brazil, Puerto Rico, Japan) have been known to be poinsettia pathogens for some time (Farr and Rossman 2015; Kanto et al. 2007; Estevez de Jensen et al. 2006). In Taiwan, *P. cryptogea* as well as *P. nicotianae* occurred on rotted poinsettia roots (Ann 1992), and Orlikowski also described *P. cryptogea* on poinsettia in Poland (Orlikowski and Ptaszek 2013). Some additional species have been seen on *Euphorbia* relatives of poinsettia, including *P. cactorum*, *P. citricola*, and *P. palmivora*. The disease is lethal to infected plants. Its impact depends upon the production system: in greenhouses with recirculating irrigation systems, the potential for extensive crop loss is high. Poinsettia cultivars vary in their susceptibility to Phytophthora root rot (Woodworth and Hausbeck 2003).

Symptoms/signs. The part of the poinsettia plant affected by *Phytophthora* varies from outbreak to outbreak: whereas Engelhard and Ploetz (1979) described *P. nicotianae* causing a crown and stem rot, in Hawaii a short time later,



Fig. 13 Root rot caused by *Phytophthora drechsleri* (M. Daughtrey © 2017. All Rights Reserved.)

P. nicotianae and *P. drechsleri* were found causing symptoms on leaves, cyathia, and bracts (Yoshimura et al. 1985). In the United States, *P. drechsleri* has been a significant problem in cutting production in ebb-and-flood benches, causing root rot and stem rot (Lamour et al. 2003) (Fig. 13). *P. cryptogea* caused extensive stem base rot as well as root rot and wilt on stunted poinsettias in an outbreak in Poland (Orlikowski and Ptaszek 2013).

Biology and epidemiology. The pathogen is thought to be moved from greenhouse to greenhouse on cuttings (Lamour et al. 2003). Infected stock plants may lead to extensive loss of cuttings during propagation. Because of the production of sporangia and zoospores, dispersal of *Phytophthora* inoculum during production is especially likely in plants grown on a subirrigated recirculating irrigation system (Strong et al. 1997). In a US study, the populations of *P. drechsleri* affecting two poinsettia greenhouses were unique to the two locations and showed no genetic diversity within a greenhouse (Lamour et al. 2003), indicating asexual reproduction within the two facilities.

Management. Removing the obviously diseased plants from a recirculating irrigation system is not sufficient to remove all of the inoculum when attempting to halt an epidemic. A system for filtering beneath each pot would be ideal (Van der Gaag et al. 2001), or alternatively treating water to be toxic to zoospores would be useful (von Broembsen and Deacon 1997). Trials have shown the benefit of certain fungicide treatments to prevent Phytophthora disease on poinsettias (Daughtrey and Tobiasz 2003; Hausbeck and Harlan 2009). Current methods in the production industry rely heavily upon chemical control in response to an outbreak, with fungicides registered for this use in the United States including cyazofamid (FRAC 21), dimethomorph and mandipropamid (FRAC 40), etridiazole (FRAC 14), fluopicolide (FRAC 43), fosetyl-Al and other phosphorous acid compounds (FRAC 33), mefenoxam (FRAC 4), and strobilurins (FRAC 11) (Daughtrey et al. 2015). There is little published data on the use of biopesticides for Phytophthora disease suppression in poinsettias (Hausbeck et al. 2004a).

2.11 Rhizoctonia Cutting Rot, Crown Rot, and Root Rot [*Rhizoctonia solani* Kuhn (syn. *Thanatephorus cucumeris* (A. B. Frank) Donk)]

Geographic occurrence and impact. *Rhizoctonia solani* is widely distributed, has a wide host range, and is very common in propagation of poinsettias.

Symptoms/signs. When *R. solani* causes cutting rot, stems become darkened and mushy at the soil line (Fig. 14). Under the warm, humid conditions typical of propagation, the fungus will grow over the rooting cube as well as the entire cutting (Fig. 15). When this occurs, a light brown mycelium, closely appressed to the plant tissues and soil, is sometimes visible. The fungus grows out more or less radially from the point of contamination, giving the damage a circular or arc-shaped pattern. The mycelium can also cause a web blight and appears as a brown, cobweb-like growth on the leaves. Leaves in contact with the growing medium can develop roughly circular lesions (Fig. 16).

Root rot caused by *R. solani* usually shows discrete, brown lesions and rotting of the cortical tissues, leading to decreased plant development. *R. solani* causes cutting



Fig. 14 Rhizoctonia stem rot (M. Daughtrey © 2017. All Rights Reserved.)



Fig. 15 Root rot caused by *Rhizoctonia solani*, with visible mycelium on the surface of rooting cubes (M. Daughtrey © 2017. All Rights Reserved.)



Fig. 16 Mycelium of *Rhizoctonia* solani on a blighted leaf (M. Daughtrey © 2017. All Rights Reserved.)

rot more often than root rot, although poinsettia is quite susceptible to Rhizoctonia root rot.

Crown rot commonly occurs in the absence of root rot and first appears as a brown canker at the root crown. Longitudinal cracking and a dry appearance of the rotted crown tissues often develop on older plants. Other aboveground symptoms include chlorosis, wilting, loss of lower leaves, and in severe cases stunting and plant death.

Biology and epidemiology. Poinsettias are most susceptible just before or soon after rooting and just prior to plant maturity. *R. solani* generally grows best in potting media that are evenly moist and warm: Rhizoctonia root rot increases at soil temperatures between 17 and 26 °C (62 and 79 °F) (Bateman and Dimock 1959) with a moisture holding capacity below 40%. *R. solani* also grows best in soils with high oxygen and low carbon dioxide levels (Bateman 1961).

Management. Sanitation practices to guard against soilborne fungi are applicable to *R. solani*. Methods for keeping field soil out of contact with a soilless growing

medium are effective at preventing most Rhizoctonia disease problems for poinsettias.

- ***Cultural methods*** – Composts from municipal sewage sludge have been explored for use in ornamental production. Compost cured for at least 4 months with an internal temperature <60 °C/140 °F suppressed *Pythium* but not *Rhizoctonia* spp. An additional 4 weeks of storage resulted in a medium suppressive to *Rhizoctonia* as well as *Pythium*. Trialing a potting medium amended with 25% of the aged compost showed that poinsettias had significantly less root damage caused by *R. solani* (Kuter et al. 1988).
  Suppression of Rhizoctonia crown and root rot of poinsettia was evaluated in potting mixes prepared with dark, highly decomposed sphagnum peat; with light, less-decomposed sphagnum peat; or with composted pine bark. In none of the media was the disease consistently suppressed. Inoculation of these mixes with *Chryseobacterium gleum* (C299R2) and *Trichoderma hamatum* 382 (T382) significantly reduced the severity in the composted pine bark mix. Both biocontrol agents maintained high populations over 90 days in this medium. The microorganisms were less effective in the light and dark peat mixes, in which populations of C299R2 declined. In contrast, crown and root rot was suppressed in all three types of mixes where high populations of T382 were maintained (Krause et al. 2001).
- ***Biological Control*** – A variety of antagonists/biological control organisms have been evaluated for their ability to affect Rhizoctonia cutting rot on poinsettia. Cartwright and Benson (1994) evaluated the effects of *Burkholderia* (syn. *Pseudomonas*) *cepacia* (strain 5.5B) and *Paecilomyces lilacinus* (isolate 6.2 F) applied to polyfoam rooting cubes and found that colonization of cubes by *R. solani* was reduced in cubes treated with *B. cepacia*. Colonization of the *P. lilacinus*-treated cubes by *R. solani* was significantly less than colonization of infested controls. Direct treatment of poinsettia stock plants with sprays of *B. cepacia* was also effective in preventing Rhizoctonia cutting rot (Cartwright and Benson 1995a, 1995b). Survival of *B. cepacia* and *P. lilacinus* in rooting cubes showed that *P. lilacinus* was stable over a 3-week period, while some strains of *B. cepacia* decreased after only 7 days. The population level of these antagonists determined the amount of protection of poinsettia cuttings against *R. solani*.
  Strategies for applying *Burkholderia cepacia* (strain 5.5B) and Pesta formulations of binucleate *Rhizoctonia* (BNR) isolates (BNR621 and P9023) were evaluated (Hwang and Benson 2002). *B. cepacia* suppressed *Rhizoctonia* cutting rot during propagation, while BNR isolates did not. After transplanting, BNR isolates were more effective for suppression of stem and root rot than application of *B. cepacia*. Application of different biocontrol agents during the different production phases of poinsettia was effective for disease control.
- ***Fungicides*** – Fungicides such as strobilurins (FRAC 11), fludioxonil (FRAC 12), iprodione (FRAC 2), pentachloronitrobenzene (FRAC 14), thiophanate-methyl (FRAC 1), and triflumizole (FRAC 3) are effective as drenches against *Rhizoctonia*. Treatments must be made before infection occurs.

The effect of spraying the surface of rooting cubes compared to soaking the cubes with the same fungicides was evaluated by Benson (1991). Surface sprays with benomyl (FRAC 1), flutolanil (FRAC 7), iprodione (FRAC 2), or chlorothalonil (FRAC M5) prevented *Rhizoctonia* infection. Rooting cube soaks with benomyl, flutolanil, or iprodione were as effective as the sprays of the cubes in preventing disease. Rooting cube soaks with benomyl and sprays with iprodione, chlorothalonil, or benomyl inhibited root initiation but not final growth of the plant. A rooting cube soak with iprodione did not affect rooting (Benson 1992). Azoxystrobin (FRAC 11) as a preplant soak to rooting cubes or post-plant spray was also effective against *R. solani* and not harmful to the plant (Benson and Parker 2001a).

2.12 Rhizopus Blight [*R. stolonifer* (Ehrenb.:Fr.) Vuill. (syn. *Rhizopus nigricans* Ehrenb.)]

Geographic occurrence and impact. Rhizopus blight of *Euphorbia* spp. (including poinsettia) was reported during the 1980s in Florida (Chase and Engelhard 1984). The pathogen is more commonly known as a bread mold or storage mold on ornamentals, soft fruits, and vegetables.

Symptoms/signs. Typical growing conditions in the summer in the southern United States favor infection and development. The tissue is blighted, and webs of white mycelium may develop over the dead tissue under humid conditions. The sporangia (seen as black specks) appear on the white mycelial webs (Fig. 17) and on cankered stems (Fig. 18). Poinsettias may show symptoms during propagation or soon after potting. In the northern Unites States, *R. stolonifer* causes a stem rot of poinsettia. A dark, greasy-looking discoloration extends 10 cm or more up from the stem base. Poinsettias may suddenly wilt and collapse; symptoms may be mistaken for bacterial soft rot.



Fig. 17 Rhizopus blight (A.R. Chase © 2017. All Rights Reserved.)



Fig. 18 Mycelium and sporulation of *Rhizopus stolonifer* on a canker (A.R. Chase © 2017. All Rights Reserved.)

Biology and epidemiology. *R. stolonifer* was reported as a pathogen of *Euphorbia* spp. previous to its documentation as a pathogen of *Gerbera jamesonii* (African daisy), *Crossandra infundibuliformis* (crossandra), *Catharanthus roseus* (annual vinca), and *Sinningia speciosa* (gloxinia). It is particularly aggressive on *E. milii* (crown of thorns). The fungus affects a wide range of non-ornamental hosts as well, most commonly causing a soft rot of fruits. Annette Hegg poinsettia cultivars were reported to be less susceptible than V-14 Glory or V-14 White.

Spores of *R. stolonifer* are ubiquitous and survive desiccation. The pathogen grows rapidly at 21–32 °C/70–90 °F; disease develops only at a relative humidity of more than 75%. *R. stolonifer* usually colonizes wounds as an opportunistic pathogen, often affecting crops growing under stressful conditions.

Management. Poinsettia crops should be maintained under optimal cultural conditions, because outbreaks of this disease have generally followed periods of stress. It is particularly important to maintain ideal conditions for prompt callusing and rooting of cuttings, since the cutting wound is a major point of entry for the fungus. Infected plant debris should be removed. No reports of effective fungicides have been made for Rhizopus blight on poinsettia.

2.13 Scab (Spot Anthracnose) (*Sphaceloma poinsettiae* Jenk. and Ruehle)

Geographic occurrence and impact. Poinsettia scab was initially reported on poinsettia from Florida (Ruehle 1941). The disease is distributed throughout subtropical and tropical America including Florida, Puerto Rico, and Hawaii, as well as the South Pacific, on the genus *Euphorbia*. During the late 1960s scab became less serious as disease-free cuttings became increasingly available. However, the rapid spread of scab-infected cuttings worldwide does happen occasionally.

Symptoms/signs. Tan spots (1–4 mm in diameter) with 1- to 2-mm chlorotic haloes appear. The leaf tissue may buckle, causing the leaves to distort somewhat (Fig. 19). Occasionally, larger necrotic areas may develop, with the entire leaf turning chlorotic and abscising. Scab-like lesions can form on stems and petioles; these are 1–10 mm in diameter and may be either rounded or elongated. Lesions are usually tan and may have a red or purple margin. Stem lesions, which may be sunken and can contain fungal growth (Fig. 20) and gray sporulation (Fig. 21), may coalesce to girdle stems. One interesting characteristic is the abnormal growth of infected stems, which can be markedly longer than uninfected stems on the same plant (Fig. 22).

Biology and epidemiology. Inoculation trials with isolates of *S. poinsettiae* from poinsettia showed that *E. heterophylla* (Mexican fire plant), *E. prunifolia* (painted euphorbia), *Manihot esculenta* (cassava), and *M. carthaginensis* were all susceptible. Symptoms are apparent 7 days after stem inoculation, and younger leaves are more susceptible than the older leaves. Conidia were produced 1 month after inoculation in one study; they are readily spread by splashing water from irrigation and rainfall.

Management. Disease-free cuttings should always be used: growers should make a thorough examination of all propagation material whether rooted or unrooted. A



Fig. 19 Leaf lesions of poinsettia scab (*Sphaceloma poinsettiae*) (M. Daughtrey © 2017. All Rights Reserved.)



Fig. 20 White lesions on stem due to scab (*Sphaceloma poinsettiae*) (M. Daughtrey © 2017. All Rights Reserved.)



Fig. 21 Stem lesions of scab bearing light brown sporulation (M. Daughtrey © 2017. All Rights Reserved.)



Fig. 22 Hyper-elongated stems are an indication of scab infection (M. Daughtrey © 2017. All Rights Reserved.)

grower should not retain diseased stock plants from season to season. A trial in 2001 that evaluated 42 cultivars showed that the cultivars with dark green leaves were slightly less susceptible than cultivars with leaves having lighter green coloration (Daughtrey and Tobiasz 2002). Research at Cornell University and Michigan State

University concluded that all cultivars tested (in excess of 100 over a 4-year period) were susceptible to *Sphaceloma*. The most sensitive cultivars included Champagne Punch, Cranberry Punch, Strawberry Punch, Redberry Punch, Snowberry Punch, Freedom Fireworks, Freedom Pink, Freedom Coral, Jester Red, Peterstar White, Peterstar Pink, Peterstar Orange, Peterstar Marble, Winterfest Coral, Winterfest Red, Silver Star, Max Red, Eurostar, Chianti Red, Eternity Red, and Enduring Pink. On the other end of the spectrum, the lowest disease levels were seen on Classic Red, Holly Point, Jingle Bells, Strawberries and Cream, Thanksgiving Red, Winter Rose White, Winter Rose Pink, and Freedom Salmon.

Excellent prevention of scab with fungicides was found in one trial on Freedom Red with pyraclostrobin and boscalid (FRAC 7 and 11), azoxystrobin (FRAC 11), copper fungicides (FRAC M1), mancozeb (FRAC M3), and chlorothalonil (FRAC M5) (Hausbeck et al. 2004b). In a 2000 trial on Freedom Red and Pink Success, good control with a 7-day interval was found with trifloxystrobin (FRAC 11), copper pentahydrate (FRAC M1), and triflumizole (FRAC 3). Reduction of symptoms with combination products of copper hydroxide and mancozeb or thiophanate-methyl (FRAC 1) and chlorothalonil was also high, but residue after several weekly sprays was excessive. Foliar residues from wettable powder products may be reduced by adding a spreader-sticker or wetting agent (Daughtrey and Tobiasz 2001).

2.14 Thielaviopsis Black Root Rot: *Thielaviopsis basicola* (Berk. and Broome) Ferraris (syn. *Chalara elegans*)

Geographic occurrence and impact. Black root rot caused by *Thielaviopsis basicola* (syn. *Chalara elegans*) is found worldwide, with the causal fungus affecting many hosts. This was a very common and damaging disease in the early years of poinsettia production, but since the advent of growing poinsettias in soilless media, the incidence of the problem has plummeted. Sanitation efforts by poinsettia propagators have also been credited with reducing this problem (Strider and Jones 1985).

Curiously, black root rot is today a severe and common problem in production of certain bedding plants, notably pansies and calibrachoas, grown in the same greenhouse operations as poinsettias that remain free from the disease. It is unclear whether this is due to host specialization of the pathogen on the bedding plants or to reduced susceptibility of modern poinsettias.

Symptoms/signs. Black root rot darkly discolors roots (Fig. 23), but generally leaves the tissues intact – in contrast to the more common Pythium root rot, which causes a lighter brown, softer rot. Another distinguishing trait is that Pythium root rot begins at the root tip, whereas *T. basicola* infection can occur anywhere on the root system (Perry 1971). Identification of the disease is best accomplished by direct examination of discolored root tissues under a compound microscope, as the pathogen grows slowly in standard laboratory media and can be overlooked in the presence of faster growing microorganisms. The root system is diminished in size



Fig. 23 *Dark brown* root discoloration results from *Thielaviopsis basicola* attack on poinsettias (M. Daughtrey © 2017. All Rights Reserved.)

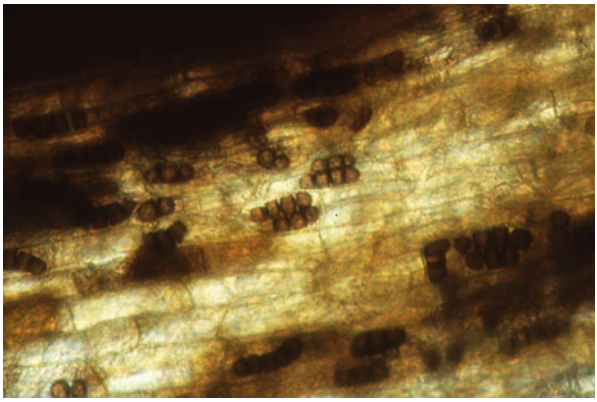


Fig. 24 Dark chlamydospores of *T. basicola* in an infected root (M. Daughtrey © 2017. All Rights Reserved.)

by early infections and will in all cases show sections that are darkened, in part by the brown chlamydospores (Fig. 24) of the fungus.

Affected plants are stunted if infection occurs before plant maturity, and foliage can be undersized and chlorotic. In particular, the lower leaves of infected plants turn yellow, curl, and defoliate, similarly to leaves on plants with Pythium root rot. Leaf spotting due to *T. basicola* was reported on one occasion (McCain and Raabe 1975).

The infection may be evident at the stem base, where dark brown chlamydospores (aleuriospores) accumulate on the stem en masse (Fig. 25). The stem may also show dark longitudinal cracks – a distinctive characteristic of black root rot (Bateman and Dimock 1959).

Biology and epidemiology. Black root rot is generally thought of as a soilborne disease, but in greenhouses there are opportunities for aerial or waterborne spread. The mycelium of *T. basicola* produces two kinds of spores, both of which are infectious. In addition to dark-walled chlamydospores, *T. basicola* forms hyaline phialospores, produced as endoconidia, on the surface of the root. In the humid, still air of petri plates, these endoconidia will develop as long chains of cylindrical spores



Fig. 25 Black discoloration at the base of the stem is an indication of *T. basicola* attack (M. Daughtrey © 2017. All Rights Reserved.)

on root lesions incubated on moist filter paper. The phialospores may be airborne in greenhouses (Graham 1994), spreading the pathogen to noncontaminated growing media, but how commonly this happens is not known. The phialospores are probably spread through recirculating irrigation systems, but it was noted in 1994 that transmission through a recirculating system had not been conclusively demonstrated (Stanghellini and Rasmussen 1994), and no subsequent studies have been conducted to our knowledge. The phialospores were initially thought to be short lived, but studies have shown that they may persist in soil for months to over a year, depending upon factors such as soil type (Schippers 1970).

The chlamydospores of *T. basicola* are generally produced in short chains of 1–8 dark brown segments supported by 2–3 hyaline cells (Patrick et al. 1965) on root surfaces or within the root cortex. When fragmented into the individual spores, they are more difficult to recognize. Fragmentation has been observed to be a necessary precursor to germination (Patrick et al. 1965). The chlamydospores are resting spores with the ability to survive years without a host (Stover 1959), so they present a challenge for greenhouse sanitation.

Bateman (1961) established that high soil moisture is conducive to poinsettia infection by *T. basicola*. The disease was very severe at 70% moisture holding capacity and above. The infective phialospores can germinate quickly in wet soils even without additional nutrients (Punja 1993). Chlamydospores also germinate readily in wet soils (Linderman and Toussoun 1967).

Black root rot is encouraged by cooler soil temperatures. High temperature suppresses disease: in inoculated mineral soil, poinsettias showed severe root rot at 13–26 °C/55–79 °F with a maximum at 17 °C/62 °F, but almost no symptoms at 30 °C/86 °F or above (Bateman and Dimock 1959).

Soil pH has a strong effect on this disease: acidic soils are disease suppressive. Growing poinsettia at a pH of 4.8 prevented disease (Keller and Shanks 1955), and

pH 5.5 or lower reduced the impact of black root rot or *Pythium* infection on poinsettias grown in mineral soil (Bateman 1962).

Fungus gnats can disseminate the pathogen (Harris 1995). Shore flies have also been shown to spread *T. basicola* chlamydospores aerially within a greenhouse (Stanghellini et al. 1999). See ▶ Chap. 4, "Insect Management for Disease Control in Florists' Crops," for additional information.

Management

- ***Sanitation*** – Strider and Jones (1985) pointed out that black root rot was not as important a disease in North Carolina as it had been in the past, and this was attributed to the propagators' efforts at improving sanitation during cutting production, the use of soilless growing media, and preventive fungicide drenches. The disease is rarely seen today. Reuse of containers after black root rot outbreaks will lead to repeated occurrences of disease (Graham 1994). A hydrogen dioxide sanitizer at recommended rates and chlorine bleach (1:9 dilution of a 5.25% solution) were shown to be highly effective disinfectants (Warfield 2003). Use of bromine or quaternary ammonium compounds did not rid greenhouse surfaces of *T. basicola* inoculum, whereas chlorine bleach at a 1:4 dilution (1.04% sodium hypochlorite) was effective on all surfaces tested (plastic, wood, and metal) (Copes and Hendrix 1996). Aerated steam heat treatment for 30 min at 50 °C/122 °F completely eliminated *T. basicola* from a soilless mix and was safe on plastic pots and trays (Linderman and Davis 2008).
- ***Cultural Control*** – Using a well-drained growing medium is important for black root rot avoidance. Avoiding a high pH that favors black root rot can be part of the overall integrated pest management plan, but should not be used to the detriment of the nutritional status of the plant. A pH low enough to suppress black root rot, pH 5.2 (Strider and Jones 1985), is lower than the recommended pH range for poinsettia cultivation, pH 5.8–6.2 (Williams 2011).
- ***Biocontrol*** – Biocontrol of black root rot has not been developed for floriculture production systems. Fluorescent pseudomonads were identified (Stutz et al. 1986) in the microflora of soil suppressive to black root rot in tobacco, with their plant-protective effect being attributed in part to production of pathogen inhibitors such as 2,4-diacetylphloroglucinol and/or hydrogen cyanide (Ramette et al. 2006). A more recent study used microarrays to identify *Azospirillum*, *Gluconacetobacter*, *Burkholderia*, *Comamonas*, and *Sphingomonadaceae* associated with a soil suppressive to black root rot of tobacco (Kyselkova et al. 2009). A commercial formulation of *Streptomyces griseoviridis* reduced black root rot on citrus when used at a high rate (0.03%, 0.32 g/L) (Graham 1994).
- ***Fungicides*** – Benzimidazole fungicides (FRAC 1) were seen as the most effective against *T. basicola* in the past (Manning et al. 1970), and today one of the fungicides in this same mode of action group, thiophanate-methyl, has the best reputation for management of the disease (Daughtrey 2006; Atwood 2013). Other materials should be used in rotation with thiophanate-methyl to fend off resistance development. Some of the effective choices would be the active ingredients polyoxin D, triflumizole, and fludioxonil, found in fungicides in FRAC groups

19, 3, and 12, respectively (Daughtrey et al. 2015). Fungicides may be used to good purpose to protect the remainder of the crop after a disease outbreak, but great effort should be expended at the end of the growing season to eliminate the fungus from the greenhouse.

3 Bacterial Diseases

3.1 Bacterial Canker [*Curtobacterium flaccumfaciens* pv. *poinsettiae* (Starr and Pirone) Collins and Jones (Previously *Corynebacterium flaccumfaciens* subsp. *poinsettiae*)]

Geographic occurrence and impact. A bacterial blight of poinsettia was originally described from New Jersey in 1942 (Starr and Pirone 1942). Widespread occurrence in old and new plants, home gardens, and nursery plantings as well as many separated localities indicated that the disease may have been present for many years prior to its initial identification (Creager 1959). It has been subsequently reported in Florida (McFadden and Creager 1960), Maryland, New York, Pennsylvania, Virginia (Salazar 1983), and New Zealand. Disease outbreaks may result in extensive damage to the crop but appear to be rare.

Symptoms/signs. Stems, leaves, and bracts may become blighted. In some cases, only branch cankers are noted, and disease incidence is often low in a particular crop. The bacterium causes elongated, water-soaked to brown streaks visible on the surface of the stem (Fig. 26). Irregular water-soaked to brown cankers that girdle the stem also can form and the stem tip may curve. Terminal leaves may become deformed; irregular brown spots develop and leaf abscission occurs. Severely cankered stems show longitudinal cracks and sometimes amber droplets containing bacteria are found (McFadden 1959).



Fig. 26 Bacterial canker caused by *Curtobacterium flaccumfaciens* pv. *poinsettiae* (Mary Ann Hansen © 2017. All Rights Reserved.)

Biology and epidemiology. Environmental sources of *C. f. poinsettiae* have not been identified. The bacterium has been recovered from asymptomatic, healthy-appearing, rooted poinsettia cuttings; 10% of asymptomatic plants tested were infected. The bacterium can ooze from infected leaves and stems and be splashed by irrigation water to adjacent plants. Infection of leaves occurs through stomates. It is probable that pruning tools and hands can transmit the bacterium from plant to plant during standard crop culture. Warm conditions and high nitrogen levels have been reported to be associated with disease occurrence. Disease is not usually evident until the plants are close to finishing.

Management. Plants showing symptoms of this disease should be removed and discarded. Due to the slow disease development, the location of the pathogen in the vascular system, and little if any plant-to-plant movement during the growing season, bactericidal sprays have not been evaluated and are not warranted. It is possible that bactericides used for other bacterial pathogens are masking low levels of infection due to the bacterial blight pathogen.

3.2 Greasy Canker [*Pseudomonas viridiflava* (Burkholder) Dowson]

Geographic Occurrence and Impact Canker on poinsettia caused by *Pseudomonas viridiflava* was described and named greasy canker in 1981 (Suslow and McCain 1981). The disease was also reported in Florida (Engelhard and Jones 1990), and the pathogen has been cultured from stems and foliage of diseased poinsettia in Massachusetts.

Symptoms/signs. The first report of this disease described a canker and leaf spot as well as a bract and bud blight (Fig. 27). Cankers may originate at pruning wounds on the stem. The cankered areas are greasy in appearance with no soft rot. They eventually turn light tan to brown and, as the cuticle lifts from the stem, they take on a papery texture. Leaf spots are angular and randomly distributed.

Biology and epidemiology. *P. viridiflava*, first described from green beans in 1927, is an opportunistic pathogen, reported to be an epiphyte on some plants and a secondary invader. Marginal leaf necrosis and necrotic spots on stems form on potted plant genera including *Euphorbia*, *Hibiscus*, *Capsicum*, and *Hydrangea.* Inoculations of other hosts of *P. viridiflava* from poinsettia strains were successful, showing no evidence of host specificity.

Inoculations of seven poinsettia cultivars showed no resistance to the bacterium. There are conflicting reports of the optimum temperature for disease development in poinsettia. Suslow and McCain (1981) reported that severity was greater at 27–32 °C/80–90 °F than at 25 °C/77 °F. However, tests in Florida indicated that disease was severe at 10 and 15 °C (50–59 °F), mild at 27.7 °C/82 °F, and absent at 32.2 °C/90 °F. Because of this variability, it does not appear that temperature can be



Fig. 27 Poinsettia bract inoculated with *Pseudomonas viridiflava* (A.R. Chase © 2017. All Rights Reserved.)

used to predict disease occurrence or severity for *P. viridiflava*. No other studies of greasy canker on poinsettia have clarified this discrepancy of results, which may have been due to the cultivar of the host chosen and the specific strain of *P. viridiflava* employed.

Management. Control measures outlined for *Xanthomonas* leaf spot (below) are appropriate for greasy canker. Plants known to be infected should be discarded and not used for propagation. Although no bactericides have been demonstrated effective against diseases caused by *P. viridiflava* on poinsettia, it is likely that those effective on other bacterial diseases will have an impact.

3.3 Soft Rot and Stem Rot [*Dickeya chrysanthemi* (syn. *Pectobacterium chrysanthemi*) and *Pectobacterium carotovorum* (syn. *Erwinia carotovora* (Jones)) Holland.]

Geographic occurrence and impact. Soft rot on poinsettia cuttings was first reported from Missouri and attributed to *Erwinia carotovora* by Rogers (1959); the pathogen was later renamed *Pectobacterium carotovorum*. Another soft rot bacterium causing stem rot in poinsettia (Hoitink and Daft 1972), previously named *Erwinia chrysanthemi* or *E. carotovora* subsp. *chrysanthemi*, has now been moved to the genus *Dickeya* (Ma et al. 2007). *Dickeya chrysanthemi* was recently reported from China (Rungnapha et al. 2008).

Symptoms/signs. One of the most common symptoms on poinsettia is a cutting rot (Fig. 28). During propagation, cuttings develop a water-soaked decay, beginning at the cutting wound. In other cases, symptoms may be delayed, so that extensive symptoms appear suddenly in hot weather, presumably due to latent infections.



Fig. 28 Bacterial soft rot in propagation (A.R. Chase © 2017. All Rights Reserved.)

Chlorotic spots anywhere along the stem and purplish-black petioles are the first noticeable symptoms. The spots rapidly coalesce, forming large, irregular, chlorotic areas. Petioles turn black and shrivel and affected leaves wilt. Infected tissues are soft and water soaked. Mature plants may also collapse from latent infections with *D. chrysanthemi* in hot weather.

Management. Maintenance of proper shipping environment (temperature and moisture) minimizes subsequent development of soft rot on unrooted cuttings. Shading cuttings during rooting is important for reducing stress, thus promoting fast rooting and reducing bacterial cutting rot. A waterlogged growing medium is conducive to disease development. Growers report that water management during propagation is the key to preventing cutting rot due to soft rot bacteria. Typically, no bactericides are used.

3.4 *Xanthomonas* Leaf Spot [*X. axonopodis* pv. *poinsettiicola* (Patel et al. 1951) Vauterin, Hoste, Kersters, and Swings 1995 (syn. *X. campestris* pv. *poinsettiicola*)]

Geographic occurrence and impact. Leaf spot of poinsettia caused by a *Xanthomonas* species was first observed in India in 1951 (Patel 1951). The disease was later described in Florida in 1962, and in 1985 a xanthomonad was reported on *Codiaeum* (croton – another euphorbiaceous plant) that was able to infect both croton and poinsettia. The pathogen previously named *Xanthomonas campestris* pv. *poinsettiicola* was separated into three species in 1995 on the basis of DNA:DNA homology, with the pathogen most often associated with poinsettia separated out as *X. axonopodis* pv. *poinsettiicola*. Classification of these bacteria is not yet entirely resolved. Two other pathovars of *X. campestris* have also been reported to cause leaf spot on poinsettia: *X. c. euphorbiae* (Sabet, Ishag, and Khalil) Dye and *X. c. manihotis* (Berthet and Bondar) Dye have been reported to cause poinsettia leaf spot upon inoculation. Although *Xanthomonas* leaf spot appeared rarely in poinsettia



Fig. 29 *Xanthomonas* leaf spot (*Xanthomonas axonopodis* pv. *poinsettiicola*) (A.R. Chase © 2017. All Rights Reserved.)

production in the past, in the late 1990s and continuing today, it is found in many locations worldwide: Slovenia (Dreo et al. 2011), Norway (Perminow et al. 2011), China (Li et al 2006), Austria (Gottsberger and Plenk 2009), Taiwan (Lee et al. 2006), and Italy (Stravato et al. 2004).

Symptoms/signs. Spots are at first visible on the undersurface of the leaf as gray to brown, water-soaked lesions (Fig. 29). As they enlarge to 2–3 mm, they become visible on the top surface of the leaf and are chocolate brown to rust colored and may or may not have pale green halos. Spots may coalesce into large areas of blighted tissue. Severely spotted leaves turn yellow and abscise.

Biology and epidemiology. In a Florida inoculation trial, *X. campestris* pv. *poinsettiicola* was shown to be pathogenic to poinsettia and to croton, *Codiaeum variegatum* (L.) Blume (Chase 1985). The latest taxonomic revision separates out the bacteria pathogenic to *Codiaeum* as *X. codiaei* and uses the name *X. axonopodis* pv. *poinsettiicola* for the poinsettia pathogen, but there is likely some overlap in their host ranges. *X. c. euphorbiae* causes leaf spot on *E. acalyphoides* (a weed from Sudan) and will spot poinsettia upon inoculation. *X. c. manihotis* is a non-pigmented xanthomonad and is pathogenic to *Manihot esculenta* Crantz (cassava) and to poinsettia upon inoculation. Recently, a study of xanthomonads associated with poinsettia leaf spot over a 64-year period has uncovered additional genetic diversity in the bacteria able to cause symptoms on poinsettia (Rockey et al. 2015); now we ascribe what we have long considered one bacterial leaf spot disease of euphorbs to multiple pathogens within the genus *Xanthomonas* and anticipate further clarification with additional research.

Little has been published on the epidemiology of bacterial leaf spot on poinsettia, but it has been reported to spread rapidly, presumably from splashing water and from worker handling. If epiphytic populations occur, as is known with other diseases caused by xanthomonads, then colonized, asymptomatic cuttings may develop disease under favorable conditions.

Management. Affected plants should be discarded, and no cuttings should be taken from plants with leaf spots. Wetting of foliage should be avoided, especially during periods that would extend the leaf wetness duration.

Some work has been conducted to identify potential leaf-associated bacterial strains for biological control of the pathogen (Li et al. 2008) from China. Two strains with promise were identified as *Bacillus amyloliquefaciens* (PAB241 and PAB242).

Poinsettia plants treated with titanium dioxide (TiO_2 at 25 and 75 mM) showed 85–87% and 92–93% reductions in lesions, respectively. No phytotoxicity was observed (Norman and Chen 2011). Studies with conventional products have been generally less successful. In one trial acibenzolar drench and mancozeb (FRAC M3) provided the best control, followed by acibenzolar foliar spray (which was moderately phytotoxic) and cupric hydroxide (FRAC M1). In another trial, copper pentahydrate (FRAC M1) and didecyl dimethyl ammonium chloride (DDAC) (FRAC NC) provided excellent prevention.

3.5 Poinsettia Branch-Inducing Phytoplasma (*Candidatus* Phytoplasma sp.)

Phytoplasmas are plant pathogenic bacteria in the class *Mollicutes* that are restricted to phloem, resulting in symptoms that include yellowing, stunting, flower malformation, and witches' brooms (Lee et al. 2000; Chaturvedi et al. 2010). Although a disease, infection of poinsettias with the poinsettia branch-inducing phytoplasma (PoiBI) introduces welcome rather than undesirable traits because it promotes branching to give the potted poinsettia a fuller and more compact form.

Geographic occurrence and impact. This phytoplasma affects poinsettias all over the world. Chen et al. (2007) examined multiple poinsettia cultivars originating from the United States and Europe and found all of them to carry phytoplasmas.

Symptoms/signs. Poinsettias infected with this phytoplasma have less apical dominance and show more growth from axillary branches than those lacking the pathogen (Fig. 30). Some side effects of the phytoplasma infection include shorter internodes, shallower leaf lobes, thinner stems, paler bract color, earlier anthesis, and cyathia abortion and lower fertility (Preil 1994).

Biology and epidemiology. The main poinsettia phytoplasma belongs to 16SrIII-H, a subgroup of the X-disease group (Chaturvedi et al. 2010). However, there are three additional types of phytoplasma that have been found in poinsettia (Pondrelli et al. 2002): the B and C subgroups of 16Sr1 and 16SrXII-A are also sometimes found by PCR. The 1967 cultivar 'Annette Hegg' was the first to show the desirable trait of free branching. It was discovered that the trait was graft transmissible (Stimart 1983). Many attempts were made to identify the branching factor. In 1997 a phytoplasma was determined to be the branch-inducing factor (Lee et al. 1997),



Fig. 30 The plant at left exhibits the morphology induced by the poinsettia branch-inducing phytoplasma, in contrast to a phytoplasma-free plant of the same cultivar at right (Michael Klopmeyer © 2017. All Rights Reserved.)

and it was shown that there were multiple phytoplasmas occurring in poinsettia (Abad et al. 1997). PCR tests may be used to identify the presence of phytoplasmas in poinsettia. A comparison of gene expression in infected vs. healthy poinsettia (Nicolaisen and Christensen 2007) indicated increased expression of cytokinin biosynthesis genes in plants containing phytoplasma.

Management. Tetracycline has been used to cause reversion of poinsettia from the free-branching form to the healthy form (Bradel et al. 2000a). Since the diseased form is more desirable in the trade, however, the tetracycline treatment is primarily useful for research purposes.

4 Viruses

4.1 Poinsettia Mosaic (*Poinsettia mosaic virus* PnMV)

Geographic occurrence and impact. This virus is likely to be found in commercially grown poinsettias all over the world and also affects *Euphorbia fulgens* (Koenig et al. 1986). In a survey in British Columbia (Chiko 1983) in 1981, PnMV was found in eight of nine cultivars tested (all Annette Hegg types) and in 94% of the 65 samples.

Symptoms/signs. Poinsettias with PnMV may show malformed leaves or bracts and a systemic mosaic of the foliage (Fig. 31) (Fulton and Fulton 1980). Expression of symptoms is thought to be greater at cooler temperatures (Fulton et al. 1978); symptoms were noted in winter but not in summer in Queensland, Australia (Gordon et al. 1996).

Biology and epidemiology. Particles of PnMV are icosahedrons 26–29 nm in diameter. Although when initially characterized PnMV was thought to be a



Fig. 31 Mosaic caused by *Poinsettia mosaic virus* (Dag-Ragnar Blystad © 2017. All Rights Reserved.)

tymovirus (Fulton and Fulton 1980), studies on PnMV recently provided good evidence that the virus is instead a *Marafivirus*, and it has now been designated a species of the genus *Marafivirus* (Bradel et al. 2000b; Koenig et al. 1986). PnMV was transmitted by grafting to two other euphorbias: *E. cornastra* (Dressler) A. Radcliffe-Smith, which was symptomless, and *E. bubalina* Boiss., which showed a mild mosaic (Fløistad and Blystad 1999). There is no evidence of seed transmission; the virus is transmitted by mechanical inoculation. Curiously, although no vector is known, plants freed from PnMV have been seen to become reinfected quickly (Blystad and Fløistad 2002).

Standard production practices did not allow disease spread in one trial (Pfannenstiel et al. 1982), yet commercial propagators have had difficulty ridding their poinsettias of the virus. Pfannenstiel et al. (1982) were able to cure shoots of the viral infection with heat therapy (32 °C/90 °F for at least 9 weeks), and then to graft these shoots to disease-free rootstocks, so it seems that clean stock should be obtainable.

Management. Efforts by Bech and Rasmussen (1996) were effective at curing poinsettias of PnMV. They used meristem-tip culture to eliminate PnMV from 'Freedom' poinsettia, but the branching of the resulting plant indicated Freedom from the poinsettia branching-inducing phytoplasma as well and thus was unacceptable. A temperature treatment of 'Lilo' resulted in plants free from PnMV but branching normally. Using RNA silencing methodology, transgenic poinsettias were developed that were resistant to mechanical inoculation of PnMV (Clarke et al. 2008).

4.2 *Poinsettia Latent Virus* (PnLV)

Poinsettia latent virus (PnLV) causes no symptoms in poinsettia and was earlier named poinsettia cryptic virus (Koenig and Lesemann 1980). It has icosahedral particles and is a chimera, with genetic features of both poleroviruses and sobemoviruses (Aus dem Siepen et al. 2005). It is found in many commercial lines of poinsettia today, where it occurs along with *Poinsettia mosaic virus* (PnMV) and the phytoplasma that causes free branching.

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Part IV

Florists' Crops Diseases: Flowering Geophytes

Diseases of Caladium

38

H. M. Bowman, J. E. Polston, and Robert J. McGovern

Abstract

Caladium (*Caladium* x *hortulanum* Birdsey, Araceae; syn. *C. bicolor* Vent.) is a popular interior and exterior florists' crop known for its multicolored foliage. Approximately 95% of the world's caladiums are produced in Florida, USA. Pathogen infection and disease can occur during all stages of caladium development: production (division and in the field) and storage. Common diseases of caladium are caused by *Fusarium solani*, *Pythium* spp., *Rhizoctonia solani*, *Sclerotium rolfsii, Pectobacterium carotovorum*, *Xanthomonas axonopodis*, *Dasheen mosaic virus*, and *Meloidogyne* spp.

Keywords

Aspergillus niger • *Botrytis cinerea* • *Hinomyces moricola* • *Fusarium solani* • *Pythium* spp • *Rhizoctonia solani* • *Sclerotium rolfsii* • Grassy tuber • *Xanthomonas axonopodis* • *Pectobacterium carotovorum* • *Dickeya padantii* • *Caladium virus X* • *Dasheen mosaic virus* • *Konjac mosaic virus* • *Meloidogyne* spp

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R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_40

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1 Introduction

Caladium (*Caladium x hortulanum* Birdsey, fam. Araceae; syn. *C. bicolor* Vent.), a native to the tropical Americas and the West Indies, is grown as an ornamental for its arrow-shaped, dramatic multicolored foliage, and tolerance to heat, shade, and clay/sand and acidic soils. Caladiums are used in interior and exterior landscaping (Evans et al. 1992). Approximately 95% of the world's caladiums are produced in Florida, USA (Deng and Harbaugh 2007) in highly organic muck soil (80–90%) and sandy soils (10–20%). Pathogen infection and disease can occur during all stages of caladium development: production (tuber division and in the field) and storage. Caladium production involves vegetative propagation through the division of tubers and rarely by seed or tissue culture (Wilfret 1993). This method of propagation facilitates the spread of disease-causing pathogens from season to season and field to field (Harbaugh et al. 1988).

Where data are available, specific information on caladium disease management is presented in this chapter. General disease management strategies may be found in the introductory part "Components of Integrated Disease Management."

2 Fungal and Fungus-Like Diseases

2.1 Aspergillus Tuber Rot (*Aspergillus niger* van Tieghem; *Aspergillus parasiticus* Speare)

Geographic occurrence and impact. *Aspergillus niger* is a common pathogen of many stored plant products (cashew, dates, figs, garlic, onions, etc.) and is ubiquitous in the environment. Both *Aspergillus niger* and *A. parasiticus* have been reported to cause tuber rot in caladium (Alfieri et al. 1994; Varga et al. 2004). Mixed infections with other pathogens such as *Fusarium* spp. are common.

Symptoms/signs. Symptoms of Aspergillus tuber rot include small brown sunken and rotted areas on the tuber. Under warm conditions, the fungus sporulates from these lesions giving infected tubers a fuzzy black appearance. The black sporangia of *Aspergillus* are slightly larger than pepper grains and are easy to see with the naked eye. Severe Aspergillus rot will result in loss of tuber viability.

Biology and epidemiology. Moist and humid storage environments favor disease development and sporulation, and conidia are easily spread through air currents and water splash. Wounded and senescent tissues are most susceptible to invasion. The optimum temperature for growth of *A. niger* in culture is 30–35 °C (86–95 °F) (Hayden and Maude 1994).

Management

- ***Cultural practices*** – Avoid wounding the tubers during harvest and postharvest operations as much as possible.
- ***Sanitation*** – Hot water treatment at 50 °C (122 °F) for 15 min significantly reduced recovery of *A. niger* and other fungi from corn seed and increased seed germination (Rahman et al. 2008). Hot water treatment of caladium seed tubers at 50 °C for 30 min is routinely used by growers as a preplant practice for control of root-knot nematodes (*Meloidogyne* spp.) (Rhoades 1961).

2.2 Botrytis Blight or Gray Mold (*Botrytis cinerea* Pers.:Fr.); Tuber Rot (*Amphobotrys ricini* (N.F. Buchw.) Hennebert (syn. *Botrytis ricini*))

Geographic occurrence and impact. *Botrytis cinerea* has a very broad host range that includes most cultivated crops. *Amphobotrys ricini* has a narrower host range including castor bean, poinsettia, and strawberry. Botrytis blight can be a problem for potted caladium production in cool humid environments, while tuber rot can occur during storage (Hansen and Bega 1955).

Symptoms/signs. As the name gray mold implies, a prolific mass of gray, fuzzy fungal growth (masses of hyphae, conidiophores, and conidia) can be observed on

infected tissues under humid conditions. *Botrytis cinerea* can cause spots and blight on leaves as well as crown rot of caladium. Symptoms will initially appear on leaves and petioles as small tan to brown spots. As they enlarge leaf lesions will often develop a zonate pattern. If disease development is favored, blighting can occur on shoots, leaves, and petioles and, if left unchecked, entire plants.

Biology and epidemiology. *Botrytis cinerea* can survive as a pathogen on a very wide range of plant hosts including many florists' crops, as hyphae in plant residue and stress-resistant sclerotia in the soil (Daughtrey et al. 2000). Wounded or senescent tissues are most susceptible to invasion; however, healthy tissues can be infected as well. Conidia are released during rapid changes in relative humidity and dispersed by air currents and water splash. Botrytis blight will occur most often during wet, cloudy weather. Tubers in storage can also be infected and damaged.

Management

- ***Cultural practices*** – Avoid over fertilization and reduce relative humidity by increasing spacing between plants, avoiding overhead irrigation, and, specifically for greenhouse production, by venting and heating the greenhouse late in the day.
- ***Chemical control*** – Preventive application of fungicides can provide effective control; however, they work best when used in conjunction with cultural practices. To avoid buildup of resistant fungi, tank-mix and/or alternate fungicides with different modes of action. Widespread resistance to benzimidazole fungicides (including benomyl and thiophanate-methyl) and dicarboximides (iprodione and vinclozolin) has been observed in Europe and North America (Daughtrey et al. 2000; Moorman and Lease 1992; Pappas 1982; Vali and Moorman 1992).

2.3 Redhead Leaf Spot [*Hinomyces moricola* (Hino) Narumi & Y. Harada syn. *Cristulariella moricola* (Hino) (Teleomorph: *Sclerotium cinnamomi* Swada)]

Geographic occurrence and impact. Redhead leaf spot has been reported on many woody and annual herbaceous plants (Alfieri et al. 1994).

Symptoms/signs. Symptoms can develop rapidly during cool and moist conditions which favor disease development. Initially, water-soaked, greenish gray, circular, or irregularly shaped leaf spots will develop, often with a yellow halo. Spots will eventually become zonate with alternating concentric bands of lighter and darker tissue. Leaf spots may coalesce which will give infected leaves a scorched appearance. *Hinomyces* conidia can readily be observed on both sides of the infected leaves with a hand lens. The conidia are pyramidal or cone-shaped and off-white in color. The sexual stage or teleomorph and sclerotial stages of the fungus can be observed on older infected foliage or fallen leaves and serve as the overwintering

structures (Cline et al. 1983). Sclerotia are black, crusty, irregularly shaped, and approximately 2–5 mm in diameter (Cline et al. 1983; Leahy 1995).

Biology and epidemiology. Cool, wet weather is optimal for disease development. The fungus survives in infected plant tissue and sclerotia, and spores are spread by air and splashing water.

Management

- ***Cultural practices*** – Avoid overhead irrigation or water early in the morning to reduce the duration of leaf wetness.
- ***Sanitation*** – Remove and destroy infected leaf litter which serves as a source of inoculum for new disease outbreaks.
- ***Chemical control*** – Protective sprays of the systemic fungicide thiophanate methyl or mancozeb products have been recommended (Leahy 1995; Simone et al. 1993).

2.4 Fusarium Tuber Rot [*Fusarium solani* (Mart.) Sacc. (Teleomorph: *Haematonectria haematococca* (Berk & Broome) Samuels and Rossman (1999))]

Geographic occurrence and impact. This disease has been reported in the USA (Florida) (Knauss 1975). Fusarium tuber rot, caused by *F. solani*, is one of the major limiting factors for caladium tuber production and has resulted in the elimination of several susceptible commercial cultivars. A survey of six important caladium cvs. produced by six companies conducted in 1997 detected an 80–100% incidence of the pathogen (McGovern et al. 2004). *Fusarium solani* is widely distributed in soils and water and exists as either a saprophyte or pathogen, and therefore, it is commonly encountered in the field, greenhouse, and storage.

Symptoms/signs. In general, diseased plants are wilted and unthrifty with a reduction in stand, growth, and tuber yield. *Fusarium solani* first invades the roots and basal plate of the caladium tuber, and the infection progresses upward to the bases of the petioles. Initially, diseased areas on the tubers, roots, and petioles will appear light tan, and as time progresses, the affected areas will darken and become rotted. The roots and basal plate may appear brown to black, sunken, and decayed. The appearance of brown flecks in the tuber flesh is a characteristic intermediate symptom. In the final stage of tuber rot, infected tubers become totally desiccated or mummified and have a hard chalky consistency. Diseased tubers will also develop a strong fruity odor. The progression of tuber rot symptoms is shown in Fig. 1. Random patches of infected plants occur in the field. The foliage of infected plants becomes yellow then brown and necrotic. During wet and humid conditions, tubers weakened by *F. solani* may be invaded by other fungi and soft rotting bacteria.

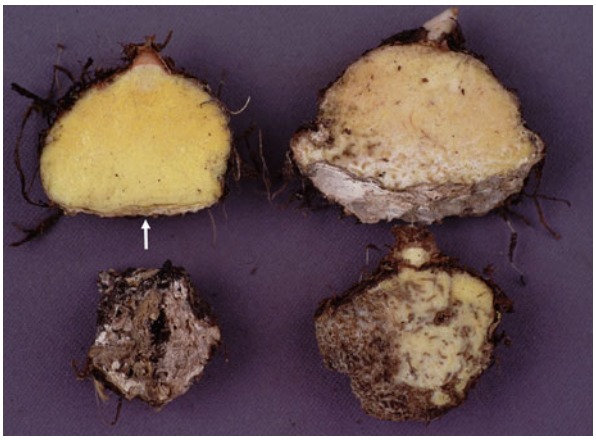


Fig. 1 The progression of symptoms of *Fusarium solani* tuber infection: light tan colored, early basal plate infection (*arrow*) (*top left*); light tan to whitish discoloration with a few brown flecks and increased basal plate infection (*top right*); darker brown discoloration with many brown flecks and some desiccation (*bottom right*); and complete dark brown desiccation (*bottom left*) (R.J. McGovern © 2017. All Rights Reserved.)

Biology and epidemiology. The fungus produces three types of spores: microconidia, macroconidia, and chlamydospores. The first two spore types may be aerially disseminated, but their role in Fusarium tuber rot is not known. The disease occurs in the field and storage. The most pertinent mode of *F. solani* survival is as mycelium and chlamydospores in seed tubers. The pathogen can also survive between crops in soil, plant debris, and on tools, equipment, and surfaces (especially wood or abraded plastics). This is possible due to the ability of the pathogen to persist for years in the absence of a living host as resistive chlamydospores. Wounds created during harvesting practices may serve as infection courts for the pathogen.

The optimum temperature range for growth of *F. solani* in culture is 25–30.5 °C (77–87 °F) (Goktepe et al. 2007; Gupta et al. 2010; Jensen and Reynolds 1971), and soil temperatures of 26–28 °C (79–82 °F) favor maximum pathogen development (Booth and Waterson 1964). Interestingly, Deng et al. (2007) found that temperatures of 13–18 °C (55–64 °F) enhanced rot in experimentally inoculated tubers. As a tropical crop, caladium sustains damage if stored below 15 °C (59 °F). Such low temperatures were found to inhibit suberinization in caladium (Marousky and Raulston 1973), a normal defense response in plants to wounds and pathogen invasion, thus making cold-treated tubers more susceptible to infection. *Fusarium solani* causing tuber rot in caladium is primarily disseminated in infected tubers and infested soil and possibly through contaminated tools and equipment.

Interaction of *F. solani* with other soil-borne pathogens like *Pythium*, *Rhizoctonia*, and *Meloidogyne* spp. in a caladium tuber rotting complex is a possibility. *Pythium myriotylum, F. solani*, and root-knot nematodes *Meloidogyne* spp. (*M. javanica* and *M. indica)* were associated with rhizome rot of ginger in India

(Mathur et al. 2002). In addition, Garcia and Mitchell (1975) identified a synergistic interaction between *F. solani*, *P. myriotylum*, and *Meloidogyne arenaria* in pod rot of peanut in the USA.

Management

- ***Cultural practices*** – Cull diseased tubers, select the largest and most vigorous tubers for propagation, and use optimal field production practices. The benefit of periodic tissue culture of caladium cvs. to produce more vigorous, pathogen-free propagative tubers has been demonstrated in the field (Harbaugh et al. 1988).
- ***Chemical control*** – Preplant fungicide application to seed tubers is necessary because of the uniformly high incidence of *F. solani*. Knauss (1975) indicated that treatment of tubers with the fungicides chlorothalonil, mancozeb, or thiophanate methyl provided effective disease control. In a series of laboratory and field experiments McGovern et al. (2001a; 2002; 2004; McGovern and Singh 2003) evaluated various combinations of fungicides applied to tubers in conjunction with hot water immersion [50 °C (122 °F)] for 30 min. In the laboratory, chlorothalonil alone and in combination with thiophanate methyl and azoxystrobin, fludioxonil, or triflumizole alone and in combination with chlorothalonil significantly reduced the incidence and severity of tuber infection by *F. solani*, while thiophanate methyl or hot water alone were generally ineffective. Chlorothalonil + thiophanate methyl increased plant emergence and the number of jumbo tubers in comparison to thiophanate methyl alone in sub-irrigated caladiums grown on muck soil. This same fungicide combination increased plant emergence and total number of marketable tubers compared with hot water treatment or thiophanate methyl alone in caladiums grown in sand on raised beds with drip irrigation.

In vitro assays of *F. solani* isolates from caladium indicated that about 16% were moderately to highly resistant to the benzimidazole fungicide thiophanate methyl, most likely due to its continuous use by growers (McGovern et al. 2004). To avoid buildup of resistant fungi, tank-mix and/or alternate fungicides with different modes of action.

In the past, caladium growers in Florida relied on preplant application of various combinations of methyl bromide and chloropicrin (MBC) to control soil-borne pests including fungi, nematodes, and weeds. However, because of the progressive phase out of methyl bromide in developed countries mandated by international environmental agreements, much research has been conducted to identify effective chemical and nonchemical alternatives to MBC. Refer to ▶ Chap. 8, "Soil/Media Disinfestation for Management of Florists' Crops Diseases."

- ***Resistance*** – Five caladium cultivars are known to be resistant to *F. solani*: Candidum, Rosebud, White Christmas, Florida Sweetheart, and Aaron. Cultivars with moderate resistance are White Wing and Red Flash. Cultivars found to be susceptible include: Candidum Jr., White Queen, Red Frill, Florida Cardinal, Miss Muffet, and Postman Joyner. Cultivars found to have the highest susceptibility are Fannie Munson, Gingerland, Frieda Hemple, and Carolyn Whorton with the last being the most susceptible (Deng et al. 2007; Goktepe et al. 2007).

2.5 Pythium Root Rot (*Pythium* spp., *Pythium myriotylum* Drechsler)

Geographic occurrence and impact. Several species of the soil-borne pathogen *Pythium* are commonly encountered in caladium production. *Pythium myriotylum* is considered to be the most virulent caladium pathogen of the group (Ridings and Hartman 1976a). Caladium tubers infected with *P. myriotylum* are 3 to 5 weeks slower to sprout. Once infected tubers sprout, a 70% reduction in plant growth and up to a 40% tuber loss can be observed (Chase 1988; Ridings and Hartman 1976a).

Symptoms/signs. The disease occurs in field and storage, and infected plants appear as random patches with irregular borders. Above ground symptoms include leaf discoloration, stunting, epinasty, wilting, and eventual collapse (Fig. 2). Below ground, roots may have discrete lesions which progressively appear water soaked, brown, and shriveled and the lateral roots may lack white growing tips or be totally rotted (Fig. 3). Often, the outer tissue of the root (cortex) can be pulled away from the



Fig. 2 Aboveground symptoms of caladium infected with *Pythium*: leaf discoloration, stunting, epinasty, and wilting (*left*) and healthy plants (*right*) (R.J. McGovern © 2017. All Rights Reserved.)



Fig. 3 Below ground symptoms of caladium infected with *Pythium;* healthy root system (*left*) and rotted and discolored root system with decayed lateral roots (*right*) (R.J. McGovern © 2017. All Rights Reserved.)

inner core (stele), a symptom known as "root sloughing." Usually by the time the above ground symptoms are evident, large portions of the root system have already decayed. Under moist conditions, whitish-grey fungal mycelia can be observed on or around the diseased roots.

Biology and epidemiology. *Pythium*, a fungus-like "water mold" or oomycete, may survive between crops in infected tubers, alternate hosts which include many horticultural crops and weeds, soil, plant debris, and seed tubers. A survey of six major caladium cvs. conducted in 1997 detected a 10% incidence of *Pythium* spp. in tubers (McGovern unpublished data). The severity of the disease is a function of host and environmental factors. Abundant soil moisture is conducive to Pythium root rot development and a soil/media moisture holding capacity greater than 70% will favor pathogen growth. Inocula may be disseminated by irrigation water, shore flies and fungus gnats, infected seed tubers and plant debris, unsanitary storage facilities, and contaminated potting media, soil, and tools (Agrios 2005; Garcia and Mitchell 1975). A soil temperature of 32 °C (90 °F) and high soil moisture favor Pythium root rot development in caladium; the disease is most severe in Florida during the hot and wet weather of late summer. *Pythium* infection can create favorable conditions for other soil-borne pathogens, such as *Fusarium solani*, to infect roots (Daughtrey et al. 1995; Garcia and Mitchell 1975; Mathur et al. 2002).

Management

- ***Cultural practices*** – Cull diseased tubers and choose the largest and most vigorous tubers for propagation. Select well-drained planting sites or improve site drainage. Carefully monitor irrigation practices to avoid saturated soil. Avoid introducing contaminated soils to fields on tools and equipment. In the greenhouse, avoid water-logged soil, control shore flies and fungus gnats, and use pathogen-free growing media.
- ***Chemical control*** – Ridings and Hartman (1976b) recommended a soil drench with the fungicide etridiazole. McGovern et al. (2001b) reported that a soil drench of the fungicides azoxystrobin or mefenoxam significantly reduced the incidence and severity of root rot in caladium caused by *P. myriotylum* and increased the fresh weight of shoots and roots and that a soil drench of the biocontrol *Trichoderma harzianum* and spray application of azoxystrobin were ineffective. In the field, use a preplant fumigant or other soil disinfestation practice.
- ***Physical control*** – Hot water treatment of seed tubers at 50 °C for 30 min should help to reduce *Pythium* infection in propagative stock (Ridings and Hartman 1976b). Solarization of a 25 cm-high mound of a nursery potting soil with a double layer of clear polyethylene mulch eliminated *Pythium myriotylum* at 10 and 25 cm within 7 d in Australia (Duff and Barnaar 1992).
- ***Resistance*** – The majority of caladium cultivars currently in production are moderately or highly susceptible to Pythium root rot. Cultivars with moderate levels of resistance are: Candidum, Candidum Jr., Frieda Hemple, and White Christmas. Cultivars with moderate susceptibility are: Aaron, Carolyn Whorton, Florida Elise, Florida Fantasy, Florida Red Ruffles, Florida Sweetheart, Pink

Beauty, and Miss Muffet. Cultivars found to have the highest susceptibility include: Fannie Munson, Gingerland, Gray Ghost, Red Flash, Texas Beauty, White Queen, and Red Frill (Deng et al. 2004, 2005).

2.6 Rhizoctonia Root Rot and Blight [*Rhizoctonia solani* Kühn (Teleomorph: *Thanatephorus cucumeris* (A.B. Frank) Donk)]

Geographic occurrence and impact. Rhizoctonia root rot and blight of caladium has been reported in the USA (Florida) (Alfieri et al. 1994).

Symptoms/signs. Given a conducive environment, full disease development can occur in less than a week. Besides discoloration and rot of the root system, brown, irregularly shaped spots may form anywhere on the foliage but are most common at the base of caladium petioles, where moisture levels and humidity are high. The disease spreads rapidly, and the entire plant can become covered with the brown web-like mycelium of the pathogen, blighting leaves under moist conditions (Chase 1991).

Biology and epidemiology. *Rhizoctonia solani* has a very wide host range including many horticultural crops and weed species and exhibits some host specialization (Mordue 1974a). The disease is most severe during warm and humid or wet weather. *R. solani* survives in the soil, in growing media, and in infected plant debris as hyphae and stress-resistant sclerotia. Spores are not produced; however, *R. solani* can be readily disseminated through infected seed tubers, water splashing, and by airborne particulate matter (Agrios 2005). A survey of six major caladium cvs. conducted in 1997 detected *R. solani* at an incidence of 8% (McGovern unpublished data).

Management

- ***Cultural practices*** – Cull diseased tubers and select the largest and most vigorous tubers for propagation.
- ***Chemical control*** – Although there is no specific data on its efficacy, the same fungicide-hot water combinations used for control of *F. solani* should help to reduce *R. solani* infection of caladium tubers. In the field, use a preplant fumigant or other soil disinfestation practice.

2.7 Sclerotium Blight or Southern Blight [*Sclerotium rolfsii* Sac. (Teleomorph: *Athelia rolfsii* (Curzi) C.C. Tu & Kimbr.)]

Geographic occurrence and impact. Sclerotium blight is a worldwide problem on many horticultural crops primarily in warm climates (Punja 1985; Singh and Dwivedi 1991).

Symptoms/signs. Caladium plants affected by *S. rolfsii* are usually attacked at the soil line or below (roots and tubers). Often this disease is not recognized until plants are wilted in the field. Initially, symptoms are manifested as discrete light brown lesions on petioles at the soil surface. The disease can rapidly progress causing leaf yellowing, wilting, plant collapse, and eventual death (Fig. 4). Under warm and humid conditions, very robust white mycelial growth can be observed approximately 3–4 days after infection on the soil, plant parts such as petioles and senescing leaves at the soil line, and tubers (Figs. 4 and 5). Very characteristic survival structures (sclerotia) form on the mycelia, infected plant parts, and in the soil approximately 7 days after infection. The sclerotia are comparable in size to mustard seeds (2–3 mm in diameter), are initially white, and mature to a light tan to brown color. Early wilt symptoms of Sclerotium blight can be confused with those caused by *Fusarium*, *Pythium*, *Rhizoctonia* spp., and other soil-borne pathogens.

Biology and epidemiology. Sclerotium blight is a common problem in warm and wet tropical and subtropical climates and can develop rapidly (within days). Often outbreaks are associated with the rainy season. *Sclerotium rolfsii* survives as mycelium on dead organic material or sclerotia in the soil or other growing media. However, sclerotia are the principle overwintering structures in the field and serve as the initial inoculum for the following season. Temperatures of 25 °C to 35 °C (81 °C to 95 °F) favor disease development, and near 100% humidity is required for sclerotial germination (Mordue 1974b). Mycelial growth occurs over a wide range of temperatures, 8 °C to 40 °C (46 to 104 °F) (Punja 1985). The sclerotia are disseminated from field to field through infected seed tubers, water, wind, and movement of soil on tools and equipment. Dense plantings that increase high canopy humidity, high soil moisture, and frequent irrigation promote disease spread (Chase 1988). Sclerotia germinate in the upper regions of the soil and growth is promoted by



Fig. 4 Aboveground symptoms/signs of caladium infected with *Sclerotium rolfsii*. Initially, symptoms include leaf yellowing and wilting and plant collapse. A robust, fast growing white mycelium and small, round immature sclerotia can be observed on diseased petioles and leaves at the soil line (R.J. McGovern © 2017. All Rights Reserved.)



Fig. 5 *Sclerotium rolfsii* root rot of caladium. White mycelial growth can be observed on necrotic roots and tuber (R.J. McGovern © 2017. All Rights Reserved.)

sandy soils. Germination progressively decreases with time and depth of burial in soil. Mycelial growth will occur in a broad pH range but is highest in the acidic range (Agrios 2005).

Management

- ***Cultural practices*** – Hot water treatment of seed tubers at 50 °C for 30 min may be effective in eliminating *S. rolfsii* from propagative stock. To reduce inoculum in the soil, deep plow using a moldboard plow to bury sclerotia and organic material below the root zone and promote their disintegration. Deep plowing alone may not provide adequate control and therefore should be combined with other cultural and chemical control practices (Subbarao 1996). Rotation with nonsusceptible crops is known to lower the initial inoculum and thereby decrease disease incidence; however, this may not be practical due to the wide host range of *S. rolfsii*.
- ***Physical control*** – Soil solarization conducted for 40–55 days using clear plastic mulch applied to raised beds significantly reduced the incidence of Sclerotium blight in tomato in the USA (Florida) (Chellemi et al. 1997). Solarization of a 25 cm-high mound of a nursery potting soil with a double layer of clear polyethylene mulch eliminated *S. rolfsii* at 10 cm 25 cm within 7 and 10 days, respectively, in Australia (Duff and Barnaar 1992).
- ***Chemical control*** – Soil fumigation and pre- and post-plant fungicide drenches may be effective in controlling this disease. Chlorothalonil, iprodione, and thiophanate methyl may be effective in inhibiting sclerotial germination and mycelial growth (Mullen 2001). In a growth chamber experiment conducted

with potted caladiums inoculated with *S. rolfsii*, McGovern and Seijo (2003) found that an ammonium bicarbonate-based fungicide (ABF) and the fungicides fluazinam and fludioxonil significantly decreased the time of shoot emergence compared with the nontreated control. Fluazinam and fludioxonil significantly increased plant survival. All fungicides significantly increased foliage fresh weight, with ABF being of intermediate effectiveness. Fluazinam and fludioxonil significantly increased tuber weight, and fluazinam and ABF significantly increased root weight. The biocontrol *Streptomyces lydicus* was ineffective.

The same fungicide-hot water combinations used for control of *F. solani* should help to reduce *S. rolfsii* infection of caladium tubers.

2.8 Grassy Tuber [Various]

Geographic occurrence and impact. Grassy tuber of caladium was first identifiedin 1990 in Florida (USA) affecting the caladium variety "Postman Joyner" (Polston et al. 1991). While seven cultivars were initially affected by the disease (Postman Joyner, Frieda Hemple, Buck, Carolyn Whorton, Rosebud, Caloosahatchee, and Pink Beauty) the disease can now be found in all standard cultivars. Grassy tuber results in reductions in quantity and quality of tuber yield, as shown in "Rosebud" (Deng and Harbaugh 2013). A further reduction in the diversity of commercially produced caladium cultivars and a decline in the caladium industry have been attributed to the disease (Pinnell 2014).

Symptoms/signs. Symptoms of grassy tuber include stunting, increase in the number of meristems, reduction in leaf size, reduction of anthocyanin pigmentation, alterations in pigmentation, lack of flower production, and formation of small, spindly tubers (Fig. 6). Caladiums with symptoms of grassy tuber disease are less vigorous.

Biology and epidemiology. Polston et al. (1991) first described the disease with an unknown etiology. Recent studies have shown that grassy tuber is a set of symptoms



Fig. 6 Symptoms of grassy tuber: severe stunting and decreased pink pigmentation (*arrows*) (R.J. McGovern © 2017. All Rights Reserved.)

that are the result of damage to the tubers. Tuber may be damaged by rots caused by fungi (e.g., *Fusarium* spp.) or damage from high temperatures or other physiological stresses (Polston unpublished). Tuber rots put stress on the tuber by killing tuber tissue as well as the meristems in the diseased regions. This results in (1) reduction in the storage capacity of the plant and (2) can cause de-eyeing that produces the proliferation. Physiological damages such as from high temperatures can result in similar symptoms (Polston unpublished).

Management

- ***Chemical practices*** – Select fungicides that are effective against tuber rotting fungi, such as *Fusarium* and *Rhizoctonia* spp., and use (1) as a dip to treat tubers before planting, (2) at planting in the row, (3) apply again if needed within the first 2 months. Be sure to rotate classes to prevent the selection of resistance in the fungi.
- ***Cultural practices*** – As for many asexually propagated crops, cull diseased tubers (rotted, small, and spindly) at harvest and again before planting; use those tubers that are rot free and vigorous for propagation. Scout regularly and remove and destroy symptomatic plants. Use cultivation practices that result in canopy closure before the onset of high light intensity and high temperatures of summer. Plant tuber pieces deep enough so that high soil temperatures of summer do not affect the developing tubers. If the crop is damaged by extreme stresses (flooding, high temperatures, etc.) be prepared to rogue high numbers of damaged plants for the next several seasons or consider replacing with disease-free tissue cultured plants.

When diligently applied by growers, these tactics have reduced the incidence of grassy tuber in subsequent seasons. If the incidence of grassy tuber is very high, then it may be more cost effective for growers to discard most or all of their stock and replace it with tissue cultured plants that are free of fungi (available as of 2016). Tissue cultured plants should be treated as above (with timely applications of fungicides, and rouging for best plants) to insure a high quality crop in future years.

Management

Additional Fungal Diseases

The following fungal diseases of caladium have also been reported in the USA (Alfieri et al. 1994):

Alternaria sp., leaf spot

Bipolaris sp., leaf necrosis

Botryosporium sp., leaf and petiole necrosis

Choanephora cucurbitarum (Berk. & Ravenel) Thaxt., leaf necrosis

Colletotrichum spp., anthracnose, leaf spot

Cylindrocladiella peruviana (Batista, Bezerra & Herrera) Boesewinkel, root and corm rot

F. oxysporum Schlechtend.:Fr., corm rot

Gloeosporium sp., leaf spot

Helminthosporium sp., leaf spot
Meliola sp., black mildew
Myrothecium sp., crown rot
Penicillium sp., corm rot
Phyllosticta sp., leaf spot
Rhizopus stolonifer (Ehrenb.:Fr.) Vuill. (=*R. nigricans* Ehrenb.), corm rot
Sclerotinia sclerotiorum (Lib.) de Bary, cottony rot

3 Bacterial Diseases

3.1 Bacterial Leaf Spot, Blight [*Xanthomonas axonopodis* pv. *dieffenbachiae* (McCulloch and Pirone) Vauterine, Hoste, Kersters, and Swings]

Geographic occurrence and impact. This pathogen has been reported from many ornamental and edible members of the family *Araceae. Xanthomonas axonopodis* pv. *dieffenbachiae* (*Xad*) can be found in the Americas, Oceania, South Africa, and Asia (Jouen et al. 2007; Robene-Soustrade et al. 2006).

Symptoms/signs. Symptoms typically occur on leaves but may also appear on petioles. The bacterium invades through stomates and hydathodes at the leaf margin and through wounds. Initial disease symptoms are small water-soaked lesions. These lesions rapidly enlarge and turn dark brown to black under warm, humid conditions. The lesions are impeded by major leaf veins giving them an angular appearance, and they are often surrounded by a yellow halo (Fig. 7). In humid or wet weather, total blighting of the foliage occurs, and bacterial ooze may be observed on infected tissue (Chase et al. 1992).

Biology and epidemiology. Dissemination of the bacteria occurs by overhead irrigation, rain, runoff, plant to plant contact, insects, handling of plants, and tools. Extended periods of leaf wetness promote disease development. The bacterium persists in debris from infected plants in the soil. Once the debris deteriorates, the bacterium will no longer survive.

Management

- ***Cultural practices*** – This pathogen can be difficult to control once it is present in a production area. Sanitation and exclusion of *Xad* is important. As feasible in greenhouse production, remove caladium leaves/plants with leaf spot symptoms and volunteer caladiums and other possible *Xad* reservoir plants in the Aroid family from around the production site.
- ***Chemical control*** – Preventive use of copper bactericides mixed with dithiocarbamate fungicides may be effective.

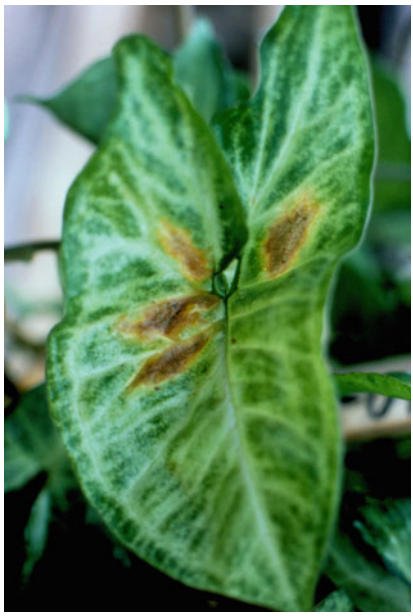


Fig. 7 Symptom of bacterial blight (R.J. McGovern © 2017. All Rights Reserved.)

- ***Resistance*** – Cultivars White Queen, Florida Red Ruffles, Florida Sweetheart, Candidum Jr., and Mrs. Arno Nehrling have been identified as resistant to bacterial blight in greenhouse or field evaluations (Seijo et al. 2010).

3.2 Bacterial Soft Rot [*Pectobacterium carotovorum* (syn. *Erwinia carotovora*); *Dickeya dadantii* (Syn. *Erwinia chrysanthemi*)]

Geographic occurrence and impact. This disease has been reported in the USA (Florida) (Alfieri et al. 1994) and leads to significant losses when tubers are harvested during cool, wet weather.

Symptoms/signs. The initial symptoms of soft rot are small, water-soaked, tan discolored areas within the tuber. As the disease progresses the entire interior of the tuber becomes macerated, turning into a cream to gray watery mass (Fig. 8). Severely rotted tubers develop a foul odor caused by the invasion of secondary bacteria.

Biology and epidemiology. These bacteria have a broad host range of cultivated and wild plants. Dissemination is by rain, irrigation, soil particles, tools, and the hands and clothing of workers. Carryover of soft rot bacteria in tubers from season to season is also possible. The disease was detected at a very low incidence (<0.01%) in a survey of six major caladium cvs. in 1997 and at an average incidence of 11% in two field trials in caladium production sites during the 1998–1999 season (McGovern unpublished data). The incidence of soft rot varies with the condition



Fig. 8 Symptoms of bacterial soft rot in tuber (R.J. McGovern © 2017. All Rights Reserved.)

of the tubers and time of harvest. The soft rot bacteria enter tubers through wounds and natural openings. Infection by other pathogens, cold injury, and wet soils predispose tubers to the disease.

Management

- ***Cultural practices*** – Choose the most vigorous tubers for propagation. Avoid injuring tubers during the harvesting process as much as possible. Harvest tubers before the onset of cold weather and avoid harvesting during wet periods. Improve soil drainage.
- Increasing the calcium nutrition of potato tubers reduced their susceptibility to soft rot caused by *Pectobacterium carotovorum* (McGuire and Kelman 1984). Form of nitrogen applied to chrysanthemum affected its susceptibility to soft rot caused by *Dickeya padantii*; a high rate (400 ppm N) of $(NH_4)_2SO_4$ and a complete fertilizer containing NH_4NO_3, KNO_3, and $NaNO_3$ increased disease severity, while an equivalent rate of N supplied by NH_4NO_3 or $Ca(NO_3)_2$ decreased severity (McGovern et al. 1985).
- ***Chemical*** – Application of labeled products containing the antibiotic streptomycin may help to manage bacterial soft rot. Control of other soilborne tuber diseases reduces the potential infection sites for soft rot bacteria.

4 Viral Diseases

4.1 *Caladium virus X* (CalVX)

Geographic occurrence and impact. *Caladium virus* X (CalVX), although not fully sequenced, is most likely a virus in the genus *Potexvirus*, family Flexiviridae. CalVX was described in Brazil by Rivas et al. (2005) from caladium. Synergistic coinfections with *Dasheen mosaic virus* (DsMV) are known to occur.

Symptoms/signs. Symptoms of CalVX in caladium are chlorotic spots, green rings, and mosaic. When co-infected with DsMV, symptoms of chlorotic and necrotic spots and rings can be observed.

Biology and epidemiology. CalVX can be mechanically transmitted and is able to infect and cause symptoms in an array of species in at least six families including several species within the Araceae.

Management

- ***Cultural*** – Use sanitation and avoidance. Scout regularly and remove diseased plants.

4.2 *Dasheen mosaic virus* (DsMV)

Geographic occurrence and impact. Dasheen mosaic of caladium is caused by *Dasheen mosaic virus* (DsMV, genus: *Potyvirus*, family Potyviridae). DsMV has a worldwide distribution, being most prevalent in tropical and subtropical regions (the Caribbean, Egypt, USA, Europe, India, Japan, and Oceania) (Alconcero and Zettler 1971; Ram et al. 2003; Zettler and Hartman 1995). DsMV infects a wide range of aroids, and hosts can be found in the following genera: *Aglonema, Arum spp., Caladium, Colocasia, Cryptocoryne, Dieffenbachia, Dractontium, Philodendron. Xanthosomas, Zantedeschia*, and many others (Hartman 1974, Leseman and Winter 2002, Pham et al. 2002). Infection with DsMV can result in yield losses of 40 to 61% (Hartman and Zettler 1974). Fresh tuber weights may be reduced by 40% and leaf area by 53% (Hartman and Zettler 1974). Vegetative propagation has played a major role in turning DsMV into the most prevalent virus in caladium in Florida and the USA (Chase 1988; Hartman and Zettler 1974).

Symptoms/signs. A foliar mosaic can appear as localized feathering of white tissue along leaf veins. However, symptom expression can be intermittent or seasonal and as a result, symptomatic and asymptomatic leaves can occur on the same plant (Elliott et al. 1997). DsMV feathering is often observed in white caladium cultivars such as White Christmas, Candidum, and Candidum Junior and less often in colored cultivars such as Frieda Hemple and Carolyn Whorton (Zettler and Hartman 1986). Leaf distortion and other changes in normal coloration patterns can also occur.

Biology and epidemiology. DsMV can be spread through vegetative propagation of diseased tubers on tools contaminated with infected plant sap and can be vectored in a non-persistent manner by several species of aphids (Hemiptera: Aphididae) (Zettler et al. 1970). Virus particles are picked up on the aphid's stylet during probing. Known aphid vectors of DsMV include: the green peach aphid (*Myzus persicae*) and the cowpea aphid (*Aphis craccivora*) but not the banana aphid (*Pentalonia nigronervosa* Coquerel) (Morales and Zettler 1977; Zettler et al. 1970).

Management

- ***Cultural practices*** – Sanitation and avoidance. Scout regularly and remove diseased plants. Eliminate infected araceous hosts, caladium volunteers, and weed species that may serve as virus and/or aphid reservoirs. The virus may be eliminated by tissue culture (Hartman 1974).
- ***Chemical*** – In general, chemical management is often ineffective for reducing the spread of nonpersistently transmitted viruses. Foliar application of mineral oil has been shown to greatly reduce transmission of another potyvirus, *Tobacco etch virus*, by *M. persicae* by interfering with the binding of the virus particles to the aphid stylet (Wang and Pirone 1996).

4.3 *Konjac mosaic virus* (KoMV)

Geographic occurrence and impact. *Konjac mosaic virus* (KoMV, genus: *Potyvirus*, family Potyviridae) is a more recently described virus (Nishiguchi et al. 2006; Shimoyama et al. 1992). Two other viruses, *Zantedeschia mosaic virus* (ZaMV) and *Japanese hornwort mosaic virus* (JHMV) are currently recognized as isolates of KoMV (Nishiguchi et al. 2006). KoMV has been reported in caladium and a number of other araceous species and has a worldwide distribution having been reported from Germany, Japan, Korea, India, Taiwan, the Netherlands, and the USA, (Alcala-Briseno and Polston unpublished; Kwon et al. 2002; Lesemann and Winter 2002; Manikonda et al. 2011; Nishiguchi et al. 2006; Pham et al. 2002; Shimoyama et al. 1992). However, the reports of KoMV in caladium have only come from India and the USA (Alcala-Briseno and Polston unpublished; Manikonda et al. 2011).

Symptoms/signs. KoMV has been reported to cause symptoms of chlorotic spots and blotches and leaf deformation in caladium in India (Manikonda et al. 2011). These symptoms were distinct from those of DsMV. KoMV often occurs in mixed infection with DsMV so symptoms may not be a reliable means of recognition (Alcala-Briseno and Polston unpublished).

Biology and epidemiology. KoMV can be mechanically transmitted on tools contaminated with infected plant sap and can be spread through vegetative propagation of diseased tubers. Seed or pollen transmission has not been reported. To date only one vector of KoMV has been reported, the cotton-melon aphid, *Aphis gosspyi* (Shimoyama et al. 1992). The virus presumably can be vectored nonpersistently by aphids (Hemiptera: *Aphididae*), in a similar manner to that of DSMV and other potyviruses. KoMV has been found infecting other araceous hosts: a number of *Amorphophallus* species*, Arisaema serratum, Arum italicum, Colocasia esculenta, Dieffenbachia* spp, *Dracontium gigas*, *Philodendron oxycardium P. selloum, P. verrucosum, Zamioculcas zamiifolia Zantedeschia aethiopica, Zantedeschia* sp. hybrids (Alexandre et al. 2013; Lesemann and Winter 2002; Manikonda et al. 2011; Nishiguchi et al. 2006; Shimoyama et al. 1992).

Management

- ***Cultural practices*** – Sanitation and avoidance. Scout regularly and remove diseased plants. Eliminate infected araceous hosts, caladium volunteers, and weed species that may serve as virus and/or aphid reservoirs. The virus may be eliminated by tissue culture. For additional management information, refer to the introductory (▶ Chap. 4, "Insect Management for Disease Control in Florists' Crops").
- ***Chemical*** – Foliar application of mineral oil has been shown to greatly reduce transmission of another potyvirus, *Tobacco etch virus*, by *M. persicae* to tobacco by reducing the retention of virus particles on the aphid stylet (Wang and Pirone 1996).

5 Nematode Diseases

5.1 Root-Knot (*Meloidogyne* spp.)

Geographic occurrence and impact. Root-knot of caladium is caused by obligate plant-parasitic nematodes within the genus *Meloidogyne* (family *Meloidogynidae*). The genus name *Meloidogyne* is derived from two Greek words meaning "apple-shaped" and "female." Three root-knot nematode (RKN) species are known to parasitize caladium: *M. arenaria*, *M. floridensis*, and *M. incognita* (Kokalis-Burelle et al. 2010; McSorely et al. 1999; 2004; Rhoades 1964). Depending on the severity, RKN infestation can result in reduced tuber yields and plant death.

Symptoms/signs. Most often, the damage caused by RKN is signaled by above ground symptoms including stunting, yellowing, wilting, and leaf dieback. These symptoms are similar to those caused by a number of soil-borne pathogens; therefore, it is best to check the caladium tubers and roots for root-knot symptoms and the presence of the pest before making a diagnosis. Below ground symptoms include galled, stunted roots with few lateral roots and root hairs, and a characteristic swelling of the shoot base (Fig. 9). Galling is the product of RKN feeding and can range from root swelling to large knots. RKN egg masses can be seen with the naked eye when stained with red food color (Fig. 10) (Thies et al. 2002). Fleshy white swollen female nematodes can be observed with a hand lens when the galls are cut. Sometimes tubers can be highly infested with RKN but remain symptomless or exhibit nondescript corky lesions (Kokalis-Burelle et al. 2010).

Biology and epidemiology. The RKN lifecycle begins as eggs in root tissues or the soil and pass through four juvenile stages (J1–J4) before reaching maturity. The infective stage is the J2 stage which invades roots, migrates intracellularly to developing vascular cells, and injects secretions that stimulate the formation of giant cells from which they feed, enlarge, and undergo their final developmental stages. The characteristic galling or "root knots" are caused by the division and swelling of cells around the RKN feeder cells. The female RKN will enlarge into a pear-shape and lay numerous eggs in a gelatinous matrix outside of the posterior end of its body. The egg mass may be within the root or wholly or partially exposed on

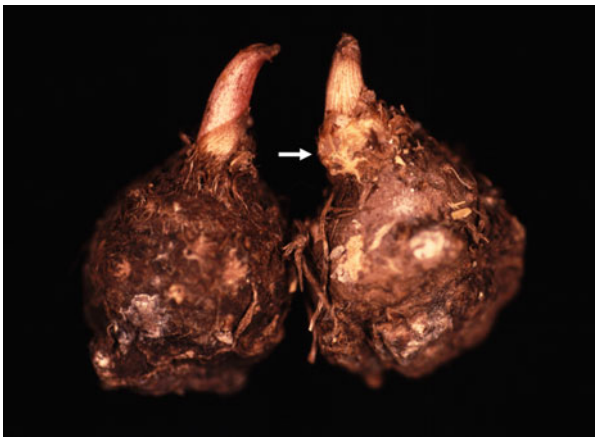


Fig. 9 Root-knot symptoms on caladium tuber caused by *Meloidogyne* spp.: healthy tuber (*left*), infected tuber with swollen shoot base (*arrow*) (*right*) (R.J. McGovern © 2017. All Rights Reserved.)

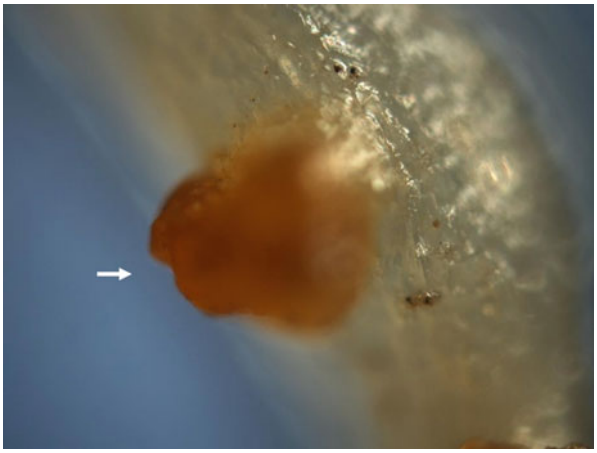


Fig. 10 Root-knot nematode egg mass stained with red food dye (Janet Brito, Florida Department of Agriculture and Consumer Services, Division of Plant Industry, Gainesville, Florida © 2017. All Rights Reserved.)

the root surface. Several RKN generations may be completed over the course of a growing season.

The RKN can be disseminated in contaminated soil, roots, and seed tubers. A survey conducted in 1997 detected RKN at an incidence of about 40% in tubers of six major caladium cvs. (McSorley and McGovern unpublished data). RKN may also be spread by contaminated tools, machinery, and irrigation water. As the RKN is highly polyphagous, it can survive between caladium crops on alternate hosts that include multiple floriculture crops and weed species. Quiescent RKN eggs can survive in the absence of a host and during periods of unfavorable temperatures for growth.

The degree of root-knot nematode infection of caladium depends on the nematode population density, temperature, the presence of free water, and caladium cultivar. The required time for the RKN to complete a life cycle is approximately 16 to 31 day with a reported temperature range of 15 °C to 30 °C (59 °C to 86 °F) (Taylor and Sasser 1978). The optimal temperatures for development fall within 21 °C (69.8 °F)

to 30 °C (86 °F) (Dávila-Negrón and Dickson 2013). Free water is necessary for nematodes to move from host to host. Nematodes are commonly a problem in well-structured soil with large pore spaces or coarse (sandy) soils. For information on cultivar susceptibility, refer to the Management section below.

Management

- ***Cultural practices*** – Cull diseased tubers and select the largest and most vigorous for propagation. If possible, adjust the planting date to when the infective juveniles are not active in the soil. This tactic may require multiple soil analyses by a nematology diagnostic clinic. If feasible, the use of fallow periods, rotations with resistant or nonhost plants, antagonistic plants, and trap crops can reduce RKN populations in the field. When selecting resistant or nonhost plants, it is important to know what species of *Meloidogyne* are infesting your caladiums; submit a sample to a nematode diagnostic clinic for identification. Rotation with nonhost crops for 2–3 years can provide sufficient control of RKN. Antagonistic crops (e.g., *Tagetes* spp., *Crotalaria juncea*, and *Crotalaria spectabilis*) may be used to control RKN. Trap cropping to control RKN involves planting host plants in an infested field, the RKN will infect the trap host, and the trap host is killed before the infecting RKN can lay eggs.
- ***Chemical control*** – Chemical management can protect crops from infection by lowering the nematode population density to below an economic threshold. Soil fumigants particularly MBC formulations in the past, drip chemigation with dimethyl disulfide (DMDS) (Kokalis-Burelle et al. 2010), preplant nonfumigants, as well as taking advantage of the soil's natural buffering capacity (Gilreath et al. 1999; Overman and Harbaugh 1983) can provide effective management. Refer to introductory (▶ Chap. 8, "Soil/Media Disinfestation for Management of Florists' Crops Diseases").
- ***Physical control*** – Hot water treatment of tubers at 50 °C for 30 min before planting has been shown to eliminate RKN infestation (Kokalis-Burelle et al. 2010; Brcka et al. 2000; Rhoades 1961). Solarization may be as effective as some fumigant nematicides for nematode management and may occasionally be equivalent to MBC in nematode suppression, especially in hot, arid areas. Although reduction of RKN has been demonstrated in humid subtropical and tropical climates, solarization has generally not been as effective against *Meloidogyne* spp. in these environments as fumigants containing methyl bromide (McGovern and McSorley 1997). Solarization reduced the densities of RKN but not the severity of galling in tomato in two experiments conducted in the USA (Florida) (Sotomayor et al. 1999). However, continuous flooding of the soil significantly reduced both RKN densities and galling severity in these same experiments.
- ***Resistance*** – Cultivars with varying levels of susceptibility to *M. incognita* have been identified. The cultivars White Christmas and Pink Glow are considered the most resistant. The most susceptible cultivars are Crimson Wave and Autumn Beauty. Cultivars with intermediate to low resistance are Fire Nymph, Avalon Rose, Dawn, and Fire Chief (McSorley et al. 2004).

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Diseases of Cyclamen

39

Wade H. Elmer and Margery L. Daughtrey

Abstract

Cyclamen (*Cyclamen persicum* Mill.) is a major potted flower in the horticultural trade. As a result of more intensive production over the past 20 years, many new disease problems have emerged as breeders have exchanged germplasm and seed. Fusarium wilt and tospovirus diseases have become particularly challenging to manage. The following chapter offers an array of management strategies for more than 15 disease conditions based on current research.

Keywords

Botrytis cinerea • Fusarium wilt • Root rots • Leaf spots • *Tospovirus* • Integrated disease management

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R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_42

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1 Introduction

Cyclamen is a genus that contains over 23 species in the family Primulaceae. Its name was coined from the Greek word, "Kyklos," meaning circle, in reference to the corm. The ancestral region for the species is unknown, but many species are native to parts of Europe, Asia, and North Africa where they flourish in diverse habitats ranging from woodlands to alpine meadows. The species most commonly cultivated by florists is *C. persicum*, which originated in the Middle East. Although the plant was brought to Europe over 300 years ago, popularity of cyclamen did not increase until the 1900s when selected variants exhibiting larger flowers appeared. Today, F1 hybrids are marketed, providing consumers with over 200 distinct cultivars. Cultivation of cyclamen is commonly done from seed germinated in the dark under tightly controlled conditions, since temperature fluctuations are very detrimental to uniform germination. Seedling plugs are usually marketed to growers who produce a finished flowering product. Many times, the initial flower stalks (pedicels) are removed to allow a uniform stand to develop among all plants. A listing of the specific diseases that attack cyclamen and a description of their management are presented in the following sections. The reader is also referred to the introductory chapters for more information on integrated disease management practices.

2 Fungal and Fungus-Like Diseases

2.1 Anthracnose (*Colletotrichum gloeosporioides* Penz.) Penz. & Sacc. in Penz, *C. fragariae* J.H. Simmonds, *Cryptocline cyclaminis* (Sibilia) von Arx

Geographic occurrence and impact. Two different anthracnose diseases have been reported to be problematic on cyclamen. One caused by *Cryptocline cyclaminis* is reported more frequently from Europe (Krebs 1985). The other, caused by *Colletotrichum gloeosporioides* or *C. fragariae*, is more commonly observed elsewhere (Daughtrey et al. 1995; MacKenzie et al. 2008). The disease was reported on florist's cyclamen in South America (Marque et al. 2006; Wright et al. 2006) and North Carolina (Liu et al. 2011). It is difficult to assess its overall economic impact since it is still appearing in many new areas. All cultivars appear to be susceptible.

Symptoms/signs. *Cryptocline cyclaminis* affects the young petioles and pedicels beneath the canopy, stunting and killing them (Fig. 1). Vascular discoloration can be observed in the petioles and in the corm. Eventually the collapsed tissue and lesions will give rise to the orange spore masses called acervuli. Leaf spots are also common (Fig. 2). The other anthracnose, caused by *Colletotrichum gloeosporioides*, causes the round, brown leaf spots and the occasional petal spot. The lesions are more distinct when viewed from the leaf's underside. These spots can resemble *Impatiens necrotic spot virus* lesions but, unlike the virus, eventually give rise to orange spore masses similar to those of *C. cyclaminis* (Fig. 3).

Biology and epidemiology. Conidia from anthracnose lesions are spread by rain or irrigation water to new infection sites (Daughtrey et al. 1995). Inoculum density of *C. cyclaminis* is very important as low densities rarely cause symptoms (Krebs 1986). Disease develops at temperatures from 15–26 °C (59–79 °F) and tends to be most severe at 26 °C (79 °F) (Krebs 1985). Both fungi can survive on compatible host plants and on plant debris. Besides cyclamen, other ornamentals, such as *Cissus*



Fig. 1 *Cryptocline cyclaminis* attacks the young petioles and pedicels beneath the canopy, stunting and killing them Margery Daughtrey © 2017. All Rights Reserved.)



Fig. 2 Foliar lesions of *Cryptocline cyclaminis* (Margery Daughtrey © 2017. All Rights Reserved.)



Fig. 3 Leaf spots caused by *Colletotrichum gloeosporioides* (Margery Daughtrey © 2017. All Rights Reserved.)

rhombifolia, *Erica gracilis*, *Lathyrus odoratus*, and *Pelargonium zonale*, were also susceptible to *C. cyclaminis* when inoculated (Brielmaier-Liebetanz and Bohmer 1988). The potential for spread to these plants from infected cyclamen should be recognized.

Management

- ***Cultural practices*** – Begin with healthy transplants in a suitable soilless potting mix and provide balanced nutrition (Krebs 1987). Always use new or disinfested pots, flats, and benches, especially following an outbreak. Both anthracnose

pathogens spread by spores splashing from plant to plant, so overhead irrigation should be done in the morning to allow leaves to dry. Plants should be spaced properly to provide air movement and decrease the time leaves are wet, and corms should be planted high in the pot. Although cyclamen is the only known natural host, *Cissus rhombifolia*, *Erica gracilis*, *Lathyrus odoratus*, and *Pelargonium zonale* were experimentally inoculated with the pathogen, so certain crops brought into contact with diseased cyclamen could subsequently be potential sources of inoculum in production areas.

- ***Fungicides*** – The strobilurin fungicides pyraclostrobin, pyraclostrobin plus boscalid and fluoxastrobin provided excellent suppression on cyclamen leaves (Chase 2009). In addition, the thiophanate-methyl and mancozeb fungicides whose labels list treatment for cyclamen will also be needed for rotation with the above products for anthracnose control.
- ***Biological control*** – Maya and Matsubara (2013) achieved an 80% reduction in anthracnose following inoculation of cyclamen with mycorrhizae. Later studies showed that mycorrhizal inoculation also countered the damaging effect of heat stress on cyclamen (Maya et al. 2014).

2.2 Botrytis Blight (*Botrytis cinerea* Pers.: Fr.)

Geographic occurrence and impact. Botrytis blight occurs wherever humid conditions (>93% relative humidity) prevail, making it a common occurrence in the greenhouse. The fungus, *Botrytis cinerea*, is ubiquitous and infects most aboveground plant tissue. It infects almost all florist crops (Daughtrey et al. 1995). On cyclamen, it is considered one of the most important diseases of the crop (Kessel et al. 1999) and can reduce profitability directly by destroying the flowers and making them unmarketable.

Symptoms/signs. Symptoms on petals begin as small, bleached spots (Fig. 4) that enlarge, coalesce, and eventually cause the petals to blight. Similarly, the fungus can infect petioles causing them to collapse. *B. cinerea* can often be observed in the



Fig. 4 Botrytis blight lesions on the petals (Missouri Botantic Garden © 2017. All Rights Reserved.)



Fig. 5 *Botrytis cinerea* sporulating on cyclamen stems (Diffusion Morel Co. © 2017. All Rights Reserved.)

lower canopy sporulating on dead and senescing tissue (Fig. 5). These sites become major focal points for the production of inoculum. The symptoms of Botrytis blight can be diverse, manifesting as blight or as distinct lesions. Leaves tend to be less susceptible than the flowers.

Biology and epidemiology. Survival of the pathogen occurs as mycelium on plant debris (Yunis and Elad 1989) and as sclerotia (Araújo et al. 2005). The pathogen produces spores, borne on stalks, which are released with air currents and water splashing. A factor that is crucial to Botrytis infection is high humidity (>93%), and this is commonly achieved in microclimates within the canopy in a greenhouse. Initial infections begin following a buildup of inoculum on dead and senescing leaves within the canopy. Once sufficient inoculum has been released, healthy leaves and flowers are infected and the epidemic begins, provided susceptible tissue is available and high humidity prevails.

Management. Successful management of Botrytis blight on cyclamen must include cultural practices and sanitation along with chemical and/or biological practices to suppress inoculum buildup.

- ***Cultural practices*** – Given that Botrytis blight occurs when the relative humidity reaches above 93%, one major component of disease management is to reduce the humidity. Proper spacing and ventilation can help eliminate the humid microclimates within the canopy where infection can occur. Heating and venting greenhouses at dusk can expunge large amounts of humid air and suppress infection (Daughtrey et al. 1995). Any efforts to keep the foliage dry by using subirrigation systems can also reduce humidity in the canopy. When subirrigation is not available, overhead watering should be done in the morning to allow time for the foliage and flowers to dry. Proper spacing of plants favors rapid drying of leaves and flowers. When possible, computer-controlled environmental systems can be used to increase ventilation and raise temperature to avoid high humidity. Postharvest conditions can also affect Botrytis blight development in cyclamen. On *C. persicum,* the presence of ethylene increased disease, while applying the

ethylene antagonist 1-methylcyclopropene extended the aesthetic life of inoculated flowers by 25 d (Seglie et al. 2009).

- ***Sanitation*** – Although the fungus is ubiquitous, any effort to prevent inoculum from increasing in and around the production facility will result in fewer infections. Efforts should be made to remove dead tissue on the bench or greenhouse floor, which the fungus will use as a substrate to sporulate. Efforts should be made to remove all pruned flowers as they, along with defoliating leaves, can land on healthy tissue and quickly lead to new infections.
- ***Fungicides and biocontrols*** – A considerable amount of research has been done on chemical control of Botrytis blight, in part due to the propensity of the pathogen to develop resistance (Leroux 2007; Moorman and Lease 1992). There are many products in many different families of fungicides that can provide excellent suppression providing tolerant strains have not been allowed to proliferate. The fungicides commonly used today include chlorothalonil, singly and in combinations, along with iprodione, fludioxonil, fenhexamid, strobilurins, and triflumizole. Additional combination products including cyprodinil + fludioxonil and pyraclostrobin + boscalid are also employed in Botrytis blight management. Fungicide resistance is common for thiophanate-methyl and iprodione due to the extensive movement of plant materials in the horticultural trade. Once resistant strains are present in an operation, alternate classes of fungicides need to be used. More environmentally safe active ingredients are also labeled for control of Botrytis blight, such as bicarbonates, *Bacillus subtilis*, and reduced risk materials like polyoxin D. There has been other work using the biological control *Ulocladium atrum*, which performed as well as chemical fungicides in suppressing Botrytis blight on cyclamen (Köhl et al. 2000). The chitinolytic bacterium *Serratia marcescens* also suppressed Botrytis blight on cyclamen petals by 60% and was equal in effect to the dicarboximide fungicide iprodione (Iyozumi et al. 1996).

2.3 Fusarium Wilt of Cyclamen (*Fusarium oxysporum* Schlechtend.:Fr. f. cyclaminissp. Gerlach)

Geographic occurrence and impact. Fusarium wilt was first reported in 1935 in Germany (Wollenweber and Reinking 1935). Outbreaks were subsequently reported from France (Barthelet and Gaudineau 1936), Italy (Bongini 1940), England (Moore 1947), the United States in 1949 (Tompkins and Snyder 1972), Bulgaria (Khristova 1958), Belgium (Rouxel and Grouet 1974), and Portugal (Pitta and Teranishi 1979). The fungus was described and labeled *F. oxysporum* f. sp. *cyclaminis* in 1954 by Gerlach (1954). In 1977, Fusarium wilt was reported for the first time in the Netherlands, where it was particularly destructive due to the recycling of contaminated irrigation water (Rattink 1986). No races of the pathogen exist, but some clonal lineages have been observed (Woudt et al. 1995). The disease can be extremely destructive and has resulted in losses of over 50% of the crop in greenhouses (Elmer and Daughtrey 2012).

Symptoms/signs. All reports of Fusarium wilt of cyclamen have described the same symptoms regardless of locale. In young plants, the disease first appears as bright-yellow chlorosis (Fig. 6), which begins at the base of the leaf and then expands outward toward the perimeter. Alternatively, and more often in older plants, chlorotic patches may develop on the leaf blade or at the leaf edge. In both situations, the affected leaf portions become flaccid and loose turgidity (Fig. 7). Symptoms may appear at all stages of growth and can be observed on roots, corms, and aboveground parts. Infected roots exhibit vascular discoloration and may be totally discolored and darkened.

Reddish-brown vascular discoloration is frequently observed in the corm (Fig. 8). Fusarium wilt can be distinguished from the bacterial soft rot diseases (caused by *Pectobacterium* spp. and *Dickeya* spp.; see below) largely because the corm remains firm and shows vascular discoloration in the case of Fusarium wilt. The vascular discoloration can turn to brownish black, dark red, or purplish over time. Many times, no symptoms are apparent when the plant is vegetative, but then it quickly develops wilting and chlorosis when flowering occurs. The timing is most unfortunate since growers have expended considerable resources only to lose the crop when it becomes marketable. The latent expression of symptoms (Gerlach 1954; Rouxel and Grouet 1974) has cost the industry considerable profits. Molecular assays that quickly detect the pathogen in pot leachates might allow growers to identify and discard contaminated seedlings. Recently, a set of polymerase chain reaction (PCR)



Fig. 6 Chlorosis associated with Fusarium wilt (Margery Daughtrey © 2017. All Rights Reserved.)



Fig. 7 Collapsed plant due to Fusarium wilt (Margery Daughtrey © 2017. All Rights Reserved.)



Fig. 8 Vascular discoloration in the cyclamen corm due to Fusarium wilt (Margery Daughtrey © 2017. All Rights Reserved.)

primers proved effective experimental tools for identification and detection of *F. oxysporum* f. sp. *cyclaminis,* distinguishing the pathogen from nonpathogenic strains of *F. oxysporum* often associated with cyclamen. However, these molecular tools have not been commercialized for use.

Biology and epidemiology. Infection results from invading hyphae that can originate from a microconidium, macroconidium, chlamydospore, or mycelium. Most studies on inoculum density revealed disease severity is a function of inoculum load (Elmer 2002b; Garibaldi 1988; Rattink 1986, 1990). Although seed transmission is strongly suspected, it has been very difficult to routinely detect the pathogen from seeds (Tompkins and Snyder 1972). The pathogen can be spread from plant to plant by overhead watering, contaminated tools, soil, pots, and trays. Although insect vectoring is strongly suspected, definitive experiments demonstrating transmission by fungus gnats and/or shore flies have yet to be done (Gillespie and Menzies 1993). Spread by windblown conidia may also be a means for short-distance spread of *F. oxysporum* f. sp. *cyclaminis*, and this should be considered a possible means of dissemination in greenhouses. Short-distance transmission can also occur via irrigation water, despite reports stating *F. oxysporum* f. sp. *cyclaminis* does not spread in water as effectively as other *Fusarium* fungi (Krebs 1985). Ebb and flow recycling systems have been associated with major outbreaks in the Netherlands (Rattink 1986). However, disease thresholds are rarely reached if scouting and removal of symptomatic plants are done promptly. Long-distance transmission of the *Fusarium* pathogen on seedling plugs is a recognized problem within the cyclamen industry. Rattink (1986) found that the time of the onset of symptoms became shorter as temperatures increased from 15 °C (59 °F). The ideal temperature for infection was 27.5 °C (81.5 °F). Wide fluctuations in temperature have also been associated with increased disease, possibly by placing heat and drought stress on the host.

Management

- ***Cultural practices*** – Fusarium wilt of cyclamen is one of the more difficult diseases to manage (Elmer and Daughtrey 2012). All attempts to identify horticulturally acceptable resistance in the host have been unsuccessful. Orlicz-

Luthardt (1998) and Ewald et al. (2000) have identified tolerance to Fusarium wilt in related hybrids of *C. persicum* Mill. × *C. purpurascens* Mill., but no further breeding has been advanced for disease resistance. Steam sterilization of the soil can greatly reduce disease. However, growers should exercise caution when using soil amendments with steam-sterilized soil. Garibaldi (1988) reported that steam-sterilized local medium made from soil, peat, beech leaves, and manure (30:40:20:10) was highly conducive to disease. Growers should always experiment with small batches before incorporating new amendments. Fertilizing with nitrates and adding lime to achieve a 6.5–7.0 pH in the rooting medium will suppress Fusarium wilt of cyclamen. Fertilizers that acidify the soil and lower pH, such as ammoniacal sources of N [NH_4NO_3, $(NH_4)_2SO_4$], should be avoided. Amending the soil with well-rotted composts is also effective at suppressing Fusarium wilt, because the dominant form of N in these composts is nitrate. In addition, chloride salts can reduce the severity of disease, but growers should exercise caution and test on a small number of plants before applying salts (Elmer 2002b).

- ***Fungicides*** – Fungicides have been useful in protecting against Fusarium wilt if applied preventively and during early onset but generally fail in providing acceptable disease control in infected plants. Soil drenches with benomyl, thiophanate-methyl, chlorothalonil, and tolylfluanid provide little or no suppression. Gullino et al. (2002) found that soil drenches with azoxystrobin were effective, whereas the other strobilurins tested, such as kresoxim-methyl and trifloxystrobin, were phytotoxic. Elmer and McGovern (2004) found that some combinations of biological fungicides with the registered fungicide fludioxonil were more effective than the fungicide alone in reducing disease progress. Disease onset was delayed 3 weeks when acibenzolar-S-methyl, a product that induces resistance to Fusarium wilt, was applied, but marked phytotoxicity was also observed (Elmer 2006).
- ***Biological control*** – Migheli and Garibaldi (1995) and Minuto et al. (1995) reported that disease incidence could be reduced by supplementing the growing media with nonpathogenic antagonistic strains of *Fusarium* species applied alone or in mixtures. Later, Minuto et al. (2004) showed that nonpathogenic *Fusarium oxysporum* strains were superior to benomyl in protecting cyclamen from Fusarium wilt. Elmer (2001, 2002a) found combinations of nonpathogenic *Fusarium oxysporum, Pseudomonas fluorescens*, and other agents that could suppress disease, but these products have not been made commercially available. Other attempts at biological control have resulted in some success in reducing disease incidence and severity. A strain of the bacterium *Serratia marcescens* that has chitinolytic activity reduced Fusarium wilt for up to 3 weeks after inoculation with the pathogen; at harvest, only 20% of the plants were diseased compared to 90% incidence in the untreated control (Someya et al. 2000). Unfortunately, experimentation with biological fungicides has not yielded a commercially acceptable management strategy. Even when combinations of biological products were applied preventively, disease control in inoculated plants never reached an acceptable level, even when the plants were subsequently returned to a

conventional chemical fungicide program (Elmer and McGovern 2004). Disease suppression is sometimes improved by combining biological products with fungicides (Minuto et al. 1995; Elmer and McGovern 2004; Daughtrey and Tobiasz 2005). Growers should read the labels and consult resources to determine if products are compatible with one another.

- ***Integrative strategies*** – Until host resistance becomes available, growers must rely on a combination of strategies. The more effective practices for management of Fusarium wilt include strict sanitation, crop monitoring, and immediate fungicide application.

2.4 Phyllosticta Leaf Spot (*Phyllosticta cyclaminis* Brun)

Geographic occurrence and impact. The fungus was first described in 1890 by Brun, but no reports of economic damage were recorded until 1929 when a report of its occurrence was made in Italy, where the disease caused defoliation (Bongini 1930). The disease is also listed as a potential disease to manage in California, but outbreaks appear to be sporadic.

Symptoms/signs. The symptoms are first observed as yellowish-brown spots that appear near the margins. Lesions later develop concentric light and dark zones approximately 3 cm in diameter. Lesions can coalesce and, when a sufficient area of the leaf has become infected, the leaf will abscise. In the center of the lesion, signs of the fungus appear as immersed pycnidia, which liberate long, dense cirrhi of hyaline, ovoid spores.

Biology and epidemiology. The fungus overwinters on infected plant tissue. Splashing rain or irrigation favors spread. It is known that high humidity is necessary for disease to occur. Bongini (1930) discovered disease would not occur when minimum and maximum temperatures were 10°–11 °C (50–52 °F) and 18–19 °C (64–66 °F), respectively.

Management. Management strategies for Phyllosticta leaf spot are the same as for all leaf diseases. Practice sanitation and clean up from last year's crop; scout and apply registered fungicides when disease is suspected. Chlorothalonil and mancozeb fungicides have preventive activity.

2.5 Pythium Root Rot (*Pythium* spp.)

Geographic occurrence and impact. Most reports on cyclamen have identified *P. irregulare* as the most commonly identified species (Moorman et al. 2002), but, in Germany, *P. debaryanum* was reported (Reimherr 1985). A report from Ohio found that *P. aphanidermatum* was the cause of root rot (Kuter et al. 1988). Regardless of the species, the disease can be very destructive when wet soil conditions prevail (Reimherr 1985).

Symptoms/signs. Leaves initially develop a dull green color, lose turgor and then collapse. Many times they twist and curl. The root system darkens and feeder roots collapse. Roots frequently slough off the outer cortical tissue leaving the inner vascular stele, which gives rise to a rat-tail appearance. When seedlings are attacked, the symptoms can appear as damping-off disease.

Biology and epidemiology. *Pythium* spp., like *Phytophthora* spp., are not true fungi but oomycetes, requiring free water to release zoospores. Consequently, the disease is favored by wet conditions, and spread of the pathogen requires irrigation water (Moorman et al. 2002). *Pythium* spp. commonly persist on the floors of greenhouses in organic debris; their long-term survival spores called chlamydospores and sexually produced oospores can remain dormant without a susceptible host. These propagules germinate in response to plant root exudates and invade susceptible roots. Fungus gnats (*Bradysia* spp.) likely assist *Pythium* by creating infection sites through their root feeding. (See ▶ Chap. 4, "Insect Management for Disease Control in Florists' Crops" for additional information.)

Management

- ***Cultural practices*** – Pythium root rot can be suppressed effectively by improving water drainage. Providing partial saturation of the growing medium can also reduce Pythium root rot (Elmer et al. 2011). Growers should plant in pasteurized media and immediately discard infected plants. Avoiding excess nitrogen has been shown to be important for *Pythium* control in geraniums (Gladstone and Moorman 1989, 1990).
- ***Fungicides*** – When outbreaks appear, many fungicides are effective. In California, suppression of root rot was achieved following drenches with metalaxyl, followed by metalaxyl + benomyl and fenaminosulf (Raabe and Hurlimann 1981). Spengler (1979) found that furalaxyl successfully controlled *P. debaryanum* on cyclamen. Growers should rotate fungicides to prevent resistant strains of *Pythium* pathogens from developing. Mirkova (1995) reported in Bulgaria that *Pythium* isolates from Cyclamen showed tolerance to metalaxyl in 1988–1990. Growers using mefenoxam (the biologically active enantiomer of metalaxyl) against Pythium diseases in potted plant production today will want to rotate with other effective fungicides such as etridiazole and cyazofamid.
- ***Biological control*** – Some advances have been made with biological control of Pythium root rot of ornamentals, but information on cyclamen is not available.

2.6 Phytophthora Root Rot of Cyclamen (*Phytophthora tropicalis* Aragaki) & Uchida, syn. *Phytophthora capsici*, Leonian

Geographic occurrence and impact. The pathogen was first described as *Phytophthora capsici* but was redescribed as *P. tropicalis* (Aragaki and Uchida 2001; Donahoo and Lamour 2008; Oudemans and Coffey 1991). The disease is not commonly found in cyclamen operations but can be devastating when it appears.



Fig. 9 Phytophthora blight (Diffusion Morel Co. © 2017. All Rights Reserved.)

Outbreaks have been reported from the United States, the Netherlands, and Germany (Gerlach and Schubert 2001). The disease caused economic losses in the late 1990s in Europe when it first appeared. The pathogen presumably entered the European operations on plant material since the fungus has no reported hosts in Europe. In the United States, *P. tropicalis* has been reported causing root rots on greenhouse and woody ornamentals (Olsen and Benson 2010).

Symptoms/signs. When the seeds are infested or are planted into infested soils, damping-off can occur. In older plants, the first symptom noticed is a change in leaf color, which turns from a bright, dark green to an off-colored, olive green (Fig. 9). Leaves become chlorotic and wilt followed by dark browning at the leaf base. The roots and stems eventually turn black and collapse even though the corms usually do not exhibit vascular discoloration.

Biology and epidemiology. *Phytophthora* spp., like *Pythium* spp., are not true fungi but are oomycetes that will release their spores (zoospores) in free water. Consequently, a major requirement for spread and disease development is wet conditions. *P. tropicalis* can spread very effectively in irrigation water (Hong et al. 2006). Infection occurs on young roots where the pathogen invades and grows intercellularly, collapsing the cells while it grows. Optimal temperatures for disease development are between 20 °C and 25 °C (68–77 °F), but the disease can also develop in cool conditions (15 °C/59 °F). During periods of optimal environmental conditions, the pathogen remains asexual. When moisture is limited, these spores germinate to produce infecting hyphae, whereas if free water is available, then sporangia and zoospores are produced. Once the disease has killed the plant or when conditions become unfavorable, the pathogen begins producing its sexual oospores. Species of *Phytophthora* are not recovered on greenhouse floors as frequently as *Pythium*, but the pathogen can persist in infested soils and on infested tissue for extended periods of time as oospores, chlamydospores, or mycelium. In the United States, the pathogen is reportedly causing disease on other ornamentals besides cyclamen, so growers should be aware of new sources of inoculum for cyclamen that may enter their greenhouse on other plants (Hong et al. 2006; Olsen and Benson 2010).

Management. Management of Phytophthora root rot is the same as for Pythium root rot. Avoid overwatering and improve soil drainage to prevent development of the disease. Inspection and frequent scouting of new material are important given that the European outbreak presumably occurred from the importation of infested material. Fungicides that are effective against Pythium root rot will also suppress Phytophthora root rot (Aragaki and Uchida 2001). Strobilurins and phosphorous acid compounds are more effective against *Phytophthora* than against *Pythium* spp.

2.7 Powdery Mildew of Cyclamen (*Pseudoidium cyclaminis* (Wenzl) U. Braun & R.T.A. Cook)

Geographic occurrence and impact. Powdery mildew is rarely observed on cyclamen. However, breeding programs developing new hybrids could alter susceptibility to powdery mildew, so the disease may become more important in the future. Powdery mildew on cyclamen has been reported from Europe.

Symptoms/signs. The disease appears as small powdery lesions that appear on petals only (Fig. 10). Infections can reduce or destroy the value of the plant.

Biology and epidemiology. The spores of this fungus spread by the wind during dry periods. The infection occurs during warm, humid weather. Temperatures of 21–25 °C (70–77 °F) are favorable to disease development, fostering spore formation and germination.

Management. This disease is rare; it can be suppressed with chemicals when encountered. Test chemicals for phytotoxicity to cyclamen before using.

2.8 Ramularia Leaf Spot, Cyclamen Stunt Disease (*Ramularia cyclaminicola* Trel.)

Geographic occurrence and impact. The disease was first called cyclamen stunt, but Ramularia leaf disease was later shown to be caused by the same pathogen,



Fig. 10 Powdery mildew of cyclamen caused by *Pseudoidium cyclaminis* (Wade Elmer © 2017. All Rights Reserved.)

Ramularia cyclaminicola. The fungus was first described as *Cladosporium cyclaminis* but was reclassified in 1950 as *Ramularia cyclaminicola* (Baker et al. 1950; Davis 1950). It was first observed in Illinois in 1914 (Trelease 1916) then in New York in 1926. Since then, outbreaks were reported in California, Colorado, Massachusetts, Ohio, Pennsylvania, New Jersey, and Quebec (Massey and Tilford 1932). Before 1950, the disease was widely distributed in North America causing moderate to severe losses, but the occurrence of disease is not significant in the horticultural trade today. However, since the original source of inoculum was speculated to be a native plant in the Primulaceae, growers should be aware of the possibility of its reoccurrence.

Symptoms/signs. *Ramularia cyclaminicola* is a fungus causing systemic infection. The disease can manifest as a stunt, a leaf spot, or both. Smaller plants with small chlorotic leaves, short petioles, and short peduncles that force the flower to open within the plant canopy are typical of the stunt disease. Reddish-brown necrotic areas occur in the corm tissues and can extend up into the petioles, peduncles, and down into the primary roots. The older leaves of mature plants can develop diffuse yellow lesions and eventually wilt. The leaf spot disease is usually most obvious on the lower surface of leaves, where brown spots with indefinite margins that coalesce are seen. The fungus usually sporulates on the undersurfaces of the lower leaves but can also be recovered from the vascular tissue.

Biology and epidemiology. The fungus survives as sclerotia (long-term survival structures) in soil and on plant debris. The pathogen can be spread by soil on infested pots, on airborne spores, and on seedlings and seeds. Following infection, the leaves die and sclerotia form and serve as the overwintering or survival structure. They germinate when susceptible tissue is available and the infection cycle begins again. The disease is favored by hot, humid weather .

Management. Destroy affected plants and use new or disinfested growing medium. The fungicides effective against Phyllosticta and Septoria leaf spots should be effective against cyclamen leaf spot/stunt.

2.9 Rhizoctonia Crown and Stem Rot [*Rhizoctonia solani* Kühn (Teleomorph: *Thanatephorus cucumeris* (A.B. Frank) Donk)]

Geographic occurrence and impact. Rhizoctonia root rot can be a serious disease, but it appears to be restricted to isolated outbreaks. The pathogen is found globally so the disease can be common in all cyclamen-producing areas.

Symptoms/signs. The fungus can infect all stages of the plant, beginning with a seedling damping-off and extending to a web blight that can colonize and infect mature aboveground tissues when humidity levels are very high. The most commonly observed symptom, however, is a root and stem rot. Once the pathogen invades the tissue, reddish-brown lesions appear, which over time can expand.

Infected stems and petioles frequently are girdled and fall over. Portions of root systems may also turn brown. A common appearance on foliage of plants affected by Rhizoctonia attack is the look of nitrogen deficiency (yellowing of older leaves), often accompanied by the collapse of the aboveground tissue.

Biology and epidemiology. *R. solani* can attack many different plants, and it can be a common resident in greenhouses. It is found in field soil, where it persists for years as resting spores called sclerotia or as a saprophyte on plant debris. *R. solani* has strong competitive saprophytic ability, meaning that it can grow quickly through soil if plant debris is present and the environmental conditions are suitable. The fungus does not spread in irrigation water or wind unless contaminated soil particles are being distributed: It does not have spores. Insect transmission is not common. The main sources of inoculum are non-pasteurized soil and infected plants. *R. solani* infects a wide range of ornamentals so inoculum can enter the production cycle any time new plant propagules are brought into the greenhouse.

Management

- ***Cultural practices*** – Given the wide host range of *R. solani* and its propensity for survival in soil, it would be safe to assume that all unpasteurized soil is a potential source of inoculum. Sanitation should be the first line of defense.
- ***Fungicides and biocontrols*** – Fungicides also remain an essential tool for managing Rhizoctonia root rot including fludioxonil, PCNB, strobilurins, and thiophanate-methyl. Of the biological controls tested against Rhizoctonia root rot, the bacterium *Serratia marcescens* has been shown to be effective (Someya et al. 2000). Commercial formulations of *Trichoderma harzianum* have also been labeled for use on cyclamen.

2.10 Septoria Leaf Spot (*Septoria cyclaminis* Durieu & Mont.)

Geographic occurrence and impact. The fungus was first described in 1849 and has been reported from Central and Eastern Europe and the Middle East (Bacigálová et al. 2010). Septoria leaf spot is not a disease that causes major problems in the industry, but it can occasionally become problematic in some greenhouses. It was recently observed on a perennial cyclamen, *C. fatrense*, in Slovakia (Bacigálová et al. 2010).

Symptoms/signs. The fungus first infects the undersides of the leaves. From above, red concentric spots are observed, which turn gray with red borders. There can be a fairly large number of leaf blotches, and they may dry up the leaf. Within the centers of the lesions, there will appear small, peppery dots called pycnidia, which bear the spores. Symptoms can be confused with those caused by *Phyllosticta cyclaminis*, which also produces spores in pycnidia. Spore shape distinguishes the two pathogens.

Biology and epidemiology. The fungus spreads similarly to *Phyllosticta cyclaminis*. When spores land on the leaf and germinate, hyphae invade the plant through the stomata on the undersides of the leaves. Once established, the fungus develops in the intercellular spaces. In damp weather, the fruiting bodies exude elongated, multicelled conidia, which are spread by water, on tools and clothes. This disease spreads very fast. The fungus can survive as fruiting bodies on plant debris. Following a 12-h period when free water is on the leaf, spores will germinate in about 20 h at 18 °C (64 °F). Disease develops quickly when humid conditions prevail. Optimal temperatures for disease occur between 20 °C (68 °F) and 27 °C (81 °F).

Management. Management of Septoria leaf spot of cyclamen is the same as for Phyllosticta leaf spot. For cultural control, reduce the length of time that the leaf surface sits wet.

2.11 Thielaviopsis Black Root Rot [*Thielaviopsis basicola* (Berk. & Broome) Ferraris (1912)]

Geographic occurrence and impact. Like Rhizoctonia root rot, Thielaviopsis root rot occurs sporadically. The disease has been reported in Europe and the United States. Thielaviopsis root rot appears to be a seedling problem, and, like many soilborne problems, outbreaks may be restricted to certain batches of seedlings. When the disease is present, losses can be severe.

Symptoms/signs. A distinct black discoloration occurs in the small feeder roots, with a blackened root system seen in more advanced cases (Fig. 11). Infected plants are stunted, turn yellow, wilt, and die. Yellowing of the young leaves is seen when Thielaviopsis attacks the young roots.

Biology and epidemiology. The fungus infects young roots and penetrates between cells. Discontinuous regions of root discoloration can be observed on the root system. *T. basicola* produces black, thick-walled, resistant chlamydospores as well as endoconidia that function as propagules for dissemination. The fungus grows best between 13 °C (55 °F) and 18 °C (64 °F) and enjoys very damp soil conditions; it is inhibited by high temperatures, ammonium fertilization, and acid (pH $<$ 5.5) soils (Harrison and Shew 2001). The fungus is worldwide in distribution and infects a broad range of cultivated plants in many unrelated families. Fungus gnats and shore flies can spread black root rot.

Management. Cultural practices can be effective in suppressing Thielaviopsis root rot of cyclamen. Soil pH greater than 5.5 is more conducive to disease, but acid soils can cause other problems for cyclamen, so growers should not rely on pH management. Proper diagnosis should be done to ensure the pathogen is *T. basicola* before



Fig. 11 Symptoms of Thielaviopsis black root rot on cyclamen roots (Diffusion Morel Co. © 2017. All Rights Reserved.)

taking control actions. Sanitation is also critically important in eliminating black root rot following outbreaks. Disinfesting seed trays and pots and using pasteurized soil mixes should be done following an infestation. Fungicides work best when used as protectants in combination with cultural controls and when applied early in the production cycle. Fungicides that have activity against *T. basicola* are fludioxonil, polyoxins, strobilurins, triflumizole, and thiophanate-methyl. Thiophanate-methyl has provided the best control in many studies, but this material should be used in rotation with others to avoid the development of fungicide resistance in the *T. basicola* population within a greenhouse.

3 Bacterial and Phytoplasma Diseases

3.1 Bacterial Corm and Leaf Rots *Pantoea agglomerans* (Ewing & Fife) Gavini, Mergaert *syn. Erwinia herbicola* (Gavini, Mergaert); *Pectobacterium carotovorum* (Jones) Waldee *syn. Erwinia carotovora; Dickeya dadantii subsp. dieffenbachiae* (Samson et al.; Brady et al.), *syn. Erwinia chrysanthemi; and Erwinia rhapontici*

Geographic occurrence and impact. Erwinia corm rot is observed in most production facilities. Reports of severe losses have come from Australia (Chandrashekar

and Diriwaechter 1984), Europe (Bonifacio 1960; Carta 1993; Lemattre 1973; Panagopoulos and Psallida 1970), Asia (Amani 1967), and North and South America (Beamont 1953; Romero and Rivera 2005). This disease can become widespread in a production facility and cause considerable damage if not contained.

Symptoms/signs. Symptoms are first observed in the leaves, but this is usually a reflection of advanced infection in the corm tissue. Leaves wither and droop abruptly onto the pot with blackened, flaccid stems as the transport of water and nutrients is blocked (Fig. 12). The petiole can also develop oozing lesions that form at the junction of the leaf and the petiole. Eventually, the disease advances and colonizes the whole plant as it travels upward. Many times, an unpleasant fishy odor accompanies diseased plants.

Biology and epidemiology. Many bacteria in the genus *Erwinia* have been assigned new names, but the problems they cause are the same that were encountered in the last century. In general, all of these soft rot bacteria have the ability to dissolve pectin and other structural components in the plant, so infected tissues collapse and the bacteria utilize the plant material as food. The bacteria cannot directly penetrate plant tissue so they enter through stomata, wounds, and cracks in the corm – and at the sites where leaves or buds have been removed. Once inside the plant, tissues are soft rotted as the bacteria multiply and produce enzymes to break down the plant material; they may also spread throughout the plant through the vascular system. The bacteria can survive in the soil, in irrigation water, and in plant debris. Cyclamens that are planted too deeply are especially prone to bacterial soft rot, because the



Fig. 12 Bacterial corm rot of cyclamen (University of Maryland Extension Service © 2017. All Rights Reserved.)

pathogens do well in an anaerobic environment. Fungus gnat infestations have been seen to aggravate problems with bacterial soft rot, as larvae feeding on the corms make wounds that facilitate entry of the bacteria.

Management. Following an outbreak, all infected tissue should be discarded. A sanitary cleanup should be undertaken, since the pathogen has the ability to persist in soil and within plant debris. Once the operation has been sanitized, close inspection of incoming seedlings should be done to minimize reentry into the establishment. The disease tends to be more severe in warm, humid weather, since the bacterium multiplies more readily at high temperatures (between 25 °C (77 °F) and 30 °C (86 °F). Overfertilizing the plant increases disease severity by producing more young, succulent, susceptible tissue. Careful roguing of infected plants is important since billions of bacteria are released into the soil from infected plants. Minuto et al. (2004) had success using copper sulfate sprays to suppress *P. carotovorum*.

3.2 Ralstonia Wilt (*Ralstonia solanacearum* (Smith 1896) Yabuuchi et al. 1996, comb. nov.)

Geographic occurrence and impact. Bacterial wilt caused by *Ralstonia solanacearum* (formerly known as *Pseudomonas solanacearum* and now considered a heterogeneous species complex) has a very wide host range including cyclamen. Although outbreaks on cyclamen are rare, the potential exists for losses. Were this pathogen to appear in cyclamen, it could be a matter of regulatory concern, as some pathogen strains (race 3, biovar 2) also cause losses in important vegetable crops. *R. solanacearum* causes latent infections, which may slow its detection in new or uncommon hosts.

Biology and epidemiology. Much of the information on the biology and epidemiology for bacterial corm and leaf rot diseases of cyclamen applies to the Ralstonia disease.

3.3 Phytoplasma Diseases

Geographic occurrence and impact. Phytoplasma diseases on cyclamen are rare. One report in 1990 cited an occurrence in northern Italy and Germany (Bertaccini 1990). However, it is likely that the disease has appeared many times before and merely gone unreported.

Symptoms/signs. Symptoms on cyclamen were observed as phyllody (the abnormal development of floral parts into leafy structures) and virescence (green coloration in flowers) (Fig. 13). Stunting with thickened, rolled leaves has also been associated with the disease (Fig. 14).

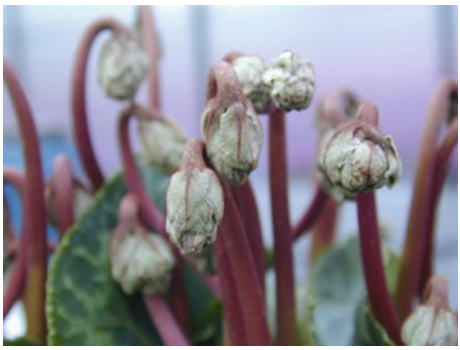


Fig. 13 Symptoms of phyllody (the abnormal development of floral parts into leafy structures and virescence (*green* coloration in flowers) (Diffusion Morel Co. © 2017. All Rights Reserved.)



Fig. 14 Phytoplasma disease of cyclamen causing stunting with thickened, rolled, cup-shaped leaves (Diffusion Morel Co. © 2017. All Rights Reserved.)

Biology and epidemiology. Phytoplasmas are prokaryotes like bacteria, but they are obligate plant parasites vectored by phloem-feeding leafhoppers. Based on molecular analysis, the cyclamen phytoplasmas belong to 16S rDNA subgroups 1B and 1C (Alma et al. 2000). According to RFLP molecular analysis, the cyclamen phytoplasmas are in the same subgroup (1B) as aster yellows phytoplasmas. The cyclamen phytoplasma has been placed in the stolbur phytoplasma group along with the phytoplasmas detected in apricot, grape, lisianthus, papaya, and pepper (Weintraub et al. 2007). The disease is only transmitted by leafhopper feeding, and there is only one report of cyclamen serving as a reservoir for inoculum. In that study only one plant out of 366 plants tested was infected by leafhoppers that had fed on infected cyclamens. The phytoplasma disease on cyclamen was considered a dead end for the pathogen, since transmission appeared to be extremely rare (Alma et al. 2000).

Management. Given the rarity of the disease, it is not likely that a major outbreak will occur. Management, however, is achieved through monitoring and the reduction of the insect vector, which would not typically be found in greenhouses. Additional information on vector management may be found in introductory (► Chap. 4, "Insect Management for Disease Control in Florists' Crops").

Symptoms/signs. Symptoms are similar to bacterial corm rot. Wilting and stunting are first observed in the aboveground tissue, but this reflects advanced infection in the corm tissue. Leaves wither and droop abruptly onto the pot.

Management. Refer to bacterial corm and leaf rot diseases of cyclamen.

4 Viral Diseases

4.1 Tomato Spotted Wilt Virus: TSWV

Geographic occurrence and impact. *Tomato spotted wilt virus* (TSWV) is considered to be one of the worst viral threats on cyclamen. It belongs to the genus *Tospovirus* in the family Bunyaviridae and has a very wide host range. The virus was reported in Europe in 1932 but seemed to have disappeared. It reappeared in the ornamentals industry in 1986 when the thrips *Frankliniella occidentalis*, one of its most effective vectors, became widespread in the horticultural trade. It still constitutes one of the most prevalent viruses in the global greenhouse industry (Daughtrey et al. 1997; Kamińska 1975; Resende et al. 1996; Vozelj et al. 2003).

Symptoms/signs. The level of damage can vary widely depending on the environmental temperatures and cyclamen cultivar. In general, plants are stunted with leaf mosaics, discoloration, and necrotic patches (Fig. 15). Round brown spots are typical on more mature cyclamen. Line patterns and various chlorotic and necrotic symptoms associated with veins are seen. It is impossible to distinguish TSWV from *Impatiens necrotic spot virus*, another tospovirus, by symptoms, so proper diagnosis needs to be done by a professional laboratory using serological or molecular assays.

Biology and epidemiology. Over 500 species play host to this virus, including ornamentals, vegetables, and weeds. Contamination is only through the feeding of the thrips vectors, since the virus is not seed transmitted. Only six species of thrips are known to carry the virus; of these, the Western flower thrips (*Frankliniella*



Fig. 15 *Tomato spotted wilt virus* on cyclamen (Diffusion Morel Co. © 2017. All Rights Reserved.)

occidentalis) is the most common one in most greenhouses. Only the first and early second larval stages are able to acquire tospoviruses, and only immature thrips that acquire these viruses or adults derived from such immatures are vectors. Adult thrips remain viruliferous for life; but tospoviruses are not transovarial. Incubation of the virus in the cyclamen plant takes around 2 months, weeks longer than for some other flower hosts.

Management. Control of the thrips vector is the single most important strategy, which should be focused on while maintaining effective scouting and sanitation practices. In the most stringent management program, one should quarantine new stock and seedlings for at least 2 months, until plants appear to be clean; prevent thrips from developing on these plants while they are in quarantine. Remove old flowers as they serve as a source of pollen for thrips. Sterilize tools used in cutting old flower peduncles as plant sap could possibly transmit the virus (it is not known to be easily sap transmitted, however). All infected plants must be destroyed and removed from the greenhouse promptly. Eradicate weed hosts and any other potentially infected host crops to eliminate inoculum reservoirs. More information can be found in the introductory (▶ Chap. 4, "Insect Management for Disease Control in Florists' Crops").

4.2 Impatiens Necrotic Spot Virus: INSV

Geographic occurrence and impact. *Impatiens necrotic spot virus* (INSV) is a very common viral disease in greenhouses, with a wide host range encompassing hundreds of flower crops. It is reported from Europe (Bellardi and Vicchi 1998); Japan (Goto et al. 2001), South America (Resende et al. 1996), New Zealand (Elliott et al. 2009), and North America (Daughtrey et al. 1997). Within North America, this is the tospovirus most often encountered in greenhouses, whereas the closely related *Tomato spotted wilt virus* (TSWV) is more often found outdoors.

Symptoms/signs. Leaf symptoms on cyclamen are sometimes very distinctive, with the production of ring spots, chestnut, or yellow in color, sometimes seen at the margin of the leaf as "fingerprints" (Fig. 16). The virus can also manifest these symptoms on the flower petals. As with TSWV, infected and damaged cells do not expand and grow at the same rate as healthy cells so distortion and leaf deformity develop over time. The virus can cause wilting, stem death, stunting, yellowing, poor flowering, and sunken round, brown spots on leaves along with the characteristic ringspots. In some situations, the plant may remain asymptomatic. Positive diagnosis is achieved only after samples are assayed using diagnostic kits or serological assays in a laboratory that can specifically identify the virus.

Biology and epidemiology. INSV belongs to the genus *Tospovirus* in the family Bunyaviridae and acquisition and transmission of the virus by thrips is the same as with TSWV. The incubation period within the plant is 2 months.

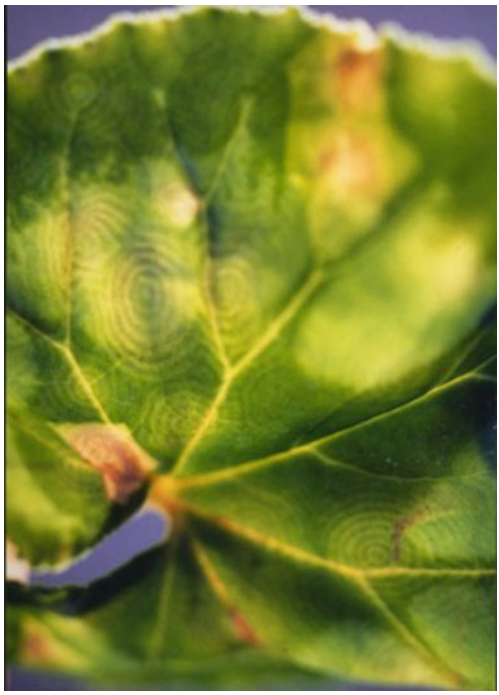


Fig. 16 *Impatiens necrotic spot virus* on cyclamen (Diffusion Morel Co. © 2017. All Rights Reserved.)

Management. Controlling the thrips vector is a key management strategy. Efforts to strongly suppress the thrips population will reduce the threat of viral infection. Eradicating weed hosts and making certain that other crops are not infected will reduce inoculum reservoirs. Scout regularly and destroy unnecessary flowers, so as to limit potential refuges and pollen sources for thrips. More information can be found in the introductory (▶ Chap. 4, "Insect Management for Disease Control in Florists' Crops").

4.3 Cucumber Mosaic Virus

Geographic occurrence and impact. *Cucumber mosaic virus* (CMV) infects a wide range of host plants including over 100 ornamentals (Korbin and Kaminńska 1998). It can cause problems in isolated greenhouses, but it is not as devastating as the tospoviruses.

Symptoms/signs. Cyclamen plants infected with CMV frequently look stunted and chlorotic, with mosaic patterns of yellow and green. Leaves can also be asymmetric (Kamińska 1975; Minuto et al. 2011).

Biology and epidemiology. CMV is a single-strand RNA virus that belongs to the genus *Cucumovirus* in the family *Bromoviridae*. It is transmitted by certain species

of aphids in the non-circulative (nonpersistent) mode, meaning the virus does not survive for long periods of time in the insect. Once the aphid has acquired the virus, it can transmit it to any susceptible host for a limited amount of time. Seed transmission of CMV also occurs, but data on seed transmission in cyclamen is not available. Transmission of CMV through seed can be as much as 50% in some plants (Gallitelli 2000).

Management. Management of viruses, including CMV and other species in the *Bromoviridae*, is based on monitoring and reduction of vector populations through chemical or biological measures, excluding vectors by the use of physical barriers such as nets or screens and eliminating alternate hosts – especially established, infected crops. Aphid populations are generally not tolerated during cyclamen production, so scouting to find the rare occurrences of the disease should be the cornerstone of managing this disease. Test kits (and diagnostic laboratory analysis) are available to test for the presence of CMV, allowing the grower to distinguish it from the more common thrips-vectored TSWV and INSV. Resistance to CMV has been introduced into a wide variety of vegetables through genetic modification. Additional information on integrated disease management of viruses may be found in introductory (▶ Chap. 4, "Insect Management for Disease Control in Florists' Crops").

4.4 Tobacco Mosaic Virus (TMV)

Geographic occurrence and impact. *Tobacco mosaic virus* (TMV) has a global distribution and can infect a wide range of plants. This virus is a single-strand RNA virus belonging to the genus *Tobamovirus* and family *Virgaviridae*. Outbreaks are rare and localized in cyclamen.

Symptoms/signs. TMV causes mosaic patterns on leaves.

Biology and epidemiology. TMV is easily mechanically transmitted and can persist in plant debris for 50 years.

Management. Inspect incoming stock and keep it segregated from older plants. Rogue out infected plants and practice strict sanitation. Workers should frequently wash hands and disinfest cutting tools. Powdered milk solutions may be used to inactivate TMV in some instances.

5 Nematodes Cyst [*Heterodera radicicola* (Greeff) Mull.], Root knot (*Meloidogyne* spp.), Stem and bulb (*Ditylenchus* spp.), Lesion (*Pratylenchus* spp.), and Foliar nematodes (*Aphelenchoides* spp.)

Geographic occurrence and impact. Different nematodes can attack cyclamen depending on region and cultivation practices. However, the movement by the

industry to use soilless potting mixes has essentially eliminated damage from soilborne nematodes (cyst nematodes, root-knot nematodes, lesion nematodes, and stem and bulb nematodes). Isolated outbreaks have been reported but seem to be associated with nonsterilized soil (Butcher 1934; Reimherr 1985). However, outbreaks of foliar nematodes (*Aphelenchoides* spp.) may still cause damage in cyclamen, although this has been rarely reported.

Symptoms/signs. Roots infected with nematodes can exhibit a range of symptoms depending on the pathogen. Aboveground symptoms appear as stunting and yellowing and in severe cases wilt and death. Root symptoms can also vary. Root-knot nematodes cause galls, while lesion nematodes cause stunted roots that have distinct reddish-brown lesions. Stem and bulb nematodes attack the corm and can cause wilt. Foliar nematodes (*Aphelenchoides* spp.) can invade leaves, leaf buds, and meristems. Due to cyclamen's thick, dense leaves, foliar nematodes do not show the clear interveinal demarcation that is commonly seen in plants with thin leaves. Instead, cyclamen infestation with *Aphelenchoides* shows as yellowish patches, which over time turn brown or blackish. The leaves dry up and often remain attached to the petioles. Affected plants are unmarketable.

Biology and epidemiology. Parasitic nematodes are microscopic worms that possess stylets for piercing plant cells and extracting nutrients. Different types of nematode may infect the roots, stems, or leaves of their host plants. Root-knot nematodes (*Meloidogyne* spp.) and cyst nematodes (*Heterodera* spp.) infect roots and establish feeding sites (nurse cells) that develop into visible galls and cysts, respectively. Root-knot and cyst nematodes do not migrate and are considered sedentary endoparasites. Lesion nematodes invade roots but migrate within the root system feeding on cells (migratory endoparasite). Stem and bulb nematodes (*Ditylenchus* spp.) invade at the soil line and migrate within the tissue (migratory endoparasite). Foliar nematodes (*Aphelenchoides* spp.) are also migratory endoparasites. Nematodes survive as eggs that overwinter in soil or can be introduced on host material. Handling and splashing water can spread the foliar nematode and stem and bulb nematode between crops or within an infested crop. The movement of infected crops or contaminated soil is usually necessary to spread the other kinds of nematodes.

Management. If unsterilized soil is used in the potting medium, the potential for root infection by nematodes is high. Growers should be vigilant in examining roots for stunting and poor growth. Although the use of soilless potting mixes will eliminate most soilborne nematode threats, foliar nematodes usually enter a production facility on other crop hosts. Careful examination of incoming plant material should be practiced. Common hosts of foliar nematodes are ferns and African violets (Daughtrey et al. 1995), as well as numerous herbaceous perennials.

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Diseases of Daffodil (*Narcissus*)

40

Gordon R. Hanks and Gary A. Chastagner

Abstract

Daffodils (*Narcissus*) are popular spring flowerbulbs in Europe, North America, Australia, and New Zealand, and tazetta narcissi are widely grown in China, Israel, and Japan. Large volumes of field-grown cut-flowers are traded, along with forced flowers and pot-grown plants from greenhouses, as well as the bulbs themselves. Basal rot (due to *Fusarium oxysporum* f. sp. *narcissi*) causes substantial losses, while the stem nematode (*Ditylenchus dipsaci*) is capable of devastating crops and is an ever-present concern; both are controlled by a hot-water treatment incorporating biocide and/or fungicide, but husbandry and cultural factors also play a major role. Biological control is in its infancy, but some progress is being made against basal rot using soil applications of composts that increase the population of beneficial soil microorganisms. Fungal foliar diseases reduce yields and can affect flower quality – smolder (*Botrytis narcissicola*), white mold (*Ramularia vallisumbrosae*), fire (*Botrytis (Botryotinia) polyblastis*), and leaf scorch (*Peyronellaea (Stagonospora) curtisii)* – and are controlled by a fungicide spray program. Bulb rots are also caused by species of *Penicillium* and *Rhizopus*. Instances of bacterial disease are rare, but *Pectobacterium (Erwinia) carotovorum* subsp. *carotovorum* attacks bulbs of tazetta narcissi. Daffodils are widely infested with multiple aphid-borne potyviruses, in particular Narcissus Late Season Yellows Virus, Narcissus Yellow

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R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_43

Stripe Virus, and (in tazetta narcissi) Narcissus Degeneration Virus, reducing bulb and flower yields and flower quality. Control of viruses is largely limited to crop inspection and the removal of affected plants.

Keywords

Basal rot • Biocide • *Botryotinia* • *Botrytis* • Daffodil • *Ditylenchus* • Fungicide • *Fusarium* • Hot-water treatment • *Narcissus* • Nematode • *Penicillium* • *Peyronellaea* • *Ramularia* • *Rhizopus* • *Stagonospora* • Virus

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1 Introduction

Plants of the genus *Narcissus* (daffodils and narcissi) were known in antiquity, mainly as medicinal plants (Dweck 2002). Although the Chinese sacred lily (*Narcissus tazetta* var. *chinensis*) was appreciated in the Far East and the Levant from much earlier times, in the sixteenth century daffodils were imported to the Netherlands from Constantinople and thence to England by horticulturally astute Huguenots (Willis 2012). A "daffodil revival" in the nineteenth century led to the start of substantial hybridization in the British Isles (with Peter Barr and the Royal Horticultural Society greatly raising the profile of the genus) and the Netherlands (notably at de Graaff's nursery): daffodils soon became a major floricultural crop. In the USA, the Plant Quarantine Act 1919 was extended in 1926 to ban the import of daffodils, creating a pre-emptive flood of imports, further hybridization, and a new crop for the Pacific Northwest (PNW) (Willis 2012). Currently some 4,000 ha of daffodils are field-grown in England (2008 figures; Defra/National Statistics 2009), 1,500 ha in the Netherlands (Bloembollenkeuringsdienst (BKD) 2016), 600 ha in Scotland (authors' estimate from Scottish Government (2015) and commercial information), and 370 ha in the USA (authors' estimate from USDA NASS (2016) and commercial information). Significant areas are also grown in Australia, Canada, New Zealand, Poland, and the Republic of Ireland and of mainly tazetta daffodils in China, Israel and Japan.

Much of the information here is derived from advisory publications in the Netherlands (Bergman et al. 1978; van Aartrijk et al. 1995; van der Zwet et al. 1990), UK (ADAS 1984, 1986; Hanks 2013a; Hanks and Linfield 1994; Lane 1984; Moore et al. 1979), and USA (Byther et al. 1998; Gould and Byther 1979; Pscheidt and Ocamb 2015). Further material derives from standard textbooks (Brunt 1995; Byther and Chastagner 1993; Chastagner and Byther 1985; De Hertogh and le Nard 1993; Hanks 1993a, 2002; Kamenetsky and Okubo 2013; Melville 1980; Rees 1972, 1992; Smith et al. 1988). Databases consulted include "Descriptions of Plant Viruses" http://www.dpvweb.net, "Distribution Maps of Plant Diseases"

http://www.cabi.org/dmpd/, "Fungal Databases" http://nt.ars-grin.gov/fungaldatabases/ (Farr and Rossman 2016), "Plant Viruses Online" http://srs.im.ac.cn/vide/refs.htm, and "Species Fungorum" http://www.speciesfungorum.org/Names/Names.asp. Note that the term "daffodil" is used to include all types of *Narcissus*: in most cases the reference is to the typical or mainstream cultivars of commerce, but when referring to a particular type of daffodil (e.g., tazetta species and cultivars) this is specified. Referred to by their active ingredient (a.i.) names, pesticides and other chemicals mentioned in the text are used to report the findings of research and trials – they are neither recommendations nor statements of current approval, availability, or legality.

2 Fungal Diseases

2.1 Basal Rot (Base Rot, Fusarium Rot) [*Fusarium oxysporum* f. sp. *narcissi* Snyder & Hansen (syn. *F. bulbigenum* Cooke & Massee)]

Geographic occurrence and impact. Basal rot is a major problem in daffodil-producing countries (Chastagner and Byther 1985; Moore et al. 1979; van Aartrijk et al. 1995) and probably wherever daffodils are grown. Basal rot has also been reported from China (Xu et al. 1987) and *Fusarium oxysporum* f. sp. *narcissi* has been reported on daffodils in Australia, Bulgaria, Canada, Germany, Mexico, New Zealand, South Africa, and Zimbabwe (Farr and Rossman 2016).

In the late-nineteenth century, daffodil bulb rot was a frequent subject of the horticultural press and attracted attention in the Netherlands and UK, especially in the exceptionally hot summer of 1911 when large numbers of bulbs rotted in both countries. Moore et al. (1979) provide an interesting history of the earlier investigations and how they were beset by poor interpretation and mischance, giving full references. Confusion was rife – was the rot caused by a nematode, a fungus, or something else? By the 1920s, UK researchers concluded that *Fusarium* was not involved or was at most a secondary phase of a nematode disease. Starting in 1926 large numbers of daffodils imported to the UK suffered from a rot that was apparently free of nematodes and similar findings emerged from Canada and the USA. In 1927–1932 research in the Netherlands, UK and USA finally showed that a *Fusarium* species – *F. bulbigenum*, now known to be *F. oxysporum* f. sp. *narcissi* – was the sole cause of basal rot, though it and stem nematode frequently occurred together. Weiss (1929, 1932) confirmed *Fusarium* as the cause of basal rot, and it was shown to be widely distributed, and of economic importance, across the USA, going on to investigate its management through hot-water treatment (HWT) or separate fungicide treatments. Similar conclusions were reached by others in the USA, Netherlands, and UK.

The impact of basal rot has become more severe since the 1970s, perhaps due to changes in husbandry. Many aspects of contemporary bulb-growing exacerbate

damage, such as reduced sorting by hand (more infested material planted), handling in bulk bins (rather than flats or trays), higher planting densities (20 t/ha rather than 10 t/ha), longer growing cycles (for 2 or more years instead of lifting, treating, and replanting annually), early lifting (before the bulb skins have "ripened"), bulb washing (when necessary to produce soil-free bulbs for export), and early planting (before soil temperatures have fallen) (Hanks 2013a). Now, increasingly robust restrictions and withdrawals of fungicides and biocides limit the opportunities for chemical management. Finally, basal rot is favored by warmer temperatures: once associated with warm summers, it now appears to be a permanent problem, perhaps exacerbated by climate change.

The obvious effect of basal rot is reduced bulb yield, but the health of the bulb crop naturally has consequences for cut-flower and pot-plant production. In the UK and USA, where most flowers are picked from field crops, their yield and health will be directly dependent on that of the field crop. Bulbs lifted and sold for flower forcing, pot-plant production and retail sales do not have the HWT afforded to bulbs intended for growing-on as field crops, and bulb rot sometimes becomes apparent a few weeks after dispatch, damaging the perception of the product. Gaps due to rotting bulbs will reduce the yield of forced flowers, exacerbated by greenhouse temperatures that favor the development of *Fusarium*. Where pot-grown daffodils are produced, the failure or poor appearance of one bulb will downgrade the whole pot.

Basal rot is a great threat to daffodil production because of its ubiquity and aggression and a long-term failure to find acceptable, effective methods for its management. In the UK, 30% losses in bulb yield have occasionally been known in seriously affected bulb stocks of basal rot-susceptible cultivars. Even when husbandry has been optimized in such bulb stocks over a number of years, it is difficult to reduce infection rates to below 2% (Hanks 1996). For calculating the benefits of effective basal rot management, an increase in yield of about 10% has been considered reasonable.

Symptoms/signs. After planting, depending on the extent of damage, roots may fail to emerge from the base rot or may emerge only to be affected later, becoming dark in color. In growth, affected bulbs produce short-lived, crooked shoots, or increasingly weak foliage that senesces prematurely several weeks before that of healthy plants. In the early stages, basal rot may be seen as a small dark spot in the base plate when the bulb is cut lengthways. The rot spreads through the base plate and then upwards through the bulb scales, which become characteristically moist and dark chocolate to reddish-brown in color – the color may vary with the cultivar (Fig. 1). It is important to distinguish these symptoms from those of stem nematode (brown rings when the bulb is cut through transversely) and the normal die-back of leaf bases (which does not show the typical leading edge of basal or neck rot). Once a significant portion of the bulb has been affected, its softness is apparent on handling. A pinkish-white spore and mycelium mass forms near the base plate (Fig. 2). As the bulb becomes dehydrated, perhaps aided by bulb mites moving in to feed on damaged tissue, it becomes brittle and mummified.



Fig. 1 Typical upwards progression of daffodil bulb rot due to *Fusarium oxysporum* f. sp. *narcissi* (University of Warwick © 2017. All Rights Reserved.)



Fig. 2 Daffodil bulb with basal rot showing eruption of mold from base plate (*Fusarium oxysporum* f. sp. *narcissi*) (University of Warwick © 2017. All Rights Reserved.)

Several weeks before bulb lifting, infected plants may already be obvious as their leaves die-down earlier than the rest. At lifting infected bulbs may have no sign of roots, and at or soon after lifting feel soft, especially around the base, and show a discolored area extending upwards if the outer scale is removed.

Biology and epidemiology. *F. oxysporum* f. sp. *narcissi* is found in most soils where daffodils have been grown and sometimes in soils where they have not, despite the pathogen being limited to this host (Apt 1958; Price 1975a, b). In soil the pathogen can remain viable for up to 10 years, at least under artificial conditions (CA Linfield, personal communication, 1990). Spores of both pathogenic and nonpathogenic strains have been found on healthy bulbs in storage (Price 1973). The disease can be bulb- or soil-borne and spread can occur in both stored and planted bulbs (Hawker 1940; Price 1977a). Spread of spores on air currents and via water (during bulb washing, dipping or HWT) would be expected. Variation in the pathogenicity of *F. oxysporum* f. sp. *narcissi* isolates was studied by Linfield (1997) and the cross-infectivity of its *formae speciales* by Rataj-Guranowska et al. (2007).

Control is important during the first months after planting as the pathogen is active only above 13 °C/55 °F (Langerak and Haanstra-Verbeek 1977). In planted bulbs, infection occurs when the integrity of the bulb surface is compromised: when new roots are emerging, when old roots are dying-down, or when there is mechanical damage (Gregory 1932; Hawker 1935; Langerak and Haanstra-Verbeek 1977; Price 1975a, b, 1977a). Root infection and disease development are closely related to soil temperature above 13 °C/55 °F, reaching a maximum at 29 °C/84 °F (McClellan 1952), explaining the finding that the base plate can be penetrated via the senescing roots in summer if the soil is wet (Hawker 1935, 1943a). Root senescence starts in late-May and lasts until September, corresponding to the time when temperatures favor the pathogen's active growth (Hawker 1943a; Price 1977a). Soil-borne infection is encouraged by high temperatures (Hawker 1935) and can occur late in the season when moribund roots are present (Hawker 1943a; Price 1977a), though it has also been reported in temperatures as low as 5 °C/41 °F (Price 1982).

Little infection was recorded when inoculum was placed 30 cm below bulbs (Price 1975c). When healthy bulbs were placed 0, 10, or 20 cm laterally from infected bulbs, 60, 27, and 6% of bulbs, respectively, were affected after one growing season (Linfield 1987). It has been suggested that because of the relatively sparse spread of daffodil roots and the high concentrations of chlamydospores needed to produce infection, bulb-borne inoculum is more important than soil-borne inoculum, especially with high planting rates, reduced sorting by hand, and 2-year-down growing (Price 1977a, b). While the pathogen can move only a little through soil, it can move along or in roots for some distance to infect bulbs (Price 1975a, b). The pathogen advances intercellularly before invading damaged cells (Bald et al. 1971).

The nutritional state of the soil influences infection. Using excessive N fertilizer increased the incidence of basal rots, splits, and bruises in daffodil bulbs (Biekart 1930; McClellan and Stuart 1947). Applying no more than about 120 kgN/ha was considered satisfactory (Rikhter 1976). In trials, bulbs of 'Golden Harvest' (basal rot-susceptible) and 'St Keverne' (resistant) were planted in soil amended with the equivalent of up to 300 kgN/ha, together with no, low or high rates of basal rot inoculum. Using a split-dose of 150 kgN/ha at planting and 150 kgN/ha top-dressed in April increased basal rot in 'Golden Harvest' and, in combination with the higher rate of inoculum, in 'St Keverne' also (Hanks et al. 1998).

The incidence of rotting bulbs, and the numbers of pathogen propagules isolated from the base rots of healthy bulbs, increased as storage temperature was raised from 15 to 24 °C/59 to 75 °F and then declined somewhat at 30 °C/86 °F (Price 1975a, b). When apparently healthy bulbs were taken from storage and grown for a year, the percentage rotting was similar in all treatments. Pathogenic isolates were found on the base rots of 21% of samples. Because of these effects of temperature, it is not surprising that storage of bulbs at 15 °C/59 °F after lifting prevents the development of many fungal lesions. The overall optimal temperature for bulb storage is 17 °C/62 °F; storage at 20 to 30 °C/68 to 86 °F should be avoided.

Management. Extension workers have long stressed that the available fungicide treatments alone are insufficient for managing basal rot and must be combined with good bulb handling and husbandry which exert a substantial effect in reducing the incidence of basal rot if used consistently (e.g., Hanks 2002, 2013a). The "physical" management of basal rot through correct bulb drying, storage, and HWT, cultural management through using appropriate planting sites, depth and timings, and the limited chemical controls available make an interesting example of integrated crop management (ICM), but one that needs to be extended by the development of biological controls and using the potential of plant breeding.

HWT, a key element in basal rot management, is even more important for the control of stem nematode (*Ditylenchus dipsaci*), such that it is routine to treat all daffodil stocks in the run-up to planting. Bulbs are immersed in a tank containing a biocide and/or fungicide for, typically, 3 h at 44.4 °C/111.9 °F. Because HWT is a key to managing stem nematode as well as basal rot, the general features of HWT (such as the temperature, duration and timing of treatment) are discussed in Sect. 7 to save repetition, and only information specific to managing basal rot is given here. HWT also contributes incidentally to the management of other daffodil pathogens (e.g., leaf scorch) and pests (e.g., narcissus flies (*Merodon equestris* and *Eumerus* spp.), bulb mites (*Rhizoglyphus* and *Histiostoma* spp.), and bulb-scale mites (*Steneotarsonemus laticeps*). Colloquially, HWT is referred to as "sterilizing," "bulb-dipping" or even "boiling," terms avoided in this chapter! In disease management short, bulb-dip treatments in water at ambient temperatures may also be used, and to distinguish such treatments from HWT, they are referred to as "cold dips."

- ***Cultural*** – Alongside their work on bulb-dipping, both Gregory (1932) and Hawker (1940) demonstrated the importance of appropriate bulb storage as a means of alleviating basal rot. Both recommended that bulbs should be stored at temperatures <25 °C/<77 °F (today 18 °C/64 °F would be preferred) and planted only when soil temperatures fell below those in storage; and they emphasized the need for rapid drying of bulbs following bulb-lifting and again after HWT. Hanks (2001) used basal rot-prone bulb stocks ('Carlton' and 'Golden Harvest') to evaluate four key elements of good bulb handling: (a) a postlifting thiabendazole bulb spray, (b) high-temperature first-stage drying at 35 °C/95 °F for 3 days, (c) second-stage drying and bulb storage at 17 °C/62 °F, and (d) HWT with thiabendazole and formaldehyde (formalin); the controls for each element were (a) no postlifting spray, (b) first-stage drying at ambient temperatures, (c) second-

stage drying and bulb storage at ambient temperatures, and (d) HWT with formaldehyde only. Six treatments were tested, bulbs receiving either all four treatments, treatment a, b, c, or d with its control regimes, or control regimes throughout. Prior to HWT, bulb samples were stored for 8 weeks at 25 °C/77 °F and assessed for rots: in treatments with 17 °C/62 °F storage in common there were few rotted bulbs; the remaining treatments had a high proportion of rotted bulbs. In growing-on for 2 y, the same treatments – those including 17 °C/62 °F storage – continued to give the best results, with higher bulb yields and more marketable bulbs.

Hanks (1992a) used a stock of 'Golden Harvest' with high levels of basal rot, imposing good bulb drying and storage regimes (b and c above) and adding either a postlifting cold dip in thiabendazole, HWT with thiabendazole (as well as formaldehyde), both of these, or neither. The bulbs were grown for four, 1-year cycles, the same treatments being applied each year. The cumulative yield increase of marketable bulbs over 4 years was 399% when thiabendazole had been applied only as a postlifting cold dip, 236% when thiabendazole had been applied post-lifting and in HWT, but only 136% when thiabendazole had been applied in HWT only. These results pointed to the great value of the prompt, postlifting cold dip, and showed a mildly phytotoxic effect from the annual double thiabendazole treatment. Interestingly, the treatment with no thiabendazole gave an increase of 249%, an effect attributed solely to the imposition of good physical handling.

Good cultural practices include many actions, summarized below (Byther and Chastagner 1993; Chastagner and Byther 1985; Gould and Byther 1979; Hanks 2013a; Moore et al. 1979). A long rotation – at least 8 years – is necessary between daffodil crops to reduce inoculum in the soil. Daffodils should be left down for no longer than 2 or 3 years, to reduce the build-up of diseases before HWT can be applied again. Warm sites and situations, such as south-facing slopes that expose bulbs to warm soils, should be avoided, and the bulbs should be planted relatively deeply to keep them cooler in summer. Using a relatively low planting density reduces bulb-to-bulb transfer of pathogens. Bulbs should be planted once summer soil temperatures have begun to fall and lifted early to avoid warming soils.

- ***Sanitation*** – It is important at all stages to avoid contamination by the sometimes durable and easily spread bulb pathogens. Bulb equipment, stores, and other facilities should be cleaned and treated with a suitable farm disinfectant before use (Lole 2001). Operations should be planned to reduce bulb damage during lifting and handling. After lifting, dry bulbs promptly at 17 °C/62 °F for as long as required, or at 35 °C/95 °F for 3 days followed by rapid cooling and continued drying at 17 °C/62 °F; aim to achieve first-stage drying (surface drying, especially of the bulb necks and base) quickly to reduce the spread of the pathogen. Storing bulbs at 17 °C/62 °F (not higher) has a major effect in reducing basal rot, but temperatures <17 °C/<62 °F may slow development. Where drying at 17 °C/62 °F or 35 °C/95 °F is not an option, try to keep the bulbs below 18 °C/64 °F by ventilation and shading. Maintain air circulation around the bulbs at all times, and

when drying and storing bulbs use the recommended air-flows, air exchange rates, and relative humidity (RH). Checking temperatures inside the mass of bulbs is useful, it may be warmer than the ambient air would suggest and a mass of warm bulbs cools slowly unless ventilated. Containers of bulbs should not be left in direct sunlight. HWT should be applied according to the best practice, cooling the bulbs rapidly afterwards and either drying and storing as previously described, or planting immediately.

Bulbs should be inspected at every opportunity, rejecting and destroying any that are soft, lightweight, or damaged. Infected bulbs returned to the soil, or their inclusion in tanks used for bulb dipping, are major sources of inoculum. Manual inspection of bulbs is slow and labor-intensive, requires experience, and may still be inaccurate. There should be potential to develop non-destructive, automated bulb sorting. Because healthy bulbs are denser than those with basal rot, a floater/sinker method of separation was suggested, but when tested proved inaccurate (Tompsett 1976). Hills (2011) evaluated a prototype MRI sensor on a mixture of healthy and diseased bulbs. Under ideal conditions – with the bulbs stationary and using multiple scans per bulb – the method gave a very reliable measure of the internal condition of bulbs as seen when they were subsequently cut through. But developing an apparatus capable of making a single-scan assessment on bulbs moving along a conveyor would be challenging.

- ***Fungicides, biocides and hot-water treatment*** – Experiments by Gregory (1932) and Hawker (1935, 1940, 1944) established the value of adding formaldehyde to the water of the HWT tank to control the basal rot pathogen. Later, as well as formaldehyde, a fungicide was added to the HWT tank to improve the control of fungal pathogens (see below for fungicides). In the USA, however, formaldehyde is considered more important for basal rot control than for stem nematode control, because nematicides are available for application in the field. HWT for 4 h at 44 °C/111 °F without formaldehyde was not sufficiently robust to control the spread of basal rot from diseased to healthy bulbs in the tank, although it did reduce the number of fungal colonies of *F. oxysporum* f. sp. *narcissi*, *Penicillium* species, and *Mucor plumbeus* recovered from bulbs (Qiu et al. 1993). Similarly, in trials where a chlamydospore inoculum of *F. oxysporum* f. sp. *narcissi* was added to the treatment tanks and the water sampled for viable inoculum, the number of spores recovered from HWT tanks with plain hot water was reduced by almost 99%, but some viable spores were recovered even after a 4 h treatment (GA Chastagner, unpublished data). Hence, the high temperature used in HWT does not, per se, offer complete control of the basal rot (and other fungal) pathogens. To achieve total control, longer and (or) hotter HWT would be needed, which is impractical; alternatively (if available) formaldehyde or another suitable biocide should be added to the tank, or the survival of a small percentage of pathogen propagules has to be accepted.

  Other biocides were evaluated as alternatives to formaldehyde for controlling the basal rot pathogen. In the UK, peroxyacetic acid and glutaraldehyde were effective at HWT conditions for controlling the basal rot pathogen and were crop-safe (Hanks and Linfield 1997, 1999; Lole 1990) but did not become available for

bulb growers. Lole et al. (2010) evaluated several biocides for their potential in HWT and cold dips against basal rot chlamydospores in vitro. At 18 °C/64 °F, it was found that ascorbic acid, citric acid, didecyldimethyl-ammonium chloride, chlorine dioxide, and a silicone wetter were ineffective or only partly effective, while an iodophor disinfectant ("FAM 30") and hydrogen cyanamide each achieved a 100% kill, but hydrogen cyanamide was found to have phytotoxic effects on daffodils. The crop safety of "FAM 30" was confirmed in field trials, with the proviso that higher concentrations of "FAM 30" slightly reduced yields in the year after treatment, with growth compensating in the next year (Hanks 2011a), and it has been used in the UK instead of formaldehyde since 2010. "FAM 30" does have some disadvantages, however (Sect. 7.3), and a more suitable biocide is still needed.

Chlorine dioxide is a biocide used in a number of agricultural production systems to prevent the spread of human and plant pathogens in water (Copes et al. 2014). It was shown by Chastagner and Riley (2002, 2005) to be very effective in HWT against the basal rot and other fungal pathogens, and better than sodium hypochlorite, iodine, quaternary ammonium compounds, glutaraldehyde, and phenols. Because of this body of research, when restrictions were placed on the agricultural use of formaldehyde in the USA in the 1990s, growers were able to add chlorine dioxide to their HWT tanks instead. Chlorine dioxide was used for several years without any observed crop toxicity or other issues, but once the registration and availability issues of formaldehyde were resolved growers generally returned to using formaldehyde because it was less costly. The evidence from Chastagner and Riley's (2002, 2005) work on chlorine dioxide outweighed the negative result reported by Lole et al. (2010), and the practicalities of using chlorine dioxide in HWT were subsequently investigated on-farm in the UK (Hanks 2011b, 2013b). When its concentration was maintained at ca. 5 ppm no practical difficulties were encountered, the substantial bioload already present in the HWT tanks was effectively neutralized, and treated stocks of two daffodil cultivars showed no adverse effects over the next 2 years when grown alongside controls treated with "FAM 30." Unfortunately, circumstances prevented the measurement of bulb yields, so further research is needed.

Although several other fungicides have shown useful control of the basal rot pathogen, in the UK thiabendazole has been the most consistently effective (e.g., Hanks and Carder 1998; Hanks and Linfield 1994, 1996); thiabendazole has been the material of choice in HWT and cold-dips for many years. In use, however, thiabendazole raised concerns about the amount of a.i. precipitated on the tank floor despite the usual vigorous HWT circulation. The initial recommended concentration of 1,100 ppm a.i. fell to ca. 200 ppm during a 3 h-HWT (concentrations here refer to the combined amount of thiabendazole in solution and suspension). Because thiabendazole is only slightly soluble at a pH >4, an acidifier, sodium bisulfate, was added to the tank to maintain a pH between 2.5 and ca. 3.0 (Hanks and Carder 2000, 2004). Initially sodium bisulfate was added at 1.38 kg/1,000 L water and thereafter it was topped-up after each HWT period to maintain the required pH; after 5 days of use, 72% of the original a.i. remained

in the tank. In 2008, restrictions were imposed on the bulb-dip use of thiabendazole fungicides in the UK, including a cut of 75% in the approved rate, the restriction to one use per year per crop, and withdrawal of use in the Isles of Scilly, UK. Trials, however, indicated that the reduced rate of thiabendazole remained effective against basal rot, and it was advised that sodium bisulfate be added routinely as described. In the Netherlands, Vreeburg and Korsuize (2014) investigated fungicide alternatives to formaldehyde for increasing bulb yields and managing Fusarium bulb rots (tested with 'Dutch Master'), "skin diseases" ('Barrett Browning') and *Penicillium* ('Tête-à-Tête') in HWT. Captan and combinations of prochloraz + thiophanate-methyl appeared to be effective replacements for formaldehyde with respect to bulb yield and skin quality (though captan may carry some exposure risks), whereas they were ineffective against Fusarium rots.

At the time of writing, only thiabendazole, chlorothalonil, and a formulation of fludioxonil + cyprodinil are approved in the UK for use in bulb dips (HWT and cold-dips), though each has its problems. These fungicides and others were tested by Clarkson and Hanks (2012, 2014). It was shown that thiabendazole may have limited use because resistant strains of the pathogen were common, having been first recorded in 1991 (Carder 1999b). The effectiveness of chlorothalonil was poorer than expected. No efficacy data are presently available for fludioxonil + cyprodinil. In vitro tests and small-scale practical trials of tebuconazole and of prochloraz, which had been previously approved for this purpose, were more effective than either thiabendazole or chlorothalonil, while copper oxychloride, although less effective, nevertheless had a useful effect and has the advantage of being from a completely different mode-of-action group. To date it has not been possible to gain approvals from the regulatory authorities in the UK for these uses. There is concern that the use of pesticides in large volumes of hot water will deter the agrochemicals industry from supporting applications of this type.

As a biocide is used, fungicides are not generally added to the HWT tank in the PNW, though bulbs are sometimes given postharvest or preplant fungicide dips (see below). There are very few products registered for use as bulb dips, with registrants concerned about disposal issues. The limited amount of product that would be sold for this use is another reason why registrants may be reluctant to register products.

Where a basal rot problem is serious enough and the stock valuable enough to justify the collateral damage caused, it may be more effective to lift an affected bulb stock early and follow this by prompt HWT (ADAS 1974; Millar 1976; Price and Briggs 1976).

- ***Fungicides and biocides applied other than in HWT*** – There are other options, besides HWT, for the application of fungicides and biocides. Hawker (1940) showed that postlifting cold dips with formaldehyde were effective, sometimes adding a fungicide, and cold dips were developed further in the UK and USA (Miller and Gould 1967; Wallace 1940); later, benzimidazole fungicides were used (Gould and Miller 1970, 1971a, b). An optimal treatment was a 15-min dip within 1–2 days of lifting, using formaldehyde, a fungicide or both, and this was

favored by many growers as a simple, effective treatment. While fungicides are not generally added to the HWT tank in the PNW, bulbs are sometimes given postharvest or preplant fungicide dip treatments, often a tank-mix of captan and thiabendazole. "FAM 30" is generally effective against basal rot chlamydospores and will, to an extent, control basal rot in bulbs as a 15–25 min cold dip though the effectiveness of a short, cold dip could not be guaranteed. Cold, 15-min dips in thiabendazole will also generally kill basal rot chlamydospores, but it was found that where spores were clumped together, it was not always effective (Wedgwood 2010).

Fungicides have also been applied to daffodil bulbs as on-line postlifting sprays over the bulbs soon after lifting when they are on conveyor belts *en route* to the drying facility. Using a thiabendazole fungicide, this was reasonably effective using either flat belts or a roller table, despite achieving a less-than-uniform application because daffodil bulbs do not turn readily (ADAS 1987; Hanks 1992a). As well as conventional, high-volume spray nozzles, low-volume ultrasonic or electrostatic applicators were as effective (MSG-Agvet and GR Hanks, unpublished data, 1992). Anecdotally, over-bulb fungicide sprays have been used as the bulbs are lifted or re-planted, but this does not appear to have been critically tested.

Might fungicides be applied to control basal rot as a conventional crop spray in the field? Few trials appear to have been done, and none has produced any evidence of beneficial effects. In UK trials, crops were sprayed up to seven times with a benomyl fungicide at 2-week intervals, inoculated with *F. oxysporum* f. sp. *narcissi*, and stored before assessment (ADAS 1983; Tompsett 1972). None of the treatments reduced basal rot development. In other work, crops were drenched with benomyl 3 weeks before lifting (Briggs 1973). Drenching failed to control basal rot, unlike postharvest and HWT applications.

With few fungicides to choose from, there is little opportunity for daffodil growers to develop strategies for combating fungicide resistance. The use of thiabendazole, the fungicide of choice, is currently restricted in the UK to once per year. Growing daffodils for at least 2 years-down, the fungicide used in HWT could be alternated after each growing cycle. If using two treatments per year, different fungicides could be used postlifting and in HWT.

- ***Biological controls*** – Some progress has been made towards the biological control of basal rot in daffodils. Following successful pot-based trials using microorganisms antagonistic to the basal rot pathogen (Beale and Pitt 1990), granular formulations of a *Trichoderma* species, *Minimedusa polyspora*, a *Streptomyces* species, and *Penicillium rubrum* were field-tested. Bulbs of the susceptible cultivar 'Golden Harvest', which had previously received (or not received) thiabendazole in HWT, were sprinkled with granules after placing in furrows and then covered. After 2 years, there was a small reduction in the number of bulbs developing basal rot in some biocontrol treatments, the more so where thiabendazole had also been given. But the effects were too insubstantial to warrant commercial development (Hanks and Linfield 1997; Pitt 1991).

Subsequently, commercial biocontrol agents have been registered for use on a range of crops for controlling soil-borne plant pathogens, and it is known that soil application of organic amendments such as composts can increase the soil population of beneficial microorganisms such as naturally occurring *Trichoderma* species. In a daffodil pot experiment, Noble (2015) tested several biocontrol agents as granules, preplanting bulb sprays or dips, or by addition to green waste compost. None of the compost treatments, with or without biocontrol agents, affected the frequency of basal rot symptoms, but granules of two biocontrol agents and a bulb spray application of another significantly reduced basal rot and significantly increased the weight of healthy bulbs. These materials are yet to be tested on a field scale.

- ***Cultivar selection and plant breeding*** – It is unfortunate that some of the most popular, typical large-flowered yellow daffodils, such as 'Carlton', 'Dutch Master', 'Golden Ducat', and (particularly) 'Golden Harvest', are susceptible to basal rot. Many UK growers have altered their portfolio of cultivars, taking-on the newer 'Rosewarne hybrids', though even recently 'Carlton' still accounted for some 25% of the bulbs produced (UK growers, personal communication, 2014). In the Netherlands, 'Golden Harvest' and 'Carlton' now account for only 0.3 and 1.7% of the Dutch daffodil area, respectively, compared with 3 and 9% 15 years ago, the area being largely taken up by the dwarf cultivar 'Tête-à-Tête', used for pot-plant production on a large scale and expanding from 26 to 39% of the area over the same period (BKD 2016; Productschap Tuinbouw (PT)/BKD 1999).

  The 'Rosewarne hybrids' were bred at Rosewarne Experimental Horticulture Station (EHS) in the 1970s and 1980s, using especially 'St Keverne', also 'Malvern City', 'Toorak Gold', and 'Dawley', selected for their tolerance to basal rot and early flowering in the field (Fry 1975; Hanks 2013a; Pollock 1985; Tompsett 1984). The tranche released over 1979–1989 included many typical yellow trumpet and long-cup cultivars such as 'Armynel', 'Banallan', 'Barenwyn', 'Dellan', 'Emblyn', 'Feena', 'Jedna', 'Kerensa', 'Millgreen', 'Rosenwyn', 'Talwyn', 'Tamara', 'Tamsyn', and 'Veryan', as well as more varied species × cultivar crosses. Many of these are now growing in commercial quantities, but some recent indications have confirmed an impression that the tolerance of these cultivars is breaking down (Hanks and Withers 1998; UK growers, personal communications, 2010). The mechanism controlling resistance to basal rot is not well understood, though under some circumstances even normally highly resistant cultivars may succumb to basal rot, such as 'St Keverne' growing in a high-N substrate (Hanks et al. 1998) and in supposedly resistant poetaz cultivars (Hargreaves and Lyon 1977). From a study of 'Golden Harvest' and 'St Keverne', Nicholson et al. (1989) concluded that disease-resistance involves an interaction between the accumulation of antifungal compounds, cell wall modification, and the formation of lignituber-like structures. A better understanding of these mechanisms is needed, including the development of a nondestructive test for determining resistance or susceptibility, for which attempts so far have been unsuccessful (Carder 1999b, 2003b).

2.2 Fire [*Botrytis polyblastis* Dowson (Teleomorph: *Botryotinia polyblastis* (Greg.) Buchw.) (syn. *Sclerotinia polyblastis* Greg.)]

Geographic occurrence and impact. Fire occurs on the Pacific coast of the USA and across the bulb-producing areas of the British Isles (references in Moore et al. 1979). Fire also occurs in the Netherlands, where *Engels vuur* seems to be a more recent problem (van Aartrijk et al. 1995). Before daffodils were picked and marketed in tight bud, fire was prevalent in south-west England, where 'Grand Soleil d'Or' and other tazetta cultivars grown in the mild, humid climate of the Isles of Scilly were most susceptible to attack and mainstream cultivars less so. Doucette's (1953) list of resistant cultivars included the five most widely grown in south-west England at that time ('Carlton', 'Golden Harvest', 'King Alfred', 'Minister Talma', and 'Rembrandt') (Moore et al. 1979).

Its limited distribution, increased prevalence of tolerant cultivars, and changes in the flower-picking stage mean that fire is now of less concern in the UK than other foliar diseases (leaf scorch, smolder and white mold). In the PNW, fire is infrequent, probably due to the limited number of susceptible cultivars grown and the fact that very few flowers are left unharvested in the field, limiting the number of primary infection sites. But infection might be a problem if, for some reason, a crop was not picked or de-headed (Moore et al. 1979), and it has been reported that, uncontrolled, fire can destroy a crop in 3–6 weeks (Chastagner 1983). Given that most flowers are now picked while in tight bud, when fire occurs its greatest impact is a reduced yield of bulbs due to the premature death of the foliage.

Symptoms/signs. In late-winter/spring, spores from germinating sclerotia infect the flowers of whatever daffodils are out at the time, producing water-soaked spots that turn brown. As the flowers wither, spore masses are produced and infect the foliage, resulting in small, elliptical, pale brown spots, often near the leaf-tips and accompanied by yellow streaking on either side of the lesions (Fig. 3). As with the other fungal foliar diseases of daffodil (leaf scorch, smolder, and white mold), fire results in premature die-back of the foliage, reducing bulb and subsequent flower yields, clearly seen in the absence of a fungicide spray program) (Fig. 4).

Biology and epidemiology. The primary inoculum is the ascospores produced by apothecia arising from overwintered sclerotia of *Botryotinia polyblastis*. Ascospore infection is limited to flowers. The *Botrytis* stage of the pathogen then sporulates on infected flowers, the conidia being spread via wind to the foliage. Additional conidia are produced on the infected foliage, resulting in rapid disease build-up. As infected leaves die-down under warmer, drier conditions, sclerotia (black, 3–8 mm long, flattened, elongated-oval and embedded in the leaf tissues) are produced in the leaf spots (Moore et al. 1979). Sclerotia overwinter in soil or leaf debris and bulbs do not become infected (Moore et al. 1979). Gregory (1938, 1941) established the link between *Botrytis polyblastis* and its perfect form, *Botryotinia* (or *Sclerotinia) polyblastis.* This is a host-specific species that produces large conidia (30–60 μm in diameter) with multiple germ tubes at germination.



Fig. 3 Daffodil leaf symptoms due to fire, *Botrytis polyblastis* (Ralph S. Byther © 2017. All Rights Reserved.)



Fig. 4 Daffodil crop showing early foliage senescence in the front half of field, due to inadequate fungicide program; far side of field with regular spray program and foliage still green (Gordon R. Hanks © 2017. All Rights Reserved.)

Management. Where necessary fire is managed by growing-season fungicide sprays. As fire is not carried in the bulb, bulb dip treatments are inappropriate. Cultural practices and sanitation are important. As far as is known biological controls for daffodil fire have not been reported.

- ***Cultural practices*** – Debris from flowers is the main source of infection, so de-heading or flower picking are the main controls, along with crop rotation and general sanitation.
- ***Fungicides*** – At one time, daffodil fire control and improved bulb and flower yields were achieved using post-flowering Bordeaux mixture sprays on 'Grand Soleil d'Or' (Gregory and Gibson 1946). Current information relating to the fungicidal control of fire is limited. Foliage can be protected from infection with a single application of fungicide applied immediately after flowering. While there are many fungicides used to control other *Botrytis* diseases, many are not very effective against *Botrytis polyblastis*. In the PNW, the most effective materials included benomyl and vinclozolin (Chastagner 1983). A later trial indicated that iprodione was the most effective fungicide. Applications of cyprodinil plus fludioxonil, chlorothalonil, fludioxonil, cyprodinil, pyraclostrobin, and mancozeb reduced disease development, but were less effective than iprodione (Chastagner and DeBauw 2011a). In the UK, control of fire relies on "generic" fungicide spray programs described at the end of the section on smolder (Sect. 2.6).

2.3 Leaf Scorch [*Peyronellaea curtisii* (Berk.) Aveskamp, Gruyter & Verkley (syn. *Stagonospora curtisii* (Berk.) Sacc.)]

Geographic occurrence and impact. The fungus first collected from daffodils in the USA and later called *Stagonospora curtisii* was shown to be responsible for leaf scorch in the Netherlands, Hungary, and England between 1901 and 1926 (references in Moore et al. 1979). In 2010, it was placed in the genus *Peyronellaea* (Aveskamp et al. 2010). A widespread disease in daffodil-growing countries, leaf scorch occurs in many parts of Europe, North America, Australia and New Zealand. It is associated with warmer areas such as the south-west of England, where the climate is relatively mild with few frosts (Moore et al. 1979).

In the UK, leaf scorch has caused significant damage in some years since 1927 but rarely other than in south-west England (Moore et al. 1979). Leaf scorch damages flowers by causing unsightly brown spotting and can develop on apparently healthy flowers during storage and distribution, rendering them worthless, so leaf scorch is of serious concern where crops are grown primarily for flowers. As with other leaf diseases, when the loss of green-leaf area (GLA) is extensive and swift, bulb yields and subsequent flower yields will be reduced. Leaf scorch is exacerbated by growing the crop for 2 or more years between lifting, causing a build-up of inoculum in the bulbs, debris, and soil, but it is seldom damaging in the Netherlands where more 1-year-down growing is practiced. Most cultivars are attacked, but it is



Fig. 5 Reddish leaf-tip lesions of due to leaf scorch of daffodil, *Peyronellaea curtisii* (Gordon R. Hanks © 2017. All Rights Reserved.)

most common on 'Grand Soleil d'Or' and 'Magnificence'. Leaf scorch attacks many other ornamental amaryllids (including *Amaryllis*, *Crinum*, *Galanthus*, *Hippeastrum*, *Nerine*, *Sprekelia*, and *Sternbergia*).

Symptoms/signs. Soon after emergence, damage appears as scorched or reddish-brown leaf tips, sometimes mistaken for frost or herbicide damage (Fig. 5). Lesions may also occur further down the leaf. Damaged areas are usually separated from healthy tissue by a yellowish zone. In damp weather, the lesions spread downwards, producing eye-shaped spots. The lesions can turn scabby and coalesce and rapid foliar die-back can occur, the lesions remaining visible even in withered leaves (Figs. 6 and 7). Within the lesions, many minute pycnidia appear, just visible to the unaided eye as brown or dark-brown swellings just below the leaf surface, a useful diagnostic feature distinguishing leaf scorch from fire (Fig. 8). The pycnidia exude large numbers of spores, in turn producing water-soaked spots on leaves, flower stalks, spathes, and flowers. As the disease continues to develop after flower picking the flower crop can be ruined, but in daffodils forced in the greenhouse leaf scorch is not usually progressive (Moore et al. 1979).

Biology and epidemiology. In the earlier studies, spores recovered from affected plants were very variable, which may have led to the confusion of names applied to a single causative fungus. Since the name leaf scorch was applied (Pethybridge 1929), its etiology and epidemiology have been well described (Beaumont 1950; Creager



Fig. 6 Leaf scorch of daffodil (*Peyronellaea curtisii*) showing spread from leaf-tip lesions (Ralph S. Byther © 2017. All Rights Reserved.)



Fig. 7 Leaf scorch of daffodil (*Peyronellaea curtisii*) showing loss of green-leaf area (Gary A. Chastagner © 2017. All Rights Reserved.)



Fig. 8 Leaf scorch (*Peyronellaea curtisii*) lesion on daffodil showing the minute pycnidia (Gary A. Chastagner © 2017. All Rights Reserved.)

1933; Feekes 1931). The pathogen persists in both the bulb neck and leaf debris. In the bulb neck, the pathogen lies between the fleshy scales or between the outer fleshy scales and the dry, papery "skin," and shoots are infected as they grow out. Spores from the pycnidia are spread by rain-splash (Moore et al. 1979). The fungus in the bulb neck can also spread down into the bulb, but is not generally regarded as causing bulb rot – though this has been disputed (Bergman and Noordermeer-Luyk 1974). Studies on the etiology and control of leaf scorch were published by Bergman and Muller (1965) and Bergman and Noordemeer-Luyk (1974).

Management. Although cultural practices and sanitation are likely to be important, they do not appear to have been tested experimentally for their effect on leaf scorch, and its management is largely through a fungicide spray program and HWT. The development of biological controls against leaf scorch is at an early stage. Saniewska et al. (1993) demonstrated that a *Bacillus* strain isolated from *Hippeastrum* was antagonistic to *Peyronellaea curtisii* and other plant pathogens in culture and could inhibit the development of leaf scorch symptoms caused by *Peyronellaea curtisii* on narcissus bulbs. Chen et al. (2003b) reported that strains of *Bacillus subtilis* and *Pseudomonas fluorescens* inhibited the growth of *Peyronellaea curtisii* in culture, while the liquid culture supernatant from the *Pseudomonas* strain could suppress leaf scorch caused by *Peyronellaea curtisii* in detached leaves of *Narcissus tazetta*. It was shown that bacteria of these strains could colonize and be recovered from the root system of *N. tazetta*.

- ***Fungicides*** – Tompsett (1981) reviewed the effects of HWT and fungicide sprays on leaf scorch and white mold carried out largely with 'Grand Soleil d'Or' on the Isles of Scilly since 1967. There were some beneficial effects on disease levels, senescence dates or bulb and flower yields, though all did not necessarily occur together. Formaldehyde alone or with benomyl reduced the inoculum carried on bulbs, eliminating leaf scorch symptoms in first-year crops. Effective foliar fungicides included Bordeaux mixture (which has a number of issues around cultivar tolerance and downgraded leaf appearance), zineb, benomyl, carbendazim, chlorothalonil, and captafol. Since then, many pesticides have been superseded or withdrawn, and some impose harvest intervals (HI) unsuited to flower-picking. Although there have been no recent trials on leaf scorch control in the UK, O'Neill (2013) considered the options for extrapolating the results of trials on the management of *Stagonospora nodorum* on wheat, suggesting that fungicides likely to provide good control of daffodil leaf scorch include azoxystrobin, boscalid + pyraclostrobin, boscalid + epoxyconazole, and pyraclostrobin, with some control also from chlorothalonil. Guideline spray programs for use against leaf scorch, smolder, and white mold, based on one or two sprays before flowering and three or five sprays postflowering, were suggested: they incorporated chlorothalonil (pre- or post-flowering), boscalid + pyraclostrobin (preflowering only), and (post-flowering only) tebuconazole, pyraclostrobin or boscalid + epoxiconazole.

2.4 Neck Rot [*Fusarium oxysporum* f. sp. *narcissi* Snyder & Hansen (syn. *F. bulbigenum* Cooke & Massee), and Other Fungi]

Geographic occurrence and impact. Neck rot has been recognized in the UK since the 1930s (Hawker 1935) but has caused serious concern only since the 1980s, perhaps because it was then seen more often as a result of cutting through bulbs as part of pre-export bulb inspections. In the Netherlands, top-rot (*koprot*) appears similar but is associated with *Botrytis narcissicola* and *Peyronellaea curtisii* (van Aartrijk et al. 1995).

Symptoms/signs. Neck rot symptoms usually start after harvest and can be seen in the remains of the old flower stalk when the bulbs are cut through. The rot can progress downwards, even invading the base plate, with the tissues becoming dark-chocolate to reddish-brown as in the case of basal rot (Fig. 9). Eventually the new shoot is attacked, and seriously affected bulbs become blind or produce weak shoots. Neck rot appears to affect a wider range of cultivars than basal rot (Price 1982). In a nonspreading forms of the condition, the damaged tissue may become dry and ginger or brown in color (Fig. 10) (JMLl Davies, personal communication, 1990).



Fig. 9 Daffodil bulbs showing examples of the downward progression of neck rot caused by *Fusarium oxysporum* f. sp. *narcissi* (University of Warwick © 2017. All Rights Reserved.)

It is important to distinguish neck rot from so-called "physiological neck rot," which, in the UK, was sometimes considered to have been mistaken for true neck rot, causing bulbs to fail inspections (Fig. 11). On lengthwise cutting of affected bulbs, true neck rot is seen as a rot spreading downwards from the bulb neck with a leading edge typical of a fungal rot; physiological neck rot is simply the presence of the dead bases of leaf laminae in the bulb neck.

Biology and epidemiology. Neck rot is often associated with the basal rot pathogen *F. oxysporum* f. sp. *narcissi*, and in this case neck and basal rot can be considered expressions of the same disease ("Fusarium rots"); other data suggest that neck rot can be due to a complex of pathogens. Thus, neck rot can be induced by inoculating the bases of senescing leaves with spores of *F. oxysporum* f. sp. *narcissi* or plain water, the latter implying there is a predisposition to the condition (Price 1982). But other fungal pathogens, including *Penicillium hirsutum* Dierckx (syn. *Penicillium corymbiferum* Westling), *Botrytis narcissicola* Kleb., and

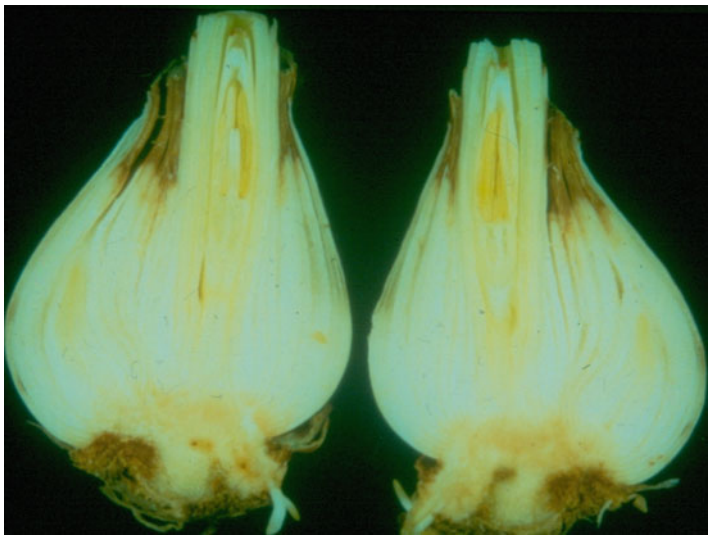


Fig. 10 Gingery, nonspreading form of neck rot of daffodil bulbs (University of Warwick © 2017. All Rights Reserved.)



Fig. 11 Natural die-back of tissues in the neck of a daffodil bulb, sometimes referred to as "physiological neck rot" (University of Warwick © 2017. All Rights Reserved.)

Peyronellaea (*Stagonospora) curtisii* (Berk.) Sacc., have also been associated with neck rot, as have "greasy skin" condition and mites (Carder 1999a, 2003a; Chastagner and DeBauw 2011b; Davies 1994; Davies et al. 1998; van Aartrijk et al. 1995).

A low incidence of neck rot was induced by inoculating defoliated, damaged shoots with high concentrations of spore and mycelia suspensions of *F. oxysporum* f. sp. *narcissi*, *B. narcissicola* or *P. hirsutum*, the *Fusarium* being the most virulent (Hanks and Carder 1998). Another study considered the incidence of the same pathogens in neck- and basal-rot tissue (Davies 1994; Davies et al. 1998). *Fusarium*, *Botrytis* and *Penicillium* were found in 51, 76, and 92%, respectively, of 101 bulb stocks sampled. The cultural factors that may have favored the incidence of these rots were examined through a questionnaire, but this revealed few useful associations between these factors and the incidence of pathogens.

Management. All the procedures used to manage basal rot, such as correct bulb drying, postlifting fungicide application and HWT, should be applied (Sect. 2.1). Cultural practices are also important.

- ***Cultural practices*** – A survey of husbandry and bulb handling practices associated with neck rot showed that it was associated with flailing-off foliage in advance of bulb lifting and flailing-off too deeply (Linfield 1990). Both practices, which produce tissue damage and so enhance the likelihood of infection, should be avoided.
- ***Fungicides*** – Hanks and Linfield (1996) evaluated a number of fungicides in vitro against the neck rot pathogens *F. oxysporum* f. sp. *narcissi*, *B. narcissicola*, and *P. hirsutum*. Captan, imazalil, prochloraz, and thiabendazole were effective, while fludioxonil, guazatine, and quintozene were ineffective against *F. oxysporum f.* sp. *narcissi*. These fungicides, plus chlorothalonil and iprodione, were applied as postlifting online bulb sprays or in HWT (bulbs receiving post-lifting sprays also received HWT but with formaldehyde only), and the bulbs were assessed over a 2-year period. As postlifting sprays, all except iprodione gave some benefits in terms of reduced bulbs with rots and higher bulb and flower yields. With HWT applications, the benefits were small, but all except iprodione and imazalil had positive effects. A cocktail of prochloraz and a reduced rate captan and thiabendazole was also tested in HWT, but resulted in short flower stalks and reduced flower and bulb yields. This test was repeated at a milder HWT regime of 2 h at 43.5 °C/110.3 °F, and the adverse effects were much less. Caution is needed when using fungicide mixtures in HWT.
  A further trial with *F. oxysporum* f. sp. *narcissi* involved different types of inoculum, inoculation timing, applications of benomyl during the growing season, different flailing depths to remove foliage prior to harvest, and a postharvest cold-dip in thiabendazole and formaldehyde (Hanks and Carder 1998). A higher incidence of neck rot occurred when (a) *Fusarium* inoculation was applied early (April and May), (b) flowers were not cropped, (c) plots were sprayed with benomyl fungicide, (d) foliage was deeply flailed-off, (e) the cut shoots were exposed to *Fusarium* inoculum, and (f) no postlifting fungicide dip was applied. The postlifting fungicide dip reduced the incidence of bulb rots by 80–90% and

could be adopted where neck rot is a problem. The trial also emphasized the need for less damaging flailing-off of foliage. Other results were less easy to explain: possibly leaving flowers intact (rather than having cut-stems dry-out some weeks earlier) and using a prolonged fungicide program (delaying foliar senescence and the development of the abscission zone) resulted in softer foliage at lifting, facilitating pathogen entry.

2.5 Penicillium Rot (*Penicillium* spp.)

Geographic occurrence and impact. *Penicillium* species are widespread opportunistic pathogens that cause mold on daffodil bulbs. Infection occurs through wounds and mechanical damage. For example, *P. corymbiferum* occurs on damaged tissues after lifting where the outer scales are torn or where offsets have recently been removed.

For many years, *Penicillium* species on daffodil bulbs was regarded as saprophytic, but aggressiveness varies by species (Dugan et al. 2014). Disease development can occur rapidly under damp conditions (Figs. 12 and 13). Dwarf cultivars such as 'Tête-à-Tête' are prone to *Penicillium* and other fungal pathogens, which could be potentially serious because of the large scale on which this cultivar is



Fig. 12 General rot of daffodil bulb caused by *Penicillium* sp. (University of Warwick © 2017. All Rights Reserved.)



Fig. 13 Surface growth of *Penicillium* sp. on daffodil bulbs under poor storage conditions (Gordon R. Hanks © 2017. All Rights Reserved.)

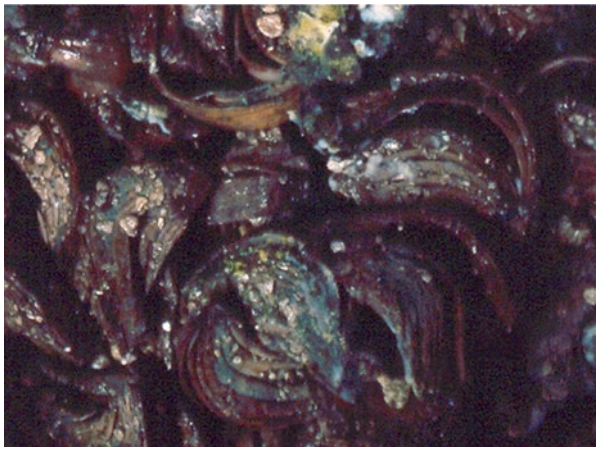


Fig. 14 Damage due to *Penicillium* sp. during daffodil propagation by "chipping" (University of Warwick © 2017. All Rights Reserved.)

grown. *Penicillium* is a particular problem in bulb propagation ("chipping"; Fig. 14) (Price and Linfield 1982).

Symptoms/signs. The outer scales of daffodils often show blistery markings when bulbs are kept in moist conditions, and species of *Penicillium* are commonly associated with this symptom, often with no adverse effect on growth. Bulb rots due to a *Penicillium* species were reported in the 1920s but do not appear to have been followed up on at that time (Moore et al. 1979). Aggressive strains of *Penicillium* can cause a dark-brown rot, though often the symptoms are limited to

the damaged outer scale (Dugan et al. 2014). A grey-green or blue mold can be found under the tunic; a "puffer" is a completely rotted bulb under an intact skin, that, when broken, releases a cloud of spores (Plate and Schnieder 1967). In some cases, *P. corymbiferum* has been linked with basal rot-like symptoms and has caused necrosis when inoculated to healthy bulbs (Davies 1994; Nicholson and Ingram 1989).

Biology and epidemiology. *Penicillium* species are wound pathogens. Harvesting wounds, insect injuries, bruises, sunburn, and natural wounds are avenues of entry. Spores are ubiquitous and dispersed by air. Prolonged cool, moist storage conditions favor disease development.

Management. Control generally consists of avoiding wounding, proper storage conditions, and using appropriate fungicides (van der Weijden 1989). Fungicide resistance is a significant problem with this pathogen.

2.6 Smolder [*Botrytis narcissicola* Kleb. (Teleomorph: *Botryotinia narcissicola* (Greg.) Buchw.; syn. *Sclerotinia narcissicola* Greg.)]

Geographic occurrence and impact. Descriptions of smolder came from Germany and the Netherlands in the early-twentieth century, and the disease was later reported on bulbs imported into England and in Denmark and the USA (references in Moore et al. 1979). Smolder is common on daffodils wherever they are grown, including North America, Europe, Asia, Australia, and New Zealand (Distribution Maps of Plant Diseases 1989).

In the Netherlands, smolder is seldom of economic importance (van der Zwet et al. 1990); and in the UK, despite its being common in all daffodil-growing areas, Moore et al. (1979) questioned its economic importance. However, where foliar diseases are now exacerbated by growing daffodils for 2 or more years-down, with a build-up of inoculum in bulbs, debris, and soil, smolder was estimated by UK growers in the 1990s to exact a loss of bulb and flower yield of around 10%. In the Netherlands, large quantities of dwarf cultivars are marketed as pot-plants, a trade dominated by 'Tête-à-Tête' which is prone to attack by *Botrytis narcissicola*, *B. cinerea*, and *Penicillium* spp. (van der Weijden 1989).

Symptoms/signs. Smolder becomes evident at or soon after shoot emergence, when diseased shoots ("primaries") emerge, having become infected with the pathogen by contact with germinating sclerotia (small, black, flattened, and 1–2 mm across) in or on the bulb or in the soil. Infected leaf-tips may be dark brown or black, with a yellowing zone below, stuck together, crooked, and torn (Figs. 15 and 16). Leaf lesions characteristically affect one side of the leaf, and the consequent tissue death there results in uneven growth leading to a curved leaf described as like a sickle or shepherd's crook. The damage may be associated with earlier feeding damage due to bulb-scale mite (*Steneotarsonemus laticeps*) (Fig. 17). In severe



Fig. 15 Smolder (*Botrytis narcissicola*) primary with shoot rot, curved leaf, and stunted flower stem (Gordon R. Hanks © 2017. All Rights Reserved.)



Fig. 16 Daffodil with curled leaves (one-sided damage) due to smolder (*Botrytis narcissicola*) (also showing virus symptoms and snail predation) (Gordon R. Hanks © 2017. All Rights Reserved.)

cases, the plants go on to produce pale, broken leaves, dwarf flower stalks, and misshapen flower buds. Under damp conditions, a mass of grey conidia forms on the primaries and the spores are spread by wind and rain-splash causing secondary leaf and flower spotting through the crop (Fig. 18), sometimes late in the season. The pathogen colonizes the cut-end of stems when the flower has been cropped. As with other foliar diseases, smolder results in premature die-back of the foliage, reducing bulb and subsequent flower yields. More sclerotia germinate at this time and produce ascospores that cause further leaf and flower spotting. As the leaves die-back, sclerotia form and remain in leaf debris on the ground. The pathogen also grows



Fig. 17 Daffodil foliage with one-sided, saw-tooth damage due to feeding activity of bulb-scale mite (*Steneotarsonemus laticeps*) – often found in association with smolder (*Botrytis narcissicola*) (Gordon R. Hanks © 2017. All Rights Reserved.)



Fig. 18 Spotting on daffodil tepals due to smoulder, *Botrytis narcissicola* (Gordon R. Hanks © 2017. All Rights Reserved.)

down into the bulb and forms sclerotia there, over-wintering particularly on the thin, papery bulb scales in the neck of the bulb (Fig. 19). The bulb skins may become "greasy," the base rot corky, and the bulb may partly rot (Moore 1939; Moore et al. 1979).



Fig. 19 Daffodil bulb with sclerotia of smoulder (*Botrytis narcissicola*) (University of Warwick © 2017. All Rights Reserved.)

The ubiquitous grey mold (*Botrytis cinerea*) also occurs on bulbs as a secondary pathogen. It forms grey sporulating areas on leaf and stem bases (O'Neill and Mansfield, 1982a) and sclerotia similar to those of smolder. Morphologically, it is difficult to distinguish *B. cinerea* from the smolder pathogen.

Biology and epidemiology. The development of smolder lesions as the leaves grow through the infected neck of the bulb is enhanced by wet soil conditions and cold wet weather at emergence and is more likely in multinosed bulbs (Gray 1971; Gray and Shiel 1975). In a study in Scotland using a stock with a low level of infection, smolder incidence did not increase over 4 years, but where flower-heads were removed or the flowers cropped, smolder had increased by the third and fourth year (Dixon 1985, 1986).

The fungus does not easily enter undamaged tissue, and infection is enhanced by damage caused by frost, hail, or flower cropping (Hanks et al. 2003). It has been suggested that infection is linked to tissues already colonized by the bulb-scale mite, a primary pest of daffodils that feeds on the margins of young leaves, though controlling bulb mites (larger mites that feed only on bulbs that are already damaged) did not reduce the incidence of smolder (Gray and Shiel 1987; Gray et al. 1975). The spores are spread by wind and rain-splash.

The effect of crop damage was investigated in Scotland (O'Neill and Mansfield 1982a; O'Neill et al. 1982). Conidial infection by both *B. narcissicola* and *B. cinerea* occurred more readily later in the growing season, and infection by *B. narcissicola* was enhanced by the light mechanical wounding of leaf and bulb tissues. The addition of nutrients (such as daffodil pollen grains) enhanced infection, particularly

at higher temperatures (18 °C/64 °F). In this study, eight cultivars were tested for infection by the smolder fungus, and all were susceptible, with 'Golden Harvest' the most susceptible and 'Dutch Master' and 'Geranium' the least. Conidial inoculations of bulb scales with either *B. narcissicola* or *B. cinerea* resulted in cell wall lignification and phytoalexin accumulation, but neither was detected at the time when *B. narcissicola* was forming spreading lesions (O'Neill and Mansfield 1982b). Further information on the life cycle is given in Moore et al. (1979).

In the UK, a disease-forecasting model/spray-timing system was developed to improve the targeting of sprays. Key factors in the development of smolder included crop damage (confirmed experimentally for hail, simulated rain at terminal velocity, frost or mechanical damage) and temperatures of 4–16 °C/39–61 °F with a leaf-wetness period of ca. 24 h or 12 °C/54 °C with a short leaf-wetness period of 6 h (Hanks et al. 2003). A decision-support system was developed relating temperature and leaf wetness duration to the number of smolder lesions per leaf.

Management. Smolder management combines cultural practices, sanitation, HWT (with biocide and fungicide), and a fungicide spray program. The decision-support systems referred to have not been taken up by growers on the grounds that the number of days when crop spraying is practical (in a wet, windy climate) limit the ability of growers to spray on the target dates; however, this is true of any decision about when a crop-spray should be applied. As far as is known, biological controls for smolder have not been developed.

- ***Cultural practices*** – The pathogen persists in crop debris, so repeatedly removing primaries, re-ridging crops between growing seasons, and burning debris are sensible measures. In trials across the UK, however, these treatments were not consistently effective (ADAS 1976; Gray 1971; Millar 1978, 1979). Where foliar disease is of concern, new plantings should be sited away from existing ones to reduce the chance of infection. Other measures include rotation, early bulb lifting, avoiding low, wet areas of fields, and removing rotted bulbs and loose bulb skins. Some growers say there is less smolder if they remove the thin outer bulb scale before replanting (Moore et al. 1979).
  In growing dwarf daffodil cultivars for pot-plant production, several steps are needed to avert damage from foliar fungal diseases, to which they are prone. Following the proper regimes for bulb drying and storage are important, a preplanting fungicide soak should be used, bulbs should be planted at the commencement of cold-storage, and a low temperature should be used in the greenhouse phase (van der Weijden 1985, 1989; van der Weijden and Vreeburg 1988; Vreeburg and Korsuize 1991; Vreeburg and Schipper 1990).
- ***Cultivar selection*** – There is little information about the relative susceptibility of cultivars to smolder. In trials in Poland, three *B. narcissicola* isolates were tested on five cultivars (Piwoni 2009): all isolates were pathogenic and no cultivar was totally resistant, though 'Dutch Master' was least susceptible and 'Ice Follies' most susceptible.

- ***Fungicides*** – Using infested bulb stocks Gray (1971) and Gray and Shiel (1975) showed that yield increases were obtained when bulbs were dipped in benzimidazole fungicides, so the current use of thiabendazole in HWT should provide some control. The effect of fungicide dips on the infection of emerging leaves by *B. narcissicola* was investigated by Chastagner and DeBauw (2011b). Preplanting bulb dips in boscalid + pyraclostrobin, fludioxonil, or the combination thiabendazole + captan significantly reduced the amount of neck rot and leaf necrosis. Although azoxystrobin and trifloxystrobin reduced disease development compared with controls, they were not as effective as the previous fungicides. Bulb dips in pyraclostrobin had no effect on disease development.

  In the UK, management is primarily through a spray program: in the 1990s, this involved applying a range of fungicide types at 2–3 weeks intervals (Hanks et al. 2003). Looking for savings by using fewer, but targeted and more effective, sprays, a decision-support system was developed and tested (Hanks et al. 2003, 2009; O'Neill et al. 2004). Fungicides offering effective disease control, delay of foliar senescence, and increased bulb yield were azoxystrobin, cyprodinil, kresoxim-methyl, tebuconazole, pyrimethanil, and the mixtures azoxystrobin + tebuconazole and carbendazim + tebuconazole. A program of two sprays of vinclozolin after shoot emergence plus two sprays of tebuconazole around flowering was also effective. These findings enabled growers to achieve better, more cost-effective disease management.

  Many pesticides have been superseded or withdrawn, and some impose HI unsuited to flower-picking: field-grown daffodils typically require picking-over a few times across a 1–2 weeks period. Recent trials showed that azoxystrobin, tebuconazole, prothioconazole + tebuconazole, boscalid + pyraclostrobin, cyprodinil + fludioxonil, and boscalid + epoxiconazole were the most effective of those tried against smolder, delaying foliar senescence and increasing bulb yield (Millar and O'Neill 2012; O'Neill 2013), and some were also shown to increase next spring's flower yield. Guideline spray programs for use against smolder and white mold, based on one or two sprays before flowering and three or four postflowering and taking account of HI requirements, were suggested, and incorporated (pre- or postflowering) mancozeb or boscalid + pyraclostrobin and (postflowering only) boscalid + epoxiconazole, tebuconazole or pyraclostrobin. The issue of HI and re-entry periods has been eased in the UK since a decision that the protective factor of suitable gloves may be taken into account during the authorization process for a plant protection chemical.

2.7 Soft Rot (*Rhizopus* spp.)

Geographic occurrence and impact. Soft rot affects only wet or damp bulbs that are stored at high temperatures, usually at a RH >80% and a temperature > 30 °C/86 °F (Moore et al. 1979). This is avoidable, but could occur in high-temperature drying or heat treatment if air exchange or humidity control were inadequate, in transit under poor conditions, or in propagation by chipping if temperatures are not



Fig. 20 Two examples of *Rhizopus* soft rot of daffodil bulbs due to warm, moist storage (University of Warwick © 2017. All Rights Reserved.)

properly controlled. Soft rot has led to bulb losses on a large scale, but should be avoidable with modern facilities that are correctly monitored so that poor conditions can be quickly rectified.

Symptoms/signs. Affected bulbs rot to a mushy, grey-white mass covered with coarse mycelial growth (Fig. 20), in which the black, globose fungal spore-heads may be seen.

Biology and epidemiology. *Rhizopus* species are common and may be normally present on bulbs without causing damage. Under warm, moist conditions, infection can take place rapidly, especially if bulbs have been mechanically injured.

Management. Appropriate bulb handling practices are used to avoid soft rot.

2.8 White Mold (*Ramularia vallisumbrosae* Cav.)

Geographic occurrence and impact. Around the start of the twentieth century, white mold was described from Italy, France, and the UK (Moore et al. 1979). It is distributed across western Europe (British Isles, Denmark, France, Italy, Norway, Portugal, and Spain) and occurs in British Columbia and the PNW (Distribution Maps of Plant Diseases 1971). Although associated with warmer bulb-growing areas, such as south-west England, it has also been recorded in the English Midlands and Scotland (Moore et al. 1979) and was later confirmed in eastern England (O'Neill et al. 2002).

In some years, white mold causes severe epidemics in south-west England (Moore et al. 1979). Late-flowering daffodils ('Actaea', 'Cheerfulness', and 'Double White') are susceptible, as well as some mainstream cultivars ('Dutch Master', 'Fortune', and 'Magnificence'). White mold appears to have become more troublesome since the 1980s (Hanks et al. 2003). When severe, the flower crop is spoiled and the extensive, premature loss of GLA reduces bulb yield, and the following year's flower yield. White mold does not appear to be a significant disease in other countries; in the PNW, white mold sometimes occurs in areas of northwestern California, southwestern Oregon, and British Columbia that are close to the ocean and have moderate rainfall with high rainfall in the spring. White mold has not been reported on other species.

Symptoms/signs. White mold results in pale, sunken spots, or streaks on leaves and stems, especially distally. The lesions enlarge and darken to yellowish-brown patches: under moist conditions, a cream or white, powdery spore mass develops (Fig. 21). In warm, wet springs the lesions spread downwards and coalesce, and if not checked by cool weather, the disease becomes epidemic and foliage may die-back in a few days (Fig.22) (Gregory 1937; Moore et al. 1979).



Fig. 21 Three examples of the rapidly coalescing lesions of white mold (*Ramularia vallisumbrosae*) on daffodil leaves (Gordon R. Hanks © 2017. All Rights Reserved.)



Fig. 22 Field of daffodils showing rapid loss of green-leaf area and premature die-down, the results of a severe infestation of white mold, *Ramularia vallisumbrosae* (Gordon R. Hanks © 2017. All Rights Reserved.)

Biology and epidemiology. Spores are spread by wind and rain-splash. For the spores to infect through the stomata, moisture is needed, and if the leaves are dry, the spores are unlikely to survive for more than a few days. Within infected leaves many minute, black sclerotia (or sclerotia-like bodies) are formed, just visible to the unaided eye, the means by which the dormant pathogen overwinters in foliage residues. In winter and spring, the sclerotia germinate, producing conidia about the same time as the bulb shoots emerge. Leaves and flower stalks can be attacked just below soil level, but the bulbs are not attacked and the infection is not carried on them. Gregory (1939, 1940) and Moore et al. (1979) described the pathogen's life-history and complexities.

In the UK, a disease-forecasting model/spray-timing system was developed to improve the scheduling of sprays. Experimentally some infection apparently occurred across undamaged leaf surfaces, but infection was enhanced by damage (such as exposure to frost or mechanical damage) and the presence of free water. Optimal conditions for infection were temperatures of 5–10 °C/41–50 °F with a leaf-wetness period of 12–24 h. The common spore type produced by lesions was phragmospores, with an optimal production temperature of 5–15 °C/41–59 °F, and scolecospores were produced at 5–10 °C/41–50 °F (Hanks et al. 2003). A decision-support system was developed relating temperature and leaf wetness duration to the number of white mold lesions per leaf.

Management. White mold management is based on a fungicide spray program. Because this disease is not carried in bulbs, fungicides applied in HWT would not be

effective. Cultural practices and sanitation are important. As far as is known biological controls have not been developed, nor is formal information available on the relative susceptibility of cultivars.

- ***Cultural practices*** – The pathogen is carried on crop debris, so sanitation is important and ideally would involve clearing and burning debris (Gregory 1936, 1940; Moore et al. 1979). However, the sclerotia are short-lived (<1 year). Bulb stocks that have previously been affected by white mold should not be planted in close proximity to other bulb stocks.
- ***Fungicides*** – The role of fungicide sprays was investigated by Gregory (1936, 1940) and Jenkins and Hawken (1969). Thereafter, few trials appear to have been directed specifically at the control of white mold, and the results of "generic" spray trials were relied on. By the 1990s, a four-spray program was commonly used for late-flowering and other susceptible cultivars, starting when shoots were 8–10 cm high and spraying at 2–3 week intervals, usually three sprays before flowering and one afterwards (the early-season sprays could be omitted in the first year of daffodils planted on clean ground). Over time both the frequency of sprays and the variety of a.i. have increased (Hanks et al. 2003).
  Looking for savings by using fewer, but targeted and more effective, sprays, a white mold decision-support system was developed and tested (Hanks et al. 2003; Millar et al. 2009). Axoxystrobin + tebuconazole and carbendazim + tebuconazole, applied at 10–15 cm shoot height, offered good control. Three sprays of axoxystrobin + tebuconazole applied at 21–28 days intervals, or six sprays of carbendazim + dithane at 10–14 days intervals, were effective treatments for established white mold. Spray programs of three sprays starting with a low level of white mold were as effective as a five-spray program starting earlier.
  Many pesticides have been superseded or withdrawn, and some impose HI unsuited to flower-picking. Recent trials showed that epiconazole + metconazole, tebuconazole, tebuconazole + trifloxystrobin, fluazinam, boscalid + pyraclostrobin, boscalid + epoxiconazole, and pyraclostrobin were the most effective of those tried against white mold (Millar and O'Neill 2012; O'Neill 2013). Guideline spray programs for use against white mold and smolder were suggested and were given in the final paragraph of Sect. 2.6.

2.9 White Root Rot (*Rosellinia necatrix* Prill.)

Geographic occurrence and impact. *Rosellinia necatrix* is widely distributed, especially in warmer climates (Distribution Maps of Plant Diseases 1987). Originally described on the outer bulb scales of daffodil in Germany in the late-nineteenth century, it was reported from bulbs in the UK in the 1920s. In the UK, it occurred sporadically on daffodils on the Isles of Scilly, sometimes with serious effects: although sometimes seen on the mainland, it appeared the islands provided the soil moisture and temperature for it to succeed (Moore et al. 1979).

Severe outbreaks of white root rot used to occur on the Isles of Scilly, but have not been seen for many years (AA Tompsett, personal communication, 2000). *R. necatrix* has potential to infect other bulbous ornamentals and has been recorded on the Isles of Scilly and mainland England on *Arum*, *Gladiolus byzantinus, Iris* × *hollandica*, *Iris stylosa*, *Ixia, Montbretia*, and *Tulipa* (Moore et al. 1979).

Symptoms/signs. Bare patches occur in bulb fields, and examination shows the bulbs there were completely rotted, with blackened outer scales, deep brown inner scales, and no roots. If bulbs were lifted from the edge of patches, the bulbs had few or no roots and blackened outer scales, with white strands of mycelium clearly visible between them. If moist, a mass of wooly mycelium appears on the base of the bulbs, and the mycelial strands can grow through the soil and infect other plants (Hodson and Beaumont 1930; Mantell and Wheeler 1973; Moore et al. 1979).

Biology and epidemiology. On the Isles of Scilly, the hedging plant *Pittosporum crassifolium* and the weed *Oxalis pes-caprae* grow in close proximity to daffodil crops and both are hosts to *R. necatrix* (Mantell and Wheeler 1973; Pethybridge 1929; Pethybridge et al. 1934).

Management. Cultural practices and soil fumigation are the chief options for managing white root rot.

- ***Cultural practices*** – Lifted bulbs should be examined carefully and any doubtful ones removed. Some growers believe that the frequent cultivation of infested land causes it to dry out, killing the fungus (Moore et al. 1979).
- ***Soil fumigation*** – Mantell and Wheeler (1973) found that soil treatment with formaldehyde and metam-sodium were effective. Later work showed that chloropicrin was also effective (Moore et al. 1979).

2.10 Other Fungal Diseases

Black slime disease is caused by S*clerotinia bulborum* Kleb. It has been observed only in the Netherlands, when daffodils are planted in fields following a heavily infested crop of another host, such as hyacinth or *Muscari* (Boerema 1961). In black slime disease, the underground parts of the leaves become soft and black, and the bulb is superficially or completely attacked, with the development of large sclerotia. The disease was investigated on the dwarf cultivar 'Tête-à-Tête' in the Netherlands (Koster et al. 1987), finding that the application of procymidone, either in a cold dip or in HWT, controlled black slime, but with some loss of yield.

Other *Sclerotinia* species have been recorded on daffodils (Moore et al. 1979). *S. sativa* Drayton and Groves was found in rotted daffodil and tulip bulbs (Drayton and Groves 1943), producing sclerotia 1.0–1.5 mm in diameter on the outer papery scales. Drayton and Groves (1952) also described and named the saphrophyte

Stromatinia narcissi on daffodils (now called *Sclerotium narcissi* (Sacc.) Boerema and Hamers [teleomorph: *Stromatinia narcissi* Dayton and Groves, syn. *Sclerotium ambiguum* var. *narcissi* Sacc.]).

Rusts include *Aecidium narcissi* Liou, a pathogen of the grass *Phlaris arundinacea* sometimes used as a cover crop for daffodils in the Netherlands (Boerema 1961). It can infect daffodils, resulting in abundant, typical rust pustules growing on senescing leaves, but does not attack the bulbs and is not transmitted in them. *A. narcissi* has also been reported from France (Boerema et al. 1961; Liou 1929). Other rust species have been recorded on daffodils, without evidence of their causing any detriment, including *Puccinia schroeteri* Pass. (syn. *Dicaeoma schroeteri*) (Plowright 1898; Wilson and Henderson 1966) and *Coleosporium tussilaginis* (Pers.) Lév. (syn. *Coleosporium narcissi*) (Grove 1922) in the UK and *Puccinia narcissi* Laundon in New Zealand (Laundon 1965).

The smuts *Urocystis colchici* (Schlecht.) Rabenh. and *U. narcissi* (Gonz. Frag.) Vánky (syn. *Urocystis colchici* f. *narcissi*) have been recorded on *Narcissus* species in Spain (Liro 1935; Moore et al. 1979). *Sclerotium rolfsii* Sacc. [teleomorph: *Athelia rolfsii* (Curzi) Tu and Kimbr. (syn. *Corticium rolfsii*)] and/or *Sclerotium rolfsii* var. *delphinii* (Welch) Boerema and Hamers have been reported to cause crown rot occasionally, with a reddish-brown rot of the outer scale of daffodils found in the PNW, California, and southeast USA (Aycock 1966; McWhorter and Weiss 1932; Moore et al. 1979). The presence of a white mycelial mat and small reddish-brown sclerotia are diagnostic for the disease (Chastagner and Byther 1985); see the chapter on lily diseases for potential controls.

Dry-scale rot, *Stromatinia gladioli* (Drayt.) Whetzel. (syn. *Sclerotinia gladioli* Drayton and *Sclerotium gladioli* Massey) is reported to cause a dry rot of daffodil bulbs, with sharp margins and black sclerotia (Sakaguchi 1950). In the year after infection, there are yellowing leaf-tips and sclerotia are formed on the scales. In the second, there is early leaf senescence and plant death. Dry-scale rot occurs in heavy, wet soils, and control is through HWT and planting on a better drained site (Chastagner and Byther 1985).

Grey bulb rot is caused by *Rhizoctonia tuliparum* (Kleb.) Whetzel and Arthur and is characterized by the distal ends of leaves developing grey-brown areas in early spring and becoming crooked and withered. A destructive disease of tulips and bulbous iris, it is usually mild in daffodils and the bulbs are not damaged (Moore et al. 1979).

Honey fungus, *Armillaria mellea* (Vahl) Kumm, can cause a bulb rot, particularly on newly cleared ground. The bulbs become covered with a white mold and rhizomorphs may be present (Chastagner and Byther 1985). It has been reported from the USA (McWhorter and Weiss 1932), England (Pethybridge et al. 1934), and Jersey (Phillips 1960).

Dutch texts refer to several conditions ("skin diseases") in which the surface of the bulb is degraded, the skin becoming darker (*huidziek*), greasy (*vethuidigheid*), and multilayered or irregular (van Aartrijk et al. 1985). These conditions detract from appearance and make bulb cleaning more difficult, with dwarf cultivars such as

'Tête-à-Tête' susceptible. The fungi associated with these conditions include *Ophiostoma narcissi* Limber, *Fusarium oxysporum*, *Peyronellaea* (*Stagonospora) curtisii*, *Botrytis narcissicola*, and *Phoma leveillei* Boerema and Bollen. Appropriate fungicide, HWT and hygiene treatments are needed.

Leaf blight, *Heterosporium gracile* Sacc., was described as the cause of a leaf blight in the Netherlands (Bos 1901). The leaves turned yellow from the margins, became covered with a black incrustation of the conidial stage of the fungus, and withered.

Several other fungi have been described as causing (or being associated with) miscellaneous bulb or root rots in daffodils, including species of *Phytophthora* and *Pythium* (Moore et al. 1979).

3 Bacterial and Phytoplasma Diseases

3.1 Bacterial Rot [*Pectobacterium carotovorum* subsp. *carotovorum* (Jones) Hauben et al. (syn. *Erwinia carotovora* subsp. *carotovora* (Jones) Bergey et al.)]

Bacterial diseases are not normally associated with daffodils, though rots due to *Pectobacterium carotovorum* subsp. *carotovorum* have occasionally been seen in bulbs of tazetta cultivars 'Paper White' and 'Grand Soleil d'Or' from the Isles of Scilly. Infection can result in complete destruction of the bulbs into a stinking black mass (A Jansen and J Snowden, personal communication, 2010). No specific control is available, so management depends on awareness, visual inspection, and the sorting of bulbs.

3.2 Bacterial Streak (*Pseudomonas* sp.)

A bacterial streak caused by a *Pseudomonas* species has been reported in the PNW (Gould 1957). In the field, flower stalks collapse from the base. A vascular discoloration can be seen in infected bulb tissues. With stored bunches of cut-flowers, a dark, soft decay of the flower stalk begins near the rubber band used to hold the bunch together, though this does not normally occur if the stems have been correctly cold-stored. The condition can be controlled by dipping in an antibiotic solution or storage at a low temperature (2 °C/36 °F).

3.3 Diseases Due to Phytoplasmas

A phytoplasma (mycoplasma-like organism) has been reported in unhealthy daffodil plants, but there is no information as to the wider significance of this discovery (Bellardi et al. 1990).

4 Virus Diseases

4.1 Introduction

Over 20 distinct viruses have been identified in daffodils and there is general agreement that the aphid-transmitted potyviruses are the most damaging as well as the most specific. These include *Narcissus degeneration virus* (NDV), *Narcissus latent virus* (NLV), *Narcissus yellow stripe virus* (NYSV), *Narcissus late season yellows virus* (NLSYV) and *Narcissus white streak virus* (NWSV), and typical foliar symptoms are shown in Figs. 23, 24, 25, and 26. *Narcissus mosaic virus* (NMV) and *Narcissus tip necrosis virus* (NTNV), with no known vectors, are damaging and also specific to daffodils. The list of most commonly found viruses in the Netherlands comprises NLV, NYSV, *Narcissus silver stripe virus* (equivalent to NWSV), NMV, NTNV, *Arabis mosaic virus* (ArMV), *Tobacco rattle virus* (TRV), and *Tobacco ringspot virus* (TRSV) (Asjes 1996). With no practical, cost-effective way of managing virus infestations, they are arguably a greater threat to commercial daffodil production than basal rot or stem nematode.

Most daffodil stocks are infested with viruses, and it is common for older cultivars (say 'King Alfred', 'Golden Harvest', or 'Grand Soleil d'Or') to be infested with up to five viruses (Brunt 1966a, b, 1977a, b). As well as detracting from the appearance of foliage and flowers, virus infection leads to serious loss of vigor. It has been estimated that virus infection might decrease bulb yields by up to two-thirds (van Slogteren and de Bruyn Ouboter 1946), while in stocks seriously infected with



Fig. 23 Foliar symptoms of *Narcissus yellow streak virus*, a common and widespread virus of daffodils causing mosaic patterns soon after emergence (University of Warwick © 2017. All Rights Reserved.)



Fig. 24 Foliar symptoms of *Narcissus white streak virus*, another common daffodil virus (University of Warwick © 2017. All Rights Reserved.)



Fig. 25 Daffodil leaf showing mild chlorotic streaks due to *Narcissus late season yellows virus* (Gordon R. Hanks © 2017. All Rights Reserved.)

NYSV and NWSV yields were reduced by between 10% and 35% (Asjes 1990). In contrast, however, Rees (1966, 1972) found little or no difference in the net assimilation rates of healthy and virus-infected daffodils, suggesting the losses due to virus might sometimes be much less than previously reported.



Fig. 26 Yellow stripes and chlorosis as commonly seen in many daffodil stocks (Gordon R. Hanks © 2017. All Rights Reserved.)

Management of viruses is largely dependent on the control of their aphid, nematode, and other vectors and is usually targeted against mixed viruses rather than individual species. Hence, virus management will be discussed in Sects. 4.1.1 and 4.1.2 rather than under the individual viruses. Several of the viruses that occur in daffodils are known to be seed-borne in many other hosts, daffodils not being specifically mentioned, though Brunt (1995) stated that in daffodils "only the ringspot viruses are transmitted through the seed"; this is of interest mainly to hybridizers. All the aphid- and nematode-borne viruses are insufficiently infective to be transferred mechanically by alternately handling infected and healthy material (Moore et al. 1979).

4.1.1 Management of Vector Aphids

The spread of aphid-transmitted viruses is slow, localized, and typical of "nonpersistent" transmission. Spread is proportional to the population of aphids and the initial number of infected plants: for NYSV with 10, 20, and 50% of plants infested, after 3 years, infection had increased to 16, 46 and 90%, respectively (Broadbent et al. 1962). Although there are instances of aphids colonizing daffodils under favorable conditions – reported for *Macrosiphum euphorbiae* in Long Island, NY (Blanton and Haasis 1942), and *Aphis fabae* in the Netherlands on daffodils growing in late-summer for export to the Southern Hemisphere (van Slogteren and de Bruyn Ouboter 1946) – aphids rarely colonize daffodils. Virus transmission is therefore dependent on chance landings and exploratory probing, with the risk period limited to between the first appearance of aphids and foliar senescence or bulb harvesting. Whereas this could formerly have meant from, say, mid-May to mid-July in southern

England, climate change has resulted in earlier aphid appearance. While some changes in bulb husbandry, such as earlier harvesting, probably reduce the risk of virus spread, others, such as leaving bulbs in situ for 2 or 3 years, likely increases the risk. Van Slogteren and de Bruyn Ouboter (1946) reported infection rates for NYSV to be 41–72% higher if bulbs were lifted 4–8 weeks later than usual. Common aphid species known to infest daffodils in the UK include *Macrosiphum euphorbiae, Macrosiphum rosae, Myzus persicae, Myzus cerasi, Aphis fabae, Aulacorthum solani, Aulacorthum pisum, Aulacorthum circumflexum, Dysaphis plantagina*, and *Rhopalosiphoninus staphylae*.

Alatae of vector aphids are able to transmit virus particles within minutes of feeding on an infected plant, so it would be impractical to apply insecticides quickly enough to control incoming aphids totally. When protecting high-health stocks, aphid control has used sprays of antifeedant pyrethroids (e.g., cypermethrin or deltamethrin) at 7–10 days intervals (Hanks 1998), though such insecticide use has been argued to be counterproductive since it can increase aphid irritability and probing (Broadbent et al. 1957) – and would be environmentally unacceptable today. In a trial, virus-tested (VT) mother stocks of 'Carlton' and 'Fortune' were planted in ridges between highly infested commercial stocks and regular pyrethroid applications made through the growing season for 6 years. For 'Fortune', 1 in 10 ELISA tests for aphid-borne potyviruses were positive after only 4 years, but there were no visual symptoms; for 'Carlton', 1 in 20 tests were positive after 6 years and mild visual symptoms were seen. Growing in isolation was more effective: there were no positive tests or visual symptoms in either cultivar after 6 years when grown in sterilized, isolated plots in the open or in a simple mesh tunnel, or growing in sterilized growing medium in a vector-proof tunnel (Hanks 1998). We do not know how these dynamics would apply to large bulb fields: would a temporary increase in the movement of alatae result in more virus transmission than uncontrolled, prolonged visits? (Moore et al. 1979). Alternatively, mineral oil sprays, which disrupt normal virus transmission, have been sprayed weekly on some stocks. These treatments sometimes caused stunting or loss of yield or were sometimes ineffective (ADAS 1982a, b; Hanks 1998; Mowat et al. 1984; Vreeburg and Korsuize 1987).

4.1.2 Management of Other Vectors

Since virus-resistant cultivars or virus-tested stocks are unlikely to be available to daffodil growers for some time, the control of nematode–borne viruses invariably depends on the use of nematicidal and nematostatic chemicals (Harrison and Robinson 1986). In the past methyl bromide was used for soil fumigation, and in some areas fumigants such as metam-sodium, chloropicrin or dazomet may be available; they are all nematicidal and provide varying levels of control of other pests, fungi, and weeds and may be suitable for protecting high value virus-tested bulb stocks. Experience suggests that disease problems can be minimized by employing crop rotations that diminish nematode populations (Murant 1970a). Thus, an awareness of other nematode hosts, in combination with weed control and suitable crop rotations, might also be useful measures in limiting the spread of nematode-vectored viruses.

Tomato spotted wilt virus (TSWV) is transmitted by thrips, and its management depends largely on the control of weed hosts and avoidance of planting near infected crops (Sect. 4.5.1). *Tobacco necrosis virus* (TNV) is transmitted by zoospores of the fungus *Olpidium brassicae* (Sect. 4.8.2) and its management would probably benefit from use of the same fumigants deployed against nematodes. Additional information on insect vectors, their life cycles, and management can be found in introductory (▶ Chap. 4, "Insect Management for Disease Control in Florists' Crops").

4.1.3 Prevention of Sap Transfer

The aphid- and nematode-borne viruses are considered unlikely to be spread by bulb-to-bulb contact or by alternately handling healthy and infected plants during flower packing and other operations. However, a very low level of NYSV transmission was reported from greenhouse experiments under forcing conditions or when recovering bulbs after forcing (Haasis 1939a; McWhorter and Weiss 1932). In contrast, NMV, which is neither aphid- nor nematode-transmitted, is easily spread by the mechanical inoculation of sap (Brunt 1966b). For NTNV, no evidence was found for spread by mechanical means such as handling and flower cropping, but it appeared likely that NMV could be spread by flailing, at least at some stages of the crop (Mowat 1980a, 1987).

4.1.4 Management Through Husbandry Practices

"Roguing" – the removal and destruction of diseased plants from the field – is the basis of virus management in many crops (Broadbent et al. 1962; Moore et al. 1979). Daffodil crops need to be inspected and rogued more than once in the growing season – for example, TRV needs to be checked early in the season (before soils warm and nematodes begin breeding), NYSV in March and April, and NWSV in late-May (when the symptoms are easier to see). In a survey of grower practices for the virus management of flower-bulb crops in the PNW, Chastagner et al. (2002) reported growers relied on the removal of symptomatic plants, either by hand-digging or using herbicide. However, unlike the (more profitable) lily and tulip crops, daffodil crops were rarely rogued. In the UK, the rigorous rouging formerly practiced has become less frequent because it is labor-intensive and requires skilled workers, and it is difficult to remove bulbs and offsets completely. Rouging is probably the most important means of controlling daffodil viruses (e.g., Asjes 1996; Broadbent et al. 1962; Moore et al. 1979).

When planting newly acquired stocks, it is sensible to grow them in isolation until their health can be verified. Some growers would then select the most vigorous plants in a stock and bulk these in isolation (Mowat and Chambers 1975). Moore et al. (1979) observed the high incidence of virus symptoms occurring in new cultivars, suggesting breeders should grow them in sterilized growing medium in a simple wooden cage covered with mesh fine enough to keep out vector insects (32 mesh, 32 openings/inch).

The good control of weeds and of escapes from cultivation, in and around daffodil fields, will help to reduce sources of alternative hosts and foci of infection. Bulb stocks could be lifted early, to shorten the time they are exposed to aphids; to

minimize the associated loss in yield, cultivars prone to virus infection might be lifted before others.

4.1.5 "Virus-Free" Stocks

In the 1960s, the gradual decline in vigor of tazetta daffodil 'Grand Soleil d'Or', a specialist crop of the Isles of Scilly, was deduced to be a result of widespread virus incidence involving NDV in complexes with others. A scheme to produce "virus-free" stocks – better called virus-tested (VT) stocks – was begun (Stone 1973; Stone et al. 1975, 1978). When stocks of regular cultivars on the mainland were found to be similarly infected, the scheme was extended (Brunt 1980, 1985). Initially using meristem-tip culture to obtain "virus-free" material, and "twin-scale" and "chip" propagation and growing in strict vector-free conditions to bulk numbers, much of the tazetta population of the Isles of Scilly was replaced. In Scotland, ordinary bulbs were indexed for virus content, and the occasional "virus-free" bulbs found in some cultivars were propagated and bulked the same way (Mowat 1980b, 1986; Mowat et al. 1986). A sizeable area of the Scottish crop was replanted with virus-indexed stocks.

The initial appreciation of VT stocks was high, often involving manifestly brighter, larger flowers and modest (ca. 10%) increases in yield. Ultimately these schemes stalled due to their high, ongoing costs, the re-infection of stocks when not grown in isolation, and the lack of any increase in returns for high-health bulbs, so it is unlikely that such schemes will be revisited. The schemes have been described elsewhere (Hanks 1993a, 2013a). In New Zealand, low-virus stocks compared favorably with standard commercial stocks (Allen and McIntosh 1994). In Poland, a scheme to produce potyvirus-free virus-indexed cultivars through adventitious shoot cultures in tissue culture from infected plants is showing good results (Sochacki 2011; Sochacki and Orlikowska 2005; Sochacki et al. 1997). In the Netherlands, VT stocks of other flowerbulbs have been produced through tissue culture, but it was reported that work with daffodils has made less progress (Asjes 1990). The heat treatment of twin-scale propagules (8 h/day at 40 °C/104 °F for 10 weeks) produced some "virus-free" plants, though mortality rates were high (Phillips and Brunt 1980). The use of anti-viral agents (such as vidarabine and ribavirine) to achieve virus freedom had variable results (Phillips 1990; Sockacki 2011).

4.1.6 Breeding for Resistance

The conventional plant breeding of commercial daffodil cultivars seems to have been restricted to the improvement of floral and plant attributes and breeding for resistance to basal rot. However, genetic material may be available for enhancing resistance to viruses. Daffodil cultivars originating from *Narcissus tazetta* are susceptible to viruses, while those derived from *N. pseudonarcissus*, as well as several of the *Narcissus* species themselves, are relatively resistant, with *N. jonquilla* having an intermediate rating (Brunt 1971a; Caldwell and Wallace 1955).

4.1.7 Other Strategies

Significant advances have been made in the development and use of serological tests, such as enzyme-linked immunosorbent assay (ELISA), and nucleic acid-based

tests, such as the polymerase chain reaction (PCR), to identify and detect virus-infected plants. The sensitivity of these detection methods provides opportunities to monitor the spread of viruses and develop special certification schemes associated with the production of "virus-free" stock.

4.2 Alphaflexiviridae

4.2.1 Narcissus Mosaic Virus (NMV, Genus: Potexvirus)

Geographic occurrence and impact. NMV was described by van Slogteren and de Bruyn Ouboter (1946), van Slogteren (1955), and Cremer and van der Veken (1964). Further information is found in Brunt (1966b, 1995) and Mowat (1971). NMV is prevalent in many commercial stocks of daffodils, its only natural host. Reported from the Netherlands and UK, it probably occurs in all countries to which the bulbs are exported. In a survey, Brunt (1966b) isolated NMV from some 25 out of 50 commercial UK stocks; stocks of 'Fortune', 'Golden Harvest', 'King Alfred', and 'Magnificence' were all infected and incidence in the others was high. Most of these stocks were symptomless or showed only an inconspicuous mosaic at the base of leaves around flowering time. Also examined were stocks of tazetta narcissus 'Grand Soleil d'Or' and jonquil cultivars, which were free of NMV. Seedlings of "virus-free" 'King Alfred' and 'Sulphur' were inoculated with NMV: after 8 months they were still symptomless and the virus could not be recovered, and after 17 months all showed inconspicuous mosaic and the virus was recovered.

Symptoms/signs. NMV results in indistinct to severe mosaic symptoms in leaves of many cultivars (Brunt 1966b). It can also produce dark brown, oval spots, mainly on the distal half of leaves (CJ Asjes, personal communication, cited in Brunt 1995). Comparing infected and symptomless (virus-indexed) bulbs of 'Minister Talma', Rees (1966) found the effect of NMV on growth and productivity was less than previously suggested. Interestingly he noted that one effect of infection was to delay the main growth period by about 2 weeks, raising the possibility of interactions with climate.

'Reverse bicolor' daffodils are prone to "color-break," with the yellow color of the perianth appearing to be "broken" by patches of white. This was thought to be due to the presence of a potyvirus, though no other symptoms of virus infection are seen. Hunter et al. (2011) showed that color-break was associated with the presence of NMV.

Biology and epidemiology. NMV isolates vary in virulence, some producing more severe symptoms in some hosts than Brunt's (1966b) type isolate. Some common species of vector aphids failed to transmit the virus, and no vector is known. NMV was readily transmissible by sap to species in several plant families, and when so inoculated the leaves of most were symptomless or produced slight symptoms; few hosts became systemically infected. The virus is not found in daffodil seedlings and so is not seed-transmitted (Brunt 1966b). It appeared likely that NMV could be spread by flailing at some crop stage, so it may be transmitted mechanically in

commercial stocks during flower harvesting. NMV frequently occurs together with other viruses in severely affected plants.

4.3 Betaflexiviridae

4.3.1 Carnation Latent Virus (CLV, Genus: Carlavirus)

Geographic occurrence and impact. CLV was described by Kassanis (1954, 1955) and is found occasionally in cultivated carnations plants in the UK and continental Europe. It was reported in daffodil by Brunt (1977a, 1995). Further information is found in Wetter (1971).

Symptoms/signs. CLV causes few or no symptoms in carnation and multiplies in test species without causing symptoms. Little is known of its host range, though it can infect a number of plants outside the Caryophyllaceae.

Biology and epidemiology. CLV is transmissible by aphids (including *Myzus persicae*) in the nonpersistent manner. It is also transmissible by inoculation of sap, though it is difficult to transmit from carnation to other species because of inhibitors in the carnation sap.

4.4 Bromoviridae

4.4.1 Cucumber Mosaic Virus (CMV, Genus: Cucumovirus)

Geographic occurrence and impact. Recent accounts of CMV include Palukaitis and García-Arenal (2003) and, for its occurrence in daffodils, Brunt (1966a, 1995) and Iwaki and Komuro (1972). CMV has the widest host range of any known plant virus – more than 1,200 species in over 100 plant families. It occurs worldwide in tropical and temperate climates causing significant disease and economic loss in many crops, particularly vegetables and bananas. CMV is common in tazetta daffodils in the UK and Japan, but rare in "mainstream" daffodil cultivars.

Symptoms/signs. CMV induces a wide variety symptoms that differ between host species and virus strain. It causes mosaic in cucurbits, and symptoms on other hosts include blight, necrosis, ringspot, stunting, chlorosis, and rot. In tazetta daffodils, it invariably occurs with other viruses: its symptoms alone are unknown. The virus can be difficult to identify from its symptoms, as many symptom variants occur. Strains may be differentiated by symptoms, host range, transmission, and serology, and many strains have a restricted geographic distribution.

Biology and epidemiology. CMV is transmitted in a nonpersistent manner by over 80 aphid species, of which *Myzus persicae* and *Aphis gossypii* are important. CMV

isolates are known that have lost the ability to be aphid-transmissible; it is easily transmitted by mechanical inoculation and the loss of transmissibility through repeated inoculation is rare. Seed transmission has been reported in many species, and it is transmitted by several species of dodder (*Cuscuta* spp.).

4.5 Bunyaviridae

4.5.1 Tomato Spotted Wilt Virus (TSWV, Genus: Tospovirus)

Geographic occurrence and impact. TSWV was first described by Samuel et al. (1930) and has been reported in daffodil only by Guy (personal communication cited in Brunt 1995). The following information derives largely from Kormelink (2005) and Best (1968). TSWV is found worldwide due to its wide host range (over 800 plant families) and the global distribution of western flower thrips (WFT) *Frankliniella occidentalis*, its principal vector. The virus occurs widely in tropical and subtropical zones and in greenhouses in temperate zones. TSWV causes significant yield loss in important crops including groundnut, tobacco, and many vegetables and ornamentals (e.g., alstroemeria, chrysanthemum, and gerbera).

Symptoms/signs. The symptoms of TSWV generally include chlorosis, mottling, stunting, wilting, and necrosis, varying according to environmental conditions and the state and age of the host. Its symptoms in daffodils do not appear to have been reported.

Biology and epidemiology. Several strains of TSWV have been described, but most information has been obtained using strain BR-01 from tomato. Besides WFT, TSWV is transmitted by other *Frankliniella* species and by *Thrips* species. The rapid spread of *F. occidentalis* since the 1980s led to an explosion of TSWV due to its hidden, brief life-cycle, its colonization of many weed and cultivated species, increased tolerance to insecticides, and the global trading of plant material. Transmission is in a circulative (persistent) and propagative manner, and once acquired thrips remain infectious for life. Seed-transmission is considered unlikely. Weeds play an important role in the spread and survival of TSWV, forming a reservoir for infected thrips.

Management. Thrips have become resistant to many formerly effective insecticides, so TSWV management depends largely on weed control and the use of resistant crop cultivars. Natural resistance genes and transgenic resistance strategies are being developed.

4.6 Potyviridae

4.6.1 Narcissus Degeneration Virus (NDV, Genus: Potyvirus)

NDV was reported by Brunt (1966a, 1969, 1995) and further information is available in Moore et al. (1979).

Geographic occurrence and impact. NDV was identified in the tazetta daffodil 'Grand Soleil d'Or', a specialty crop on the Isles of Scilly, where stocks in general have shown a decline in vigor and productivity since at least the 1950s. The impact on this rural industry was severe.

Symptoms/signs. NDV is probably the most serious of the complex of viruses found in tazetta daffodils. Infected leaves show conspicuous yellow striping and general chlorosis, with declining bulb and flower quality and yield. NDV is found only in complexes with up to four other viruses – ArMV, CMV, Strawberry Latent Ringspot Virus (SLRSV), and Tomato Black Ring Virus (TBRV) – and their individual contributions to symptomatology are not known.

Biology and epidemiology. NDV is transmitted by aphids such as *Myzus persicae*. A scheme was put in place to replace the stocks on the Isles of Scilly with virus-tested bulbs (see Sect. 4.1.5).

4.6.2 Narcissus Latent Virus (NLV, Genus: Macluravirus)

Geographical occurrence and impact. NLV was described by Brunt and Atkey (1967) and Brunt (1976a, b, 1977b, 1995) and further information is found in Moore et al. (1979).

As well as daffodils, in Western Europe NLV is found in *Acidanthera*, *Gladiolus, Iris* × *hollandica*, and *Nerine*, and it probably occurs wherever these genera are grown. It has been reported on one or more of these hosts in the UK, the Netherlands, and Germany. NLV alone is probably less damaging than most other viruses infecting daffodils, but this has allowed it to spread and it probably contributes substantially to the gradual decline of daffodils when present with other viruses.

Symptoms/signs. In daffodils, NLV induces light and dark green mottling near the leaf tips of intolerant cultivars (among which 'Boswin', 'Cheerfulness', 'Criterion', 'Dutch Master', 'Lucifer', 'Magnificence', 'Minister Talma', 'Rembrandt', 'Scarlet Elegance', 'Spanish Gold', and 'Tinker' have been listed). These symptoms can at first resemble the chlorosis induced by NYSV, but occur only on the distal 6–10 cm of leaves. Other cultivars are symptomless or have barely discernible tip necrosis masked by the host's waxy bloom. In daffodils forced in the greenhouse, the mottling can be more extensive, though still not conspicuous. Virus symptoms in some test species, such as those previously attributed to NYSV, are now known to be caused by NLV.

Biology and epidemiology. NLV is transmitted effectively by aphids – *Acyrthosiphon pisum*, *Aphis gossypii*, and *Myzus persicae* have been tested – in a nonpersistent manner. Besides the flowerbulbs mentioned above, NLV was able to infect only some species from the families tested. In test species, some isolates produced local infections and others were systemic, though the isolates were

serologically indistinguishable and can be regarded as minor variants. NLV can readily be transmitted by the mechanical inoculation of sap. It is not seed-borne.

4.6.3 Narcissus Late Season Yellows Virus (NLSYV, Genus: Potyvirus)

Geographical occurrence and impact. NLSYV was described by Mowat et al. (1988a, 1998) and further information is found in Brunt (1995). NLSYV is known only in daffodil, manual inoculation failing to infect several other species. It has been identified in the UK and Netherlands, where it is the most common infection in commercial stocks, though not all cultivars are susceptible. Cultivars vary in their field infection, some seeming to be immune, but the situation in 'Carlton' is less clear: some stocks seem to be immune, whereas others show partial infection. Stocks of several important cultivars (e.g., 'Dutch Master', 'Golden Harvest', and 'Grand Soleil d'Or') are largely or totally infected; worldwide distribution can be assumed from the thriving international trade in bulbs.

Symptoms/signs. NLSYV symptoms comprise narrow chlorotic streaks and elliptical markings restricted to the leaves and flower stems. Lesions are usually sharply defined, angular, pale green to yellow and can occur in part or all of the leaf or flower stalk. Affected plants produce normal flowers, there is no malformation, and plant stature is not obviously affected. In all cultivars, symptoms appear at the earliest about 2 to 3 weeks after flowering and may then develop at any time up to the onset of senescence. Although plants remain infected, symptoms may not be seen every year. NLSYV may be responsible for the white/silver stripes/streaks formerly attributed to NWSV (Sect. 4.6.6).

Biology and epidemiology. The virus was transmissible by *Myzus persicae* when alatae were allowed access to virus source and test plants for 10 days. Transmission by apterous *M. persicae* given brief acquisition and inoculation feeds was unsuccessful. There appear to be some serological variations among isolates.

4.6.4 Narcissus Yellow Stripe Virus (NYSV, Genus: *Potyvirus*)

Geographical occurrence and impact. The symptoms of NYSV were recognized by Haasis (1939a), Blanton and Haasis (1939, 1942), and van Slogteren and de Bruyn Ouboter (1946). The information in this account is compiled from these references and Hawker (1943b), Caldwell (1946), Caldwell and Kissick (1950), Broadbent et al. (1962), Brunt (1968, 1971b, 1995), and Moore et al. (1979). In some of the older literature, the name "narcissus mosaic virus" was used for NYSV.

The natural host range of NYSV is principally a few members of the Amaryllidaceae including daffodil and *Nerine*. Its distribution is worldwide and especially in temperate regions, and, through trading, it probably occurs wherever daffodils are grown. In northern Europe, NYSV spreads slowly because apterous aphids rarely colonize daffodils and migratory alatae are usually present for only a

few weeks before normal plant senescence. NYSV is often considered the most damaging virus infecting daffodils.

Symptoms/signs. Most daffodil cultivars infected with NYSV have conspicuous chlorotic stripes on their sometimes also distorted leaves and flower stalks, especially on the upper two-thirds. The roughened chlorotic surfaces result from the abnormal division and growth of parenchyma cells, which eventually rupture the overlying epidermis. Often flowers are poor and show color-breaking, the plants are stunted and bulb yields severely reduced. In some cultivars ('King Alfred'), the chlorosis is very conspicuous. 'Magnificence' usually develops only green stripes. The stripes are present on leaves in early spring, and once the ambient temperatures are higher they become less severe. If infected by aphid transfer or sap inoculation early in the season, they may produce symptoms within 4 months, but if infected later the symptoms may not show for 15 months. In jonquil cultivars, the chlorotic stripes are narrow, sometimes coalescing to an almost complete chlorosis. The chlorotic and mottled leaf symptoms might be confused with damage from HWT, and the severe leaf distortion of 'King Alfred' with stem nematode damage.

Biology and epidemiology. Known to be transmissible by inoculation of sap and in the nonpersistent manner by several aphid species (including *Aphis fabae, Acyrthosiphon pisum*, and *Macrosiphum euphorbiae*, but not *Myzus persicae*). NYSV spreads slowly to neighboring plants as is typical of nonpersistent transmission (Sect. 4.1.1). It is not seed-borne.

4.6.5 Onion Yellow Dwarf Virus (OYDV, Genus: Potyvirus)

Geographic occurrence and impact. OYDV was described by Melhus et al. (1929), reported in daffodil by Brierley and Smith (1946), and further information is available in Bos (1976). OYDV has been reported from most countries where onions and shallots are cultivated and is probably distributed worldwide. OYDV has also been reported in other *Allium* species including some ornamental alliums. In the USA, daffodils were found to be naturally infected, while tazetta and jonquil daffodils showed symptoms after inoculation from which OYDV could be recovered.

Symptoms/signs. In onions OYDV causes plant stunting and yellow striping, yellowing, distortion, and flaccidity of the leaves and flower stalks, reduction in the number of flowers and seeds and impairment of seed quality, as well as deterioration and premature sprouting during bulb storage. In other *Allium* species, the symptoms were variable. The symptoms in daffodils do not seem to have been described.

Biology and epidemiology. There is some variation among isolates, with the garlic and daffodil isolates less infectious to onion and producing milder

symptoms. OYDV is readily transmitted experimentally by several aphid species in the nonpersistent manner, and perhaps in the field when probing in passing (aphids are not prevalent on onion crops). *Myzus ascalonicus* may spread the virus among onion bulbs and shoots during storage. The virus was not detected in onion seeds and that is considered improbable because the disease was controlled in New Zealand by introducing an *Allium*-free period, though elsewhere 6–29% seed transmission was reported in onion 'Stuttgarter Riesen' on the basis of field observations.

4.6.6 Narcissus White Streak Virus (NWSV, Genus: Potyvirus)

Geographic occurrence and impact. NWSV, at times called narcissus white stripe virus, silver streak virus and silver leaf virus, was described by Blanton (1939), Haasis (1939b), McWhorter (1939, 1940, 1957) and van Slogteren and de Bruyn Ouboter (1946). Further information is given in Moore et al. (1979) and Brunt (1995). It is likely that the symptoms attributed to NWSV may be due to NLSYV (Sect. 4.6.2), to which it is serologically related (CJ Asjes, personal communication cited in Brunt 1995; Mowat et al. 1988b, 1991). Langeveld et al. (1997), investigating silver streak symptoms, showed by sequencing that they were also due to NLSYV. NWSV has long been known in the USA, UK, and Netherlands, but because of global trade it is probably present anywhere narcissus are grown. It has a potentially high impact because of the extent of yield loss caused.

Symptoms/signs. Daffodil is the only known host, plants infected with NWSV usually remaining symptomless until 3 or more weeks after flowering, after which the leaves and flower stalk develop dark green to purple longitudinal streaks. These streaks soon become white, yellowish-white, or gray and coalesce, in a few days becoming necrotic, with the appearance of abnormally early senescence that can result in a yield loss of 30–40% over 2–4 years. Laboratory tests showed that these symptoms did not develop until the temperature exceeded 18 °C/64 °F.

Biology and epidemiology. NWSV can be transmitted by sap, but it is reported that it is not highly infectious. It is transmitted by aphids, as shown for *Aphis fabae, Acyrthosiphon pisum*, *Macrosiphum euphorbiae* and *Myzus persicae*.

4.7 Secoviridae

4.7.1 Arabis Mosaic Virus (ArMV, Genus: Nepovirus)

Geographic occurrence and impact. ArMV was described by Smith and Markham (1944) and reported in daffodils by Broadbent et al. (1962); further information is found in Murant (1970b) and Brunt (1966a, 1995).

ArMV is readily transmitted by sap and when inoculated it can infect many species in numerous plant families. Its natural spread has not been reported outside Europe, though ArMV has been found in stocks of rhubarb in Canada. ArMV was detected in Scotland and the south-west England in both mainstream daffodil cultivars and tazetta 'Grand Soleil d'Or' and is now known to be common in daffodils in north-west Europe and probably occurs wherever daffodils are grown.

Symptoms/signs. ArMV causes many diseases in soft-fruit, rhubarb, vegetables, and forsythia and occurs in many other crop plants and ornamentals, including daffodils. Following mechanical inoculation to daffodils, they showed no conspicuous symptoms, but when it occurs in complexes ArMV may exacerbate the effects of other nepoviruses.

Biology and epidemiology. The type strain is transmitted by free-living, soil-inhabiting nematodes *Xiphinema diversicaudatum* and *X. coxi.* Diseases caused by ArMV are distributed patchily in crops because of the slow migration of *Xiphinema* species. ArMV is known to be seed-borne in plants of several families. It is frequently transmitted by dodder (*Cuscuta* spp.). In general, most isolates differ little from the type strain, apart from minor differences in virulence. ArMV occurs in complexes with other viruses and may serve as an infection source for more susceptible crops. None of these studies has been made on daffodils.

4.7.2 Broad Bean Wilt Virus (BBWV, Genus: Fabavirus)

Geographic occurrence and impact. BBWV was described by Stubbs (1947) and Taylor and Stubbs (1972). It was reported in Japan in narcissus (Iwaki and Komuro 1972) and although found worldwide has not been reported from daffodils elsewhere (Moore et al. 1979). Two serotypes of BBWV described previously are now regarded as distinct viruses (BBWV-1 and BBWV-2) (Wellink et al. 2000). BBWV has a wide host range and can infect many species across some 40 plant families. Besides broad bean, its hosts include many other legumes and vegetables, many ornamentals, wild plants, and weeds. It is reported from Europe, North and South America, Asia, Africa, and Australia, probably occurring worldwide (references in Zhou 2002). BBWV-2 is economically important in China (Qi et al. 2000). On daffodils, BBWV has been reported only from tazetta daffodils in Japan, possibly its only monocotyledonous host.

Symptoms/signs. The symptoms of BBWV on naturally infected plants include vein clearing, mottle, mosaic, ringspots, blight, distortion, wilting, necrotic spots and streaks, stunting, and apical or stem necrosis, though in other cases infection is symptomless (Lisa and Boccardo 1996). Symptoms on daffodils appear to be unreported.

Biology and epidemiology. No strains of BBWV-2 have been distinguished. BBWV is readily transmissible by aphids in a nonpersistent manner, *Myzus persicae*,

Aphis craccivora, and *Acyrthosiphon pisum* being the most efficient vectors, while *Macrosiphum euphorbiae*, *Aphis fabae*, and *Acyrthosiphon onobrychis* have also been reported as vectors. BBWV is transmitted by mechanical inoculation of sap. In broad bean and pea, seed-transmission has not generally been reported.

4.7.3 Raspberry Ringspot Virus (RpRSV, Genus: Nepovirus)

Geographic occurrence and impact. RpRSV was described by Cadman (1956) and reported in daffodils by Mowat (1968). Further information was taken from Murant (1978) and Brunt (1995). As well as soft-fruit, RpRSV occurs naturally in many wild and cultivated species across several plant families, including grapevine and daffodils. It has been reported from Europe, Turkey, and the former-USSR. In daffodils, RpRSV was originally reported only in Scotland, but now may occur wherever daffodils are grown.

Symptoms/signs. RpRSV causes ringspot diseases of raspberry and strawberry and other soft- and top-fruits. As with other nematode-borne viruses, plants infected with RpRSV are patchily distributed because the vectors migrate only slowly in the soil. Symptoms in daffodils do not seem to have been described.

Biology and epidemiology. As well as being readily sap-transmissible, RpRSV infects the seed of many host plants and is transmitted by free-living soil nematodes, *Longidorus* spp. Some strains often occur in soils with strains of TBRV, with which they share the vector *L. elongatus*. In raspberries, many minor variants of RpRSV occur and they may differ significantly in serology, vector interactions, and virulence. Seed transmission is thought to occur in most host plants, though many plants so infected showed no symptoms. Infection of the seed seems to provide an important means of survival for the virus in the soil.

4.7.4 Strawberry Latent Ringspot Virus (SLRSV, Genus: Unassigned)

Geographic occurrence and impact. SLRSV was described by Lister (1964) and reported in daffodils by Broadbent et al. (1962). Further information is available in Murant (1974) and Brunt (1966a, 1995). SLRSV has a wide host range and naturally infects many species of wild and cultivated plants. It occurs throughout Western Europe and was also reported in Canada, though it was not known to spread there naturally. SLRSV was initially found in the UK in Scotland and south-west England on both mainstream daffodil cultivars and tazetta 'Grand Soleil d'Or' and became common in north-west Europe.

Symptoms/signs. SLRSV infects strawberry and raspberry causing mottle and decline in varying degrees depending on the cultivar. It causes mosaic, mottle, ring-spotting, and stunting in several horticultural crops and is found in many others, including daffodil. Diseases caused by this virus typically show a patchy

distribution. In daffodils, it produces no conspicuous symptoms on its own, but often occurs in complexes.

Biology and epidemiology. SLRSV is transmitted by the nematodes *Xiphinema diversicaudatum* and *X. coxi*. Symptoms have a patchy distribution because of the slow lateral migration in soils of its nematode vectors. SLRSV may occur in soils with ArMV which is also transmitted by *X. diversicaudatum*. Readily sap-transmissible, one isolate was found to infect 126 out of 167 species in 27 plant families after mechanical inoculation, though most were infected systemically without showing symptoms. SLRSV infects the seed of several host plants including various weed species.

4.7.5 Tobacco Ringspot Virus (TRSV, Genus: Nepovirus)

Geographic occurrence and impact. TRSV was described in 1927 by Fromme et al. and reported in daffodils by Asjes (1968, 1974). Much information in this account was taken from Stace-Smith (1985). The virus is endemic in central and eastern North America, causing serious diseases in areas where its nematode vector (*Xiphinem*a spp.) occurs, including the tobacco- and blueberry-growing areas where it is widespread and makes both crops unproductive. Soybean and cucurbits are also seriously affected, and besides these, it has a wide host range including anemone and gladiolus. Elsewhere the virus has been disseminated to many countries in infected planting material, but causes a problem only if its vector is present. TRSV has been isolated from diverse horticultural crops worldwide and was identified in daffodils and other flowerbulbs (tulip, iris, and hyacinth) in the Netherlands.

Symptoms/signs. The most severe disease caused by TRSV is bud blight of soybean, with curving of the terminal bud, other buds becoming brown and brittle, with brown streaks on the stems and petioles, underdeveloped or aborted pods or pods with dark blotches. In tobacco, it causes ring and line patterns on the foliage, dwarfing and small leaves of poor quality. In other crops, it causes ringspot, mottling, necrotic or chlorotic spots, and ring or line patterns on the foliage, stunting, and yield loss. In daffodils, it causes leaf twisting and pale chlorosis of the center of the leaf.

Biology and epidemiology. Based on symptomatology and serology, distinct isolates of TRSV have been reported. TRSV is transmitted by *Xiphinema americanum* and closely related species (*X. americanum sensu lato* is now considered a complex of many species). *Thrips tabaci* nymphs are capable of acquiring and transmitting the virus and may be important. Possible other vectors include spider mites (*Tetranychus* spp.), grasshoppers (*Melanoplus* spp.), tobacco flea beetle (*Epitrix hirtipennis*), and aphids. Seed-transmission has been reported in several crop and weed hosts.

4.7.6 Tomato Black Ring Virus (TBRV, Genus: Nepovirus)

Geographic occurrence and impact. TBRV was described by Smith (1946) and reported in daffodils by Brunt (1966a) and Iwaki and Komura (1973); further information is available in Murant (1970c) and Brunt (1995). TBRV has a wide host range and naturally infects many species of wild and cultivated plants (one isolate was found to infect 76 species in 29 dicotyledonous families after mechanical inoculation). TBRV was found in daffodils in Scotland and south-west England and is now common in daffodils in north-west Europe. It occurs on daffodils in Japan and possibly occurs wherever daffodils are grown.

Symptoms. In addition to black ring disease of tomato, TBRV causes ringspot in many species and other varied symptoms such as shoot-stunting and mosaic. In daffodils, it causes no conspicuous symptoms alone but often occurs in complexes.

Biology and epidemiology. The tomato black ring strain is the type strain, with numerous other strains causing a wide variety of diseases across many horticultural crops and not reliably distinguished by symptoms. TBRV is readily sap-transmissible, infects the seed of many host plants, and is transmitted in soils by free-living, soil-inhabiting nematodes, *Longidorus* spp. Seed transmission is thought to occur in many host plants, though many plants infected through seed are symptomless; infection of the seed may provide a means of survival for the virus in the soil.

4.7.7 Tomato Ringspot Virus (ToRSV, Genus: Nepovirus)

Geographic occurrence and impact. ToRSV was described by Price (1936) and reported in daffodils by Iwaki and Komuro (1971). Further information was taken from Brunt (1995) and Stace-Smith (1984). ToRSV is associated mostly with ornamental and woody/semi-woody hosts, and it seems to be endemic to North America around the Great Lakes and along the Pacific seaboard, probably related to populations of its nematode vectors. There has been a serious decline in commercial fruit and ornamental crop production in the USA as a result of infections with ToRSV, making it one of the more damaging plant viruses on the continent, where its natural spread appears to be largely confined, though the virus has been spread to many other countries in infected plant material. ToRSV is prevalent in dandelion and may be disseminated over considerable distances by the wind-blown seeds. There may be field spread in Japan and the former-USSR, and isolations have been made from daffodils in Japan.

Symptoms/signs. Serious symptoms as a result of ToRSV include mosaic, decline, necrosis and ringspot in tree- and bush-fruits. Infected plants may show distinctive symptoms as a shock reaction, but chronically infected plants usually show a general decline in productivity without specific symptoms. In Japan, it was isolated from daffodils showing mild mosaic symptoms.

Biology and epidemiology. Variants giving slightly different symptoms have been found, but the best known variants are distinguished by the diseases they cause in the field. ToRSV is transmitted by the nematode *Xiphinema americanum sensu lato* and other *Xiphinema* spp. (*X. rivesi* in the eastern USA and *X. californicum* in the west). Experimentally it is transmissible to a wide host range of herbaceous and woody plants by inoculation with sap. Varying degrees of seed transmission have been reported in test species.

4.8 Tombusviridae

4.8.1 Narcissus Tip Necrosis Virus (NTNV, Genus: Carmovirus)

Geographic occurrence and impact. NTNV was first described in the Netherlands and then found to occur in England and Scotland (Asjes 1972; CJ Asjes, personal communication and WP Mowat and AA Brunt, personal communications cited in Moore et al. 1979; Mowat et al. 1977). Further information is available in Barnett and Murant (1970), Brunt (1975, 1995), Home and Ronchetti (1974), and Mowat et al. (1976). Daffodil is the only known host of NTNV, in which it occurs at high concentrations in important commercial cultivars in the Netherlands and UK; it probably occurs in all countries to which bulbs are exported.

Symptoms/signs. Symptoms appear after flowering. In some cultivars (e.g., 'Barrett Browning', 'Goblet', and 'Redmarley'), large necrotic lesions appear near the leaf tips and then spread basipetally. The tip necrosis symptom does not occur in all cultivars but is consistently associated with the virus in some; it may be suppressed by prolonged dry, sunny weather in which only mild symptoms develop. In 'Sempre Avanti', NTNV induces chlorotic and necrotic streaks mainly in the mid and upper parts of the abaxial leaf surface, and both symptoms are followed by premature foliar senescence. Some cultivars (e.g. 'Fortune' and 'Dutch Master') are infected but remain symptomless.

Biology and epidemiology. Virus isolates that differ slightly in serological tests have been found in naturally infected daffodils but no significant strain differences have been described. 'Barrett Browning' and 'Sempre Avanti' are reliable sources. No vector is known, but NTNV is readily transmissible to daffodils by inoculation; however, it failed to infect 46 species from fourteen families and the lack of suitable test species has inhibited its study. There is no evidence for spread by mechanical means, such as handling and flower cropping.

4.8.2 Tobacco Necrosis Virus (TNV, Genus: Unassigned)

Geographic occurrence and impact. TNV was described by Smith and Bald (1935) and reported in daffodil by Asjes (1974). Further information is available in Kassanis (1970). TNV occurs worldwide and is common in irrigated fields and

unsterilized soil in greenhouses. It infects a wide range of plants, including some vegetables and fruit. Experimentally it has been transmitted to numerous species in some 40 plant families.

Symptoms/signs. TNV usually causes necrotic lesions, rarely infecting systemically. The symptoms of TNV can be described by the resultant diseases and include tulip necrosis ("Augusta disease"), bean stipple streak, and one form of cucumber necrosis. It has also been obtained from pear trees showing ring pattern and bark split symptoms. Its symptoms in daffodils, however, do not appear to have been described.

Biology and epidemiology. Many strains of TNV have been distinguished by their symptomatology, host range, and serology. TNV is soil-borne and transmitted by zoospores of the root-infecting chytrid fungus *Olpidium brassicae*. The virus been obtained from the naturally infected roots of many species, but infects few species systemically. It does not survive in the resting spore of the fungus.

Management. In crops seriously affected by TNV, management strategies are likely to concentrate on controlling *O. brassicae* by fungicide, biocide, and surfactant treatments.

4.9 Virgaviridae

4.9.1 Tobacco Rattle Virus (TRV, Genus: Tobravirus)

Geographic occurrence and impact. First described as Mauche disease of tobacco by Behrens (1899), the causal virus was described by Quanjer (1943). TRV was identified in daffodils by van Slogteren (1958). Further information is available in Brunt (1966a, 1995) and Robinson (2003). The host range of TRV is very wide, including major crops (tobacco, potato, and sugar beet), many other cultivated species and weeds. More than 400 species in over 50 families can be infected experimentally, though in many cases the infection does not become systemic. TRV is known to occur in Europe, Japan, New Zealand, and North America. It was described in daffodils and other flowerbulbs (gladiolus, tulip, and hyacinth) in the Netherlands; later in the UK, it was found in daffodils in a small percentage of commercial crops and new cultivars.

Symptoms/signs. TRV causes a wide range of symptoms in the many species it attacks, including necrosis, mottle, mosaic, and stunting. In infected daffodils, some cultivars remained symptomless ('Minister Talma'), others ('Golden Harvest' and 'Thalia') showed a leaf mottling similar to that described by Broadbent et al. (1962), and the tazetta daffodil 'Grand Soleil d'Or' appeared immune, with the virus not being detected despite planting bulbs in soil with vectors and infested bulbs, even though mainstream cultivars succumbed.

Biology and epidemiology. Many weed species become naturally infected in their roots or systemically, while remaining symptomless act as a source of infection. The distribution of TRV is a reflection of its trichodorid vectors' preference for light sandy or peaty soils. At least 12 species of *Paratrichodorus* and *Trichodorus* are natural vectors of TRV, and there is specificity between the virus strain and the vector species. Up to 10% seed transmission occurs in *Viola arvensis* and lower frequencies in some other weeds. At least six dodder (*Cuscuta*) species can transmit the virus. 'Grand Soleil d'Or' appears to be immune, with the virus not being detected despite planting bulbs in soil with the vectors and infected bulbs, though mainstream cultivars succumbed.

4.10 Other Viruses

In addition to the viruses just described, several others have been identified in daffodils, largely through sequencing (Table 1). In some cases, no information is available about the symptoms or effects caused.

5 Nematode Diseases

5.1 Stem Nematode [*Ditylenchus dipsaci* (Kühn) Filipjev]

Geographic occurrence and impact. Stem nematode (also called stem and bulb nematode) is one of the most damaging plant-parasitic nematodes of temperate regions, attacking >450 plant species across most temperate regions and in the tropics where there is a high-altitude temperate climate (Hooper and Southey 1978; Lane 1984).

Stem nematode was well established in both the UK and Netherlands by the end of WWI: daffodil production might have died out but for the pioneering HWT work of Ramsbottom in 1918 (Gratwick and Southey 1986). Despite its largely successful management through routine HWT, stem nematode remains the daffodil crop's greatest threat. The risk lies in failing to treat all bulb stocks with HWT every time before re-planting, and in not using HWT according to best practice (such as using the optimum temperature and duration). Stem nematode infestation has been seen to destroy a bulb stock in 3 years. Multiplying rapidly at warmer temperatures in summer or in bulb storage, four nematodes can seriously damage a bulb in 1 year, while 50 can destroy it (Hesling 1967a, 1971).

Stem nematode is the only quarantine pest of daffodils within the EU, and statutory inspections of the growing crop and dry bulb samples are carried out by plant health inspectors before Plant Passports can be issued. UK crops that fail these inspections may be condemned to destruction, made subject to further HWT, or restricted to retail sales only. Bulbs for export outside the EU may be subject to additional inspections to satisfy the importing country's phytosanitary requirements.

Table 1 Further viruses reported for daffodils

Virus name	Daffodil name (and origin)	Symptoms (if reported)	Reference
Chinese narcissus virus	Daffodil (India)	Severe mottle, light green to yellow leaf stripes, loss of yield and quality	Yadav and Khan 2008
Cyrtanthus elatus virus A	Tazetta daffodil (India)	Severe chlorotic leaf stripe on ca. 40% of plants	Kumar et al. 2015
Lily mottle virus	Daffodil 'Pink Charm' (Netherlands) Daffodil, two cultivars (China)	–	Bo et al. 2008; Liu et al. 2008
Narcissus common latent virus (NCLV)	Daffodil (UK)	–	Chen et al. 2003a
	Narcissus tazetta var. *chinensis* (China)	Mosaic, mottle, white and yellow stripes; NCLV, NMAV, Narcissus White Stripe-Associated Virus and NDV also detected, symptoms may be due to mixed infection	Yan et al. 2008
Narcissus mottling-associated virus (NMAV)	*Narcissus tazetta* var. *chinensis* (China)	As under Narcissus Common Latent Virus	Yan et al. 2008
Narcissus Q virus	Daffodil (northern Europe)	–	S Phillips, WP Mowat and AA Brunt, unpublished information cited in Brunt 1995
Narcissus symptomless virus	Daffodil (New Zealand)	Leaf mottling, flower distortion and color break, with or without stunting; NLSYV and NDV also detected, symptoms may be due to mixed infection	Ward et al. 2009
	Daffodil (China)	–	Chen et al. 2006
Narcissus white stripe-associated virus	*Narcissus tazetta* var. *chinensis* (China)	As under Narcissus Common Latent Virus	Yan et al. 2008
Nerine latent virus	Daffodil (Australia)	–	Wylie and Jones 2012
Ornithogalum mosaic virus	*Narcissus bulbocodium* (UK) Daffodils (China, and imported samples)	–	HongYing et al. 2009
Shallot virus x	Daffodil (Russia)	–	Baikalova et al. 2003
Vallota speciosa virus	Daffodil (UK)	–	Chen et al. 2003a



Fig. 27 Daffodil bulb cut transversely to shown "brown ring" symptoms due to stem nematode, *Ditylenchus dipsaci* (compare with internal damage due to basal rot and bulb-scale mite) (Gordon R. Hanks © 2017. All Rights Reserved.)

In the USA, the stem nematode is also a regulated pest, and bulbs shipped out of state have to be inspected before shipment.

Symptoms/signs. The stem nematode eats its way through one bulb scale at a time, resulting in "brown ring" symptoms (Fig. 27). When the bulb is cut transversely, distinct rings can be seen, consisting of brown, degraded scale tissues among still healthy white scales, though eventually the whole bulb rots. The brown rings are unlike basal or neck rot, which spread through several scales simultaneously, or damage due to the earlier feeding of bulb-scale mites (*Steneotarsonemus laticeps*) which occurs at specific points (Fig. 28). When nematode larvae enter the shoots, they result in the characteristic lesions known as "spikkels": small, broken, roughened, swollen, yellow patches, usually along the leaf margin, or the keel of the flower stalk (Fig. 29). If slight, the spikkels may only be felt as an unevenness of the surface; if severe, they may surround dead tissue. In more heavily affected shoots where lesions coalesce, a spongy, broad, and thickened area can appear on a twisted, distorted, discolored leaf, especially below ground level. In the field, the first symptom may be a small patch of weak plants (Fig. 30), with the infected plants often retarded, producing a late-flowering patch in the field, but a light infestation may not show until late in the first season or during the next year. In forced bulbs, stem nematodes can destroy a lightly infected bulb in a few weeks. The roots are unaffected. Secondary infestations of bulb-scale mites, small narcissus flies, and infections by various fungi are likely to follow (Lane 1984).

If spikkels are not plainly evident, suspect bulbs should be dug-up for examination. A small piece of brown, infested bulb tissue, or tissue taken from a spikkel, macerated



Fig. 28 Daffodil bulb cut transversely to shown the characteristic feeding marks due to bulb-scale mite, *Steneotarsonemus laticeps* (compare with internal damage due to stem nematode) (Gordon R. Hanks © 2017. All Rights Reserved.)

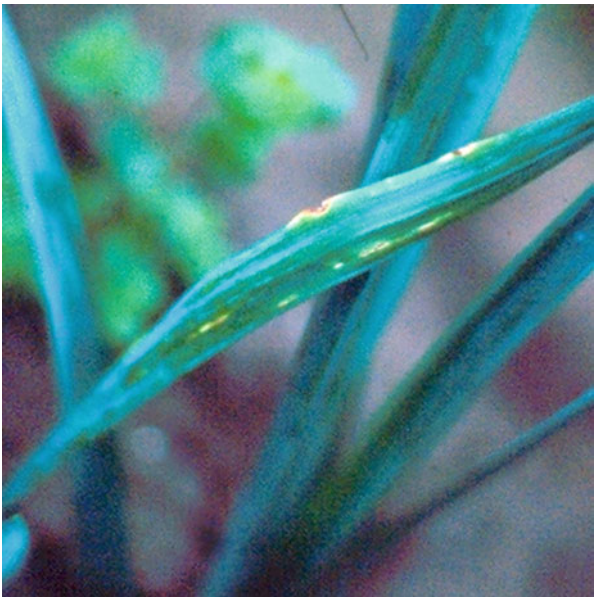


Fig. 29 "Spikkels" on daffodil leaves due to the stem nematode *Ditylenchus dipsaci* (University of Warwick © 2017. All Rights Reserved.)

in a drop of water and examined under a low-power dissecting microscope, will reveal large numbers of nematodes moving with a characteristic thrashing motion. Saprophytic nematodes may also occur, and since the species can only properly be identified through laboratory examination, expert diagnosis is necessary.



Fig. 30 Patch of weakly growing daffodils caused by a stem nematode (*Ditylenchus dipsaci*) infection (Gordon R. Hanks © 2017. All Rights Reserved.)



Fig. 31 Stem nematodes (*Ditylenchus dipsaci*) exuding from desiccated daffodil bulb as two globules of off-white "nematode wool" (University of Warwick © 2017. All Rights Reserved.)

If an infected bulb becomes dehydrated, nematode "wool" exudes from around the base plate, which sometimes becomes detached (Figs. 31 and 32), or occasionally from around the bulb neck; it appears as pinhead-sized blobs of off-white, wool-like material. Mycelium of the basal rot pathogen (*Fusarium oxysporum* f. sp. *narcissi*) may also be present, but this is fluffy and pure white. Wool, a desiccated survival stage of the nematode, is rapidly re-hydrated on contact with water and generally soon becoming active. After some treatments, however, the wool may at first remain inactive but nevertheless revives following overnight soaking, so the sample must be checked the next day (Hague and Kondrollochis 1969; Hastings et al.1952).



Fig. 32 Daffodil bulbs infested by stem nematode (*Ditylenchus dipsaci*), showing poor rooting and base plates coming away from the bulb scales (Ralph S. Byther © 2017. All Rights Reserved.)

Biology and epidemiology. In the soil, stem nematodes can generally move up to 1 m/year in moisture films, more in sandy or waterlogged soil, or in the standing water of flooded furrows. Infested patches spread more in the direction of cultivation (with cross-cultivation making matters worse) or surface water movement. In the field or in storage, spread can occur easily from infested debris and dust or flailed-off leaves, on wind, vehicles, and implements.

The transparent nematodes, up to 1.2 mm-long and generally not visible to the unaided eye, enter the bulb neck from the soil, usually via emerging leaves, or, sometimes, late in the growing season (Hesling 1967b; Lane 1984). The stem nematode completes its life-cycle within the bulb, in storage or in the field, laying up to 500 eggs and going through four larval stages. The first, second, and third molts progress rapidly, while the fourth-stage larvae are the infective stage that can survive adverse conditions such as desiccation. These larvae either enter the shoots or remain in the bulb scales, finally molting to adults that begin to reproduce. The life cycle takes about 20 days at 10–20 °C/50–68 °F and (if the host remains alive) can be continuous at this temperature. Reproduction continues in bulbs, either in the field or storage. In stored or decayed bulbs, the fourth-stage (pre-adult) larvae move to the outside of the bulb, forming wool, a mass of dehydrated larvae. The formation of wool occurs when the bulbs become desiccated during warm periods or prolonged storage. Wool is easily dispersed, resistant to desiccation and high temperatures, and can survive for 10 years or more without a host (Woodville and Morgan 1961). In the soil, stem nematodes are unlikely to remain dry for long: active stem nematodes can survive in the absence of host plants for about a year.

The biotypes of stem nematode have specific host preferences, but the daffodil, tulip, and hyacinth races all attack daffodils. The tulip race is highly pathogenic and has a wide host range, while the hyacinth race causes only minor lesions. Despite these distinctions, stem nematodes of most races can survive, or even breed slowly, in a wide range of plants: the narcissus race breeds slowly in onions, broad, French and runner beans, peas, and strawberries. Although brassica seedlings are a poor

host, the propagation and distribution of plug-grown transplants can spread nematodes. The narcissus race is not sustained in cereals and grasses, but may be maintained in many of the common weeds seen in bulb fields, such as black bindweed (*Fallopia convolvulus*), cleavers (*Galium aparine*), common chickweed (*Stellaria media*), common field speedwell (*Veronica persicae*), common groundsel (*Senecio vulgaris*), common knotgrass (*Polygonum aviculare*), and scarlet pimpernel (*Anagallis arvensis*) (Lane 1984).

Management. Because stem nematodes are so aggressive and (in the wool form) durable, their management requires strict adherence to an integrated crop management (ICM) approach involving appropriate cultural practices (including hygiene and crop inspection) and HWT (usually with a biocide) (Lane 1984). Management also needs to take account of basal rot and other fungal diseases, as many elements of cultural practices and HWT are common to both nematodes and fungal diseases. Sanitation is important at all stages to avoid contamination by the sometimes durable and easily spread bulb pathogens. The biological control of stem nematode does not appear to have been investigated. In cultivar tests, none of the varieties included proved immune to stem nematode (Hesling 1971), discouraging a plant breeding approach.

- ***Cultural practices*** – A rotation of at least 5 years (after volunteer plants from previous crops have been removed) should be used between daffodil crops. Regular crop inspections are important, with the isolation, removal, and safe disposal of infected and surrounding plants and soil within a 1 m–radius of the outbreak. Attention should be paid to maintaining sensible hygiene throughout, including cleaning and disinfecting equipment, tools, and work areas, and preventing spread on bulbs or crop debris. When infested plants are found, their handling, treatment or disposal must be arranged to avoid spread to other bulbs, and early-lifting and prompt HWT is usually recommended (alternatively, the area might be quarantined and all other bulb stocks lifted and treated first). Everything should be done to minimize wool formation: bulbs should not be allowed to dry out and should not be stored in warm conditions; if bulbs do dry out, presoak them before HWT.
- ***Hot-water treatment*** – General information on HWT, common to the management of stem nematode and basal rot, is given in Sect. 7, and only some nematode-specific points are discussed here. HWT was developed for the control of stem nematodes and should be applied vigorously, following the best current recommendations that will also control basal rot. The temperature, duration, and timing of HWT and the addition of a biocide are key factors, but despite the rigorous use of HWT since the 1930s, a few cases of stem nematode still occur, suggesting that some nematodes survive the treatment, re-infestation is occurring or, more plausibly, the treatment is failing in some way (Sect. 7.2). An important factor is that HWT is usually given only to daffodil bulbs intended for commercial

growing-on – those intended for retail sale or forcing are not treated, because HWT can damage the developing flowers; this practice must, however, have implications for the overall survival and spread of stem nematodes. There is contradictory information whether stem nematodes have become adapted to high temperatures (Hesling 1971; Wilkin et al. 1976; Winfield 1968, 1969, 1970; Woodville 1965; Woodville and Morgan 1961). Van Dam et al. (2014) investigated the number of stem nematodes in infested bulbs that survived various HWT regimes (presumably without formaldehyde). Three weeks after lifting, replicated bulb lots received warm-storage for 1 week at 30, 34 or 38 °C (86, 93 or 100 °F) before a 4-h HWT at 47, 48 or 49 °C (116, 118 or 120 °F). In 23 out of 27 cases, no nematodes survived (the number of nematodes in each untreated bulb varied from ca. 5,000 to ca. 28,000), and in the other four cases between 2 and 99 nematodes survived per bulb, but there were no clear trends between survival and treatment temperatures. In a further trial, HWT for 2, 3, or 4 h at 41, 43, or 45 °C (106, 109 or 113 °F) was given immediately after lifting; generally, this was less effective in killing nematodes.

With the likelihood of tighter regulation of the use of formaldehyde, alternative biocides were tested (Lole 1993; Hanks and Linfield 1997, 1999). Peroxyacetic acid, a biocide used in horticulture, was effective under HWT conditions for controlling stem nematodes and was crop-safe, but it did not become available to the industry for this purpose. Glutaraldehyde was also effective, but poses issues for human health. The fungicide thiabendazole, used to control basal rot but first introduced as an antihelminthic, was itself shown to be effective against stem nematodes (Hanks and Linfield 1997). In 2007, Lole et al. (2010) evaluated several biocides for their potential use in HWT and cold dips against stem nematode wool in vitro. Contrary to some earlier reports, HWT alone (3 h at 44.4 °C/111.9 °F) – with no biocide added – appeared sufficient to kill nematode wool (1 and 2 h treatments appeared to be effective initially, but some nematode revival subsequently occurred); the contrasting findings may have been the result of different test protocols. Subsequently, in testing biocide activity against nematode wool in a 3-h–cold dip (18 °C/64 °F), it was found that ascorbic acid, citric acid, a natural product didecyldimethyl-ammonium chloride, chlorine dioxide, and a silicone wetter were ineffective or only partly effective, while an iodophor disinfectant ("FAM 30") and hydrogen cyanamide each achieved a 100% kill. Hydrogen cyanamide was found to have phytotoxic effects on daffodil plants. The crop safety of "FAM 30" in HWT was subsequently confirmed in field trials (Lole et al. 2007, 2010; Hanks 2011a), and it has been used regularly in HWT in the UK since 2010, apparently successfully. However, "FAM 30" is not an ideal formaldehyde-replacement, because (a) the iodophor breaks down as the iodine reacts with the steel of the tank (GR Hanks, unpublished data, 2010) so there is a need for regular topping-up, (b) the use of dip-sticks to measure iodine concentration was unreliable, and (c) like any acidic material, there is the possibility of corrosive effects on HWT equipment. Several other formulations of phenolic iodophor biocides are available but do not appear to have been evaluated. As far as the authors are aware,

chlorine dioxide has not been tested specifically against stem nematodes in HWT.

- ***Nematicides and soil drenches*** – No nematicides are currently approved for use in bulb dips or HWT. In the USA, in-furrow applications of the nematicide fenamiphos to the bulbs at the time of planting have proved effective against the stem nematode (Chastagner 1997). On infested patches, soil treatment with a fumigant/sterilant such as dichloropropene, metham-sodium, or dazomet or drenching a small patch with xylenol, cresylic acid or another phenol may provide some reduction in nematode populations (Haglund 1991).

5.2 *Aphelenchoides subtenuis* (Cobb) Steiner & Buhrer

Geographic occurrence and impact. *Aphelenchoides subtenuis* (known as narcissus bulb and leaf nematode in the UK) was reported from daffodils on the Isles of Scilly in 1934 and was at first confined there and in Cornwall (Hodson and Gibson 1936). The extent of damage varied from year to year, and it had not been seen in the UK for many years until found on imported daffodils in 1974 (Lane 1984). It has been recorded in the Netherlands on daffodils 'Geranium' and 'Van Sion' (*N. telamonius* var. *plenus*) (Bergman et al. 1978; Lane 1984) and has caused damage on *Narcissus tazetta* var. *chinensis* in China (Lin 1987) and widespread damage on *Narcissus tazetta papyraceus* ('Paper White') in Israel (Vigodsky-Haas et al. 1985; Vigodsky-Haas and Lavi 1986). In the Netherlands it is a pest of crocus, and outside the UK it has occurred on several other flowerbulbs including ornamental *Allium*, *Colchicum*, *Iris*, *Scilla*, and tulip (Lane 1984).

Symptoms/signs. *A. subtenuis* infestation may show as blistering and crinkling of the outer bulb scales, and in bulbs cut across symptoms like those of stem nematode are found, though grayish and waxy. The leaves become heavily infested and turn prematurely yellow, but no spikkels are seen. After storage, the bulb scales are grey or brown, dry, and granular, and subsequent growth is poor. During the growing season, the leaves become heavily infected and turn yellowish and may die-down prematurely (Lane 1984). On tazetta daffodils in Israel, symptoms reported include a premature yellowing of the foliage, accompanied by a red-brown root rot and a dark, sunken rot of the basal plate (Vigodsky-Haas et al. 1985; Vigodsky-Haas and Lavi 1986).

Biology and epidemiology. Unlike other *Aphelenchoides* species which feed on flower stalks or leaves, *A. subtenuis* infects the roots. It is smaller than stem nematode and laboratory examination is required to identify it. Its spread is slow and infestations do not usually persist unless the stock is left down for several years or the same land is repeatedly used without a break (Lane 1984).

Mor and Spiegel (1993) found that *A. subtenuis* lays eggs in the root parenchyma during the winter. Juveniles spread throughout the parenchyma of the root cortex during the spring, coinciding with a rapid collapse of the root system and decay of

the basal plate at the peak of bulb and foliage production. Secondary pathogens infected the damaged bulb, causing it to rot. The disease was slow to spread and not usually persistent.

Management. *A. subtenuis* is managed through fumigation, rotation, hygiene, inspection and regular HWT (Lane 1984; Lavi et al. 1985). In China, a short HWT, for 45 min at 50 °C/122 °F or 25 min at 55 °C/131 °F, was partially effective and did not cause bulb damage (Lin 1987).

In Israel it was found that a fungicide dip reduced infections, despite being unable to link any fungal or bacterial pathogen to the disease, and HWT with a fungicide was more effective (Lavi et al. 1985). More extreme HWT regimes were more effective, 4 h at 45 °C or 2 h at 48 °C, as was late HWT (in September rather than August).

5.3 Root Rot and Soil Sickness: Root-Lesion Nematode [*Pratylenchus penetrans* (Cobb) Chitwood & Oteifa]

Geographic occurrence and impact. *Pratylenchus penetrans* attacks daffodils in association with the fungus *Ilyonectria destructans* (Zinssm.) Rossman, L. Lombard and Crous [syn. *Ilyonectria radicicola* (Gerlach and L. Nilsson) P. Chaverri and Salgado and *Nectria radicicola* Gerlach and L. Nilsson)], anamorph *Cylindrocarpon destructans* (Zinssm.) Scholten (Lombard et al. 2015). *P. penetrans* is the best known member of the genus and is found damaging a wide variety of crops (Corbett 1978). *Pratylenchus penetrans* and *Ilyonectria destructans* cause a root rot in daffodils, variously called "decline," "replant disease," or "soil sickness." It has been suggested that other genera of root-lesion nematodes, such as *Haplolaimus*, which feed on the roots from the outside, may be involved in the disease (Slootweg 1956). While the nematode is considered the primary cause, the fungus greatly extends the damage caused, and it has also been reported that the root rot can occur in the absence of nematodes (Lane 1984; Moore et al. 1979; Morgan 1970) – see "Root plate rot" (below).

The disease was recognized in the Netherlands in about 1914 (Westerdijk 1915) and in the UK from 1926 (Pethybridge et al. 1934). At one time, it was widespread in the Netherlands and sometimes serious in the UK, occurring most frequently on the Isles of Scilly where it is not easy to find un-cropped land for new plantings, but it was also reported from Cornwall, Devon and Lincolnshire. Similar "root declines" were reported from British Columbia (Hastings et al. 1932; Hastings and Bosher 1938) and South Africa (Dyer 1949). The disease occurs sporadically, but its impact is lessened because the nematode is confined to the roots and sensitive to dehydration, so the disease is unlikely to spread via the bulbs. The decline is slow and becomes apparent only if a stock is left down for many years.

Symptoms/signs. Especially in cultivars with fine roots, the fungus enters through wounds on the roots caused by the nematode, spreads, and leads to patches of stunted plants with brown, rotting and easily detached roots, severe rotting, water stress, and

premature senescence (Figs. 33 and 34). Root rotting caused by the fungus greatly extends the initial damage caused by the nematode, and if lifted the roots usually break-off short. The affected plants begin to lag behind the rest, and in a few weeks distinct patches of retarded plants can be seen. Examined early in the season, roots show small, black slit-like lesions containing nematodes and eggs (Lane 1984; Moore et al. 1979; Morgan 1970).



Fig. 33 Premature die-back of daffodil crop associated with infestations of the nematode *Pratylenchus penetrans* (six central ridges) (Gary A. Chastagner © 2017. All Rights Reserved.)



Fig. 34 Healthy, well-rooted daffodil bulbs on left, bulbs affected by the nematode *Pratylenchus penetrans* on right (Andrew A. Tompsett © 2017. All Rights Reserved.)

Biology and epidemiology. *Ilyonectria destructans* was almost always present in affected roots, and Gregory (1932) was able to isolate it from basal tissues of the bulb but unable to prove pathogenicity. However, Westerdijk (1915) had isolated the fungus from roots of affected plants and using it was able to induce rotting of healthy roots and base plates. Subsequently links with manganese deficiency and soil sulfides were suggested, but Gerretson et al. (1927) ruled out a link with soil factors and ascribed it to a fungus (which they did not identify). Feeks (1931) recorded *Nectria radicicola* as the commonest fungus present and was able to use it to induce root rot and then re-isolate it.

Management. The main control of the disease is regular HWT and crop rotation. Chemical controls include soil fumigation (Apt and Gould 1961), preplanting pesticide soil application and bulb fumigation (Wood and Foot 1982). Marigolds (*Tagetes* spp.) produce secretions that suppress *Pratylenchus* growth; it can be grown and cultivated into the soil before planting bulbs (Miller and Ahrens 1969; Oostenbrink et al. 1957).

Tompsett (2004, 2009) investigated soil sickness in tazetta 'Royal Connection' on the Isles of Scilly at two sites where *P. penetrans* and *I. destructans* were present. The treatments were growing *Tagetes* preplanting, soil treatment with 1,3-dichloropropene, and the combined treatment. The *Tagetes* was cultivated in after growing for 3 months. After two growing seasons, the combined treatment gave the highest crop vigor and lowest *Pratylenchus* numbers, while the single treatments also produced measurable improvements. After a third season, improved plant vigor was still evident in only one of the trials, though effects on nematode numbers were no longer apparent. The reason for the discrepancy in results between the sites is not known, but nematode levels fluctuated considerably over the 5-year trial period.

Root plate rot. Moore et al. (1979) described not only the "root rot" or "soil sickness" caused by *Pratylenchus penetrans* associated with *Ilyonectria destructans*, but also a "root plate rot" once known in south-west England, confusingly, as "basal rot" (Hodson and Beaumont 1930). It occurred in the Tamar Valley, where daffodils were grown on the warm, steep slopes, and was characterized by a lack of roots, accompanied by a brown decay of the base plate. Affected bulbs, scattered around the stock but not forming clumps, produced at most a few short roots, while the shoots were blind, stunted, or produced a short flower stalk with the flower low in the leaves; the bulb scales remained white and sound though prone to colonization by bulb mites and fungi (McWhorter and Weiss 1932). The disorder was common in (but not confined to) "van Sion" (*N. telamonius* var. *plenus*) and *N. obvallaris* and appeared much worse in badly drained soil. It was bulb-borne, but if stocks were lifted and replanted elsewhere in better conditions, they sometimes recovered (Moore 1939; Moore et al. 1979). Various fungi, but especially *I. destructans*, were among the fungi associated with affected bulbs, and this may support Morgan's (1970) suggestion that root rot may occur in the absence of nematodes.

6 Physiological Disorders

Most crops suffer from some disorders for which, despite their often disease-like symptoms, no causal organism has been identified. Some may be caused by specific environmental conditions, while others may be the result of poor husbandry: either way, the plant's normal growth and development are disrupted. Weather can also produce a variety of "disease-like" symptoms. One particular type of damage seen in daffodils is that due to HWT: this is described in Sect. 7.5. Because the symptoms of some disorders and conditions can be confused with those of more familiar diseases, it is important to distinguish them so that the correct remedy (if there is one) can be applied and unnecessary pesticide applications avoided.

6.1 Chocolate Spot

Chocolate spot of daffodils has been known for many years in the Netherlands, USA, and UK. It is characterized by dark chocolate-brown spots and streaks on flower stalks and leaves. Its incidence and severity is usually low, and chocolate spot is not known to have ever caused any significant problem with product quality (Fig. 35). Although little research has been published, chocolate spot was investigated in the 1970s by examining lesions for potential pathogens and analyzing nutrient concentrations in affected and normal plants and their soil (Moore et al. 1979; Rees 1972). Initial symptoms typically appear late in the season after flowering, and Moore et al. (1979) suggested the appearance of chocolate spot is associated with increasing ambient temperatures. In Scotland, it was reported that "chocolate spot was observed in virus-tested [daffodil] clones . . . it may be a physiological disorder induced by environmental conditions" (Mowat 1983). In the Netherlands, virus infection was linked with sunny weather in predisposing daffodils to this disorder (Aartrijk et al. 1995).



Fig. 35 Three examples of chocolate spot, a physiological disorder of daffodils, on foliage and flower stalks ((**a**) the University of Warwick, (**b**) Ralph S. Byther, and (**c**) Gordon R. Hanks © 2017. All Rights Reserved.)

6.2 Rust

"Rust" – sometimes called "physiological rust" or "stem rust" – has raised sporadic interest in the UK since the early-1990s. Small, rust-colored lesions arise on the flower stems around the time of flowering, in most instances comprising one or a few inconspicuous lesions that would normally pass unnoticed. On rare occasions, numerous lesions arise and may be conspicuous at flower picking: in the worst cases small cracks appear across the stems, which may become bent, brittle, and, very rarely, unmarketable. A range of symptoms is shown in Fig. 36. Although sharing some features with chocolate spot, rust appears to be a distinct condition. It does not appear to be associated with a pathogen or nutritional imbalance, and the effect of adverse water relations is being investigated (GR Hanks, unpublished data).



Fig. 36 Examples of daffodil rust, a physiological disorder, on cut-flower stems ((*above*) Gordon R. Hanks and (*below*) Andrew A. Tompsett © 2017. All Rights Reserved.)

6.3 Grassiness

Grassiness occurs when the main shoot of a bulb is killed, resulting in the production of many small daughter bulbs (Fig. 37). Moore (1979) indicates that factors such as attacks by narcissus flies or bulb-scale mites, or damage associate with too severe or too late HWT, are often responsible for the death of the main shoot. The daughter bulbs are often rather elongated and are sometimes referred to as "horse's teeth." This generally occurs at a low frequency in field-grown daffodils, with affected bulbs producing very few flowers but a large number of leaves.

6.4 Disorders of Flower Development

Normal flower development can be disrupted in several ways, and this probably happens at a low level wherever daffodils are grown and despite good husbandry, though in specific cases it can be a problem. Bud death can occur shortly before spathe-splitting, leaving a dying bud within a desiccating spathe, a problem in double cultivars during greenhouse forcing and particularly of 'Golden Ducat' which is prone to the disorder even when growing in the field (Fig. 38). Dissections



Fig. 37 Extreme examples of "grassiness" in daffodils following damage to the main growing point, with (*above*) production of many small offsets ("horse's teeth") and (*below*) many thin, grassy leaves, in this case caused by HWT damage (University of Warwick © 2017. All Rights Reserved.)



Fig. 38 Dead buds, a common disorder of flower development in double cultivars, especially when forced in greenhouses (University of Warwick © 2017. All Rights Reserved.)



Fig. 39 Late death of flower bud with failure to emerge from spathe, common in *Narcissus poeticus* 'Flore Pleno' (University of Warwick © 2017. All Rights Reserved.)

of the flower stalk and bud suggested plants were unable to translocate sufficient water to support the rapid growth of a double cultivar's dense bud tissues, but no simple preventative measure was identified (Hanks 1992b). Bud death in *Narcissus poeticus* 'Flore Pleno' ('Tamar Double White') is probably an example of the same



Fig. 40 'Bullhead', a disorder of daffodil 'Cheerfulness': normal flowers on left, with affected flowers showing non- or partially opening buds, corolla tube that does not extend, and distorted perianth segments with green areas (Andrew A. Tompsett © 2017. All Rights Reserved.)

disorder (Fig. 39). This cultivar was a specialty crop of the Tamar Valley, UK. Extreme temperatures, hot, dry growing seasons, wet autumns, poor root development, and adverse water relations were suggested as predisposing factors (Moore et al. 1979; Rees 1972; Tompsett 1972). Irrigation, mulching, and shading treatments did not prevent bud death. 'Bullhead' results in abnormal flowering in another double cultivar, 'Cheerfulness': some 10% of flowers may fail to emerge from the spathe, and there are additional or dissected perianth segments, petalloid styles, and stunted flower stalks (Fig. 40). High temperatures were suggested as a possible cause (Moore et al. 1979; Rees 1972; Tompsett 1972). Since the 1980s, another disorder appears to have become more common on tazetta daffodils in south-west England. Known locally as "melting" or "nubbles," the terminal florets either fail to emerge from the sheath or emerge reduced or distorted (Fig. 41; Tompsett 2002). Finally, during greenhouse forcing, some cultivars (especially 'Carlton') are prone to producing perianth segments with notched margins (Fig. 42). Many of these symptoms might be confused with damage due to HWT.

6.4.1 Damage Due to Weather

There are numerous effects of adverse weather resulting in crop damage, much of which may look similar to the effects of foliar diseases (Hanks 2013a). Hail causes white flecks on the leaves and can physically damage flower buds, occasionally with substantial losses (Fig. 43). Waterlogging or frozen soil, if prolonged, can have many effects, from leaf damage (yellow banding, roughened areas) to stunting. Shepherd and Parkins (1954) described the damage from cold weather in west Cornwall, an



Fig. 41 "Melting," a disorder of tazetta daffodils, with small, distorted and nonemerging florets (Andrew A. Tompsett © 2017. All Rights Reserved.)



Fig. 42 Abnormal petal margins with notches, a disorder of flowering common in daffodil 'Carlton' (University of Warwick © 2017. All Rights Reserved.)

area with a mild climate that is usually frost-free. Frost damage can show as mild chlorotic banding across the leaves and flower stalk, corresponding to periodic episodes of cold weather causing alternate slower and faster growth during flower stalk elongation (Figs. 44 and 45); if progressively more severe, the results are damaged leaf-tips, nonopening flower buds, and collapse of the leaves and flower stalks that may be temporary or permanent. Tazetta daffodils are more seriously



Fig. 43 Daffodils showing damage due to hail: (**a**) inconspicuous white flecking, (**b**) dead leaf tips, and (**c**) wholesale withering and death of buds, stems and leaves, the result of severe hail (Gordon R. Hanks © 2017. All Rights Reserved.)

damaged by frosts. Finally, if bulbs are left exposed to strong sunlight (e.g., when lifted and left on the surface of the soil to dry "naturally"), they can be internally damaged by "sun scorch," resulting in damaged leaf-tips and even blind flowers.

6.4.2 Damage Due to Herbicides

A few adverse responses to herbicides have been reported in daffodils (Hanks 2013a). They include reduced growth, the production of small, star-like flowers and flowers with deformed tepals and coronas, and bulbs with internal damage (Fig. 46). Some of these effects on flowers particularly are reminiscent of forms of HWT damage. Herbicide recommendations and conditions of use should always be followed and any unexplained crop damage investigated.

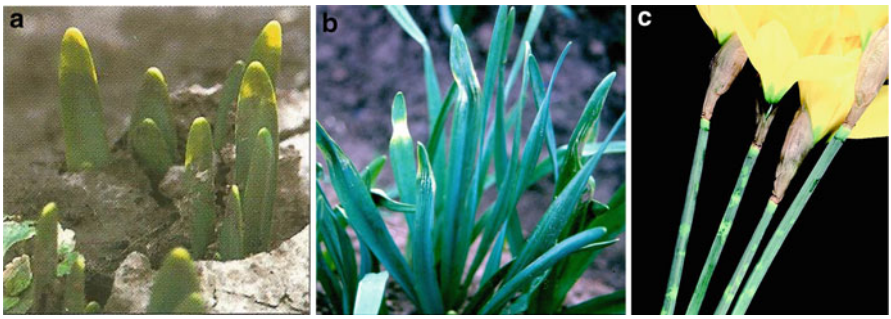


Fig. 44 Daffodils showing (**a, b**) damage due to frost at or soon after emergence, and (**c**) damage due to periodic episodes of cold weather during stem elongation ((**a, b**) the University of Warwick and (**c**) Ralph S. Byther © 2017. All Rights Reserved.)



Fig. 45 Daffodil crop showing the effects of frost just before flower picking (Gordon R. Hanks © 2017. All Rights Reserved.)

7 Hot-Water Treatment

7.1 Introduction

It has been known since the 1930s that stem nematode (*Ditylenchus dipsaci*) and basal rot (caused by *Fusarium oxysporum* f. sp. *narcissi*) can occur together in daffodil bulbs and that both can be routinely managed by HWT. In this process, stem nematodes are controlled primarily by the high temperature per se, though adding a biocide may augment nematode control. On the other hand, *F. oxysporum* f. sp. *narcissi* is controlled primarily by a biocide or fungicide added to the HWT tank



Fig. 46 Examples of herbicide damage on daffodils: (**a** and **b**) small "starry" flowers, and (**c**) damage to the base plate and bulb scales (University of Warwick © 2017. All Rights Reserved.)

(a more efficient option than having a separate fungicide dip), though the high temperature per se probably augments control (Sect. 2.1 "Fungicides, biocides and HWT"). The HWT process also controls some pests (such as bulb-scale mites, bulb mites, and large narcissus fly larvae), diseases (such as *Botrytis narcissicola*), and microorganisms (important for general sanitation). The basics of HWT are described here, and specifics for basal rot and stem nematode are given in Sects. 2.1 and 5.1.

HWT was developed in the 1920s to control nematodes in chrysanthemum stools, strawberry runners, and other plant material, but its main use now is in daffodil growing, where the HWT equipment is the major farm facility entirely devoted to the bulb crop (Gratwick and Southey 1986). Daffodil bulbs are immersed in water heated to the recommended temperature for the specified duration in a regime that seeks to achieve effective management of stem nematode without either compromising any simultaneous fungicide or biocide treatment or causing an unacceptable level of thermal injury to the bulbs. Some degree of bulb damage does appear to be inevitable, though there are ways of minimizing it, and the consequence of a stem nematode infestation is far worse. To be effective, all daffodil stocks intended for

commercial growing-on should receive the recommended HWT in the run-up to planting, and in the USA and the UK the normal practice is to treat bulbs every year they are lifted and re-planted, irrespective of whether an infestation has occurred or been suspected or not, and the relative rarity of stem nematode infections has been attributed to this practice. In the Netherlands, daffodils are more likely to be lifted annually, and a shorter, cooler regime was routine at one time, with a "full" HWT used only when an infestation was suspected or known.

7.2 Temperature and Duration of HWT

In the early-twentieth century, immersion treatments of 1–6 h at ca. 49 °C/120 °F were tested, but with the simple equipment available, such treatments were mostly as lethal to the bulbs as to the nematodes (Gratwick and Southey 1986). As equipment improved and growers sought better control, HWT was increased from 3 h at 43.5 °C/110.3 °F in the 1920s (Hastings and Newton 1934; Ramsbottom 1918, 1919; Slootweg 1962; Staniland 1933; Staniland and Barber 1937; Tompsett 1982; Turquand 1966) to 4 h at 43.5 °C or 3 h at 44.4 °C/111.9 °F in the 1930s (Chitwood and Blanton 1941; van Slogteren 1931; Woodville 1964; Woodville and Morgan 1961). Many of these regimes were based on in vitro determinations of the killing times for *Ditylenchus dipsaci*, and Southey (1978) pointed out that the great variation in results was unsurprising, considering the variations in methodology and nematode development stages used and that the researchers were attempting to determine 100% killing times, observing that "it [seems] surprising that [HWT] has been so generally successful." Winfield (1970) and Southey (1978) noted that determining the time taken to kill stem nematodes suspended in water was misleading, since desiccated nematode wool on the outside of bulbs in water was more difficult to kill than nematodes within the bulb tissue (Green 1963; Hastings 1933): adding formaldehyde was an effective way of dealing with these external nematodes. Winfield (1970) estimated that to achieve the high standard of control required, without formaldehyde, even a 4–5 h treatment at 45 °C/113 °F might not be relied on to kill nematode wool if the bulbs had been allowed to become too dry, say by warm-storage or by storing at too low a humidity. He concluded that a regime of 3 h at 43.9–44.4 °C (111–112 °F should be used, but with the proviso that it could not be relied upon to kill nematode wool unless formaldehyde were added.

HWT was evaluated with and without the addition of formaldehyde for stem nematode control in California (Qiu et al. 1993). The standard HWT regime, 4 h at 44 °C/111 °F, was confirmed as successfully controlling nematodes in the absence of formaldehyde, while with formaldehyde added a 2.5-h-long treatment was effective; both regimes were crop-safe. Using formaldehyde and elevating the temperature to 1.5 h at 46 °C/115 °F, 0.75 h at 48 °C/118 °F, or 0.5 h at 50 °C/122 °F was sufficient to control stem nematodes, but with some damage to the plants.

The 3 h, 44.4 °C/112 °F regime became the standard for the UK, with 3–4 h at 43–44 °C/109–111 °F the standard in the USA (ADAS 1985; Chastagner and Byther 1985). In the Netherlands, where many daffodils were grown on an annual basis, and

some nematode management was achieved through soil fumigation and control of the water table, a treatment of 2 h at 43 °C/109 °F became the standard. For daffodils grown on a 2-year-down basis, or where a stem nematode infection was suspected, the treatment was increased to 4 h at 47 °C/116 °F (reclaimed ex-forced bulbs, which may have been exposed to warmer temperatures and desiccation, were treated for 4 h at 48 °C/118 °F in advance of the main HWT season) (van der Zwet et al. 1990). In these cases, HWT was given within 3 weeks of lifting and followed warm-storage (1 weeks at 30 °C/86 °F) and presoaking for 24 h. In recent years, with instances of stem nematode infections still being seen occasionally in the Netherlands, the HWT-tolerance of daffodils was revisited. Vreeburg et al. (2013, 2014) tested some cultivars (mainly 'Tête-à-Tête', 'Dutch Master' and 'Tahiti') that had been treated for 4 h at 45, 47, 49 or 51 °C/113, 116, 120 or 124 °F within 3 weeks of lifting and following prewarming and presoaking as described. 'Tête-à-Tête' and 'Dutch Master' tolerated treatments at 47 and 49 °C without yield loss, but 'Tahiti' no more than 47 °C; higher HWT temperatures produced extensive damage. Using a higher warm-storage treatment, 34 or 38 °C/93 or 100 °F resulted in less damage to the leaves and flowers but had no benefit on bulb yields. Alternatively, if HWT were carried out soon after lifting and without warm-storage or presoaking, a 2-, 3- or 4-h treatment at 45 °C/113 °F was well tolerated by 'Tête-à-Tête' and less so by 'Dutch Master' and especially 'Tahiti'. Anecdotally, temperatures up to 49 °C/120 °F have been used for HWT in the UK where the outdoor cut-flower crop is very important, but it should be noted that such temperatures will increase flower damage and, although prewarming and presoaking the bulbs before HWT will reduce this damage, both warming and soaking encourage stem nematode multiplication, wool formation, and spread. There are also anecdotal reports of growers heating the HWT tanks to ca. 70 °C/158 °F between bulb treatments to improve sanitation; it would be critically important to get the dip temperature back to recommendations before immersing the next batch of bulbs. The optimum temperature needed to ensure the kill of fungal pathogens should be determined.

For optimum effect, HWT needs to be used according to the current recommendations, avoiding both economies (shorter treatment, say) and overkill (such as a higher temperature "to ensure effectiveness"). It is usual to start timing HWT from when the water in the treatment tank reaches the target temperature (this time can be minimized by slightly over-heating the water before it is pumped back from the holding tank to cover a new load of bulbs). Hanks and Collier (2012) investigated the reasons for the apparent survival of some bulb-scale mites following regular HWT, recording temperatures at the bulb centers during HWT. Bulb-center temperatures lagged behind that of the circulating water by 10–15 min in "medium-sized" bulbs, and as a result a regular regime of 3¼ h at 44.4 °C/111.9 °F was recommended. Using well-designed, modern HWT tanks, the temperature of the circulating dip was found to be uniform across the tanks and within the bulk bins in them. Apart from the temperature lag at the center of the bulb, the chief problem in achieving the desired time at the target temperature was the poor calibration and setting of temperature sensors and controls. HWT temperatures should be monitored with a suitably calibrated instrument having its bulb or sensor appropriately sited among the

immersed bulbs. At the end of HWT, it is normal to rapidly ventilate, cool, and surface-dry the bulbs under powerful fans and to avoid excessive exposure to high temperatures the bulbs should be removed promptly from the tank after pumping-out the dip and allowing a short drain-down period. It was also suggested that bulbs might be treated grade-by-grade, so that large bulbs could receive proportionally longer treatment. This point had already been made by Qiu et al. (1993), who provided an equation to calculate the additional time required. For a 44 °C/111 °F treatment, the time in minutes to be added to the basic 3 h treatment was (−15 + 3.4c), where c is the bulb circumference in cm; in this case a bulb of 16 cm circumference would require an additional 39 min of treatment.

7.3 Additives to HWT Tanks

The routine use of formaldehyde in HWT tanks – as an adjunct to the high temperature treatment itself – continued until restrictions were placed on its agricultural use in the USA in the 1990s and it was banned in the EU in 2008 (in the USA growers have since been permitted to use formaldehyde under certain conditions). Although some screening of alternative biocides has taken place, no entirely satisfactory alternative to formaldehyde has been found. The current status of other biocides for the control of the basal rot pathogen and stem nematodes, and of fungicides and pesticides used in HWT, are described in Sects. 2.1 and 5.1. While an iodophor disinfectant is now being used in the UK, it is not an ideal material: (a) the iodophor breaks down as the iodine reacts with the steel of the tank (GR Hanks, unpublished data) so there is a need for regular topping-up, (b) using dip-sticks to measure iodine concentration is difficult, and (c) as with any acidic material there is the possibility of a corrosive effect on the HWT equipment. Chlorine dioxide is a possible alternative. It is normal to add a nonionic wetter to HWT tanks and, if the production of foam is a problem, an antifoaming agent.

7.4 Timing of HWT

The date of HWT is also critical. If applied too soon, the young flowers and leaves will be damaged by the treatment, and if applied too late, the root initials will be damaged. In the UK, for example, HWT should start in mid- to late-July in south-west England and late-July to early-August in eastern England, aiming to finish by middle- to late-August. Daffodils with fine or early roots (e.g., poeticus, cyclamineus, or jonquilla cultivars) are usually treated before the standard cultivars. There is no reason to delay HWT, but if this is unavoidable for practical reasons, bulbs should be stored for 2 weeks at 18 °C/64 °F before treatment (Hanks 1993b). Earlier HWT, however, sometimes has benefits: daffodil stocks known to be infected with stem nematodes should be lifted early and treated soon after lifting, avoiding any intervening storage at elevated temperatures (Hastings and Newton 1934; Vreeburg and Korsuize 1991; Winfield and Hesling 1966).

7.5 Damage Due to HWT

Treatments that are effective in controlling stem nematodes inevitably cause some damage to the developing shoots and roots that is manifested in the next growing season. Damage can be kept to a minimum by following protocols, primarily on the temperature, duration, and timing of HWT (Figs. 47, 48, and 49). Mild thermal damage is typified by the roughened surface of leaf tips, splits, and malformations to the perianth segments and corona, and small starry flowers. More serious damage includes distortion of the leaves and flowers, death of the flower buds, and – most seriously – damage to the growing root initials. These symptoms need to be recognized and distinguished from those due to other causes: damage to the leaf surface can resemble some symptoms of virus, distortion of the perianth segments can resemble some types of herbicide damage, while death of the flower bud or roots may be caused by several other factors. Plant growth and development in subsequent years is unaffected.

HWT too late can lead to dead buds, severe leaf damage with stunting and loss of yield, and root damage also causing yield loss. Severe leaf damage may be exacerbated by storing the bulbs at too cool a temperature before HWT, or by using excessively hot or long HWT (Hanks 2013a). Overvigorous HWT can kill the



Fig. 47 Mild and moderate damage to daffodil flowers due to HWT, with (*above*) ragged margins to tepals and (*below*) small "starry" flowers with damaged coronas (University of Warwick © 2017. All Rights Reserved.)



Fig. 48 Serious damage to daffodil plants due to HWT: (*above*) leaf roughening and mottling and (*below*) flowers buds dead in spathes ((*above*) University of Warwick and (*below*) Gordon R. Hanks © 2017. All Rights Reserved.)



Fig. 49 Normal daffodil bulbs on left, bulbs on right with severe damage to roots and base plate caused by adverse HWT (University of Warwick © 2017. All Rights Reserved.)

stem apex, resulting in the abnormal production of extra bulb units and small, "grassy" plants (Edwards 1965; see Sect. 6.2). Where formaldehyde is added to HWT tanks, early treatment (while active roots are still present) or using too high a

concentration are reported to result in "corky" areas of the base plate and deformed or fewer flowers (Briggs 1988; Linfield 1991; Price and Briggs 1976).

Some procedures are available to reduce HWT-related damage and are employed in cases where the first-year flower crop is important. Before HWT the bulbs can be "prewarmed" (e.g., for 5–7 days at 30–35 °C/86–95 °F), following this by "presoaking" (immersion in water at ambient temperatures overnight or for a minimum of 3 h) and raising the HWT temperature to 46.0 °C/115 °F. Prewarming retards shoot growth – evidently only growing tissues are affected by thermal injury – and presoaking wets and reactivates stem nematodes that have changed into the "wool stage" as a result of the previous warm period, while the higher HWT temperature deals with the more heat-resistant "free-swimming" nematodes that have escaped the bulb during these processes. The lower limit of the prewarming effect is 18 °C/64 °F, and a 2-week period at this temperature (especially as an alternative to storage at cool summer temperatures) can be used to curtail HWT-related damage when HWT is unavoidably late (Hanks 1993b). Where a stem nematode infestation is suspected; however, such prewarming and presoaking procedures should be avoided.

8 Concluding Remarks

The management of diseases of daffodil faces a number of challenges. While new molecular tools have resulted in tremendous progress in our understanding of pathogen biology and ecology, host-pathogen interactions, pathogen identification, disease diagnosis, etc., there are several impediments that continue to limit the development of improved, more ecologically acceptable and economically viable disease management programs for growers (Chastagner et al. 2012; Kamenetsky et al. 2015). For example, despite a long history of R&D, two diseases with potentially serious consequences dominate any discussion of daffodil diseases: basal rot and stem nematode. Hot-water treatment (HWT) – immersing bulbs for a few hours in hot-water with biocide and/or fungicide – is the principle means of managing both. Increasing the thermal efficiency of HWT and using biopesticides and/or biodegradable biocides would be of great benefit to growers.

The bulbs industry has been a heavy user of pesticides, but the need for developing ICM in flowerbulb production has been recognized in the Netherlands (Raven and Stokkers 1992) and good prospects for integrated systems have been indicated (Wondergem et al. 1999). As indicated throughout this chapter, pesticides continue to play an important role in the management of most diseases of daffodils. However, growers have achieved substantial reductions in chemical inputs – fertilizers and pesticides – and some unpleasant chemicals are no longer used. Optimizing disease resistance through breeding programs, developing economically effective ways of stimulating natural host defense systems, and developing biopesticides that provide consistent levels of disease control under field conditions are needed.

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Diseases of Lily

41

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Abstract

Lilies (*Lilium* spp. and hybrids) are the second largest flower bulb crop in the Netherlands and the most important flower bulb crop in the world. They are grown for bulbs, as cut flowers, as container (potted) plants, or in gardens. Lilies are a vegetatively propagated crop and may be routinely increased by micropropagation (tissue culture) or through scaling (removal of bulb scales from which adventitious bulblets are produced). The propagules are subsequently grown in fields for 2 or 3 y to allow the bulbs to reach sufficient size (generally measured in circumference) for proper flowering and maximum horticultural quality. Although the majority of bulbs produced for worldwide distribution are grown mainly in the Netherlands, bulbs are also grown in other northern European countries, the USA, Asia, Israel, and southern hemisphere locations including Brazil, Australia, New Zealand, Tasmania, and Chile. There are a number of diseases caused by fungi, viruses, bacteria, and nematodes and physiological disorders that affect the production and quality of lily bulb and cut

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R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_45

flower crops worldwide. Information on the distribution, epidemiology and management, of the common diseases and disorders is presented in this chapter.

Keywords

Lilium • Cut flowers • Plant pathogens • Physiological disorders • Disease management

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1 Introduction

Lilium is a large, horticulturally and genetically complex genus comprising over 100 species (Beattie and White 1993; Okubo 2014; Wu et al. 2014). With the exception of *L. longiflorum* cvs., essentially all of the commercially important cultivars are interspecific hybrids that have been separated into a number of main cultivar groups, the most important being the Asiatic hybrids (Sinomartagon section, derived mainly from *L. bulbiferum*, *dauricum*, *davidii*, *maculatum*, and *tigrinum*), Oriental hybrids (Archelirion section, derived mainly from *L. auratum* and *speciosum*), LA-hybrids (derived from *L. longiflorum* and one or more Asiatic hybrid cultivars), and more recently, OT-hybrids (derived from Oriental hybrids and the trumpet hybrids (derived from *L. regale* and other trumpet-shaped hybrids). Numerous reviews of the genetics and breeding efforts to derive these groupings have been published (Van Tuyl and Arens 2011). The horticultural characteristics of these groups, and their individual cultivars, vary a great deal, and this has implications for susceptibility to various diseases and physiological disorders.

Lilies are the second largest flower bulb crop in the Netherlands and the most important flower bulb crop in the world (Benschop et al. 2010; Kamenetsky and Okubo 2012). They are grown for bulbs, as cut flowers, as container (potted) plants, or in gardens. Accurate statistical breakdowns within these three categories are unavailable, but worldwide probably less than 1% of all bulbs produced are used in gardens and probably less than 10% of bulbs used in pots. Thus, ca. 90% of the worldwide lily bulb crop is used as cut flowers.

Lilies are a vegetatively propagated crop and may be routinely increased by micropropagation (tissue culture) or through scaling (removal of bulb scales from which adventitious bulblets are produced). The propagules are subsequently grown in fields for 2 or 3 y to allow the bulbs to reach sufficient size (generally measured in circumference) for proper flowering and maximum horticultural quality.

Worldwide, the modern lily industry is highly concentrated within the Dutch sphere of influence, with the major breeding companies, bulb production companies, and exporters all being Dutch-controlled. Whereas the majority of bulbs produced for worldwide distribution are grown in northern European fields, many are also grown in southern hemisphere locations including Brazil, Australia, New Zealand, Tasmania, and Chile. For various horticultural reasons, southern hemisphere bulb production is advantageous for the earliest forcings in the northern hemisphere.

The worldwide increase in the production, global movement, and consumption of lilies and improved diagnostic tools has resulted in an increasing number of reported plant pathogens on this crop. The global movement of planting stock also increases the risk of spread of newly emerging pathogens. This is presently highlighted by the *Plantago asiatica mosaic virus* (PlAMV) that is currently affecting many lily cultivars and stocks worldwide. The symptoms of this virus can be, and probably were, mistaken for a severe nutrient deficiency, an unknown fungal disorder, or possibly a chemical phytotoxicity before its etiology was determined, and it had been spread around the world.

This chapter provides an overview of the most common fungal, bacterial, viral, and nematode diseases and physiological disorders of lilies. Readers are encouraged to also consult the following references: Boerema and Hamers (1988), Chastagner et al. (2012), Conijn (2014), Daughtrey and Benson (2005), Dimock and Tammen (1967), Gould (1957), Lawson (2011), Lawson and Hsu (1996), Lim and van Tuyl 2006, Linderman (1985), Loebenstein et al. (1995), McRae (1998), and Moore et al. (1979).

2 Fungal and Fungus-Like Diseases

2.1 Botrytis Blight/Fire [*Botrytis elliptica* (Berk.) Cooke] (Teleomorph *Botryotinia elliptica* Terhem, Staats, van Kan) *and Botrytis cinerea* Pers. (Fr.) [Teleomorph *Botryotinia fuckeliana* (de Bary) Whetz.]

Geographic occurrence and impact. Botrytis blight (also known as "fire") affects lilies nearly everywhere they are grown and is considered to be the most important foliar disease on this crop (Conijn 2014; Linderman 1985; McRae 1998; Moore et al. 1979; Ward 1888). *Botrytis* can affect all aboveground parts of the plant: leaves, stems, and flowers. Due to its ability to cause disease on floral tissues under storage conditions (Droby and Lichter 2007; Miller 1993), *Botrytis* is both a pre- and postharvest concern for growers. Fire can completely destroy a planting of lilies, reducing bulb growth and making cut flowers or pot-grown plants unmarketable.

Symptoms/signs. Typical initial symptoms are small water-soaked spots that expand into circular- or elliptical-shaped brown leaf spots that can appear anywhere on the leaf. The color of the spots varies with leaf texture, the lily species affected, and from the center to the edge of the spot. Yellow streaks are often associated with these spots (Doss et al. 1988b). As the spots increase in size, the foliage is killed and hangs down (Fig. 1). The severity of disease is generally greater on lower leaves. Symptoms on flowers consist of tan to brown spreading spots on buds and petals.



Fig. 1 Leaf spots (*left*), blight (*center*), and spotting of flower petals associated with *Botrytis* infections (Gary A. Chastagner © 2017. All Rights Reserved.)

Dead tissue may be covered by a woolly, gray growth during moist periods. Small black sclerotia may develop on blighted tissues, particularly late in the season.

Biology and epidemiology. *Botrytis elliptica* and *B. cinerea* are the most common *Botrytis* species to infect lily (Boerema and Hamers 1988; Staats et al. 2005, 2007; Tsukiboshi 2002; Ward 1888). *B. elliptica* is generally considered to be a host-specific pathogen, but has also been reported on autumn crocus (*Colchicum autumnale*) (Moore et al. 1979; MacLean 1948), daylily (*Hemerocallis fulva*, Hwang 2001), dogstooth violet (*Erythronium grandiflorum* var. *pallidium*, MacLean and Shaw 1949), Gladiolus (MacLean 1948), tuberose (*Polianthes tuberosa*, Ark and Maclean 1951), Taiwanese toad lily (*Tricyrtis* spp., Furukawa et al. 2005), and wax flower (*Stephanotis floribunda*, Tompkins and Hansen 1950). It is the primary pathogen observed on plants under field conditions. *B. cinerea* has a very broad host range and is more common on flower petals on both field- and greenhouse-grown plants. Other *Botrytis* species reported on lilies include *B. sphaerosperma* (Hennebert 1969), *B. squamosa* (Zhang 2006), and *B. tulipae* (Anonymous 1960).

Botrytis spp. overwinter as sclerotia in the plant debris (Coley-Smith 1980; Hsiang and Chastagner 1992; Trolinger and Strider 1985; Yunis and Elad 1989). The primary source of inoculum is generally considered to be conidia that are produced by sclerotia on or near the soil surface. Under prolonged periods of free moisture, these conidia infect leaves (Hsieh et al. 2001; Hsieh and Huang 1998). At 20 °C/68 °F, it takes 6–12 h of free moisture for infection to take place (Doss et al. 1984, 1988a; Van den Ende and Pennock-Voss 1977). Infection of leaves by *B. elliptica* occurs via stomata on the lower surface of the leaves and it then colonizes the leaf tissue (Doss et al. 1988a; Hou and Chen 2003; van Baarlen et al. 2004). *Botrytis* produces secondary conidia that can result in the rapid buildup of the disease under cool, moist conditions, giving rise to the common name of "fire" for the disease (Jarvis 1980; Moore et al. 1979; Linderman 1985; Lorbeer et al. 2007). Very conducive conditions can result in numerous, simultaneous infections giving rise to a large number of small spots on the foliage. Although rare, this condition is referred to as "leopard fire." The recently described perfect stage of *B. elliptica* has been observed under field conditions in the Netherlands, but it is unclear what role ascospores play in disease development there and other areas where it has not yet been observed (Chastagner et al. 1992; Huang et al. 2001; Terhem et al. 2015; Van den Ende and Pennock-Vos 1997).

Colonized flower petals and other plant tissues can also serve as an important inoculum source whereby mycelium directly infects healthy tissue from saprophytically-colonized tissue (Daughtrey et al. 2000; Holz et al. 2007). Injuries associated with hailstorms can also increase the incidence of infections by both B. elliptica and B. cinerea. Conidia are mostly dispersed by wind currents (Holz et al. 2007). When the crop is grown for 2 y, the increased density of plants in the field in the second year can increase disease pressure by inhibiting leaf drying after rain, irrigation, or dew events. Free moisture on the leaf surface contributes to a high level of *Botrytis* sporulation and infection. *Botrytis*, particularly *B. cinerea*, can become a problem

during cool forcing or pre-shipping storage when free water condenses on leaves and flower buds (Droby and Lichter 2007; Miller 1993; Vrind 2005).

Management

- ***Cultural practices*** – Crop rotation and annual digging reduces disease development. Reducing periods of leaf wetness is one of the most important mechanisms for limiting development of fire. Therefore, increasing plant spacing to avoid overcrowding of plants that allows for maximum air movement and sun exposure can help limit disease development. Avoiding overhead irrigation or irrigating only when the foliage will dry rapidly can also help reduce disease incidence. Managing environmental conditions during forcing is an important aspect of limiting disease development (Elad et al. 2011; Hausbeck et al. 1996; Hausbeck and Moorman 1996; Jewett and Jarvis 2001). Keeping flowers dry and reducing relative humidity and fluctuations in temperatures in storage areas helps decrease postharvest disease development (Miller 1993).
- ***Sanitation*** – Removal of all infected plant parts during the season, especially spent flowers and other dead tissues, is an important way to reduce sources of secondary inoculum. All foliage should be cut and disposed of at the end of the season to prevent the carryover of sclerotial inoculum from one season to the next.
- ***Biological control*** – A number of biological agents (biopesticides) have been tested for their effectiveness in controlling *Botrytis* on lilies (Chiou and Wu 2001, 2003; Elad and Stewart 2007; Huang et al. 2005; Kessel and de Haas 2001; Kessel et al. 1999; Fiddaman et al. 2000). Research in this area has resulted in an increasing number of biopesticides that are efficacious under controlled or greenhouse conditions (Paulitz and Belanger 2001). However, some promising agents have never been registered and disease control, particularly under field conditions, tends to be highly variable (DeBoer 2011; Chastagner and DeBauw 2014).

  Considerable research relating to the induction of disease resistance in ornamental geophytes is also underway (Chastagner et al. 2012; van Baarlen et al. 2007; Zehnder et al. 2001). Luzzatto-Knaan and Yedidia (2009) summarized the effect various elicitors have on the development of *B. elliptica* on lilies. Applications of salicylic acid delayed symptom development (Lu and Chen 2005); probenazole provided up to 14 d protection, partly due to closure of stomata (Lu et al. 2007); and *Bacillus cereus* had a long-lasting effect that reduced disease severity up to 75% (Liu et al. 2008). Balode (2011) reported that applications of the biological product Trihodermin B-J, which consists of *Trichoderma harzianum* 8–21 and *Trichoderma viride* 1–5, to the soil or directly to plants during the growing season resulted in lilies that showed significantly higher levels of resistance to *B. cinerea*.
- ***Fungicides*** – Fungicides play a critical role in an integrated *Botrytis* management program. However, their efficacy is hampered by widespread resistance to fungicides such as the dicarboximide and benzimidazole fungicides (Amiri et al. 2013; Chastagner and Riley 1990; Hsiang et al. 2001; Hsiang and Chastagner 1991, 1992; Leroux 2007; Migheli et al. 1990). Fungicides typically used to

control fire include thiophanate-methyl, chlorothalonil, copper-based products, iprodione, fenhexamid, fludioxonil, and mancozeb (Pscheidt and Ocamb 2015; Palmer and Vea 2014). Alternating or tank-mixing products from different FRAC groups with different modes of action is commonly recommended to prevent developing resistance (Brent 1995). Some products are formulated as mixtures to increase the spectrum of activity and reduce potential resistance development. There are also thermal-based products that vaporize the fungicide for use in greenhouses. Coverage of the lower leaf surface is important to obtain the best disease control. Climate-based warning systems have been developed to predict infection periods and optimize fungicide applications (de Kraker et al. 2005; de Ward 2008; Bastiannse et al. 1997). In some areas, up to 30 applications of fungicides are applied during the growing season to control fire (Chastagner 1997). Using the BoWaS system, van den Ende et al. (2000) reported a 30–80% reduction in the input of active ingredients depending on cultivar susceptibility and year.

- ***Resistance*** – Screening procedures have been developed and there are increasing efforts to breed for resistance to *Botrytis* in lilies (Balode 2009; Doss et al. 1986; Núñez de Cáceres González et al. 2015; van Tuyl and van Holsteijn 1996). Among various lily cultivar groups, resistance to *Botrytis* is present in the Oriental hybrids (Beers et al. 2005). Cultivars are known to differ in their susceptibility to fire. Daughtrey and Bridgen (2013) determined the susceptibility of 31 commercially available lilies (*Lilium* spp. and hybrids) under field conditions in New York. During high disease pressure in 2009 and 2011, 'Conca d'Or,' 'Time Out,' 'Sorbonne,' 'Casa Blanca,' 'Le Rêve,' 'Tiara,' 'African Queen,' 'Cobra,' 'Mozart Zanlazart,' and *L. regale* had foliage disease ratings <20%, while 'Sweet Kiss,' 'Royal Fantasy,' 'Red Alert,' 'White Heaven,' 'Yellow Star,' *L. tigrinum*, 'Côte d'Azur,' 'Gironde,' 'Connecticut King,' and 'Chianti' had ratings above 70%.

2.2 Root, Bulb, and Stem Rots

There are a number of soilborne fungal pathogens that cause various root, bulb, and stem rots on lilies. Infected plants typically exhibit yellow foliage, stunting, wilted growth, and root, bulb, and stem decay. These diseases can lead to dead plants in the field and greenhouse and the loss of young plants during the propagation from lily scales. Flower buds may also wither and not open. *Fusarium oxysporum* f. sp. *lilii*, *Rhizoctonia solani*, *Phytophthora* spp., and *Pythium* spp. often occur as a complex and can cause serious damage to lilies during greenhouse forcing (Miller 1993). This complex also contributes to postharvest leaf senescence, particularly when potted plants are cold stored prior to shipping (Prince et al. 1987). Miller (1993) reported that up to two-thirds of root rot-infected plants exhibited leaf yellowing 12 d after cold storage. Diseases caused by one or more of these pathogens are likely when bulbs are planted in infested field sites or in non-sterile media. Control of these diseases usually depends on the use of preplant dips of planting stock and

propagation scales in mixtures of fungicides, or post-plant soil drench applications of fungicides during forcing (McClellan and Stuart 1946; Pscheidt and Ocamb 2015; Raabe and Hurlimann 1971). Below is more detailed information about some specific pathogens.

Basal Rot (*Fusarium oxysporum* f. sp. *lilii* Imle and *Cylindrocarpon* spp.)

Geographic occurrence and impact. Basal rot is generally considered to be the most important soilborne disease on lilies (Slate 1936, 1972; Linderman 1985; Moore et al. 1979). It often occurs as a combination of *Cylindrocarpon*, *Fusarium*, and other fungi and has been reported from most areas where lilies are grown, including Bulgaria (Bobev 2009), Canada (Ginns 1986), China (Li et al. 2013), Europe (Cabral et al. 2011; Foister 1961), Israel (Lebiush–Mordechai et al. 2014), Mexico (Alvarez 1976), New Zealand (Pennycook 1989), Republic of Mauritius (Orieux and Felix 1968), South Africa (Gorter 1977; Crous et al. 2000), and the USA (Farr et al. 1989).

Fusarium oxysporum f. sp. *lilii* degrades the cortical tissues resulting in severe root rot along with destruction of the bulb's basal plate (Baayen et al. 1998; Imle 1940, 1942a, b; Linderman 1981; Löffler et al. 1990; Moore et al. 1979).

Symptoms/signs. Basal rot results in a severe root rot and destruction of the basal plate of the lily bulbs. Unless scales have been directly infected, a characteristic chocolate or bluish-gray rot extends from the basal plate into the scales (Fig. 2). Eventually, the scales detach at the basal plate and bulbs often fall to pieces after digging. Aboveground symptoms associated with basal rot consist of premature yellowing of the foliage, stunting, and premature senescence. Flower buds may wither and fail to open. These symptoms are not specific to this disease, so the bulb should be examined to see if it exhibits the typical decay of the basal plate and scales. Prior to the destruction of the main bulb, infected bulbs tend to produce new scale bulblets, which are frequently infected, and a large number of stem bulblets



Fig. 2 Typical symptoms associated with basal rot (Wageningen University and Research © 2017. All Rights Reserved.)

before it is killed. Fusarium bulb rots are particularly troublesome during winter weather when soils can be excessively wet. This is manifested by reduced growth and leaves that are pale green in color.

Biology and epidemiology. *Fusarium* produces three types of asexual spores: microconidia, macroconidia, and thick-walled, stress-resistant chlamydospores which enable long-term survival (Summerell et al. 2001). Initial infection takes place just behind the root cap (Bishop and Cooper 1983; Turlier et al. 1994). Baayen and Rijkenberg (1999) studied the early infection process and found that conidia germinated within 6 h and infection occurred via the anticlinal epidermal walls in the root elongation zone. Within 24–48 h, the hyphae spread through the intercellular spaces between the epidermis and the outermost cells of the root cortex. The cortical tissue is eventually degraded by the fungus (Baayen and Rijkenberg 1999; Imle 1942a, b; Linderman 1981; Löffler and Mouris 1992). From the roots, the pathogen spreads into the basal plate and attached scales. In addition, direct infection of the scales may also occur through stomata in the epidermis. Stem roots and bulblets can also be infected. Fusarium bulb rot on field-grown *Lilium longiflorum* is common during the winter when soils can be excessively wet.

Management

- ***Cultural practices*** – Cultural practices that help limit the risk of basal rot include destruction of diseased bulbs, propagation from rot-free scales, use of low-nitrogen fertilizers, removal of bulb and plant debris, improving soil drainage, and the use of pasteurized potting soil. Do not plant bulbs that have a rotted basal plate, and dip bulbs in fungicides prior to planting.
- ***Biological control*** – Several biological-based approaches in controlling *Fusarium* have been studied in lilies. Alabouvette et al. (1998, 2005, 2009) utilized non-pathogenic strains of *Fusarium* to protect plants from infection. Growing lilies in rotation with marigolds (*Tagetes patula*) is also beneficial in reducing disease development (Conijn 1994).
- ***Fungicides*** – Dipping bulbs or propagation scales in fungicides is the most common means of protecting lilies from basal rot and several other soilborne diseases. Products containing the following active ingredients are typically used: chlorothalonil, fludioxonil, quaternary ammonium chloride, thiabendazole, thiophanate-methyl, and thiram. While disinfectants such as quaternary ammonium compounds and chlorine dioxide, can kill *Fusarium* spores that come in direct contact with the disinfectant these products do not provide the same residual control as traditional fungicides (Chastagner and Riley 2002b; Copes et al. 2004, 2014). Growers commonly dip bulbs in a mixture of fungicides that also provides control of other soilborne pathogens like *Pythium* and *Rhizoctonia* (Bald et al. 1983; Bald and Chandler 1957; Pscheidt and Ocamb 2015). Fungicide resistance has been reported to benzimidazole products, which can limit the effectiveness of dips in thiabendazole and thiophanate-methyl (Bollen 1977; Chung et al. 2009).

- ***Resistance*** – Resistance to *Fusarium oxysporum* has been observed among some species and hybrid groups (Beers et al. 2005; Löffler et al. 1996; Straathof and Van Tuyl 1995). Baayen and Rijkenberg (1999) found that partial host resistancet was associated with a prolonged biotrophic phase of growth within the host, which supported earlier work that showed that partially resistant cultivars retarded, but did not fully inhibit, colonization of host tissues (Straathof et al. 1993; Baayen and Förch 2001).

Rhizoctonia Stem Rot [*Rhizoctonia solani* Kühn (Teleomorph: *Thanatephorus cucumeris* (A.B.. Frank) Donk)]

Geographic occurrence and impact. *Rhizoctonia solani* has been documented on lilies in the USA, Australia, Bulgaria, Canada, Greece, South Africa, and Scotland (Anonymous 1960; Cook and Dubé 1989; Bobev 2009; Ginns 1986; Holevas et al. 2000; Gorter 1977; Foister 1961).

Symptoms/signs. Bulbs colonized by *R. solani* are yellow rather than the normal white (Dijst and Schneider 1996; McWhorter 1957b) (Fig. 3). Stems and scales



Fig. 3 Lower stem decay associated with *Rhizoctonia* (Wageningen University and Research © 2017. All Rights Reserved.)

where the stem emerges develop a dark discoloration and soft rot, causing the plant to topple over. Stem root development may be retarded and damage on the lower leaves may appear as light brown spots. The pathogen may also cause pre- and postemergence damping-off of seedlings and young plants.

Biology and epidemiology. *Rhizoctonia solani* has a very large host range that includes a number of geophytes (Mordue 1974a). Growth of the fungus is favored by warm temperatures, and it colonizes and survives as hyphae and sclerotia in plant debris, soil, and other growing media. *R. solani* does not produce spores, but can be spread by infected propagative material, movement of infested soil or planting media, and water splash. The relationship of the sexual stage of *R. solani* to plant disease has not been well-studied.

Management

- ***Cultural practices*** – Use pasteurized potting soil. Avoid planting bulbs that have yellow scales.
- ***Soil disinfestation*** – Soil fumigation is effective in reducing disease development in the field but is seldom used (Bald et al. 1983)
- ***Fungicides*** – Dip bulbs in PCNB after harvest. For potted plants, drench applications of thiophanate-methyl are also recommended (Anonymous 2009; Linderman 1985; Moorman 2016).

Pythium Root Rot (*Pythium* spp.)

Geographic occurrence and impact. Pythium root rot in lilies may be caused by *P. ultimum*, *P. debaryanum*, *P. splendens*, *P. irregulare*, as well as unidentified species of the pathogen and has been reported in Scotland and the USA (Anonymous 1960; Foister 1961; French 1989). Losses exceeding 75% have occurred in greenhouses in the USA.

Symptoms. General symptoms associated with root rot consist of a overall loss of quality, stunted growth, wilting, and the premature yellowing and senescence of lower leaves on plants. Underground symptoms include rotted portions on roots and bulbs. Root rots can also lead to flower abortion and an increase in the postharvest senescence of leaves, especially when plants are placed in cold storage prior to shipping. The pathogen may also cause pre- and postemergence damping-off of seedlings and small plants during propagation.

Biology and epidemiology. The genus *Pythium* is not a true fungus and is closely related to *Phytophthora*. It is an oomycete "water mold" which is disseminated as zoospores through irrigation, surface water and rain, and the movement of contaminated soil/media. Depending on the species, it *Pythium* has a very broad host range. Long-term survival is enabled by chlamydospores and sexually produced oospores. Pythium root rot is common under warm conditions and in poorly drained soils.

Cycles of under- and overwatering which lead to root damage are very conducive to outbreaks of *Pythium*.

Management

- ***Cultural practices*** – Use pasteurized potting soil and healthy planting stock. Plant only in well-drained, pathogen-free media. Disinfect reused pots and any tools and equipment that might be used with potting media.
- ***Fungicides*** – Drench potted bulbs with oomycete fungicides such as mefenoxam. These are generally applied in combination with fungicides that are effective in controlling root rots caused by *Fusarium* and *Rhizoctonia* (Bald et al. 1983; Bald and Chandler 1957; Pscheidt and Ocamb 2015). Fungicide resistance can be a problem with some products, such as metalaxyl (Aegerter et al. 2002). Mefenoxam at high rates may cause yellowing of leaf margins of Easter lilies. (Moorman 2016; Anonymous 2009)

Stump Rot, Foot Rot, and Stem Blight (*Phytophthora* spp.)

Geographic occurrence and impact. A number of *Phytophthora* spp. have been reported on lilies. Stump rot, caused by *Phytophthora nicotianae* Breda de Haan (syn. *Phytophthora parasitica* Dastur), has been reported from China, Japan, Korea, the UK, the USA, West Indies, and Zimbabwe (Anonymous 1960; Erwin and Ribeiro 1996; Yang et al. 2010; Farr and Rossman 2015; Cho and Shin 2004). Foot rot and stem blight have been associated with either *P. nicotianae*, *P. cactorum*, or *P. cinnamomi* or unidentified *Phytophthora* spp. from Mexico, the USA, Japan, Korea, Australia, New Zealand, and Greece (Alvarez 1976; Anonymous 1960; Erwin and Ribeiro 1996; Pennycook 1989; Holevas et al. 2000; Cho and Shin 2004). These diseases can occur in the field or greenhouse and have the potential to kill large numbers of plants (Moore et al. 1979; Linderman 1985).

Symptoms/signs. Stump rot is associated with the infection of emerging shoots under field conditions (Fig. 4). This disease is sometimes referred to as mud rot, because emerging shoots are often covered with mud. In addition to rot at growing points of emerging plants, stems of older plants can be infected causing them to wilt and collapse. Roots are also frequently rotted. During forcing, infested plants often have a violet-brown discoloration at the base of the stem, with the infection moving upward and leaves becoming yellow. Stems of severely infested plants collapse.

Biology and epidemiology. *Phytophthora*s are not true fungi but fungal-like oomycetes ("water molds"). The species reported on lilies have very large host ranges, including a number of herbaceous and woody plants (Erwin and Ribeiro 1996; Farr et al. 1989; Farr and Rossman 2015; Ho et al. 1995; Lamour 2013; Ribeiro 1978, 2013). Diseases incited by *Phytophthora* are favored by overwatering, poorly drained soil and potting media. Hyphae produce sporangia, which can either germinate directly or produce swimming zoospores which infect host tissues. Depending on species, long-term survival is by chlamydospores and/or sexually-produced oospores



Fig. 4 Root decay and discoloration of stem tissue associated with *Phytophthora* damage (Wageningen University and Research © 2017. All Rights Reserved.)

(Erwin and Ribeiro 1996). *Phytophthora* spp. are generally disseminated by the movement of infected planting stock, infested soil, and the spread of zoospores through irrigation, surface water, and rain.

Management

- ***Cultural practices*** – The primary means of controlling *Phytophthora* diseases on lilies is to avoid saturated soils and standing water in the field which is favorable to pathogen development and movement of zoospores. This can be done by site selection, improving drainage and using well-drained potting media. Avoid overwatering and overhead irrigation, which can result in the splash dispersal of the pathogen within a field.
- ***Sanitation*** – Steam or chemically treat soil or potting media.
- ***Fungicides*** – Fungicides are not generally used in the field, but are applied as drench treatments during greenhouse forcing. General recommendations for fungicides to control *P. cactorum* typically include the use of metalaxyl (Anonymous 2009). Chemistries effective in managing *Phytophthora* spp. in other pathosystems include aluminum tris (O-ethyl phosphonate), captan, cyazofamid, dimethomorph, famoxadone + cymoxanil, fenamidone, fluopicolide, fluoxastrobin + myclobutanil, mandipropamid, mefenoxam, metalaxyl, and

pyraclostrobin and fungicides containing phosphorous acids (Palmer and Vea 2010). To prevent fungicide resistance, it is important to rotate or use mixtures of materials with different modes of action to manage *Phytophthora* diseases of action. Metalaxyl at high rates may cause yellowing of leaf margins of Easter lilies (see Sect. 6.5.1).

Southern Blight/Crown Rot [*Sclerotium rolfsii* Sacc. [Teleomorph; *Athelia rolfsii* (Curzi) C.C. Tu & Kimbr. (syn. *Corticium rolfsii*)] and *Sclerotium delphinii* Welch (syn. *S. rolfsii* var. *delphinii* (Welch) Boerema & Hamers)]

Geographic occurrence and impact. *Sclerotium rolfsii* and/or *Sclerotium delphinii* have occasionally been reported to cause a destructive decay of the lower stems (crowns) of lilies in North America, Europe, Japan, Korea, and China (Anonymous 1960; Cho and Shin 2004; Coinjn 2014; Okabe and Matusmoto 2003; Shaw 1973; Yang et al. 2007). Most reports are associated with field plantings, but crown rot can also occur in the plantings of cut flowers and potted plants under greenhouse conditions.

Symptoms/signs. In the field, symptomatic plants generally appear as patches of dead and dying plants. Stem infections initially appear as small, water-soaked lesions at the base of a stem that rapidly expands, girdling the stem and causing wilt and death. As the disease progresses, a white mycelial mat can be observed on the stem and on the soil surface, within which, small orange-tan to brown sclerotia are formed (Fig. 5). Depending on planting density, the fungus can spread from plant to plant down the row. Infected bulbs develop a white, chalky or cheesy rot with conspicuous strands of white mycelia. The presence of the white mycelial strands and the reddish-brown sclerotia are diagnostic for the disease (Chastagner and Byther 1985).



Fig. 5 Shoot and bulb decay caused by *Sclerotium rolfsii*. Immature white sclerotia are evident on the bulb scales (*left*). Characteristic coarse white mycelium and sclerotia on bulbous iris (*center* and *right*) ((*left*) Wageningen University and Research © 2017. All Rights Reserved.) and ((*center and right*) Gary A. Chastagner © 2017. All Rights Reserved.)

Biology and epidemiology. *Sclerotium rolfsii* (teleomorph *Athelia rolfsii*) and the closely related *S. delphinii* can cause extensive mortality on a number of hosts (Aycock 1966; Mordue 1974b; Okabe and Matsumoto 2003). *S. delphinii* is the primary cause of crown rot on ornamental geophytes (Linderman 1985). These pathogens persist in the soil on dead organic matter as mycelia or sclerotia that germinate to produce mycelia. Sclerotia can survive for several years in the soil, although in dry soil, survival is limited to a few months (Coley-Smith et al. 1974). The mycelia directly infect crown tissues of the plant host. The teleomorph is rare and probably does not play a significant role in disease development (Mullen 2001). The primary means of spread is thought to occur by mycelial growth to adjacent plants and movement of infected plant material and infested soil. *S. rolfsii* and *S. delphinii* are favored by warm, humid conditions, with *S. rolfsii* specifically limited by low temperatures (Xu et al. 2008).

Management

- ***Cultural practices*** – Plant only healthy bulbs. Avoid harvesting diseased bulbs and the movement of pathogen-infested soil on equipment. Rotate out of susceptible hosts and eliminate volunteer bulbs for 3–5 y prior to replanting. In display gardens, use only pathogen-free soil to cover bulbs.
- ***Fungicides*** – Soil applications of azoxystrobin, flutolanil, PCNB, and/or preplant dips in PCNB have been used to control crown rot (Chastagner and Riley 2002a; Chastagner et al. 1990). Soaking bulbs in hot water plus formalin is also recommended for some geophytes (Pscheidt and Ocamb 2015).
- ***Soil disinfestation*** – Preplant soil fumigation with metam sodium has been effective in limiting disease development on other geophytes. Soil solarization or steam sterilization can be effective as preplant treatments if high temperatures and humidity can be maintained for a sufficient duration to kill sclerotia (Mullen 2001). Flooding the soil for extended periods of time is also effective in killing sclerotia (Vink 2011a)

White Stem Rot (*Sclerotinia sclerotiorum* [Lib.] deBary)

Geographic occurrence and impact. White stem rot (also called "cottony stem rot," "white mold," or "Sclerotinia stem rot") has been reported on lilies in California, Connecticut, Florida, Texas, Washington (Alfieri et al. 1984; French 1989; Shaw 1973), and the Republic of Mauritius (Wiehe 1948).

Symptoms/signs. Initial symptoms consist of a wilting of emerged plants, which eventually die. Infected plants develop a rot at the base of the stem which can be covered by white, cottony mycelial growth under moist conditions. Large black sclerotia develop in and on stems. Ascospore infections result in small spots on flower buds and petals that turn brown and can also become covered in a white, fluffy mycelial growth under moist conditions (Dreistadt 2001; Moore et al. 1979; Chase et al. 1995).

Biology and epidemiology. *S. sclerotiorum* overwinters as sclerotia formed on plant tissue (Mordue and Holiday 1976). Sclerotia germinate either to produce mycelia or apothecia which contain sexual ascospores that become airborne and can infect flowers and senescent plant tissues. *S. sclerotiorum* does not produce conidia; thus, there is no repeating cycle throughout the year. Healthy plants or plant parts can be infected if they come in contact with colonized host tissue or by mycelial spread on the soil surface.

Management

- ***Cultural practices*** – Sanitation plays an important role in the management of white stem rot. Removal of infected plant material and senescent plant tissues, especially flower petals, is crucial for preventing pathogen spread. Infected plants should be removed along with surrounding soil to remove any overwintering sclerotia. Killing sclerotia by steaming or solarization of soil and potting media before planting is also beneficial. General fungal disease management strategies to decrease periods of leaf wetness, such as avoiding overhead irrigation and irrigating early in the day so plants dry quickly, are beneficial in reducing ascospore germination (Dreistadt 2001).
- ***Fungicides*** – Little information is available relating to fungicides that are effective in controlling white stem rot on lilies. On other crops, fungicides such as thiophanate-methyl, iprodione, boscalid, fludioxonil + cyprodinil, and fluazinam are recommended and can reduce the buildup of sclerotia in soil (Pscheidt and Ocamb 2015)

2.3 Black Scale Rot: [*Colletotrichum lilii* Plakidas ex Boerema & Hamers (syn. *C. gloeosporioides* Penz)]

Geographic occurrence and impact. There are scattered reports of *Colletotrichum lilii* on lilies from the USA, Japan, and the Netherlands (Anonymous 1960; Damm et al. 2009; Sobers and Plakidas 1962; Parris 1959; Plakidas (1944) Sobers 1980). Other *Colletotrichum* spp. reported on lilies include *C. capsici* (*C. truncatum*), *C. dematium*, *C. gloeosporioides*, *C. lilacearum*, and *C. spaethianum* (Farr and Rossman 2015). It is unclear what role many of the *Colletotrichum* spp. that have been found on lilies play in the development of black scale rot (Sobers and Plakidas 1962). Growers can experience heavy losses because the bulbs, blackened by disease, are unsalable (Sobers 1980).

Symptoms/signs. Sobers (1980) indicates that infections result in the formation of small, light brown lesions that are irregular in shape, slightly sunken, and near the tips of the outer bulb scales. As lesions mature, they become dark brown to black and eventually enlarge to cover the entire scale (Fig. 6). Severely diseased scales are shriveled due to collapsing epidermal tissue and bulbs become unmarketable. Lesions, that are mostly elongate, irregular, depressed, and light brown to brown in color, are occasionally found on the underground portion of the stem.



Fig. 6 Scale rot associated with *Colletotrichum lilii* (Wageningen University and Research © 2017. All Rights Reserved.)

Biology and epidemiology. Initially, black scale rot was thought to be associated with poor growing sites, suggesting that bulbs under stress may be predisposed to infection (Moore et al. 1979). Disease development is favored by damp conditions and can occur on bulbs in the field and during storage. Pathogenicity studies have shown that scales are infected only when soil or storage temperatures are greater than 22–26 °C/72–79 °F (Sobers and Plakidas 1962). Conidia of *Colletotrichum* spp. are typically spread by rain or irrigation water and survive as dormant hyphae in host debris. Hyde et al. (2009) indicate that except for an unconfirmed report that *Hemerocallis* sp. may serve as a host of *C. lilii*, *Lilium* spp. are the only known host of this pathogen. Thus, it is unlikely that alternate hosts play a role in its survival.

Management. Sanitation, including the discarding of diseased bulbs, helps reduce the risk of disease. Sobers (1958, 1980) demonstrated that *Lilium* spp. vary in their susceptibility to *C. lilii*. Magie (1966) and Sobers (1980) have shown that treatment of bulbs with thiram-based fungicides [bis (dimethylcarbamoyl) disulfide] or chlorothalonil prevents infection and improves flower and bulb production.

2.4 Blue Mold (*Penicillium* spp.)

Geographic occurrence and impact. *Penicillium* species are widespread, opportunistic pathogens that cause blue mold on a variety of geophytes, including lily bulbs (Overy et al. 2005; Moore et al. 1979; Dugan et al. 2014; Dreistadt 2001; Farr and Rossman 2015). *Penicillium* species reported on lilies include *P. albocoremium* (Frisvad) Frisvad, *P. corymbiferum* Westling (syn. *P. hirsutum* Dierckx), *P. cyclopium* Westling (syn. *P. aurantiogriseum* Dierckx), *P. gladioli* McCulloch & Thom, and *P. tulipae* Overy & Frisvad. They cause a decay of bulb scales, which can lead to bulba rot during storage.

Symptoms/signs. Initial infections may appear as small brown slightly depressed areas on the scale or at the base of scales used in propagation. Under moist

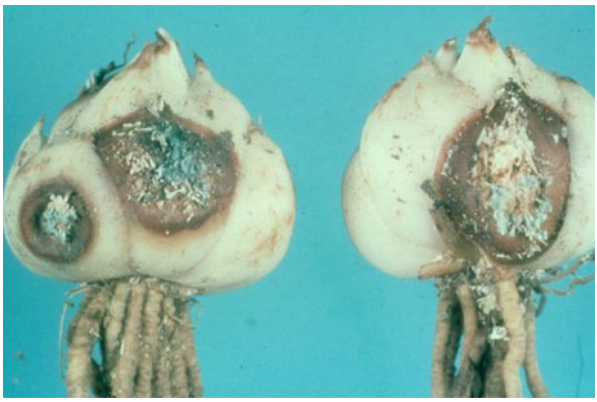


Fig. 7 Typical symptoms and signs associated with *blue* mold (Wageningen University and Research © 2017. All Rights Reserved.)

conditions, a diagnostic blue-green mold develops on infected tissues (Fig. 7). Unless freezer-stored, the severity of blue mold tends to increase with extended storage of bulbs.

Biology and epidemiology. *Penicillium* species are wound pathogens. Harvesting wounds, insect injuries, bruises, sunburn, and natural wounds, such as those that occur when propagating lilies via scaling, are avenues of entry. Spores are ubiquitous and dispersed by air. Prolonged cool, moist storage conditions favor disease development. On other geophytes, recent studies have shown the there is variation in the aggressiveness of different strains of *Penicillium* (Dugan et al. 2014).

Management

- ***Cultural practices*** – Control of blue mold generally consists of digging bulbs when they are mature, avoiding bruising and wounding, storing bulbs under cool temperatures and moderate humidity, and discarding diseased bulbs (Dreistadt 2001; Moorman 2016; Pscheidt and Ocamb 2015).
- ***Fungicides*** – Dipping geophyte bulbs in fungicides and disinfectants is commonly recommended as a way of controlling the development of blue mold (Chastagner and Byther 1985; Pscheidt and Ocamb 2015). There are a number of thiophanate-methyl-based products that are used for this purpose, but fungicide resistance is a significant problem with this pathogen (Bollen 1971). The use of disinfectants, such as didecyl dimethyl ammonium chloride and chlorine dioxide, is effective in killing spores, but does not provide any residual protection (Chastagner and Riley 2005).

2.5 Soft Rot: *Rhizopus* spp.

Geographic occurrence and impact. This storage rot has been reported in the USA and Canada (Anonymous 1960; Ginns 1986). When bulbs are stored under

moist conditions and high temperatures, soft rot can be severe. *Rhizopus* can also cause a stem rot during forcing (Vink 2011b).

Symptoms/signs. Initially brown spots develop on bulb scales, which can quickly develop into a severe, mushy rot. Stems can also be attacked by this pathogen, causing a stem rot. Under moist conditions, a gray-white mass of coarse mycelial growth with black, globose spore heads may cover the diseased bulbs or stems.

Biology and epidemiology. *Rhizopus* species are common, widespread pathogens that normally do not cause damage to bulbs. For example, *Rhizopus stolonifer*, the common bread mold, has a large host range and can cause postharvest decays on a number of fruits, vegetables, and geophytes (Sholberg and Conway 2016; Moore et al. 1979). Under warm, moist conditions, infection can take place rapidly, especially if bulbs have been mechanically injured. Spores are airborne, but can also be spread in dip tanks. Soft rot normally only occurs when wet or damp bulbs that are stored at high temperatures, usually at a RH $>$ 80% and a temperature $>$30 °C/86 °F (Moore et al. 1979). High temperatures during forcing also contribute to a stem rot.

Management. A number of approaches, including handling commodities to avoid injury, using disinfectants in dip tanks as well as fungicides and biocontrol agents, are used to limit postharvest losses on fruits and vegetables (Sholberg and Conway 2016). While some of these may have applications in the handling of bulb crops, the management of *Rhizopus* soft rot is primarily a matter of avoiding injury to bulbs and proper storage conditions. Diseased bulbs should be discarded.

2.6 Other Fungal Diseases of Lily

According to the USDA online fungal database (Farr and Rossman 2015), more than 100 fungi have been reported on lilies. The pathology of some of these on lilies is unclear. For a current list of fungi associated with lilies, visit http://nt.ars-grin.gov/fungaldatabases/fungushost/FungusHost.cfm and enter host name "lilium." Below is a brief summary of some of the other fungal diseases of lily.

Leaf Rust (*Uromyces* spp., *Puccinia* spp.)

Leaf rust has been reported on lilies in Asia, Canada, Europe, Nepal, Pakistan, Poland, Ukraine, the USA, and USSR. *Uromyces lilii* (syn. *U. holwayi*) is the most widely reported pathogen, but *U. aecidiiformis*, *U. erythronii*, *U. himalaicus*, *Uromyces* spp., *Puccinia sporoboli*, (syn. *P. sporoboli* var. *sporoboli*), *P. basiporula*, and *Puccinia* spp. have been reported sporadically (Cummins and Green 1961; Hiratsuka et al. 1992; Raabe 1985). McWhorter (1957a) reported that leaf rust was less common than had been previously reported along the west coast of the USA and was more of a "novelty than a menace." Characteristic brown to orangish powdery pustules or yellowish blotches develop on infected leaves, which may drop

prematurely. Spores are airborne and disease is favored by high humidity, rain, and overhead irrigation. If needed, sanitation and fungicides are used to manage leaf rust (Raabe 1985).

Leaf Spots

A number of fungi (*Mycosphaerella* spp., *Cercospora* spp., *Cercosporella* spp., *Cladosporium* spp. *Heterosporium* spp. *Phyllosticta* spp., *Pseudocercospora* spp., and *Ramularia* spp.) have been reported to cause leaf spots and/or stem lesions on *Lilium* spp. in Asia, Canada, Europe, the USA, and New Zealand. Symptoms can include brown, tan, or white spots, or lesion on leaves or stems that may have yellow borders. These pathogens persist in plant debris and disease development is favored by high humidity, rain, and overhead irrigation. Avoiding overhead irrigation, good sanitation, and protectant applications of fungicides are typical methods of controlling these pathogens (Conijn 2014; Moore et al. 1979).

Gray Bulb Rot: *Rhizoctonia tuliparum* (Kleb.) Whetzel and Arthur has been reported on hosts worldwide and is a destructive disease on tulips and iris (Byther and Chastagner 1993; Chastagner and Byther 1985; Dijst and Schneider 1996; Gould 1957; Moore et al. 1979; Mordue 1974a). In the Netherlands, the disease on tulips is called "Kwadegrond." This pathogen has also been reported on a number of other geophytes, including daffodils, crocus, gladiolus, and lily. There are limited reports of this pathogen from lilies in the USA and Netherlands and damage is generally minimal (Mordue 1974a; Anonymous 1960; Gould 1957). Unlike *R. solani*, which tends to be favored by warm soil temperatures, gray bulb rot is most active at cooler soil temperatures. The pathogen produces small 1–10 mm/0.04–0.4 in. in diameter dark brown sclerotia that can survive for prolonged periods in the soil, limiting the effectiveness of crop rotation. The disease tends to occur in patches in the field. Limited information is available relating to the management of this disease on lilies, but on tulips and iris, cultural practices that include sorting out and destroying infected bulbs, removing diseased plants and soil with sclerotia from affected areas, removing volunteer plants from previous bulb crops, deep plowing and shallow planting to reduce the risk of exposure of the bulbs to sclerotial inoculum, and covering bulbs with pathogen-free soil in display gardens are used to limit disease development. Flooding is also effective in killing sclerotia (Muller et al. 1988; van Zaayen et al. 1986). There are a number of fungicides that are effective against this pathogen (Chastagner and DeBauw 2011; Chastagner and Riley 2002a; Pscheidt and Ocamb 2015). Preplant soil fumigation with materials such as metam sodium is effective but probably not justified or needed to control this disease on lilies.

***Sclerotium wakkeri* Boerema and Posthumus** has been sporadically reported to cause a black leathery rot of bulbs (Fig. 8) with black discoloration and rotting of the stem bases (Moore et al. 1979; Van Kesteren 1966).

Scale rots – See Sect. 3.1.



Fig. 8 *Black* leathery rot associated with *Sclerotium wakkeri* (Wageningen University and Research © 2017. All Rights Reserved.)

3 Bacterial and Phytoplasma Diseases

3.1 Scale Rots: A Complex Interaction of *Pseudomonas* spp., *Fusarium* spp., and *Cylindrocarpon* spp.

Pseudomonas spp. have been reported as a secondary or wound pathogen that is associated with "twist," "scale tip rot," "stem lesion," "buckskin rot," and "feathery tip rot" (Bald et al. 1969; Bald 1979; Bald and Solberg 1960; Linderman 1985; McWhorter 1945, 1947; Vidaver and Lambrecht 2004). These diseases have primarily been reported on Easter lilies. Linderman (1985) indicates that control of these diseases is largely based on the control of the primary fungal pathogens (*Fusarium* spp. and *Cylindrocarpon* spp.), avoiding wounds during harvest and handling of the bulbs, the rapid drying and cooling of bulbs after harvest, and avoiding the planting of affected bulbs (Moorman 2016; Maginnes and Smith 1971).

3.2 Leafy Gall: *Rhodococcus fascians* (Tilford) Goodfellow (syn. *Corynebacterium fascians* (Tilford) Dowson)

Geographic occurrence and impact. Although limited information is available relating to the geographical distribution of *R. fascians* on lilies, Putnam and Miller (2007) indicate this pathogen is widely distributed around the world and has an ever-increasing host range that includes both monocots and dicots and woody as well as herbaceous plants. It was first associated with stem galls on *Lilium regale* in 1939 (Lacey 1939). Infections result in malformed bulbs and shoots, making plants



Fig. 9 Malformed bulbs associated with *Rhodococcus* damage (Wageningen University and Research © 2017. All Rights Reserved.)

unmarketable. Once infested, it is recommended that bulb stocks be destroyed and not used for further propagation.

Symptoms/signs. *R. fascians* induces the formation of either leafy galls or fasciations (Putnam and Miller 2007). In lily, symptoms include stem galls, severely malformed bulbs, and the formation of long side shoots (Lacey 1939; Vantomme et al. 1982) (Fig. 9).

Biology and epidemiology. *R. fascians* is a gram-positive bacterium that was previously named *Corynebacterium fascians* (Tilford) Dowson (Goodfellow 1984). It resides primarily on the exterior surfaces of plants and is able to infect plants via epidermal cells (Cornelis et al. 2001). Putnam and Miller (2007) indicate that there are a number of questions relating to the movement and survival of *R. fascians*. It is unclear to what extent the pathogen survives in soil and its potential to spread within the host. In a vegetatively-propagated host like lilies, contaminated propagation material is likely the primary means of spread. The role of insects in natural disease transmission is unknown. Disease development appears to be favored by moderate environmental temperatures. *R. fascians* can be readily spread in water, increaseing the risk of spread from diseased to healthy bulbs on wash lines and in dip tanks (Putnam and Miller 2007).

Management. Effective management of *R. fascians* is limited by the fact relatively little is known regarding the epidemiology of this disease, the up to 6 mo between infection and symptom development, and the limited efficacy data for new compounds and treatments that might offer potential control (Putnam and Miller 2007). These authors plus Daughtrey and Benson (2005) and Schnitzler (2004) provide a number of recommendations relating to the management of this pathogen. Application of some disinfectants and bactericides directly to plants and the soil, and soil

sterilization via steam has been reported to reduce the bacterial populations. Destruction of infected plants and plants that have come in contact with them, plus the sanitization of surfaces and tools are common components of disease management programs. Conijn (2014) indicates that the key to managing disease development on lilies is to protect bulbs from exposure to the pathogen by removal and destruction of infected stocks and by harvesting and processing healthy stocks prior to any potentially infested stocks. Hot water treatment of lily bulbs at 39–41 °C/102–106 °F for 2 h provided partial control and complete control, with the addition of 0.5% formalin (Kruyer and Boontjes 1982). Chastagner and Riley (2005) found that a 1-h exposure to 25 ppm chlorine dioxide killed all cells of *R. fascians* present on a glass surface, suggesting that other disinfectants could be used to limit the spread of the pathogen when bulbs are exposed to water.

3.3 Phytoplasma Diseases ("*Candidatus* Phytoplasma spp.")

Geographic occurrence and impact. Diseases of lilies caused by aster yellows-related phytoplasmas have been reported to occur in the Czech Republic, Israel, Mexico, and Poland (Bertaccini et al. 2005; Cortés-Martínez et al. 2008; Kamińska and Dziekanowska 2002; Kamińska and Korbin 2000; Kamińska and Śliwa 2007; Poncarová-Voráckova et al. 1998; Weintraub et al. 2007).

Symptoms/signs. Cortés-Martínez et al. (2008) reported that symptoms on infected plants in Mexico included a zigzag-line pattern on leaves, dwarfism, enlargement of stems, shortened internodes, leaves without petioles growing directly from bulbs, bulbils, death of young roots, atrophy of flower buds, and flower abortion. Kamińska and Śliwa (2007) noted that symptoms on greenhouse-grown plants in Poland resembled those associated with physiological leaf scorch and included leaf malformation and necrosis and flower bud abscission. Bertaccini et al. (2005) reported severe fasciation on *Lilium* spp. associated with phytoplasma infections in southern and central Bohemia during the period 1999–2003.

Biology and epidemiology. Phytoplasmas are phloem-restricted, obligate bacterial plant pathogens and insect symbionts (Agrios 2005). They are transmitted by phloem-feeding insects, such as leafhoppers and plant hoppers, and through the vegetative propagation of host material. Based on 16S rDNA analysis, the phytoplasmas detected in lilies include "Ca. Phytoplasma asteris" (Cortés-Martínez et al. 2008) and "Ca. Phytoplasma mali" (Kamińska and Śliwa 2007). There is also evidence that mixed phytoplasma infection occurs in lilies (Bertaccini et al. 2002, 2005). The spread of phytoplasmas is favored by cool moist summers. There are more than 300 hosts of "*Candidatus* Phytoplasma spp.," including edible and ornamental plants, grasses, grains, and weed hosts. Given the global movement of lily bulbs and global distribution of phytoplasmas, it is likely that additional reports of phytoplasma infection of lily will occur in the future.

Management. Integrated management strategies are needed to control phytoplasma diseases. These include removing of diseased plants, controlling and protecting plants from insect vectors, and removal of potential hosts, including weeds, near production areas that may harbor the disease.

4 Viral Diseases

4.1 General Aspects

Eleven of the most important viruses occurring in lily are listed in Table 1. *Lily symptomless virus* (LSV) and *Lily mottle virus* (LMoV) are the most common viruses in lily and have spread worldwide with the movement of infected planting material. They have a significant impact, especially for bulb growers. LSV occurs in lily as a single infection and has been reported in mixed infections with one or more viruses, such as *Arabis mosaic virus* (ArMV) (Asjes and Segers 1983), *Cucumber mosaic virus* (CMV) (Allen and Lyons 1969), *Lily mottle virus* (LMoV) (Asjes et al. 1973; Dekker et al. 1993; Derks et al. 1994), *Lily virus X* (LVX) (Stone 1980), *Strawberry latent ringspot virus* (SLRSV) (Asjes and Segers 1983), and *Tobacco rattle virus* (TRV) (Derks 1975).

Table 1 Viruses in the genus *Lilium*

Name	(Synonyms)	Genus	Vector	References
Arabis mosaic virus	(ArMV)	*Nepovirus*	Nematodes	Asjes and Segers (1983)
Cucumber mosaic virus	(CMV)	*Cucumovirus*	Aphids, mech.	Allen and Lyons (1969)
Lily mottle virus	(LMoV)	*Potyvirus*	Aphids	Dekker et al. (1993)
Lily symptomless virus	(LSV)	*Carlavirus*	Aphids	Derks and Asjes (1975)
Lily virus X	(LVX)	*Potexvirus*	Unknown, mech.	Asjes (1991)
Plantago asiatica mosaic virus	(PlAMV)	*Potexvirus*	Unknown, mech.	Anonymous (2010)
Strawberry latent ringspot virus	(SLRSV)	not assigned	Nematodes, seed	Cohen et al. (1995)
Tobacco necrosis virus	(TNV)	*Necrovirus*	Fungi	Derks et al. (2003)
Tobacco rattle virus	(TRV)	*Tobravirus*	Nematodes	Derks (1975)
Tulip breaking virus	(TBV)	*Potyvirus*	Aphids	Asjes et al. (1973)

General Management Recommendations

- ***Cultural practices*** – To control viruses, transmission should be prevented by continuous elimination of infected plants during cultivation (rogueing), elimination of weed hosts, and controlling vectors. Without these steps, the incidence of virus will increase in planting stocks during the growing season (Allen and Linderman 1976). During harvesting, processing, and multiplication (scaling), there is a high risk for transmission of potexviruses (like *Plantago asiatica mosaic virus*, PlAMV). During storage, spread of mite-borne viruses such as *Tulip virus X* (TVX) might occur.
- ***Virus-free plant material*** – Viruses can be eliminated from infected lily tissues by the use of *in vitro* culture of isolated meristems adventitiously formed on bulb scale explants (Allen 1974, 1975; Allen and Anderson 1980; Allen and Fernald 1972; Sochacki et al. 2012). The development of an *in vitro* procedure for a series of cultivars together with improved serological testing by ELISA (Beijersbergen and van der Hulst 1980; Kong et al. 2009) and the rapid propagation of plantlets by tissue culture methods (Van Aartrijk and Van der Linde 1986; Nesi et al. 2009) has enhanced the buildup of virus-free stocks in the Netherlands (Asjes 1974, 1976). Also, in the Netherlands, the Flower Bulb Inspection Service (BKD) developed and now utilizes an extensive and rigorous virus detection program to minimize the risk of virus in lilies (Asjes 2000; Conijn 2014).
- ***Pesticides*** – The routine spraying of mineral oil plus pyrethroid insecticides reduces the spread of the prevalent LMoV and LSV and has enabled growers to expand lily culture in the Netherlands from tens of hectares in the 1960s to ca. 3500 in the second half of the 1990s till now (Asjes 1997). Nematicides are used to control the nematode vectors of TRV, ArMV, and SLRSV.
- ***Resistance*** – Resistance to LMoV has been found in some cultivars of the Asiatic hybrid group. It is a monogenic traits segregating in certain populations and present in a number of Asiatic and LA-cultivars (Shahin et al. 2009, 2010; Van Heusden et al. 2002; Van Tuyl et al. 2012). Lily breeders are using these sources, and molecular marker techniques have been developed in order to make early selection possible (Van Heusden et al. 2002; Arens et al. 2014). Resistance to TBV occurs in tulip (Marasek-Ciolakowska et al. 2012).

Additional information on virus management can be found in ► Chap. 4, "Insect Management for Disease Control in Florists' Crops".

4.2 *Arabis mosaic virus* (ArMV, Genus: *Nepovirus*)

Geographic occurrence and impact. ArMV has a very broad host range and infects both monocots and dicots. Various bulbous plants are hosts of this virus, among them lily, tulip, and crocus. The geographical distribution of ArMV is worldwide.

Symptoms/signs. Mosaic and/or necrotic stripes and spots (Fig. 10). Asjes and Segers (1983) described ArMV as the causal agent of the "cream bulb disease,"

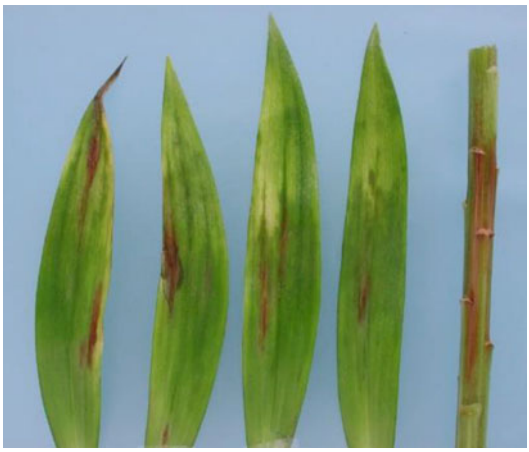


Fig. 10 Mosaic and necrotic stripes and spots associated with ArMV (Wageningen University and Research © 2017. All Rights Reserved.)

recognizable for its cream-colored scales with necrotic ring spots. ArMV can also occur symptomlessly in lily.

Biology and epidemiology. ArMV is transmitted by nematodes, more specifically *Xiphinema diversicaudatum* (Jha and Posnette 1959; Harrison and Cadman 1959) and *X. coxi* (Fritzsche and Schmidt 1963). ArMV is also transmitted via seed of various crops and weeds.

Management. Cultivate virus-free plant material on *Xiphinema*-free plots. Weed control is of utmost importance, as many are hosts for ArMV and this virus can be seed transmitted in weeds (Asjes and Segers 1983).

4.3 *Cucumber mosaic virus* (CMV, Genus: *Cucumovirus*)

Geographic occurrence and impact. CMV has a very broad host range in both monocots and dicots. CMV infects numerous crops and weeds, among them several flower bulbs, e.g., lily, tulip, *Muscari*, daffodil, and crocus. The geographical distribution of CMV is worldwide.

Symptoms/signs. A common symptom of CMV on the leaves of lily is a light-green mosaic, often evolving to gray necrotic spots in Longiflorum lilies and into brownish necrotic spots in Asiatic cultivars (Fig. 11). Brittleness of leaves and malformation of flowers and flower buds are typical symptoms of CMV infection. More severe symptoms can be observed when plants are coinfected with LSV.

Biology and epidemiology. CMV is transmitted by a large number (>80 reported) of aphid species in a non-circulative (nonpersistent) manner. This means that the virus can be acquired and inoculated by the aphid during probing. CMV can be seed



Fig. 11 Mosaic pattern on leaves associated with CMV infection (Wageningen University and Research © 2017. All Rights Reserved.)

transmitted in some hosts. Mechanical transmission through contaminated knives, machines, or water has been reported (Conijn 2014).

Management. Starting with virus-free planting material is of primary importance. Control measures consist of reduction of the transmission by aphids through monitoring and control of aphids and plant treatments with mineral oil and pyrethroids. Aphid transmission can be decreased with reflective mulches or by the use of fine mesh nets or screens. Remove virus sources by rogueing crops and thorough weed control.

4.4 *Lily mottle virus* (LMoV, Genus: *Potyvirus*)

Geographic occurrence and impact. LMoV is the most common virus in lily and has spread worldwide. The host range is mostly restricted to the Liliaceae family (Lisa et al. 2002).

Symptoms/signs. Symptoms can include vein clearing, leaf mottle, leaf mosaic, chlorotic and yellow streaking, leaf curling, and narrowing. Some cultivars can show color breaking of the flower (Fig. 12).



Fig. 12 Flower break associated with LMoV infections (Wageningen University and Research © 2017. All Rights Reserved.)

Biology and epidemiology. LMoV is transmitted by aphids in a nonpersistent way (Asjes 2000; Conijn 2014).

Management. Use virus-free or certified plant material in combination with sprays of mineral oil plus pyrethroid insecticides, to prevent virus spread. Resistance to LMoV occurs in some cultivars of the Asiatic hybrid group.

4.5 *Lily symptomless virus* (LSV, Genus: *Carlavirus*)

Geographic occurrence and impact. LSV is the most harmful virus in lily and has spread worldwide. It has a host range restricted to the Liliaceae family and is transmitted by aphids nonpersistently (Fischer 2013).

Symptoms/signs. LSV can occur symptomlessly in lily and *Alstroemeria*. The name *Lily symptomless virus* is perhaps unfortunate because other lily viruses can also sometimes infect lilies symptomlessly, and LSV itself causes symptoms in lilies in certain genotypes under certain environmental conditions. Symptoms can include mild, pale veinclearing and mottle on leaves in lilies grown for cut flowers under glasshouse conditions (Fig. 13). The leaves of LSV-infected plants generally turn yellow fairly quickly (Boontjes 1983).

Biology and epidemiology. LSV is transmitted by the aphids *Macrosiphum euphorbiae*, *Myzus persicae*, *Aphis gossypii* and others species in a nonpersistent way (Asjes 2000; Conijn 2014). In the Netherlands, aphid flights with concomitant virus spread gradually increase in April/May, with a bigger increase in June and July (Asjes 1997, 2000).



Fig. 13 Mild veinclearing and mottle on leaf (*right*) associated with LSV infection (Wageningen University and Research © 2017. All Rights Reserved.)

Management. Use of virus-free planting material in combination with sprays of mineral oil plus pyrethroid insecticides to prevent virus spread.

4.6 *Lily virus X* (LVX, Genus: *Potexvirus*)

Geographic occurrence and impact. LVX has a very narrow host range. It infects lily and some weeds (*Chenopodium quinoa* and *Tetragonia expansa* under experimental conditions).

Symptoms. LVX infects lily without causing symptoms (latent infection).

Biology and epidemiology. LVX is transmitted via contact (mechanically). There is no known vector for LVX, but vector transmission is suspected as the use of insecticides (pyrethroids) has limited transmission (Asjes 2000).

Management. Start with virus-free plant material. Hygienic measures and awareness of virus sources (infected plant material, water in dipping baths) are extremely important for contact-transmitted viruses, like potexviruses.

4.7 *Plantago asiatica mosaic virus* (PlAMV, Genus: *Potexvirus*)

Geographic occurrence and impact. PlAMV has a very broad host range, in both monocots and dicots. It was discovered in lily in recent years (Anonymous 2010). Hosts of the virus are lily, various weeds, and even shrubs like *Nandina domestica.*

Symptoms/signs. Rust-brown necrotic spots occur mostly on the backside of the leaf (Fig. 14**)**. On the upper side grayish spots are sometimes observed. Later, brown necrotic spots occur along the veins on the upper side of the leaf (mostly at the end of the vegetative phase). Leaves of infected plants are often brittle. PlAMV can also be symptomless in lily. Mixed infections of PlAMV with LSV or LMoV can cause much more severe symptoms including dwarfism.



Fig. 14 Rust-brown necrotic spots leaves from PlAMV-infected plant (Wageningen University and Research © 2017. All Rights Reserved.)

Biology and epidemiology. Transmission is mostly via contact with infested water in dipping baths. Also, transmission via soil, without a vector, has been reported (De Kock et al. 2013).

Management. Start with virus-free plant material. Hygienic measures and awareness of virus sources (infected plant material, water in washing and dipping baths) are extremely important for contact-transmitted viruses, like potexviruses. Avoid plots where PlAMV-infected plants have been grown; plots can remain contaminated with this virus through overwintering weeds and infected volunteers.

4.8 *Strawberry latent ringspot virus* (SLRSV, Not Assigned to a Genus)

Geographic occurrence and impact. SLRSV has a broad host range in both monocots and dicots. Various ornamental, vegetable, and fruit crops can be infected by SLRSV. Known flower bulb hosts are lily and daffodil.

Symptoms/signs. SLRSV in lily is symptomless; however, there is one report of asymmetric opening of flowers in the Oriental hybrid cultivar 'Stargazer' (Cohen et al. 1995).

Biology and epidemiology. SLRSV is transmitted by the free-living nematodes *Xiphinema diversicaudatum* and *X. coxi*. Also seed transmission in various crops was reported, including lily (Verbeek and Stijger 2016).

Management. Start with virus-free plant material on *Xiphinema*-free plots, and also control weeds. Nematodes can be controlled chemically with nematicides and also by biological measures such as planting certain *Tagetes* spp. and by biofumigation. Be aware of possible seed transmission in seed-propagated lilies. (Additional information on nematode management may be found in Sect. 5 below and in ▶ Chap. 8, "Soil/Media Disinfestation for Management of Florists' Crops Diseases").

4.9 *Tobacco rattle virus* (TRV, Genus: *Tobravirus*)

Geographic occurrence and impact. TRV has a very broad host range in more than 50 monocotyledonous and dicotyledonous plant families. Various food crops, ornamentals, and weeds are infected by TRV. Among the flower bulbs, lily, tulip, hyacinth, crocus, and daffodil are hosts of this virus.

Symptoms/signs. Chlorotic spots and veinal chlorosis, small necrotic spots and twisted growth, and leaf curling.

Biology and epidemiology. TRV is transmitted by nematodes from the genera *Trichodorus* and *Paratrichodorus* (family Trichodoridae). Both adult and juvenile nematodes can transmit the virus, but the virus is probably lost when the nematodes molt. Nonfeeding nematodes can retain the virus for several months. Seed transmission was reported for some weeds (shepherd's purse, poppy).

Management. Use virus-free plant material and nematode-free plots. Control weeds. Nematodes can be controlled chemically with nematicides and also by biological measures such as planting certain *Tagetes* spp. and by biofumigation. (Additional information on nematode management may be found in Sect. 5 below and in ▶ Chap. 8, "Soil/Media Disinfestation for Management of Florists' Crops Diseases".

4.10 *Tulip breaking virus* (TBV, Genus: *Potyvirus*)

Geographic occurrence and impact. TBV is the most common virus in tulip but is also found in lily. The virus has a host range restricted to the *Tulipa* and *Lilium*.

Symptoms/signs. TBV causes mild to moderate mottling in the leaves and has been associated with a brown ring formation in the Asiatic hybrid cultivar "enchantment" (Derks 1976).

Biology and epidemiology. TBV is transmitted by aphids in a nonpersistent way (Asjes 2000; Conijn 2014).

Management. Use virus-free or certified plant material in combination with sprays of mineral oil plus pyrethroid insecticides to prevent virus spread. Resistance to TBV occurs in *Tulipa fosteriana* (Marasek-Ciolakowska et al. 2012).

4.11 *Tulip virus X* (TVX, Genus: *Potexvirus*)

Geographic occurrence and impact. Hosts of the virus are tulip, lily, and various weeds.

Symptoms/signs. *Symptoms:* sunken, light green or yellow spots in between the veins, and lighter or darker stripes at the outside of the flowers (Fig. 15).

Biology and epidemiology. Transmission mostly via contact with infested water in dipping baths. Mite transmission is suspected. Transmission via soil, without a vector, has been reported (De Kock et al. 2013).

Management. Start with virus-free plant material. Hygienic measures and awareness of virus sources (infected plant material, water in washing and dipping baths)



Fig. 15 Sunken, light green to yellow spots in between veins on a TVX-infected plant (Wageningen University and Research © 2017. All Rights Reserved.)

are extremely important for contact-transmitted viruses like potexviruses. Avoid plots where TVX-infected plants have been grown; plots can remain contaminated with this virus through overwintering weeds, etc.

5 Nematode Diseases

5.1 Root Lesion Nematode (*Pratylenchus penetrans* (Cobb, 1917) Filipjev and Schuurmans Stekhoven, 1941)

Geographic occurrence and impact. *P. penetrans* is mainly found in temperate areas including the USA, Europe, Australia, Canada, Egypt, India, Japan, New Zealand, Peru, the Philippines, Zimbabwe (Rhodesia), Russia, South Africa, and Tunisia (Goodey et al. 1965; Siddiqui et al. 1973). It has a wide host range and more than 350 hosts have been recorded including apple, cherry, other fruit trees, conifers, roses, tomato, potato, corn, and sugar beets. It is considered the most economically important plant-parasitic nematode in the northeastern USA and field-grown Easter lilies in California and Oregon and in the Netherlands when lilies are grown on light and sandy soils (Conijn 2014; Westerdahl et al. 2003).

Symptoms/signs. Disease symptoms include a general devitalization of the plant: retarded top growth, chlorotic foliage, and restricted root growth (Overman 1961) (Fig. 16). Shoots may not be able to emerge from bulbs. In less serious cases, the symptoms are not evident until late in the growing season. Because these symptoms could be indicative of problems other than nematodes, soil and root samples should be sent to a nematode diagnostic laboratory for evaluation.

Biology and epidemiology. *P. penetrans* is a migratory endoparasite, with all life cycle stages found either within roots or in the soil. Reproduction is sexual. Females lay eggs singly in roots or in soil. Second-stage juveniles hatch from eggs, feed, and undergo three molts to the adult stage. The complete life cycle takes 30–86 d,



Fig. 16 Stunting and yellowing of plants due to root lesion nematode (Becky B. Westerdahl © 2017. All Rights Reserved.)

depending on temperature, and is shortest at 30 °C/86 °F. Adults, J2, J3, and J4, can all invade roots. If conditions in the root become unfavorable, any juvenile stage or the adult nematodes may leave the root and invade other nearby roots. Invasion usually takes place in the region of root elongation. Root lesion nematodes feed upon cells in the root cortex. Cells are killed and in many instances, small roots are killed. The migratory parasitism of this nematode opens up roots to secondary invasion by other soil microorganisms such as fungi and bacteria (Corbett 1973).

Management. The number of nematodes in the soil, soil characteristics, and the nematode populations in the roots of planted bulbs affects the degree of damage caused by the root lesion nematode. Conijn (2014) reports that researchers at Wageningen University Research (WUR) are developing a computer-based, decision-support system named NemaDecide to predict the probability of damage by nematodes (Elberse et al. 2012). Hot water treatment of recently harvested bulbs and a subsequent storage period at −2 °C/28 °F for 2 mo before planting is an effective method to control *Pratylenchus penetrans* in lily planting stock (Conijn 1966, 2014). This approach prevents nematode damage to bulb roots and also reduces their spread to uninfested fields.

Marigolds (*Tagetes* spp.), which are known to produce the nematicidal compound thiophene α-therthienyle (Oostenbrink et al. 1957; Conijn 1994; Pudusaini et al. 2006; Gommers and Bakker 1988), are sometimes used by Dutch lily growers who sow them directly after harvesting tulips or other spring-flowering bulb crops and have been shown to reduce populations of nematodes in the soil by 90% after 105 d (Pudasaini et al. 2006).

Easter lily growers in Oregon and California face a number of challenges in managing root lesion nematodes. Field-grown bulbs are sold to greenhouse operations for forcing to produce potted, flowering plants at Easter. The quality of field-grown bulbs is based on bulb circumference and appearance, with only bulbs with white scales that have plentiful roots being saleable. Nematode management is complicated because bulbs must be grown for 2–4 y before they are large enough for sale. Land is prepared for fumigation in May, fumigated in July, bulblets are planted from August

through October with a planting treatment of a nonfumigant nematicide, and bulbs are harvested the following August through October (D'Herde et al. 1960; Roberts et al. 1985) (Fig. 17). Bulbs not large enough for sale are replanted for an additional growing season. Avoidance of the root lesion nematode problem in Easter lilies by crop rotation or planting site selection is problematic. The nematode has a very wide host range and is supported by the pasture grasses that are commonly grown as an alternative to bulb production (Giraud et al. 2011; Westerdahl et al. 1993, 2003).

5.2 Foliar Nematode: (*Aphelenchoides* spp.)

Geographic occurrence and impact. Foliar nematodes have a wide host range and are found in temperate and tropical regions. *A. fragariae* has an extensive host range, including fern, lily, begonia, African violet, strawberry, and many aquatic plants (Goodey et al. 1965; Siddiqui et al. 1973).

Symptoms/signs. Foliar nematodes produce discolored streaks in lily foliage and will cause the bulbs to decline (Gill et al. 2006). Infested plants are stunted and distorted (Fig. 18). Infested flower buds may not bloom or flowers may be distorted (Siddiqi 1975). Because these symptoms could be indicative of problems other than nematodes, soil and plant samples should be sent to a nematode diagnostic laboratory for evaluation.

Biology and epidemiology. Foliar nematodes are migratory endoparasites in leaves, but also feed ectoparasitically on leaf and flower buds. Nematodes enter leaves through stomata or by penetrating tissues. They have been shown to move long distances on infested plant material (Handoo 2012). They reproduce sexually. The life cycle is completed in 10–13 d and females produce about 30 eggs. Unlike

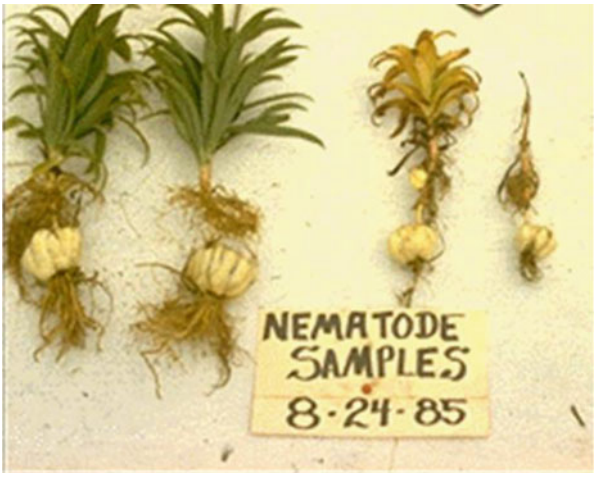


Fig. 17 Growth of Easter lilies treated with a nonfumigant nematicide (*left*) at the time of planting in a field infested with root lesion nematode (Easter Lily Research Foundation © 2017. All Rights Reserved.)



Fig. 18 Severely stunted plant (*center*) infested with foliar nematodes (Department of Entomology and Nematology, UC Davis, Slide Collection © 2017. All Rights Reserved.)

most plant parasites that appear sedentary in water, foliar nematodes are rapid swimmers. They survive in soil for about 3 mo, with longer survival in plant tissue. Weeds can harbor the foliar nematodes that infect lily. The nematode overwinters in dead plant tissues in soil and also in buds and growing points. It thrives best in moist, cool situations (Siddiqi 1975).

Management. Cultural practices include rogueing plants with symptoms, controlling weeds, using irrigation practices that reduce the incidence and duration of leaf wetness and splash dispersal of the nematode, hot water treatments of planting stock (Jagdale and Grewal 2002, 2004), burning infested material, starting/replacing with healthy stock, and general sanitation. Foliar or soil treatments with systemic chemicals are also effective (Kohl 2011; Siddiqi 1975).

5.3 Stem and Bulb Nematode: (*Ditylenchus dipsaci* (Kühn, 1857) Filipjev, 1936)

Geographic occurrence and impact. Stem nematode is widely distributed in temperate regions. It is a common crop pest with more than 450 hosts (Goodey et al. 1965; Siddiqui et al. 1973). Host range determination is complicated by there being as many as 25 host races or biotypes, some with a limited host range (Greco et al. 1991). The bulb race infests most bulbs including hybrid lilies, daffodil, narcissus, and tulip (Hooper 1972).

Symptoms/signs. Distortion and swelling of leaves and bulbs are common symptoms (Greco et al. 1991). The nematode causes individual bulb scales to rot and eventually can kill the bulb (Gill et al. 2006). Secondary invasion by bacteria and fungi may contribute to bulb rot. Because these symptoms could be indicative of problems other than nematodes, soil and plant samples should be sent to a nematode diagnostic laboratory for evaluation.

Biology and epidemiology. *D. dipsaci* is a migratory endoparasite, meaning that all life stages can be found either within a plant or in the soil. At the beginning of the crop season, fourth-stage juveniles enter young tissues through stomata or by direct penetration. The nematode is thought to secrete a pectinase enzyme that breaks down plant tissues. Migration on plant parts aboveground requires free water and may occur after rain or sprinkler irrigation. Wet conditions and mild temperatures (15–20 °C/59–68 °F) favor infestation. Under dry conditions, the nematode survives in plant tissues as a quiescent fourth-stage juvenile, and this aids spread in plant materials (Greco et al. 1991).

Management. Hot water treatment of bulbs has been a common practice for managing this nematode. However, a hot water bath is also an effective means to spread fungi and bacteria so that an additive is needed to control these pests (Qiu et al. 1993). Soil treatments with nematicides will control nematodes present in the soil but not those contaminating planting stock. Rotation for 2–3 y to nonhost crops plus weed control is an effective cultural practice. Using drip rather than overhead irrigation will help to minimize spread of the nematode within a crop (Greco et al. 1991).

5.4 Root-Knot Nematode: (*Meloidogyne* spp.)

Geographic occurrence and impact. Root-knot nematodes are found worldwide but are more common in temperate, subtropical, and tropical areas (Goodey et al. 1965; Mitkowski and Abawi 2003; Siddiqui et al. 1973; Taylor and Sasser 1978). Several species including *M. incognita*, *M. chitwoodi*, and *M. fallax* have been reported as being pathogenic to lilies (Den Nijs et al. 2004). Root-knot nematodes are a quarantine pest in many countries. The cosmopolitan distribution of root-knot nematode is the result of the movement of infested plants, water, soil, and equipment.

Symptoms/signs. Plants exhibit poor growth and roots infected with root-knot nematodes show round swellings, or galls (root knots), typically most noticeable on the small feeder roots. There is poor top growth and the foliage is frequently chlorotic (yellow) because essential elements are not taken in and transported by the impaired root system. Severe infections cause wilting of the foliage and the plants require more frequent irrigations. Because these symptoms could be indicative of problems other than nematodes, soil and root samples should be sent to a nematode diagnostic laboratory for evaluation. Female nematodes are visible under a microscope in cross sections of the root swellings and nematode infestations will usually appear as circular patches in the field.

Biology and epidemiology. Root-knot nematodes typically have very broad host ranges with some differences evident between species. *M. incognita*, for example, has more than 700 hosts. These include most cultivated crops and ornamentals.

Root-knot nematodes are sedentary endoparasites. Some species are parthenogenic, not requiring males for reproduction. Second-stage infective juveniles hatch from the eggs. These invade roots in the region of elongation near the root cap. They migrate between and through cells and position themselves with the head in the vascular tissues. Cell damage occurs as a result of the migration and if several juveniles enter the root tip, cell division stops and there is no root elongation. As feeding continues, several cells near the head begin to enlarge and become multinucleate. These are called giant cells and there are usually 3–6 associated with each female nematode. The formation of giant cells and galls is the result of cell enlargement and of increased numbers of cells. Giant cells are induced by salivary secretions that are introduced into cells and surrounding tissues during feeding by the nematode. During this process, the xylem vessels become disrupted and the roots cannot function normally with respect to water and nutrients. During the process of gall formation, the nematodes undergo second, third, and fourth molts to reach the adult stage. Mature females are saccate (pear-shaped) and lay eggs into a gelatinous matrix. This matrix may protrude from the surface of small roots or may be entirely within the gall. Eggs hatch in about 7 d. The length of the life cycle is dependent on species as well as soil temperature. For *M. incognita*, the entire life cycle is completed in 20–25 d at 21 °C (70 °F). A subsequent generation can be borne in as few as 2 wk, depending on species and environment, and eggs can remain viable in the soil for a year (Mitkowski and Abawi 2003). Males, when present, remain vermiform (Westerdahl and Frate 2007).

Management. Preplant treatment of soil with nematicides can reduce disease risk in areas known to contain root-knot nematodes (Mitkowski and Abawi 2003). Adjusting planting dates to cooler times of the season when nematodes are less active can help to reduce damage. Providing optimal conditions for plant growth including sufficient irrigation and soil amendments can help make plants more tolerant to nematode infestation.

5.5 Needle Nematode: (*Longidorus* spp.)

Geographic occurrence and impact. India, Israel, South Africa, the USA, and Zimbabwe (Goodey et al. 1965; Siddiqui et al. 1973).

Symptoms/signs. Needle nematode is devastating to seedling root systems. Plants are stunted and galls are formed at root tips (Radewald et al. 1969). Because these symptoms could be indicative of problems other than nematodes, soil and root samples should be sent to a nematode diagnostic laboratory for evaluation.

Biology and epidemiology. Needle nematodes are migratory ectoparasites with all stages being found outside of roots in the soil. They prefer coarse, well-drained soils. The nematode feeds at root tips (Fig. 19). Males are rare and reproduction is probably by parthenogenesis. A first-stage juvenile hatches from the egg. For



Fig. 19 Ectoparasitic nematode feeding on root tip (Department of Entomology and Nematology, UC Davis, Slide Collection © 2017. All Rights Reserved.)

L. africanus, the life cycle from adult to adult is 7 to 9 wk at 28 °C/82 °F, and it can survive in fallow soil for at least 3 mo at 25 °C/77 °F (Radewald et al. 1969). This genus is capable of transmitting *Strawberry latent ringspot nepovirus* and other viruses to plants (Adekunle et al. 2006), and this can result in asymmetrical opening of flowers (Cohen et al. 1995).

Management. Preplant nematicides have been shown to be effective. For *L. africanus*, timing of planting of crops to avoid temperature conditions (above 16 °C/61 °F) favorable to the nematode has been shown to reduce damage (Kolodge et al. 1979; Radewald et al. 1969).

Additional information on nematode management may be found in ▶ Chap. 8, "Soil/Media Disinfestation for Management of Florists' Crops Diseases".

6 Physiological Disorders

Physiological disorders are those not associated with pathogens such as bacteria, fungi, or viruses. They are sometimes referred to as abiotic disorders and are caused by numerous factors (nonoptimal light levels, temperatures, soil aeration, nutrition, water availability, or presence of toxins) that adversely affect plant growth. For example, the presence of ethylene in the greenhouse atmosphere may cause flower abortion, a physiological or abiotic disorder. Or suboptimal root zone aeration may interfere with normal root function, leading to reduced micronutrient uptake that is expressed as a nutrient deficiency. One must be careful, however, as it is conceivable that a root disease organism could cause similar symptoms as a result or reduced root functionality from the infection. Thus, diagnosis of abiotic or physiological disorders should include, as much as possible, the elimination of biotic causes.

Below is a summary of several specific physiological disorders that occur on lilies.

6.1 Upper Leaf Necrosis (ULN)

This disorder has long been present in forced (greenhouse grown) lilies, most especially in the Oriental hybrid group. It is characterized by necrotic, usually twisted young upper leaves associated with the buds (Fig. 20). The disorder was specifically named ULN by Chang and Miller (2003), and their extensive research has led to a much clearer understanding of this disorder (Chang and Miller 2004, 2005a; Chang et al. 2004). It is also seen on lilies in the Asiatic and LA-hybrid groups, where it presents significantly different symptoms of white-gray bands across the leaves in the upper third of the plant, with the leaf tips remaining green (Berghoef 1986) (Fig. 21). The disorder appears during greenhouse production of cut flowers or potted plants and is a calcium deficiency disorder. Chang and Miller's work demonstrated the relationship to calcium in a number of ways and included analysis of symptomatic and non-symptomatic tissue (Chang and Miller 2005a), alterations in transpiration of young leaves (Chang and Miller 2004), applications of exogenous calcium (Chang and Miller 2005b), demonstration of increased symptom severity in plants growing from bulbs depleted in calcium (Chang and Miller 2003), reduction of the problem as a result of daily foliar calcium applications (Chang et al. 2004), the relationship of severity of the disorder to bulb size (Chang and Miller 2005b), and very significant differences in cultivar susceptibility (Chang et al. 2008).

Chang's research was key in dispelling several "industry myths" about this disorder, namely, that it was caused by a warm, sunny day following an extended period of dark, cloudy weather. Onset of the disorder bore no relationship to prior weather conditions (Chang and Miller 2005a). A key realization was that plants growing from larger bulbs, which have much greater sensitivity to the disorder, have more buds and young, developing leaves associated with them. It is these very leaves that suffer from reduced transpiration and calcium uptake that eventually show calcium deficiency, expressed as upper leaf necrosis.

While foliar sprays of calcium salts (nitrate or chloride) can be effective, daily sprays were necessary (Chang et al. 2004) and are mostly impractical. Berghoef



Fig. 20 Upper leaf necrosis (ULN) on an Oriental hybrid lily cultivar (William B. Miller © 2017. All Rights Reserved.)



Fig. 21 Calcium deficiency, essentially the same disorder as ULN, as expressed on an Asiatic hybrid lily cultivar (William B. Miller © 2017. All Rights Reserved.)

(1986) similarly reported the potential for phytotoxicity as a result of the required calcium sprays.

6.2 Leaf Scorch

"Leaf scorch" is a term that should be used carefully when discussing lilies as much Dutch literature will refer to ULN (see above) as "leaf scorch". Here, however, we take this term to mean leaf injury caused by fluoride toxicity from root uptake. Specifically, this injury presents as necrotic areas on the leaf tip or margin, with a surrounding yellow (chlorotic) halo. This problem was commonly seen in Easter lily (*Lilium longiflorum*) crops in the 1960s and 1970s before Marousky and Woltz (1975) Woltz and Marousky (1975) demonstrated that fluoride in fertilizer solutions (commonly seen in superphosphate fertilizer) caused leaf scorch in lily if applied in sufficient amounts to plants grown in acid soils (pH 5.0). There are significant cultivar differences (e.g., the now-extinct cultivar "Ace" was more susceptible to the disorder than is "Nellie White"), and similar differences probably exist in modern cultivars. Fluoride-induced leaf scorch is readily controlled by manipulation of root zone pH, with a more alkaline reaction reducing fluoride availability and uptake.

6.3 Lower Leaf Yellowing

Whether grown as pot plants or cut flowers, many lilies are susceptible to chlorosis and yellowing of the lower leaves. There are two main classes of leaf yellowing in *Lilium* (Miller 2013). The first is yellowing and gradual senescence of lower leaves during crop growth in the greenhouse (Fig. 22). This is essentially a nutritional disorder (principally nitrogen deficiency) that can be caused by suboptimum fertilizer applications or by root injury from any cause, whether biotic (root pathogens) or abiotic (cold root zone, hypoxic root zone). The symptoms develop over several



Fig. 22 Lower leaf yellowing, of the "gradual" or "greenhouse" type (William B. Miller © 2017. All Rights Reserved.)

weeks and can usually be corrected by increasing fertilizer application, using a different root substrate and altering (usually reducing) irrigation frequency.

A more interesting lower leaf disorder is one that leads to rapid loss of chlorophyll from most if not all of lower to mid-leaves, usually in the retail or consumer phase, which has been described in detail and has been termed "catastrophic leaf yellowing" (Fig. 23) (Miller 2013). This disorder is exacerbated by prolonged cold storage which commonly occurs in the potted plant or cut flower postharvest chain. The disorder gained prominence with Easter lily in the late 1980s in the USA, when retailers reported that plants turned yellow within days of being placed in the retail environment. Research ultimately indicated that in addition to postharvest cold storage, the problem is exacerbated by anti-gibberellin growth regulators (used for reducing height for potted plant production) (Ranwala et al. 2000), and by growing plants in "negative DIF" environments, where night temperatures are warmer than day temperatures, also a height control technique (Ranwala et al. 2000).

While the initial work on this problem focused on *Lilium longiflorum*, subsequent research has examined lower leaf yellowing-related disorders on hybrid lilies. Ranwala and Miller (2005) found significant differences in hybrid lily cultivar susceptibility to cold-storage-induced lower leaf yellowing.

Practical solutions based on exogenous application of gibberellin$_{4+7}$ (GA_{4+7}) have been developed for the industry to reduce this problem (Fig. 24). Gibberellin$_{4+7}$ sprays are highly effective in reducing or eliminating lower leaf yellowing in the postharvest phase. In various regions of the world, registered GA products include Fascination (Valent BioSciences), Fresco (Fine Americas), BVB (Chrysal), and Bulb 100 (Floralife). While these products contain equal concentrations of GA_{4+7} and benzyladenine, research has shown that benzyladenine is essentially inert against leaf yellowing and nearly all the activity is from the GA_{4+7} (Ranwala and Miller 1998, 1999, 2000). For pot plant production, sprays of 50–100 ppm are applied one to two times during plant production. The details of how this is done are quite involved and beyond the scope of this review. The essential concerns are that the gibberellin has the potential to cause stem elongation (undesirable for pot plants); furthermore, elongation



Fig. 23 Leaf yellowing of the "catastrophic" type, after 2 wk in a 4 °C/39 °F dark cooler prior to being placed in a postharvest evaluation room. There was no leaf yellowing at the start of the cold treatment (William B. Miller © 2017. All Rights Reserved.)



Fig. 24 These plants were treated with GA_{4+7} and held in a 4 °C/39 °F cooler along with the plants in Fig. 23. The gibberellin inhibits the development of catastrophic leaf chlorosis (William B. Miller © 2017. All Rights Reserved.)

can come from GA sprayed on the aboveground part of the plant or through root uptake as a result of spray contacting the soil surface (Ranwala et al. 2003). Successful use of this product on Easter lilies in North America is predicated on careful spraying to only contact lower leaves and applying enough material to uniformly contact all leaf surfaces (to avoid green or yellow islands) while minimizing spray volume so as to reduce to runoff into the root zone.

For cut flowers, while plants may be sprayed with GA_{4+7} before harvest with excellent results, (Ranwala and Miller 2005), a more practical treatment is after cutting, while stems are being held in liquid before packing. In this case, stems are held in buckets with dilute GA_{4+7} and the GA moves into the stem and leaves with the xylem flow. For both potted plant and cut flower systems, the GA_{4+7} treatment has an added benefit of extending individual flower life and life of the entire stem (inflorescence) (Ranwala et al. 2000; Ranwala and Miller 2002, 2005).

6.4 Flower Bud Abortion and Abscission

The basis of most terminology related to floral abortion and abscission is derived from De Hertogh et al. (1971), Miller (1993), and Beattie and White (1993). Essentially, cessation of normal growth of a bud leading to its death at any stage is "abortion." Bud or flower "blast" is abortion, but only after all floral parts are visible. In common use, North American growers tend to refer to bud abortion as that occurring at a very early stage, when only a small remnant of the aborted bud is present (Fig. 25), and to "blasted" buds as those that die when larger than ca. 0.5 cm/0.2 in. (Fig. 26). Abscission is the straightforward detaching of the bud from the plant.

Many abiotic and biotic factors cause bud abortion, blasting, or abscission, and the hybrid groups show broad differences in their susceptibility to each factor. For example, Asiatic hybrid lilies are broadly susceptible to flower abscission and bud blast (buds usually less than 2 cm/0.8 in. long) from ethylene, suboptimal light, and supraoptimal temperatures in early crop growth (Durieux 1975; Durieux et al. 1982; Elgar et al. 1999). Oriental hybrid cultivars are much less susceptible to bud abortion and abscission overall, but small buds (less than ca. 1–3 cm/0.4–1.2 in. long) are readily injured by ethylene. *L. longiflorum* cultivars are susceptible to bud abortion and blast especially in early-Easter forcing years when growers are growing plants at warmer temperatures early in the crop (essentially in early January) when light levels are very low. Bud abscission is not commonly seen in *L. longiflorum*.

There are a number of management practices that help reduce the risk of bud abortion and abscission. These include avoiding forcing temperatures above 21 °C (71 °F) (especially for Asiatic hybrid lilies), forcing lilies under glass instead of plastic, and providing supplemental (assimilation) lighting during periods of low light. Greenhouse-scale treatments of the gaseous anti-ethylene compound 1-methylcyclopropene (1-MCP) are financially impractical but would likely be effective. Sprayable formulations of 1-MCP are available in agriculture but are not presently registered for use in North America or other areas of the world. Postharvest



Fig. 25 An Asiatic hybrid lily cultivar showing an aborted bud, barely visible at the base of the two top leaves. Two "blasted" buds are visible immediately beneath these two leaves (William B. Miller © 2017. All Rights Reserved.)



Fig. 26 Buds of *Lilium longiflorum* showing injury from ethylene exposure in a commercial greenhouse. Four buds are already dead or dying and a fifth (leftmost bud of the two upward-facing buds) may still fail to open (William B. Miller © 2017. All Rights Reserved.)

treatment of cut flowers with silver thiosulfate (STS) or 1-MCP also helps reduce the effects of ethylene and extends the vase life and quality of cut flowers.

6.5 Other Miscellaneous Disorders

Chemical Toxicity

There are a few specific toxicities known in lilies. One is leaf tip injury from the fungicide metalaxyl. In *L. longiflorum*, this is expressed as a whitening of the tip (ca. last 1 cm/0.4 in.) of leaves. Forced lilies have been injured by root uptake of the herbicide glyphosate in certain subirrigation regimes (Weller and Hammer 1984). Another is fluoride-induced leaf scorch (see Sect. 6.2).

Premature Flower Opening

Atmospheric ethylene contamination is a common cause of prematurely-opening buds in a number of lily cultivars (Fig. 27).

Bud "Split"

This is an uncommon disorder, with no clearly defined cause. Aphids have been attributed to this problem (Post 1941), yet split buds are typically observed to be insect-free.

Leaf Epinasty

Downward-curling leaves can be caused by a number of factors including negative DIF growing environments (i.e., warmer nights and cool days, as used for height management in pot plant crops), by ethylene (Blankenship et al. 1993) and by growing crops at very low temperatures. Epinasty is also commonly seen in mechanically-stimulated plants, a technique which is also potentially useful for height control. Epinasty is generally reversible by eliminating its cause.



Fig. 27 Two buds on this *Lilium longiflorum* plant are opening prematurely as a result of ethylene exposure in a commercial greenhouse (William B. Miller © 2017. All Rights Reserved.)

Stems Not Growing ("No Shows")

Stems may fail to grow out of the bulbs for a number of reasons, some related to preplant storage and others to conditions after planting. In the last 15–20 years, the lily industry has adopted frozen storage of bulbs to allow year-around planting and flower availability. Young shoots can be killed by too cold temperatures or by freezing the bulbs the wrong time (Gude and Kok 2005). Similarly, as the cold temperature (vernalization) tends to improve uniformity of sprouting of the population, suboptimal conditions during vernalization can lead to very slow and irregular emergence of the sprouts. Usually, the causes would be lack of oxygen, non-moist conditions or too short of a vernalization period (Miller 1992). During cooling, sprouts can grow out of the bulb and they are very susceptible to physical breakage.

Malformed Buds

Developing flower buds sometimes shows abnormalities such as malformed, twisted petals and longitudinal openings along the valve of the bud. This can be caused by high temperatures in the greenhouse after extended periods of frozen storage (Lee and Roh 2001).

Postharvest Bud Necrosis

Certain Oriental hybrid cultivars rapidly develop necrotic patches of blotches on unopened buds when placed in cold storage after harvest; essentially, chilling injury (Fig. 28). The problem is exacerbated by elevated greenhouse temperatures and high light levels before harvest and also by cold storage temperatures below approximately 4 °C/39 °F (Kim and Miller 2008).

Black Sprouts

"Black sprouts" is a problem in lily flower production especially for Oriental hybrid cultivars forced at late dates. During freezer storage, the sprouts in the lily bulb can be killed, become black, and do not flower. Delaying freezer storage



Fig. 28 Typical symptoms of "postharvest bud necrosis" which develops *during* cold storage of susceptible Oriental hybrid lily cultivars (William B. Miller © 2017. All Rights Reserved.)

until the bulbs are mature and respiration is low reduces this problem (Gude and Dijkema 2010).

Nutrient Deficiency

A number of nutrient deficiencies can occur in lily pot and flower production (IBC 2008). Barnes et al. (2011) concluded that symptoms of nitrogen (N), sulfur (S), boron (B), and iron (Fe) deficiencies and boron (B) toxicity are the first disorders to be manifested in Easter and hybrid lilies. In their experiments, they did not get visual symptoms when plants were grown under phosphate (P), potassium (K), magnesium (Mg), copper (Cu), manganese (Mn), molybdenum (Mo), and zinc (Zn) deficient conditions. Deficiency symptoms may not appear in some instances because the bulb may provide a source of these elements. The climate conditions in the greenhouse, moisture deficit, pH, and electrical conductivity (EC) of the soil are also important (Kok 2013). Lilies are sensitive to salt. Soil with an EC higher than 1.2 can give salt damage and hinder the absorption of nutrients. Iron and Mg are hard for lily plants to absorb in a high pH soils, so deficiency can occur. High relative humidity reduces mineral uptake and transport, exacerbating certain deficiencies, especially calcium (Ca, see Sect. 6.1).

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Diseases of Gladiolus

42

Wade H. Elmer and Kathryn K. Kamo

Abstract

Gladiolus (*Gladiolus* spp.) are in the Iridaceae family and are native to South Africa, and over 200 species have been described. Gladiolus have become a major crop in the florist industry. Growers of gladiolus plant their corms in the spring and harvest the flower spikes during the summer and early fall. Although the crop can be propagated sexually from seeds, most of the industry is based on movement of corms and cormels, which leads to many diseases being disseminated with the crop. Fusarium corm rot, Gladiolus rust, and Curvularia spot are the most limiting fungal diseases, whereas *Cucumber mosaic virus* and *Bean yellow mosaic virus* emerge as the more threatening viral diseases affecting gladiolus.

Keywords

Botrytis cinerea • *Fusarium* spp. • Potyvirus • Integrated disease management

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R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_47

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1 Introduction

Gladiolus (*Gladiolus* spp.) have a high economic value and play a major role in the global flower bulb industry (Benschop et al. 2010). *Gladiolus* spp. belong to the Iridaceae family, together with *Iris* and *Crocus* (Meerow 2012). Over 260 species have been described and all originate from Africa, Madagascar, and Eurasia (Goldblatt et al. 2008). Hybridization of gladiolus began in the 1800s to produce new colors when the Colvillei hybrids were first produced (Hartline 2015). Since then, hundreds of crosses have been developed. Today, gladiolus are a major crop in the florist industry. Growers of gladiolus plant their corms in the spring and harvest the flower spikes during the summer and early fall. The crop is propagated sexually from seeds, and asexually from corms and cormels, but commercial flower spikes are obtained from mature corms. In most northern climates, the corms and cormels are usually dug and stored in the fall. Where data are available, the management of major gladiolus diseases is presented in the following sections. We advise the reader to inform him/herself with the general management strategies found in the introductory chapters on integrated disease management.

2 Fungal and Fungus-Like Diseases

2.1 Botrytis Blight (*Botrytis spp.*): *Botrytis gladiolorum* Timm and *B. cinerea* Pers.

Geographic occurrence and impact. Botrytis blight develops on all florist crops. The fungi are ubiquitous and can attack gladiolus in the field when environmental conditions are conducive, but it causes more problems reducing shelf life as a postharvest disease. The first major report of the disease was in Florida in 1940 (Dimock 1940).

Symptoms/signs. Symptoms of Botrytis blight are characterized as three distinct spots: (1) very small, rust-colored spots that appear on only one side of the leaf; (2) small, yellowish brown spots that develop reddish brown margins; and (3) the large, oval spots that develop long, reddish margins (Magie 1956) (Fig. 1). Pinpoint water-soaked lesions also appear on flowers if the flower spike stays wet for at least 14 h (Magie 1956). Flower lesions enlarge and are first watery then dry and turn light



Fig. 1 Botrytis blight of *Gladiolus* (R.D. Milhollland © 2017. All Rights Reserved.)

brown unless the relative humidity is very high, and then the whole lesion becomes wet and slimy. Latent infections are common and cause postharvest losses during transit to market (Magie 1956). The fungus causes basal stem infections which can extend downward to the corm causing a neck rot. In severe cases, the fungus infects the corms and continues to rot the tissue in cold storage conditions.

Biology and epidemiology. There is much known about the Botrytis blight fungi. The fungi survive in soil as sclerotia and as mycelium on plant debris. The fungus requires very high humidity to infect and sporulate, so weather plays a key role in the development of the disease. The optimal temperature for disease development was found to be 20°± 10 °C (68 ± 18 °F), and disease severity will increase with increasing periods of leaf wetness reaching the maximum at 96 h (Sehajpal and Singh 2014).

Management

- ***Cultural practices*** – Reduction of the relative humidity and leaf wetness are critical for suppression of Botrytis blight. In field plants, growers should space plants in the row to allow for maximum airflow. Plants should be watered in the morning to allow time for leaves to dry.
- ***Sanitation*** – Careful inspection of corms for lesions can reduce the damage of Botrytis blight. Remove and discard senescing blossoms and plant debris in packing sheds and during postharvest.
- ***Fungicides*** – Botrytis blight is suppressed very effectively by fungicides. Care should be taken to rotate chemicals in different classes to delay the development of resistance. Many fungicides are effective against the disease. The fungicides chlorothalonil, iprodione, mancozeb, and strobilurins are all effective (Singh et al. 2005, 2008, 2011). The benzimidazoles may also be effective against certain species/strains, but resistance has emerged in many strains of *B. cinerea*. Copper oxychloride, triadimefon, ziram, difenoconazole, penconazole, hexaconazole, and tridemorph have also been shown to be effective. Sprays should be started before the appearance of the disease, preferably before the plants reach the flowering stage (Singh et al. 2005).

2.2 Curvularia Spot (*Curvularia gladiolus* Boerema and Hamers)

Geographic occurrence and impact. The disease was first noted in Florida, USA, in 1948 (Magie 1948) and in Canada in 1954 (Parmelee 1954). It was identified as *Curvularia lunata* (Parmelee 1956), renamed as *C. trifolii* f. sp. *gladioli*, and renamed again in 1989 as *C. gladioli* Boerema and Hamers (Boerema and Hamers 1989). It has been reported in North America (Magie 1948), South America (Torres et al. 2013b), China, Europe (Parmelee 1954), India (Singh 1968), and Philippines (Mendiola-Ela 1952). There is little information on the economic significance of Curvularia leaf spot, but some cultivars are highly susceptible (Torres et al. 2013b).

Symptoms/signs. The disease affects leaves, stems, and petals. Symptoms usually begin on leaves first as light to dark brown, oval spots. The symptomatic tissues show leaf spots that are oval to circular, brown with dark edges, and surrounded by a yellow halo. Often the lesions become necrotic, and the leaves acquire a dry and wilted appearance (Torres et al. 2013a). The fungus produces a black, powdery mass of spores in the center of the spot. Torres et al. (2013b) noted symptoms were a function of the host resistance. For example, on *G. grandiflorus* var. Red Beauty, the spots were more irregular and necrotic and surrounded by yellow halos. In other species, such as *G. callianthus*, Curvularia spots were round and always surrounded by yellow halos. More tolerant cultivars of *G. grandifloras*, such as Amsterdam, Friendship Rose, Tradehorn, Veronica, and Gold Yester, reacted differently to Curvularia leaf spot and exhibited light to dark brown spots. One report observed vascular discoloration (Forsberg 1957)

Biology and epidemiology. *C. gladioli* is favored by warm, wet conditions. Infection occurs after a 13-h dew period, but actual lesions may take 5 days after infection to appear. Torres et al. (2013a) observed lesions on susceptible cultivars after 3 days. Optimum fungal growth is from 24 to 29 °C (75 to 85 °F). Leaf spots may show up 4–5 days after infection. The fungus can survive on infected corms or in soil for at least 3 years.

Management

- ***Cultural*** – Corms should be inspected for lesions and discarded. After harvest, infected leaves should be removed from the field when practical by deep plowing or raking and burning leaves. All infected corms should be destroyed.
- ***Fungicides*** – Consult local recommendation guides for approved fungicides. Sprays are most effective when applied preventively during warm, wet weather when the spores are actively being dispersed. Fungicides known to be effective as preventative contact sprays are chlorothalonil, mancozeb, and benzimidazoles. Magie (1948) reported that mancozeb was superior to other ethylene-bis-dithiocarbamates.
- ***Resistance*** – Many cultivars listed above have moderate resistance, but Picardy is very susceptible. No cultivar is immune but a wide range of reactions are known to occur (Torres et al. 2013a). Growers should combine resistance to augment other strategies for suppressing the disease (Singh et al. 2006)

2.3 Fusarium Yellows and Fusarium Corm Rot (*Fusarium oxysporum* f. sp. *gladioli*)

Geographic occurrence and impact. Fusarium corm rot is the most common fungal disease on gladiolus and can be one of the most destructive (Nelson et al. 1981). The disease occurs wherever gladiolus is grown and can be lethal on certain cultivars. Characteristic symptoms of corm rot were first reported in 1912, but it took another 16 years before the disease and the pathogen were formerly described (Massey 1928). The pathogen is the fungus *Fusarium oxysporum* f. sp. *gladioli*, which inhabits many soils as different biotypes. As a result, the disease occurs wherever gladiolus is grown, in part, due to resident populations of the pathogen and due to latent corm infections that are frequently present (Nelson et al. 1981). About 30% annual losses have been estimated in Germany (Bruhn 1955), whereas 60–80% annual losses were noted in India (Protsenko 1958 as cited by Chandel and Deepika 2010).

Symptoms/signs. Due to the wide diversity in the pathogen populations, symptoms can be diverse and may include stunting and chlorosis (Fig. 2) and, in severe cases, wilt and death (Fig. 3). Along with the rotting of corms (Fig. 4), the disease can cause vascular discoloration. The corm rot is usually a dry corky rot that stays localized until the entire corm becomes infected.

Biology and epidemiology. The most common means for long-distance dissemination is via latent infections on the corms and cormels. However, once the pathogen is established in the soil, the fungus survives well as persistent chlamydospores and as mycelium on plant debris. The fungus is very adept at surviving in the absence of gladiolus for many years. The fungus can also endophytically colonize the roots of non-symptomatic plants. New infections result from hyphae that emerge from soil inoculum consisting primarily of chlamydospores or mycelium. The fungus invades roots intracellularly causing root rot as it advances toward the corm. The corm may



Fig. 2 Fusarium corm rot of *Gladiolus* showing yellowing and stunting (Wade Elmer © 2017. All Rights Reserved.)



Fig. 3 Fusarium corm rot of *Gladiolus* showing wilt and death (Wade Elmer © 2017. All Rights Reserved.)



Fig. 4 Fusarium corm rot of *Gladiolus* (Wade Elmer © 2017. All Rights Reserved.)

be able to compartmentalize invasion, or the infection continues becoming systemic in the vascular tissues. It is not clear whether the resistance of the host or virulence of the pathogenic biotype of *F. oxysporum* f. sp. *gladioli* governs whether vascular invasion occurs. Disease incidence is a linear function of soil inoculum and soil temperatures above 25 °C (77 °F) (Chandel 2004; Sharma and Tripathi 2008).

Management

- ***Cultural*** – While other cultural strategies are limited in their efficacy, sanitation should be highlighted as the most significant approach. Hot water treatment of

corms and cormels (30 min at 57 °C/135 °F) followed by biological and inorganic amendments at planting can be effective (Sharma and Tripathi 2008), but temperature baths should be accurately monitored to provide the best disinfestation and to prevent heat damage (Chandel 2004; Magie 1971). Gladiolus requires a well-drained soil with a pH of about 6.5 since acidity promotes Fusarium corm rot. Soil tests should routinely be performed and soil limed appropriately to achieve a soil pH of around 6.5–7.0. Although numerous papers on other plant have shown that *Fusarium* suppression occurs with nitrate fertilization (Elmer 2012; Woltz 1958), excessive nitrogen can be deleterious and result in more disease (Ashour and Gamal 1966). Fertilization with nitrate-N should be used instead of ammonium-N, which lowers soil pH and tends to promote Fusarium wilt in other crops like tomato (Woltz and Jones 1973). McClellan and Stuart (1947) found organic N promoted disease compared to inorganic N. Addition of increasing amounts of calcium superphosphate decreased corm rot (Ashour and Gamal 1966). A survey of 18 fields in Pakistan found higher levels of disease were associated with low soil potassium, manganese, and zinc indicating the importance of micronutrient availability (Riaz et al. 2009).

- ***Fungicides*** – Ram et al. (2004) found that soaking corms in acidified solutions of benomyl for 60 min gave the best protection against corm rot. However, others have found that benzimidazoles were not effective probably due to tolerant strains being transported on the corms (Magie 1974). Mishra et al. (2000) found exposing corms to combinations of *Trichoderma virens* and carboxin provides superior results in suppressing corm rot. Ramos-Garcia et al. (2009) reported that Biorend® (a biodegradable polymer) applied at 1.5% accelerated corm emergence, the number of flowers, and the vase life. Preplant corm treatments have received much attention as they reduce the cost, labor, and environmental concern compared with soil drenches. Elmer (2006) demonstrated that triflumizole and fludioxonil gave season-long suppression when applied as a corm soak. Many fungicides applied singularly or combined with other chemicals and/or biological products have often provided more suppression than when applied individually.
- ***Biological control*** – Several researchers have found biological products to be effective (Mishra et al. 2000; Mohamed and Gomaa 2000; Riaz et al. 2010), while others found no control (Elmer 2006). However, given the differences of cultivars, inoculum, and field versus greenhouse, the data are too fractionated to make consistent claims for most products. Nevertheless, a consensus has emerged demonstrating that *Trichoderma* spp. show greater efficacy than any other biocontrol product. Other reports have shown disease reduction by rhizobacteria and nonpathogenic strains of *Fusarium*. Talc-based formulations that combined two plant growth-promoting rhizobacterial strains (*Bacillus atrophaeus* and *Burkholderia cepacia*) reduced vascular wilt by 48.6% and corm rot incidences by 46.1%, respectively, when compared to the non-treated control (Shanmugam et al. 2011). These treatments were comparable to the fungicide carbendazim that provided a 51.5% reduction in vascular wilt and a 47.1% reduction in corm rot incidence. An increase in the number of spikes (58.3%) and corms (27.4%) were

also noted with this treatment when compared to control. Magie (1980) conducted field studies where corms were treated with certain isolates of *F. subglutinans* Snyd. and Hans and *F. solani* and found disease protection was equal to that obtained with benomyl as a corm dip treatment. Biologicals should be used preventatively.

- ***Resistance*** – The management of Fusarium corm rot has been difficult. Although partial resistance has been identified in some lines (Straathof et al. 1998), the wide diversity of biotypes of the pathogen and the lack of persistent screening for resistance have hindered efforts to identify highly resistant cultivars. Growers are advised to check with current information regarding resistance/tolerance of cultivars in their regions.

2.4 Gladiolus Rust (*Uromyces transversalis* Thum)

Geographic occurrence and impact. Gladiolus rust is a disease of quarantine significance in the USA since it was first detected in Florida in 2006 (Schubert et al. 2007). The disease apparently evolved along with the host in Southern Africa. The fungus spread to Southern Europe in the 1960s and reached England in 1996. Since then, major eradication efforts have been initiated to slow the spread of gladiolus rust in the USA. Reports of gladiolus rust have been made from California (Blomquist et al. 2007), South and Central America (Rodríguez-Alvarado et al. 2006; Valencia-Botín et al. 2013), and Australia (Beilharz et al. 2001).

Symptoms/signs. Symptoms are very distinct. The orange uredinia form transverse lines across gladiolus foliage. Under severe inoculum pressure, lesions can be observed on flower spikes (Fig. 5).

Biology and epidemiology. *U. transversalis* is an autoecious rust that has no alternate host. It produces only two spore types, urediniospores and teliospores. It infects *Gladiolus* spp. and *Anomatheca*, *Crocosmia*, *Melasphaerula*, *Tritonia*, and *Watsonia* spp. The rust spores are airborne (urediniospores) and infect and cause

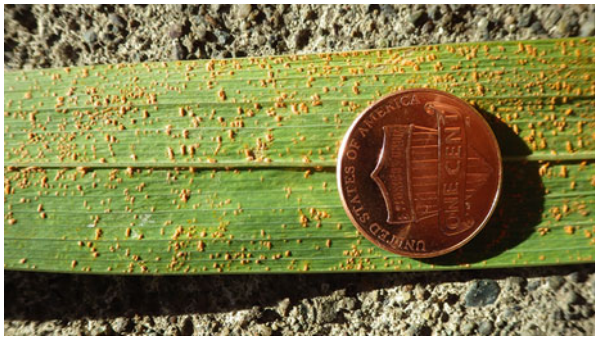


Fig. 5 *Gladiolus* rust (Tom Gulya. USDA.)

profuse sporulation on the leaf surface. Severely infected plants cannot photosynthesize sufficiently. They remain stunted and will not produce flower spikes. The fungus overwinters as teliospores on the foliage.

Management

- ***Sanitation*** – Currently, gladiolus rust is a quarantinable pathogen in the USA. Strict rust management guidelines and fallow host-free periods need to follow the appearance of the disease.
- ***Fungicides*** – In Mexico, where the disease was more common, fungicide trials found that the triazole fungicides, cyproconazole, difenoconazole, epoxiconazole, myclobutanil, propiconazole, and tebuconazole gave excellent control.

2.5 Stromatinia Rot [*Stromatinia gladioli* (Drayt.) Whet.]

Geographic occurrence and impact. The disease was recognized in 1883 in England and known to occur wherever gladiolus is grown. The fungus was first described as *Sclerotinia gladioli* (Hawker et al. 1944) but was changed to *Stromatinia gladioli*. The disease was reported along the USA Gulf Coast (Magie 1954; Drayton 1934).

Symptoms/signs. Symptoms appear as a dry rot of the corm (Fig. 6). Individual lesions may appear as superficial small, round, reddish brown spots that can have brown to black centers. Lesions more frequently appear at the point of the husk attachment. The actual husk can appear dark colored, brittle, and shredded. One diagnostic characteristic of Stromatinia rot is the presence of black sclerotia in rotted cortical tissue of the roots (McRitchie and Leahy 1988).

Biology and epidemiology. The fungus overwinters in the soil in the form of sclerotia, which can last in the absence of a host for 10 years (Nelson 1948). The disease is more severe when cool, wet conditions prevail. Young corms (1–2 years)



Fig. 6 Stromatinia rot of *Gladiolus* (R.D. Milholland © 2017. All Rights Reserved.)

tend to be more susceptible than older ones. Wounds are not required for infection (Hawker et al. 1944). The disease spreads faster when plants are planted in close proximity. Inoculation of the top of an old corm is more effective than that of the side or base. The percentage of diseased new corms produced from infected parent corms varies from 0% to 100% according to soil conditions. Disease is favored by wet soil.

Management

- ***Cultural practices*** – Crop rotation should be practiced at 3–4-year intervals to reduce soil densities of the sclerotia. Any volunteer gladiolus plants should be rogued as they can serve as sources of inoculum. Since cool, wet soil favors the disease, delaying the time of planting and improving drainage can lessen the loss due to Stromatinia rot.
  Removal of "husks" (leaf bases) increases the susceptibility of corms planted in contaminated soil, but the removal of husks is not an effective control. Dehusking did not increase the number of diseased young corms in infected stock planted in new soil. No variety was strikingly more resistant than the rest.
- ***Fungicides*** – Corm and soil treatment with fungicides should be done where there is a history of the disease. Good control was obtained when corms of a diseased stock were treated with mercuric chloride, mercurous chloride (calomel), or the proprietary mercury fungicide Aretan. Calomel gave the most consistent results. Various other treatments were tested in small-scale trials with some success. Red copper oxide, formalin, and the proprietary mercury compound, Ceresan, were harmful. Sterilization of contaminated soil with formalin, mercuric chloride, Aretan, or Uspulun gave good but not complete control. Formalin was the least effective.

3 Bacterial and Phytoplasma Diseases

3.1 Bacterial Scab *Burkholderia gladioli* pv. *gladioli* (Formerly *Pseudomonas gladioli* pv. *gladioli*)

Geographic occurrence and impact. Bacteria scab is not a major problem for gladiolus growers. Unlike the fungal corm rot diseases, bacterial scab does not kill the affected plants or affect the flower spikes.

Symptoms/signs. The lesions are very dark and appear on the surface of the corm. Scab lesions are sunken with a concentric border. Most lesions develop a shiny black layer over the surface of the lesion. Bacterial scab can be differentiated from Stromatinia by where the lesions tend to appear. Bacterial scab lesions appear more frequently at the base of corm, whereas Stromatinia lesions appear more commonly on the top of the corm (Forsberg 1965).

Biology and epidemiology. The bacteria are ubiquitous and can be introduced on previously infected corms or through wounds made from handling or insect feeding. The bacterium also infects a number of other horticultural crops including lisianthus.

Management. Inspect corms for lesions and discard infected corms. Avoid planting in areas where the disease has appeared. Efforts to suppress bulb mites will also reduce infection sites.

3.2 Aster Yellows ("*Candidatus* Phytoplasma asteris")

Geographic occurrence and impact. Aster yellows has been reported in *Gladiolus* in the USA, and it infects a wide range of plant species (Treeful and Ash 2000).

Symptoms/signs. *Gladiolu*s plants mature early, have an arrested root development, and have small or few corms (Fig. 7) during the season when they are first infected. The next year, multiple shoots that are thin and weak develop from the



Fig. 7 Phytoplasma-infected *Gladiolus* cultivars showing (**a**, **b**) stunting and spike twisting; (**c**, **d**) virescence; (**e**, **f**) malformation and reduction of floret size; and (**h**) small corms with poor root systems as compared to (**g**, **i**) healthy plants collected from the gardens of NBRI, Lucknow, India (Raj et al. 2011) – http://link.springer.com/article/10.1007/s10327-010-0259-9

corm giving the appearance of their nickname "grassy top" (Koike et al. 2007) (Fig. 7). The flower spike grows in a spiral pattern (Fig. 7).

Biology and epidemiology. This disease is caused by phytoplasmas formerly known as mycoplasma-like organisms and not by a virus. Phytoplasmas are microscopic organisms similar in size and composition to bacteria (Treeful and Ash 2000). Transmission is by *Macrosteles sexnotatus* leafhoppers (Koike et al. 2007). An infected leafhopper will remain infected throughout its life and will inject phytoplasma into the phloem cells of a plant when feeding (Missouri Botanical Garden). Plants will exhibit symptoms in 10–40 days. Aster yellows has been reported to occur in many floral crops and in a number of weed species (*Convolvulus*, *Capsella*, and *Cirsium*) (Vicchi and Bellardi 1988). Aster yellows spreads more readily in cool, wet summers because phytoplasmas and leafhoppers do not thrive as well in dry, hot weather (Missouri Botanical Garden). Massive spread of aster yellows is rare (Stein 1995).

Management. Infected plants should be destroyed (Koike et al. 2007). Weeds, particularly dandelions and plantains, should be controlled in the area where *Gladiolus* is being grown. Monitor plants for leafhoppers. Fine mesh fabrics and strips of aluminum foil between rows of plants can be used to keep leafhoppers away from the plants (Missouri Botanical Garden).

4 Viral Diseases

Twenty viruses have been reported to infect *Gladiolus* (Stein 1995; Asjes 1997). The predominant viruses that infect *Gladiolus* are *Bean yellow mosaic virus* (BYMV) and *Cucumber mosaic virus* (CMV). Other viruses that have been reported in *Gladiolus* are listed in Table 1. A number of companies offer testing services and/or ELISA kits to detect various gladiolus viruses. Nine *Gladiolus* viruses can be detected immunologically by Agdia, Inc., Elkhart, IN, USA, in their "Gladiolus Screen" (www.agdia.com/testing-services/Gladiolus.cfm). Bioreba AG, Reinach, Switzerland, can test for the presence of six gladiolus viruses by ELISA (http://www.bioreba.ch/files/Tecnical_Info/TS_Broschuere_2011_e_CHF.pdf). Neogen Europe, Ltd., Auchincruive, Scotland, UK, manufactures ELISA kits which can detect a number of gladiolus viruses.

The viruses in *Gladiolus* differ for each country, but two viruses, BYMV and CMV, are by far the most prevalent viruses found worldwide in *Gladiolus*.

4.1 *Bean yellow mosaic virus*

Geographic occurrence and impact. Almost all *Gladiolus* plants are infected with *Bean yellow mosaic virus* (BYMV) as reported for plants grown in the USA (Zettler and Abo El-nil 1977), Ontario Canada (Berkeley 1953), the Netherlands

Table 1 Viruses reported in *Gladiolus* other than BYMV and CMV

Virus	Occurrence in *Gladiolus*	Transmission	Symptoms	References
Arabis mosaic virus	Italy, India	*Xiphinema* (nematode)	ND	Bellardi et al. 1986; Katoch et al. 2003
Bearded iris mosaic virus (=*Iris severe mosaic virus*)	USA, Netherlands	Aphids	ND	Brierley and Smith 1948; Brunt et al. 1988
Broad bean wilt virus	Korea	Aphids	Symptomless	Park et al. 1998
Clover yellow vein virus	Korea	Aphids	ND	Park et al. 2002
Cowpea mosaic virus	USA	Beetles	ND	Brierley and Smith 1962
Cycas necrotic stunt virus	Japan	Nematodes and seed	ND	Hanada et al. 2006
Impatiens necrotic spot virus	France, Iran, Portugal	Thrips	ND	Marchoux et al. 1992; Ghotbi et al. 2005; Louro 1996
Narcissus latent virus	Netherlands	Aphids	ND	Stein 1995; Asjes 1997
Ornithogalum mosaic virus	India, Netherlands	Aphids	Foliar mosaic, chlorotic spots, floral deformation	Kaur et al. 2011
Plum pox virus	Germany	Aphids	ND	Kroll 1978
Soybean mosaic virus	USA	Aphids	ND	Stein 1995
Strawberry latent ringspot virus	Italy	*Xiphinema* (nematode)	ND	Bellardi et al. 1984
Tobacco mosaic virus	Japan, Netherlands, Canada	Mechanical	ND	Berkeley 1951; Fukumoto et al. 1982
Tobacco necrosis virus	Russia, Lithuania	*Olpidium brassicae* (fungus)	ND	Stein 1995; Navalinskiene and Samiutiene 2010
Tobacco rattle virus	Netherlands, Israel, Egypt, Poland, India, Korea, Japan, Czech Republic, Lithuania	Tools used during planting, low rate of transmission in *Gladiolus* seeds, nematodes of the family Trichodoridae	Notched leaf blades, severely distorted plant, chlorotic, brown or dead stripes and spots on leaves, crumpled tissue between veins, stem and	Navalinskiene and Samuitiene 2001; Univ. Illinois Extension 1983; Stein 1995; Asjes 1997; Yamaji et al. 1998; Park et al. 2002;

(*continued*)

Table 1 (continued)

Virus	Occurrence in *Gladiolus*	Transmission	Symptoms	References
			buds may stop growing	Katoch et al. 2004; Duraisamy and Pokorný 2009
Tobacco ringspot virus	Japan, India, Iran, USA, Canada, Australia, Lithuania	Cutting tools (low rate of cross contamination), *Xiphinema americanum* (nematode), thrips, mites, grasshoppers, tobacco flea, and beetles	Typically symptomless in gladiolus, sometimes necrotic or chlorotic ringspot patterns on leaves	Brierley 1952; Brierley and Smith 1960; Berkeley 1953; Randles and Francki 1965; Fukumoto et al. 1982; Kaniran and Izadpanah 1982; Univ. Illinois Extension 1983; Katoch et al. 2003; Navalinskiene and Samuitiene 2010
Tobacco streak virus	Italy	Pollen, thrips	Stunted flowers, necrotic spots on leaves	Bellardi et al. 1987
Tomato black ring virus	Poland, Italy (not reported outside of Europe)	*Longidorus* (nematode)	Symptomless	Kaminska 1978; Bellardi and Pisi 1985a, b; Stein 1995
Tomato ringspot nepovirus	USA, Iran	*Xiphinema americana* (nematode), low rate by *Gladiolus* seeds	Stunted growth, abnormally short flower spikes with only 4–5 florets. Leaves smaller, stiffer, and more erect than on healthy plants	Bozarth and Corbett 1958; Univ. Illinois Extension 1983; Stein 1995; Ghotbi et al. 2005
Tomato spotted wilt tospovirus	USA, France, S. Australia, Portugal	Thrips	Chlorosis with degeneration	Lee et al. 1979; Stein 1995; Louro 1996
Tomato aspermy virus	India	ND	Severe chlorosis along midrib	Raj et al. 2011

(Brunt 1970; Nagel et al. 1983), Israel (Stein 1995), Argentina (Arneodo et al. 2005), Iran (Kaniran and Izadpanah 1982; Dorrigiv et al. 2013), Italy (Bellardi and Pisi 1985a), Japan (Wada et al. 2000), Korea (Park et al. 2002), Russia (Gnutova et al. 1989), the Czech Republic (Duraisamy and Pokorný 2009),

Belarus (Voinilo and Burgansky 1999), Lithuania (Navalinskiene and Samuitiene 2001), New Zealand (Fry 1953), and India (Katoch et al. 2002). *Bean yellow mosaic virus* is also known as the *Pea mosaic virus*.

Symptoms/signs. BYMV-infected *Gladiolus* plants are typically symptomless or have mild symptoms. It is difficult to attribute viral symptoms to a specific virus because *Gladiolus* is generally infected with BYMV and a second virus (Stein 1995). The symptoms of BYMV have been described as faint, inconspicuous pencil-stripe break patterns that are lighter in color than the normal flower although it may be conspicuous on specific cultivars (Univ. Illinois Extension 1983). The leaves and flower stems may have light or dark green mottling seen in the early summer (Univ. Illinois Extension 1983). Symptoms will be affected by environmental conditions (Asjes 1997). Dry, high-temperature conditions will mask the symptoms (Univ. of Illinois Extension 1983). Katoch et al. (2002) examined 32 cultivars of *Gladiolus* and reported a BYMV incidence of 0–100% based on visual symptoms, but almost 100% were found to have BYMV as indicated by ELISA.

Biology and epidemiology. BYMV is of the genus *Potyvirus* and belongs to the Potyviridae family, which is the largest and most economically important group of viruses as it infects many species of vegetables, forage, fruit, ornamental, and field crops (Jordan and Hammond 1991). BYMV infects many legumes, and the virus overwinters in perennial legumes such as alfalfa, clovers, or vetch. Hampton et al. (2005) verified the non-circulative (nonpersistent) transmission of BYMV from clover, *Trifolium repens*, to an adjacent field of snap beans, *Phaseolus vulgaris*, by four aphid species (*Myzus persicae*, *Acyrthosiphon pisum*, *Aphis fabae*, and *Nearctaphis bakeri*). Over 20 species of aphids serve as vectors for BYMV transmission. Transmission of BYMV through bean seeds of legumes has been reported (Latham et al. 2000).

BYMV particles are flexuous and filamentous (750 nm in length and 15 nm in diameter) (Guyatt et al. 1996). They consist of single-stranded, positive sense RNA. Various strains of BYMV have been identified serologically using both polyclonal and monoclonal antisera (Nagel et al. 1983; Jordan and Hammond 1991). Virus is readily detectable in leaves and flowers by ELISA (Stein 1995). The low level of BYMV in corms requires that the corm be cut, and then virus can be detected by ELISA in the callus tissue that forms at the wounded areas (Stein 1995). Immunological detection of BYMV in corms of *Gladiolus* has been possible using a tissue blotting technique (Lin et al. 1990). Polymerase chain reaction (PCR) amplification of BYMV RNA has also been used to detect BYMV in corms (Rosner et al. 1992). Another method to increase the sensitivity of detection (10^5 higher than when using ELISA) was achieved by hybridizing PCR products with a radioactive ^{32}P-labeled viral riboprobe (Vunsh et al. 1991).

Management. Because aphid species spread BYMV, growing *Gladiolus* plants under fine mesh nets was found to greatly reduce virus infection (Stein 1995). BYMV as well as CMV, but not *Tobacco ringspot virus*, were found to be

transmitted on tools used when harvesting flowers and corms of *Gladiolus* (Brierley 1962). Tools should be disinfested with 70% alcohol between cuts to prevent cross contamination (Univ. Illinois Extension 1983). Logan and Zettler (1985) developed an efficient tissue culture system that had the potential to produce 50,000 gladiolus plants from a single shoot tip during a 30-week period. *Gladiolus* plants free from BYMV can be produced using meristem culture (Stein 1995). Propagating with the apical meristem of a corm grown under sterile conditions in vitro can eliminate 60–100% of the virus. Removal of three viruses from *Gladiolus* was achieved by performing meristem culture twice, adding ribavirin (5–20 ppm) to the culture medium, and treating with high (38–40 °C) temperature (Li et al. 2003). Regeneration of plants from callus was reported to be more effective than culturing shoot tips from corms for virus elimination (Park et al. 2002). Callus induced from cormels infected with BYMV and CMV was cultured on a medium containing ribavirin (virazole, 1-β-D-ribofuranosyl-a, 2, 4-triazole-3-carboxamide), and after 6–8 weeks, plants were regenerated and certified as virus-free (Singh and Dubey 2007). Virus-free plants must be grown in insect-proof conditions if the *Gladiolus* plants are to remain free of virus or else 80–100% of the crop will become infected with BYMV within one growing season (Stein 1995; Asjes 1997). It is recommended (Univ. Illinois Extension 1983; Moran 1996) that virus-free plants be grown at least 1 km from virus-infected *Gladiolus* plants and legumes, that effective insecticides be used to control aphids on the crop plants and on corms during their storage, and that plants showing viral symptoms be rogued and destroyed by burning or composting. Weeds in the vicinity of *Gladiolus* plants should be eradicated as they may serve as reservoirs of viruses and insects that can be vectors for viruses (Univ. Illinois Extension 1983). Aluminum strips placed between rows or *Gladiolus* plants can be used to repel aphids and will also decrease the number of weeds present.

4.2 *Cucumber mosaic virus*

Geographic occurrence and impact. *Cucumber mosaic virus* (CMV) is in the genus *Cucumovirus* and is a member of the family Bromoviridae. CMV consists of three spherical particles that are each 28 nm in diameter. Its genome consists of three single-stranded messenger RNA molecules in the sense orientation. There are two subgroups, I and II, of CMV that are distinguished either by their serological response to antibodies or by their nucleotide sequences (Chen 2003; Zitter and Murphy 2009). The CMV that infects *Gladiolus* plants belongs to subgroup I and has the highest nucleotide sequence homology based upon its coat protein to the CMV strain Fny (Chen 2003; Dubey et al. 2010). CMV infects 1,200 plant species that include economically important vegetable crops such as cucumbers, squash, and melons and most floral crops. CMV occurs throughout the world and has been reported in *Gladiolus* in the USA (Brierley 1952; McWhorter 1957); Iran (Dorrigiv et al. 2013); Ontario, Canada (Berkeley 1953); Tasmania (Wade 1984); Israel (Stein 1995); India (Dubey et al. 2010); the Czech Republic (Duraisamy and Pokorný 2009); and Lithuania (Navalinskiene and Samuitiene 2001).

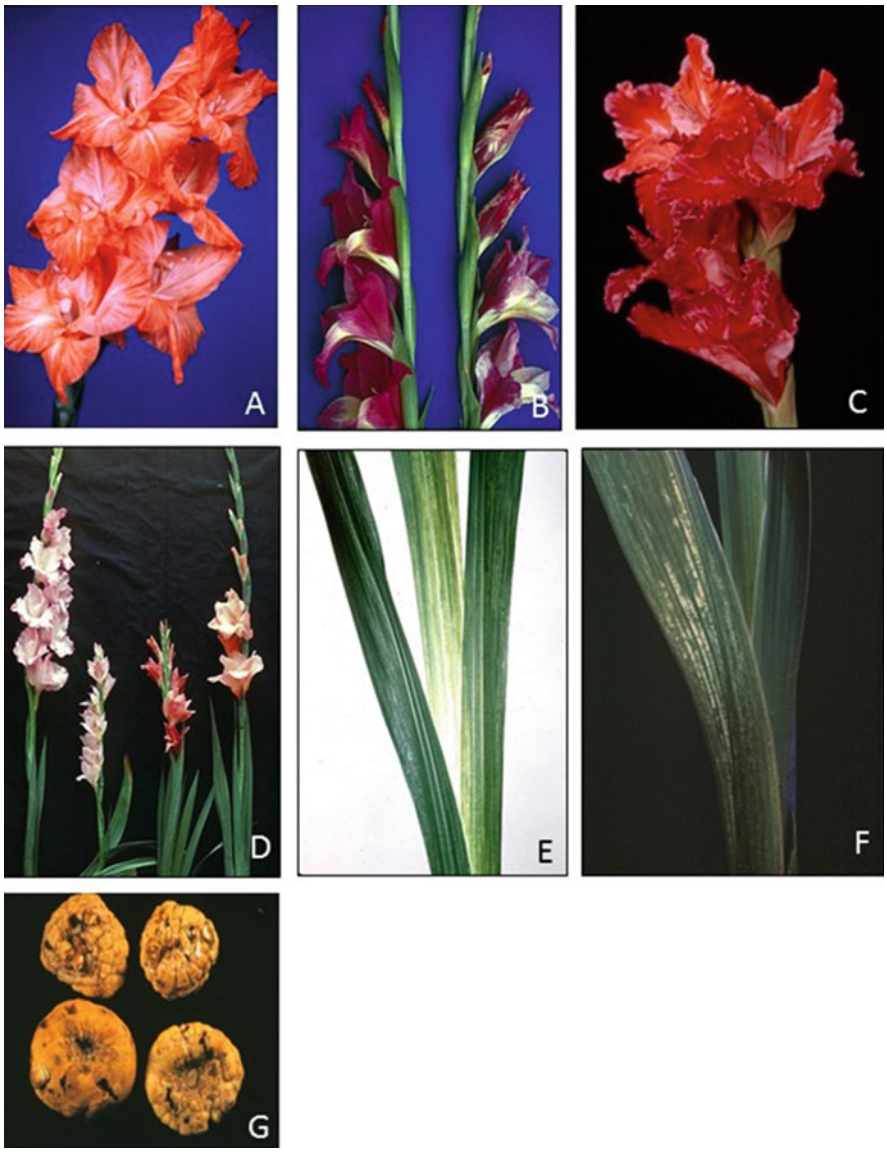


Fig. 8 Symptoms of viral infection in *Gladiolus* are (**a**–**c**) color break on flower petals. In **b** the *left* spike is a healthy spike, and the *right* spike shows color break of petals indicative of virus infection. (**d**) The two inner plants are stunted in their growth as compared to the two outer, healthy plants. (**e**) Viral symptoms on leaves (streaking or (**f**) blotching. (**g**) Corms infected with CMV are deformed (Kathy Kamo. USDA.)

Biology and epidemiology. The worldwide occurrence of CMV in *Gladiolus* can be attributed to the international trade of corms and to the virus' wide host range. When only 3% of the corms are initially infected with CMV, after one growing

season, 60% of the plants may become infected (Stein 1995). CMV transmission occurs by many aphid species in a non-circulative (nonpersistent) manner. Most commercial cultivars of *Gladiolus* are susceptible to CMV. The cultivars Peter Pears, Spic and Span, and White Friendship are readily infected by CMV whereas Trader Horn and Eurovision are more resistant (Aly et al. 1986).

Symptoms/signs. Generally, *Gladiolus* plants with CMV often appear symptomless until flowering when there may be spotting on petals; obvious color break that is more readily visible on purple, lilac, pink, and red flowers than on white or yellow flowers (Fig. 8a–c); and a reduced size of the floret accompanied by a change in the color of the petals, thickened petals, twisted and deformed florets, and flowers that will not fully open. Flowers may have crinkled petals, open slowly, imperfectly, and fade early (Univ. Illinois Extension 1983). Flower bracts can be yellow and wither early in the season. Other symptoms can be deformity of the whole plant. Stunting of infected plants often occurs (Fig. 8d) with severely affected leaves that have gray or yellow-green streaking or spots that are either gray, yellow, brown, or reddish (Fig. 8e, f). Young leaves may have a mosaic or chlorophyll break. Some cultivars have pitting and discoloration of the corms or corms that are deformed and have a warty appearance (Univ. of Illinois Extension 1983; Stein 1995).

Management. If possible, it is recommended that CMV-tested, certified corms and resistant cultivars be planted. A number of diagnostic companies manufacture rapid detection ELISA kits for CMV. The use of fine mesh nets coated with mineral oil spray or by directly spraying mineral oil on the plants in the absence of nets has been found to help with controlling spread of CMV (Aly et al. 1986). Controlling the aphid population and rogueing infected plants during flowering when symptoms are most obvious on the flowers also help in the field to prevent a large outbreak of CMV. It is advised that *Gladiolus* should not be grown near cucumbers, melons, and tomatoes because of the virus transmission that can occur between CMV-infected crops (Univ. Illinois Extension 1983). The strategies to manage CMV are the same as for BYMV (rogueing, aphid control, eliminate weeds).

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Diseases of Tulip

43

Robert J. McGovern and Wade H. Elmer

Abstract

Tulip is the most important ornamental geophyte worldwide, grown for bulbs, cut flowers, potted plants, and landscape use. Major increases in consumer demand in the first half of the twentieth century were associated with the identification of many new disease problems. Tulip is susceptible to a number of diseases caused by fungi, bacteria, viruses, and nematodes including *Botrytis tulipae*, *Fusarium oxysporum* f. sp. *tulipae*, *Pectobacterium carotovorum*, *Tulip breaking virus*, and *Ditylenchus dipsaci* that can significantly reduce flower and bulb production. Since the plant is propagated vegetatively, this factor can facilitate the spread of disease if pathogen-free propagative material is not used and integrated disease management is not followed.

Keywords

Botrytis tulipae • *Fusarium oxysporum* f. sp. *tulipae* • *Tobacco rattle virus* • *Tulip breaking virus* • *Ditylenchus dipsaci* • *Paratrichodorus* spp. • *Trichodorus* spp. • Integrated disease management

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R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5_49

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1 Introduction

Tulip (*Tulipa gesneriana* and *Tulipa* hybrids), a Eurasian and North African perennial plant, has been associated with the Netherlands since the late sixteenth century, is synonymous with Spring, and is the most important ornamental geophyte in the world. Tulip is grown for bulbs, cut flowers, potted plants, and landscape use. The Netherlands dominates tulip production on land accounting for 88% of the area devoted to the crop worldwide; Japan, France, Poland, Germany, and New Zealand are distant competitors (Buschman 2005). The country produced an estimated 4.32 billion tulip bulbs (Buschman 2005). The total sales value of tulip cut flowers in the USA in 2012 was $65.3 million (Anon. 2014).

Tulip is susceptible to a number of diseases caused by fungi, bacteria, viruses, and nematodes including *Botrytis tulipae*, *Fusarium oxysporum* f. sp. *tulipae*, *Pectobacterium carotovorum*, *Tulip breaking virus*, *Tobacco rattle virus*, *Ditylenchus dipsaci*, *Paratrichodorus* spp., and *Trichodorus* spp. that can significantly reduce flower and bulb production. Since the plant is propagated vegetatively, this factor can facilitate the spread of disease if pathogen-free propagative material is not used and integrated disease management is not followed.

2 Fungal and Fungus-Like Diseases

2.1 Tulip Fire [*Botrytis tulipae* (Lib.)]

Geographic occurrence and impact. Tulip fire is a very common and serious disease wherever tulips are grown. The pathogen can cause extensive losses in both flower and bulb production and landscape plantings.

Symptoms/signs. All parts of the tulip may be affected. As its name implies, the disease can move very rapidly through a tulip crop given a conducive environment. Yellow to brown round lesions may be seen on bulbs when the outer scales are removed. Small, black long-lasting resistive structures (sclerotia) of *B. tulipae* may form on the outer scales. Initial symptoms on leaves are small, yellowish, elongated spots that become grayish-brown blotches, which coalesce as they enlarge ("fire"); sclerotia may form in necrotic tissue (Fig. 1). Leaves may fail to emerge. Similar but more depressed lesions form on stalks and may cause stalk breakage. Shoot distortion and stunting may occur. The fungus causes brown lesions on light-colored flowers and whitish lesions on darker-colored flowers (Fig. 2); complete blighting of flowers is possible. Plant death can occur depending on the environment and tulip genotype. In general, *T. fosteriana* is more susceptible to *B. tulipae* than *T. gesneriana* (Reyes et al. 2005).



Fig. 1 Tulip fire on leaves caused by *Botrytis tulipae*; note the presence of black sclerotia (The Pennsylvania State University Department of Plant Pathology and Environmental Microbiology © 2017. All Rights Reserved.)



Fig. 2 Tulip fire on flowers caused by *Botrytis tulipae* (R. Wick © 2017. All Rights Reserved.)

Biology and epidemiology. *Botrytis tulipae* survives in infected bulbs and sclerotia in the soil and in association with plant tissue. In spring, sclerotia germinate as hyphae or give rise to conidiophores and conidia, which disseminate the fungus via water splash and air currents to foliar tissue. Germination of sclerotia is greatest at 5 °C (41 °F) (Coley-Smith and Javed 1972). Infection by the fungus is enhanced by cool soil temperatures. Doornik and Bergman (1971) found that a soil temperature of 5 °C (41 °F) and the resultant slower flowering led to higher shoot infection by *B. tulipae* than a soil temperature of 15 °C/59 °F, which resulted in faster flowering. In addition, more new bulb infections occurred at a soil temperature of 9 °C (48 °F) than 18 °C (64 °F) (Doornik and Bergman 1973). High humidity and rainfall and moderate temperatures favor the blight phase of tulip fire, and a fuzzy gray mold consisting of conidiophores and conidia may form in diseased tissue. The host range of *B. tulipae* appears to be limited to tulip (Schönbeck 1976).

Management

- ***Cultural practices*** – Use vigorous, blemish-free, and pathogen-free bulbs. Rotate to another production site each year. Researchers at Washington State University are investigating the effectiveness of growing cover crops in rotation with tulip to reduce tulip fire (MacKenzie 2015). Avoid high rates of nitrogen. Use a proper plant spacing and rigorous weed control to increase air circulation and decrease plant wetness. Harvest bulbs during dry weather and process and store them properly to avoid injury.
- ***Sanitation*** – Discard/burn infected bulbs and other diseased plant tissue when plant surfaces are dry.
- ***Soil disinfestation*** – Use a labeled fumigant or, where feasible, steam treatment to reduce the inoculum of *B. tulipae* in the soil (refer to ▶ Chap. 8, "Soil/Media Disinfestation for Management of Florists' Crops Diseases").
- ***Fungicides*** – A large number of fungicides are labeled in the USA for management of *Botrytis* on tulip (Pscheidt and Ocamb 2016). (Also refer to ▶ Chap. 7, "Fungicides and Biocontrols for Management of Florists' Crops Diseases"). Resistance to benzimidazole and dicarboximide fungicides has been identified in *B. tulipae* (Hsiang and Chastagner 1990). It is important to rotate fungicides with different modes of action to prevent the development of fungicide resistance; refer to the *Fungicide Resistance Action Committee* website (http://www.frac.info/home) for more information.
- ***Resistance*** – Absolute resistance was found in *T. tarda*, but this species cannot be crossed with the various *T. gesneriana*. Partial resistance was found in some cultivars of *T. gesneriana* and *T. kaufmanniana* (Straathof et al. 2002).

2.2 Fusarium Bulb Rot, Fusarium Basal Rot (*Fusarium oxysporum* (Schlecht.) f. sp. *tulipae* Act.)

Geographic occurrence and impact. Fusarium bulb rot was described in the 1950s when the disease was reproduced using strains of *F. oxysporum* (Slootweg 1955). In 1958, Apt (1958) assigned the *formae specialis*, *F. oxysporum* f. sp.

tulipae, to the pathogen. Although the disease can occur wherever tulips are grown, most damage is observed during bulb production and storage (Gould and Miller 1970; Schenk and Bergman 1969). There was a striking increase in the incidence of disease in the 1960s following consumer demands for increased production (Schenk and Bergman 1969). Dutch growers opted for shorter rotations that favored a buildup of inoculum. In addition, the advent of mechanization, which led to increased wounding during harvest, combined with poor grading to remove damaged bulbs favored a widespread increase in Fusarium bulb rot.

Symptoms/signs. The disease was first coined by Slootweg (1955) as "zuur" meaning sour disease due to the rank smell that infected bulbs produced. Infected bulbs tend to be whitish and the infection is clearly demarcated as dry, firm, sunken areas (Fig. 3). Depending on the relative humidity, the mycelium can be observed under the outer husk at the base of the bulb (Fig. 4) (Apt 1958; Bergman 1965). Frequently, a tan gummosis exudes, hardens, and can glue scales and occasionally entire bulbs together in storage (Liou et al. 2006). In the field, infected bulbs sprout poorly and their leaves usually turn red, wilt, and eventually die (Bergman 1965). Occasionally, atypical symptoms are associated with Fusarium bulb rot; Schenk and Bergman (1969) reported mycelium only between the parenchymatous tissues in tulip bulbs that had been forced in the greenhouse after a precooling at 5 °C/41 °F. Although variation can appear in strains of *F. oxysporum* f. sp. *tulipae*, molecular assays found close relatedness between all isolates from tulip and from lily suggesting a common progenitor (Baayen et al. 2000).

Biology and epidemiology. Latent infections by *Fusarium oxysporum* f. sp. *tulipae* commonly occur in commercial tulip production and can affect the resulting health of the daughter bulbs harvested in the following year (Bergman and Bakker-Van Der Voort 1979). This mode is also the major means for disseminating the pathogen



Fig. 3 Tulip bulbs infected with *Fusarium oxysporum* f. sp. *tulipae* (Oregon State University Plant Disease Clinic © 2017. All Rights Reserved.)



Fig. 4 Mycelium of *Fusarium oxysporum* f. sp. *tulipae* colonizing between the leaf scales of tulip bulbs (Oregon State University Plant Disease Clinic © 2017. All Rights Reserved.)

abroad (Schenk 1972). The pathogen can remain viable in the bulb during dry storage for several months but, once established in the soil, can persist for longer periods (Price 1974). Under greenhouse conditions, the pathogen can penetrate through the roots into the tissue of the planted bulbs and colonize the basal plate of the young bulb in the field. Infection more frequently occurs on the fleshy outer scale of the new bulb. This was noted to occur during the weeks before the bulb skin turns brown presumably due to the warmer soil temperatures that favor disease (Bergman and Noordermeer-Luyk 1973). Another problem comes from the fact that the *Fusarium* infection tends to produce ethylene, a plant hormone (Jarecka et al. 2008). Ethylene can cause flower abortion, stunting, poor rooting, and excessive gummosis. Infection of young growing bulbs by the pathogen occurs mainly during the last few weeks before harvest. Beijersbergen and Lemmers (1972) found that extracts of the bulb skin contained an antifungal compound called tulipalin (-α-methylene butyrolactone). The compound appears to decrease antifungal activity as soon as the tissue dries. Bergman and Beijersbergen (1971) speculated that variations in the compound may influence the wide range of reactions to Fusarium bulb rot among different cultivars at different times. Observations by researchers have concluded that the fungus is not a typical vascular parasite but tends to colonize non-vascular tissue resulting in only bulb rots (Schenk and Bergman 1969; Suazo 2012).

Management

- ***Cultural practices*** – Minimizing damage to the bulb during harvest followed by careful culling of damaged bulbs can significantly reduce disease incidence. The distinct smell that is present with infected bulbs should be employed as a tool to cull damaged bulbs. In addition, avoiding excessive nitrogen fertilizing and making an attempt to not delay harvest since warmer soil temperatures will increase disease incidence is important. A number of reports have shown that

fertilizing with nitrate nitrogen (as opposed to the ammonium form) can reduce *Fusarium* disease of ornamental bulbs (Gullino et al. 2015).

- ***Resistance*** – Considerable progress has been made in screening and breeding for resistance (Eijk et al. 1979; Straathof et al. 1996), which provides the most effective management strategy for *Fusarium* bulb rot of tulip. Van Tuyl and van Creij (2007) discovered almost complete resistance in the *T. gesneriana* assortment, but since seedling selection produced some susceptible plants, plants still need to be rescreened at the clonal level. Growers should inquire whether or not resistance is known to occur in cultivars they seek to purchase.
- ***Fungicides*** – Strategies useful for suppression of *Fusarium* diseases on Narcissus (refer to ▶ Chap. 40, "Diseases of Daffodil (*Narcissus*)") would be appropriate for *Fusarium* bulb rot of tulip (Hanks 1996) However, growers should be aware of fungicide-resistant strains of *F. oxysporum* f. sp. *tulipae* (Duineveld and Beijersbergen 1975).

2.3 Penicillium Bulb Rot, Blue Mold (*Penicillium* spp.; *Penicillium corymbiferum* Westling)

Geographic occurrence and impact. The disease is a storage rot problem and common to most production facilities. Changes in modified atmospheric conditions for the storage of tulips have favored an increase in the disease (Prince et al. 1982). It is reported in the USA and Europe (Dugan et al. 2014). The disease is caused by *Penicillium corymbiferum*. However, the pathogen has recently been subdivided into seven taxa based on pathogenicity (Overy et al. 2005a). From the original "corymbiferum group," the species *P. hirsutum*, *P. radicicola*, *P. tulipae*, and *P. venetum* were found to be the predominant pathogens on tulip *Tulipa gesneriana.*

Symptoms/signs. Bulbs in storage initially develop brown spots. The blue mold develops on the bulb basal root plate, and/or between the scales. Internally, the disease can discolor the young leaf initials. Depending on the severity of the disease, the entire bulb or only a portion of it shows symptoms. When infections are severe, the bulb will frequently dry and become very hard. Sprouted bulbs can have reddish stunted foliage.

Biology and epidemiology. These fungi generally attack bulbs that are stressed or injured and then put into storage under moist conditions. The pathogens in the *P. corymbiferum* group are unique among *Penicillium* species in that most can grow at 0 °C (32 °F) but can proliferate at 5 °C (41 °F) (Overy et al. 2005b).

Management

- ***Cultural practices*** – The pathogen has been reported on other bulb crops including crocus, gladiolus, iris, and onion (Dugan et al. 2014), so field rotations to non-susceptible crops should be practiced to limit inoculum buildup. Growers should dig their bulbs at the proper time and not delay. Care should be exercised in

harvesting bulbs to avoid bruising. Bulbs should be stored with moderate humidity and sufficient air circulation. All damaged bulbs should be destroyed.

- ***Fungicides*** – Fungicide bulb soaks can be effective. Thiabendazolein in water at 13 °C (55 °F)–24 °C (75 °F) for 15–30 min or thiophanate-methyl-based products applied at 27 °C (80 °F)–29 °C (85 °F) have been recommended (Prince et al. 1986, 1988). Bulbs should be treated within 48 h after digging. Growers should be aware that benzimidazole-resistant strains of the pathogen have been reported (Duineveld and Beijersbergen 1975).

2.4 Pythium Root and Bulb Rot (*Pythium* spp. *Pythium ultimum*, *Pythium ultimum* Trow var. *ultimum*, *P. irregulare* Buisman, *P. spinosum* Sawada)

Geographic occurrence and impact. The disease was first reported in England, Denmark, and Holland (Moore and Buddin 1937). Although different species were implicated, the disease appears to be the same (Moore and Buddin 1937). In Japan, the species *P. ultimum* var. *ultimum*, *P. irregulare*, and *P. spinosum* were isolated the most frequently (Ichitani et al. 1991). The disease can be devastating if plants are grown in heavy wet soils.

Symptoms/signs. Pythium root rot causes a browning and collapse of the roots. It can frequently be encountered with *Rhizoctonia* infection (Fig. 5). In addition, symptoms on the aboveground growth can frequently be observed as stunting, dieback of leaf tips, weak or distorted growth, and yellowing (Fig. 6).

Biology and epidemiology. *Pythium* species are ubiquitous soil species and can be devastating when soil conditions are optimal. Cool wet soil favors disease. The pathogen(s) may also be spread on bulbs into new areas.



Fig. 5 Root rot symptoms of *Pythium* and *Rhizoctonia* on tulip (Tom Creswell © 2017. All Rights Reserved.)



Fig. 6 Aboveground symptoms of Pythium root rot showing leaf tip dieback (Tom Creswell © 2017. All Rights Reserved.)

Management

- ***Cultural practices*** – Careful culling of damaged bulbs will avoid creating potentially susceptible tissue. Improving soil water drainage is paramount to managing Pythium root rot. Packing material can frequently be contaminated with *Pythium*, so recycling of packing materials should be avoided if they cannot be steam disinfested (Van Bruggen and Duineveld 1993).
- ***Fungicides*** – Soil treatments with etridiazole gave good results (Humphreys-Jones and de Rooy 1974). In Holland, the compounds etridiazole, fenaminosulf, and prothiocarb are widely used although results are not always satisfactory. In glasshouse production of tulips, furalaxyl and metalaxyl along with dichloropropene + etridiazole have been used with varying success (Koster and De Rooij 1980).
- ***Biological control*** – Weststeijn (1990) explored using fluorescent *Pseudomonas* isolates against *P. ultimum* and found suppression with one isolate when it was mixed into the soil.

2.5 Rhizoctonia Rot and Blight, Gray Bulb Rot [*Rhizoctonia tuliparum* (Whetzel and Arthur)]

Geographic occurrence and impact. The pathogen *Rhizoctonia tuliparum* is closely related to *R. solani* and has been observed in Europe and the USA (Whetzel and Arthur 1924). *R. tuliparum* has seriously threatened flower bulb production at different times. In 1974, a severe outbreak of gray bulb rot caused major losses on tulips in Lincolnshire, England.

Symptoms/signs. Infected bulbs rarely emerge and ones that do emerge are distorted, stunted, and rarely flower. Bulbs and roots turn grayish in color and progressively dry and shrivel at the site of infection. Roots and bulbs become discolored and collapse. If the soil is very wet, it is not uncommon to have *Pythium* infections along with *Rhizoctonia* (Figs. 5 and 6).

Biology and epidemiology. *Rhizoctonia tuliparum* overwinters as sclerotia and as mycelium on plant debris. One study documented that about 10% of the sclerotia were still viable after 10 years. Where severe outbreaks of disease have occurred, longer intervals between susceptible crops are suggested. Growers should avoid hosts that are also susceptible to *R. tuliparum* such as lilies and onions. *R. solani* is closely related to *R. tuliparum* and has been reported to cause bulb rot. Many times a mixture of genetically related strains may be found. In the Netherlands, the pathogens causing Rhizoctonia disease on tulips may comprise a mixture of distinct strains (Dijst and Schneider 1996). Cooler soil temperature may play a role in dictating which strains of *R. solani* dominate (Schneider et al. 2001).

Management

- ***Cultural practices*** – Care should be exercised when harvesting bulbs to minimize damage. Any damaged bulbs should be discarded.
- ***Sanitation*** – When tulips are forced in the greenhouse, sanitation should be practiced and clean disinfested soil or soilless potting mixes should be used. Field plants should be placed in areas where other susceptible plants have not been grown, e.g., lilies and onions. Once bulbs are infected, they must be discarded.
- ***Fungicides*** – Dip bulbs in PCNB after harvest. On potted plants, drench applications of thiophanate methyl are also recommended.

Other fungal or fungus-like organisms reported to be associated with *Tulipa* spp. (See Farr DF and Rossman AY Fungal Databases, Systematic Mycology and Microbiology Laboratory, ARS, USDA. Retrieved 23 May 2016 from http://nt.ars-grin.gov/fungaldatabases/):

Aecidium tulipae
Alternaria sp.
Ascochyta tulipae
Aspergillus niger
Aspergillus sp.
Botrytis cinerea
Botrytis parasitica (*Botrytis tulipae*)
Botrytis sp.
Cercospora sp.
Cercosporella sp.
Colletotrichum dematium
Colletotrichum fioriniae

Colletotrichum sp.
Corticium solani (*Rhizoctonia solani*)
Corticium vagum (*Haplotrichum curtisii*)
Curvularia sp.
Drechslera sp.
Fusarium oxysporum
Fusarium poae
Fusarium roseum
Fusarium sp.
Gibberella avenacea (*Fusarium avenaceum*)
Gloeosporium thuemenii
Gloeosporium thumenii f. *tulipae*
Ilyonectria sp.
Nectria inventa (*Acrostalagmus luteoalbus*)
Nectria sp.
Oidium sp.
Penicillium cyclopium (*Penicillium aurantiogriseum*)
Penicillium verrucosum var. *cyclopium*
Phytophthora cactorum
Phytophthora citricola
Phytophthora cryptogea
Phytophthora erythroseptica var. *erythroseptica*
Phytophthora hedraiandra
Phytophthora porri
Phytophthora sp.
Puccinia prostii
Puccinia sp.
Puccinia tulipae
Pythium intermedium (*Globisporangium intermedium*)
Pythium irregulare (*Globisporangium irregulare*)
Pythium oopapillum
Pythium ultimum (*Globisporangium ultimum*)
Rhizopus necans
Rhizopus sp.
Rhizopus stolonifer
Sclerotinia fuckeliana (*Botrytis cinerea*)
Sclerotinia sativa
Sclerotinia sclerotiorum
Sclerotinia sp.
Sclerotium perniciosum
Sclerotium rolfsii (*Athelia rolfsii*)
Sclerotium tulipae (*Botrytis tulipae*)
Sclerotium tuliparum (*Rhizoctonia tuliparum*)
Septoria sp.
Synchytrium laetum

Thanatephorus cucumeris (*Rhizoctonia solani*)
Thielaviopsis basicola
Urocystis sp.
Uromyces erythronii
Ustilago heufleri (*Vankya heufleri*)
Ustilago vaillantii (*Antherospora vaillantii*)
Vankya heufleri
Verticillium lateritium (*Acrostalagmus luteoalbus*)

3 Bacterial and Phytoplasma Diseases

3.1 Bacterial Soft Rot [*Pectobacterium carotovorum* (*Erwinia carotovora*); *Dickeya didantii* (*Erwinia chrysanthemi*)]

Geographic occurrence and impact. The disease has been reported in Australia, Belarus, China, Japan, the Netherlands, Turkey, and the USA (Boyraz et al. 2006; Fahy and Persley 1983; Fujimura et al. 2004; Gill et al. n.d.; Guolan and Zhang 1998; Gvozdyak et al. 1985) and can cause production and postharvest losses.

Symptoms/signs. Mature bulbs are most frequently attacked by soft-rotting bacteria. Infected bulb scales initially appear water-soaked and light yellow to light brown or light gray to white. As the infection advances throughout the bulb, affected tissue becomes soft and watery (macerated) due to the bacterium's pectolytic enzymes (Fig. 7). A dark, foul-smelling liquid exudes from diseased bulbs. *Pectobacterium carotovorum* has also been reported to cause small, ellipsoidal water-soaked spots which enlarge to blight leaves (Fig. 7) and bud neck rot in tulip in Konya, Turkey (Boyraz et al. 2006). The incidence of bacterial leaf blight in that area was about 27% and 17% in 2002 and 2003, respectively.

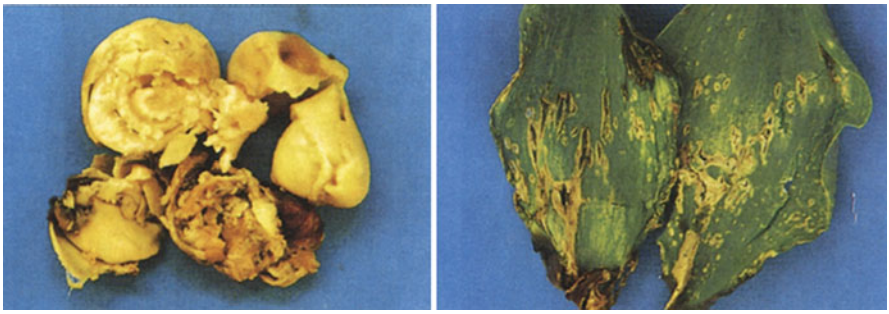


Fig. 7 Bulb soft rot (*left*) and advanced leaf blight (*right*) caused by *Pectobacterium carotovorum* (Boyraz et al. 2006)

Biology and epidemiology. *Pectobacterium* and *Dickeya* spp. attack the soft, fleshy tissue of many plant hosts. They survive in infected propagative material and in soil in association with decaying plant material and are spread by water; insects that feed on declining plant tissue; tools, hands, and clothing of workers; equipment; and airborne particles and aerosols (Bradbury 1977). The bacteria enter plants through natural openings (stomata and lenticels) and wounds. The disease is favored by poorly drained soil and air circulation.

Management

- ***Cultural*** – Use vigorous, blemish-free, and pathogen-free bulbs. Avoid planting in poorly drained areas and use adequate spacing to increase air circulation. Harvest bulbs during dry weather and process and store them properly to avoid injury. Control possible insect vectors.
- ***Sanitation*** – Discard/burn infected bulbs and other diseased plant tissue when dry. Workers should disinfest hands, tools, and equipment after processing rotted plant tissue; 10% household bleach, 70% alcohol, or a commercial material may be used to disinfest tools and equipment (Gill et al. n.d.) (refer to ▶ Chap. 9, "Sanitation for Management of Florists' Crops Diseases").
- ***Bactericides*** – refer to ▶ Chap. 7, "Fungicides and Biocontrols for Management of Florists' Crops Diseases" for general information on bactericides.

Other bacteria reported to be associated with *Tulipa* spp. include:

Bacillus sp. (Gvozdyak et al. 1985)

Burkholderia andropogonis (syn. *Pseudomonas andropogonis*) (Morikawa et al. 1996)

Burkholderia gladioli (syn. *Pseudomonas gladioli*) (Morikawa et al. 1996)

Curtobacterium flaccumfaciens pv. *oortii* (syn. *Corynebacterium flaccumfaciens* pv. *oortii*) (Fox 2014)

Rhodococcus fascians (Tilford) Goodfellow (syn. *Corynebacterium fascians* (Tilford) Dowson.) (van Aartrijk et al. 2000)

4 Viral Diseases

Geographic occurrence and impact. Viruses are among the most damaging pathogens of tulips because of the reductions in quantity and quality of flowers and bulbs that they cause. In addition, viruses are extremely difficult to control in a vegetatively propagated crop such as tulip. A wide range of viruses have been reported in tulip. The most important viruses are *Arabis mosaic virus* (ArMV), *Cucumber mosaic virus* (CMV), *Lily symptomless virus* (LSV), *Tobacco necrosis virus* (TNV), *Tobacco rattle virus* (TRV), and particularly *Tulip breaking virus* (TBV). The geographic occurrence and host range of these viruses are indicated in Table 1. Other viruses reported to infect *Tulipa* spp. include those in the following genera (Asjes 1994; Lesnaw and Ghabrial 2000; Mowat 1972):

Table 1 Selected tulip viruses

Virus, genus	Geographic occurrence	Transmission	Other natural hosts	Symptoms	References
Arabis mosaic virus (ArMV) *Nepovirus*	The Netherlands	Mechanical; dagger nematodes (*Xiphinema* spp.); seed of some plants	Many species of wild and cultivated monocotyledonous and dicotyledonous plants	Oval gray or brown ring spots primarily at the bases of lower leaves (Fig. 8); whole plants appear brittle, dwarfed and twisted, and may die	Asjes 1976; Murant 1984;
Cucumber mosaic virus (CMV) *Cucumovirus*	The Czech Republic, Japan, South Korea, New Zealand, the Netherlands, Poland	Mechanical; > 60 aphid spp.	A very large host range including many horticultural crops and weeds	Sunken brown spots or rings in bulbs which produce distorted and stunted plants; color breaking in flowers	Asjes 1994; Duraisamy and Pokorny 2009; Francki and Habili 1980/1987; Kim et al. 1995; Pearson et al. 2009; Sochacki 2013; Yamamoto 1971
Lily symptomless virus (LSV) *Carlavirus*	South Korea, the Netherlands, Poland	Mechanical; the green peach aphid (*Myzus persicae*)	*Lilium* (lily)	Color breaking in flowers and leaf mottling (Fig. 9)	Asjes 1986, 1994; Kim et al. 1995; Sochacki 2013
Tobacco necrosis virus (TNV) *Necrovirus*	The Czech Republic, New Zealand, the Netherlands, Poland	Mechanical; the fungus *Olpidium brassicae* (Chytridiales)	Depending on the strain: *Chenopodium amaranticolor*, *C. quinoa*; *Cucumis sativus* (cucumber); *Nicotiana clevelandii*, *N. tabacum* (tobacco); *Phaseolus vulgaris* (bean)	Brown necrotic streaks in leaves and stems	Asjes 1994; Pearson et al. 2009; Sochacki 2013; Teakle and Brunt 1982/1990

Tobacco rattle virus (TRV) *Tobravirus*	New Zealand, the Netherlands, Poland, South Africa	Mechanical; stubby root nematodes (*Paratrichodorus*, *Trichodorus* spp.); seed of some plants	At least nine families including Brassicaceae, Cucurbitaceae, Leguminosae, and Solanaceae	Mottling and light-colored streaks in leaves	Asjes 1994; Duraisamy and Pokorny 2009; Harrison and Robinson 1984; Pearson et al. 2009; Sochacki 2013; Whitlock 1984
Tulip breaking virus (TBV) *Potyvirus*	Worldwide, wherever tulips are grown	Mechanical; a number of aphid spp. including the green peach aphid (*Myzus persicae*) and the cotton aphid (*Aphis gossypii*)	*Lilium* (lily)	Symptoms in *Tulipa* spp. and Darwin hybrids include color breaking in flowers and leaf chlorosis	Lesnaw and Ghabrial 2000; Philips 1986
Tulip virus X (TVX) *Potexvirus*	Japan, New Zealand, the Netherlands, Norway, Poland, Sweden, UK	Mechanical	Only reported in tulip	Chlorotic or necrotic gray-brown streaks in leaves; intensified colored or necrotic streaks in flowers	Dees et al. 2011; Mowat 1982, 1990; Sochacki 2013; Ward et al. 2008

Ampellovirus – *Tulip severe mosaic virus* (TSMV)
Ophiovirus – *Tulip mild mottle mosaic virus* (TMMMV)
Potyvirus – *Lily mottle virus* (LMoV), *Rembrandt tulip-breaking virus* (ReTBV), *Tulip band-breaking virus* (TBBV), *Tulip chlorotic blotch virus* (TCBV), *Tulip top-breaking virus* (TTBV)
Tombusvirus – *Tomato bushy stunt virus* (TBSV)

Symptoms/signs. Virus symptoms in tulip vary depending on the virus, virus strain, mixed viral infections, and tulip variety. In addition, different viruses can cause similar symptoms in the same tulip variety making diagnosis of virus infection by symptoms alone unreliable. Besides TBV, other potyviruses such as TBBV, TTBV, ReTBV, and LMoV, as well as non-potyviruses such as ArMV, CMV, and LSV, can cause some degree of color-breaking symptoms (Dekker et al. 1993; Lesnaw and Ghabrial 2000). Historically, color breaking of flowers induced by viruses was viewed as an attractive and desirable trait. However, this virus-based characteristic is unstable, and virus infection leads to reduction in the quality and quantity of tulip flowers and bulbs (Bos 1970; Yamamoto 1971). Today, for the most part, flower variegation in tulips is the result of stable genetic traits produced by plant breeding. Common symptoms of selected viruses in tulip are presented in Table 1.

Biology and epidemiology. As obligate pathogens, viruses need a living host, either tulip or an alternate, for their survival and replication. A number of important tulip viruses, including all those listed in Table 1, are mechanically transmissible in plant sap. This factor is very important for virus spread in a vegetatively propagated crop such as tulip. In addition, most of these viruses also have vectors which disseminate them to new plants: CMV, LSV, and TBV are vectored in a non-propagative (nonpersistent) manner on the stylets of aphids; ArMV and TRV are spread by dagger and stubby root nematodes, respectively, while TNV is vectored by the fungus *Olpidium brassicae.*

Management. Once infected by a virus, unlike with other pathogens, only demanding tissue culture procedures can free plants of viruses, and even these stringent measures are not always successful. Therefore, management of tulip viruses must be based on prevention of infection by integrating appropriate practices:

- ***Cultural practices*** – Use certified virus-free propagative material. Isolate foundation stock and exclude aphids with fine mesh screening. Harvest early to reduce exposure to increasing aphid populations (Chastagner et al. 2002). Thomsen (1980) reported that the spread of TRV in sandy soil was greater when the bulbs were grown at 15 cm than at 7.5 cm. [The highest densities of *T. similis*, a TRV vector, in sandy and sandy-loam soils are found at a depth of 20–30 cm (Decraemer and Geraert 2006).] Spent mushroom compost added in-furrow at planting and fodder radish as a preceding crop reduced the infection percentage in

tulip under favorable conditions for TRV infection (Zoon et al. 2002). Late planting of TRV susceptible cvs. may be helpful (van Aartrijk et al. 2000).

- ***Sanitation*** – Routinely scout fields and remove and dispose of/burn diseased and volunteer bulbs. Control weeds, which may provide reservoirs of viruses and their vectors. Disinfest cutting tools, containers, and equipment (refer to ▶ Chap. 9, "Sanitation for Management of Florists' Crops Diseases").
- ***Insecticides*** – Asjes (1981) found that weekly sprays of mineral oil were more effective in reducing spread of the aphid-vectored TBV than the insecticides pirimicarb or permethrin. It was further demonstrated that the combination of mineral oil with a pyrethroid was more effective than either insecticide alone in reducing the spread by aphids of TBV and LSV in lily (Asjes 1991).
- ***Soil disinfestation*** – Use an appropriate preplant soil fumigant or other soil disinfestation practice to reduce the density of nematode and fungal vectors of ArMV, TNV, and TRV (refer to ▶ Chap. 8, "Soil/Media Disinfestation for Management of Florists' Crops Diseases").
- ***Resistance*** – Three tulip cvs., Apeldoorn, Kees Nelis, and Lustige Witwe, appeared to be susceptible to TBV, and four, Cantata, Juan, Madame Lefeber, and Princeps, showed a high degree of resistance to the virus (Romanow et al. 1990).

5 Nematode Diseases

5.1 Stem and Bulb Nematode [*Ditylenchus dipsaci* (Kühn)]

Geographic occurrence and impact. The nematode occurs in most temperate areas (Europe and the Mediterranean region, North and South America, northern and southern Africa, Asia and Oceania), but it does not appear to establish in tropical regions except at higher elevations that have temperate climates (Anon. 2017). *Ditylenchus dipsaci* infects and causes damage to a wide range of other cultivated hosts including *Scilla* spp., snowdrops (*Galanthus* spp.), *Iris* spp., *Hyacinth* spp., *Narcissus* spp., onions (*Allium* spp.), oat (*Avena sativa*), phlox, and strawberry (*Fragaria* × *ananassa*) (Agrios 2005; Slootweg 1960).

Symptoms/signs. General symptoms of *D. dipsaci* include swellings and twisting of leaves and stems and necrosis or rotting of stem bases, bulbs, tubers, and rhizomes; seedling death may also occur. In tulip, aboveground parts exhibit light yellow to white spots or swellings which may coalesce, and cracking of the epidermis occurs with white, ragged edges. Especially characteristic symptoms in tulip flowers include swellings, which become warty growths; often a portion of the petal above the stem infection site remains wholly or partly green (Fig. 10). Affected bulbs frequently dry out during storage and are secondarily affected by *Penicillium* and mites (van Aartrijk et al. 2000).



Fig. 8 Oval ring spots caused by ArMV (Asjes 1976)



Fig. 9 Color breaking in flowers (*left*) and leaf mottling (*right*) caused by LSV (Horst 2008)

Biology and epidemiology. *Ditylenchus dipsaci* is a migratory endoparasite of bulbs, stems, and leaves and normally completes its life cycle in these tissues. The nematode has a five-stage life cycle and the ability to enter into a dormant stage. *Ditylenchus dipsaci* enters the soil only during unfavorable conditions such as when extensive bulb decay occurs. Juveniles then move out of plant tissue and accumulate around the basal plates of dried bulbs as grayish-white, cottony masses called "nematode wool," through which they can remain dormant for a number of years. Nematodes may also survive in many alternate cultivated crops such as those cited above and weeds such as chickweed [*Stellaria* spp.], groundsel [*Senecio vulgaris*], shepherd's purse [*Capsella bursa-pastoris*], grasses, and wild onions (*Allium* spp.)



Fig. 10 Tulip flower on left infected with stem nematodes; note lesion on the stem (*lower arrow*) and the persistence of green coloration in the petal nearest to the infection site (*upper arrow*) (Gratwick 1992)

(Slootweg 1960). However, *D. dipsaci* occurs in more than 20 biological races, some of which have a limited host range (Anon. 2017).The nematode is spread in infected bulbs and by contaminated soil moved by water, wind, and machinery and on the clothing of workers (van Aartrijk et al. 2000).

Management

- ***Cultural practices*** – Use nematode-free bulbs, and rotate with a nonhost. Eliminate weed hosts. Flooding of infested *sandy soil* for about 10 weeks at a water temperature of about 17 °C/62 °F resulted in the elimination of *D. dipsaci* (Muller and van Aartrijk 1989).
- ***Sanitation*** – Remove and discard/destroy infected plants and bulbs. Disinfest equipment and bulb containers (refer to ▶ Chap. 9, "Sanitation for Management of Florists' Crops Diseases").

  Tulip bulbs are assumed to be sensitive to hot water treatments above 43 °C/109 °F (van Dam 2013). However, research evaluating various pretreatment temperatures (PTT) and hot water treatments (HWT) indicated that a HWT of 4 h at 47 °C/116.6 °F preceded by PTT at 27, 30, or 33 °C (80.6, 86, or 98.6 °F) for 1 week resulted in an average yield of 39, 64, and 92%, respectively, compared to the control treatment (van Dam 2013). Total elimination of *D. dipsaci* occurred with 4 h at 47 °C/116.6 °F, after PTT at 30 °C/86 °F for 1 week.
- ***Nematicides*** – Nøhr Rasmussen and Lindhardt (1980) found that a bulb dip in oxamyl and in-furrow application of aldicarb or carbofuran at planting provided satisfactory control. Multiple field application of aldicarb or oxamyl significantly

reduced the number of infected tulip bulbs but did not eradicate *D. dipsaci* from highly infected bulbs (Windrich 1985).

- ***Soil disinfestation*** – Use a preplant soil disinfestation procedure such as steam, fumigation, anaerobic soil disinfestation, etc. (Weststeijn and De Rooy 1974) (refer to ▶ Chap. 8, "Soil/Media Disinfestation for Management of Florists' Crops Diseases").

5.2 Stubby Root Nematode (*Trichodorus* spp., *Paratrichodorus* spp.)

Geographic occurrence and impact. These nematodes are of global significance and can cause direct damage to the roots of a large and very diverse array of plant hosts including agronomic crops, fruit and forest trees, grasses, ornamentals, vegetables, and weeds; a number of trichorid nematodes also cause extensive damage as virus vectors (Anon. n.d.).

Symptoms/signs. Symptoms include stubby and reduced roots, chlorosis, wilting, and stunting.

Biology and epidemiology. *Trichodorus* and *Paratrichodorus* are migratory ectoparasitic nematodes that feed on the epidermal cells at or near the root tip region, but never wholly enter the root tissue. The nematode lays eggs in the soil, which hatch to produce juveniles and then adults, completing their life cycle in 20–45 days under favorable conditions. All free juvenile stages and adults can attack and feed on plants (Agrios 2005; Decraemer and Geraert 2006). Stubby-root nematodes are obligate plant parasites; they must feed on plants in order to survive and reproduce (Crow 2014).

As mentioned previously, a number of *Trichodorus* and *Paratrichodorus* species vector TRV. However, there is a pronounced specificity between virus serotype and vector. For example, the PRV serotype of TRV from Europe is only transmitted by *P. pachydermus* (Brown et al. 2004). Adults and juveniles can transmit TRV, but the virus is probably not retained through the molt and is not transovarial (Robinson and Harrison 1989).

Management. Refer to control practices for *Trichodorus* and *Paratrichodorus* indicated in the Viral Diseases management section above and ▶ Chap. 8, "Soil/Media Disinfestation for Management of Florists' Crops Diseases."

Other nematodes reported to attack tulip:

Aphelenchoides subtenuis (Cobb) (van Aartrijk et al. 2000)
Ditylenchus destructor (Thorne) (van Aartrijk et al. 2000)
Pratylenchus penetrans (Cobb) (van Aartrijk et al. 2000)

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R.J. McGovern, W.H. Elmer (eds.), *Handbook of Florists' Crops Diseases*, Handbook of Plant Disease Management, https://doi.org/10.1007/978-3-319-39670-5

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