Diseases of Cyclamen

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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

Contents

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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).

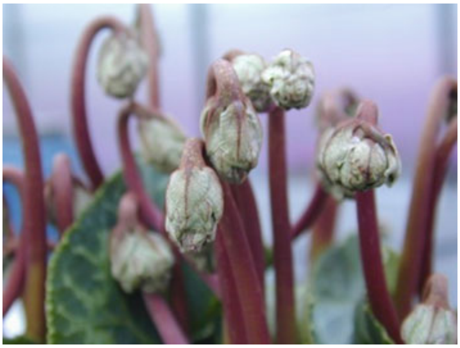

Fig. 13 Symptoms of phyllody (the abnormal development of floral parts into leafy structures and virescence (*green* coloration in flowers) (Diffusion Morel Co. © 2017. All Rights Reserved.)

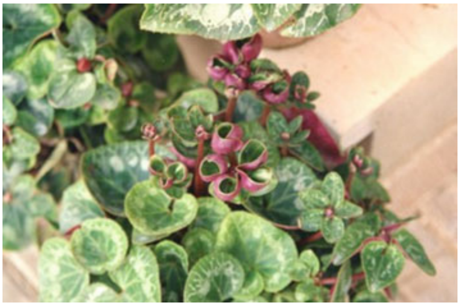

Fig. 14 Phytoplasma disease of cyclamen causing stunting with thickened, rolled, cup-shaped leaves (Diffusion Morel Co. © 2017. All Rights Reserved.)

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.

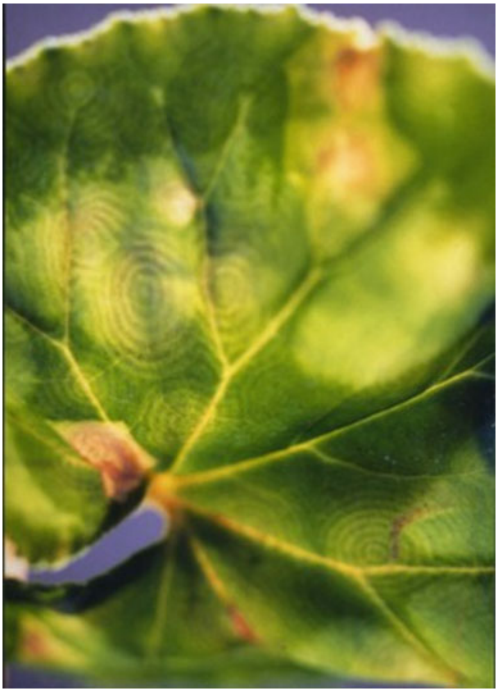

Fig. 16 *Impatiens necrotic spot virus* on cyclamen (Diffusion Morel Co. © 2017. All Rights Reserved.)

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.

References

Alma A, Marzachì C, d'Aquilio M, Bosco D (2000) Cyclamen (*Cyclamen persicum* L.): a dead end host species for 16Sr-IB and -IC subgroup phytoplasmas. Ann Appl Biol 136:173–178

Amani A (1967) Soft rot of ornamentals and vegetables. Iranian J Plant Pathol 4:1–10

Aragaki M, Uchida JY (2001) Morphological distinctions between *Phytophthora capsici* and *P. tropicalis* sp. nov. Mycologia 93:137–145

Araújo AE, Maffia LA, Mizubuti ES, Alfenas AC, Capdeville GD, Grossi JA (2005) Survival of *Botrytis cinerea* as mycelium in rose crop debris and as sclerotia in soil. Fitopatol Bras 30:516–521

Bacigálová K, Turis P, Park MJ, Shin HD (2010) First report of Septoria infection on *Cyclamen fatrense*. Thaiszia J Bot 20:109–114

Baker KF, Dimock AW, Davis LH (1950) *Ramularia cyclaminicola* Trel., the cause of cyclamen stunt disease. Phytopathology 40:1027–1034

Barthelet J, Gaudineau M (1936) Les maladies des Cyclamens. Rev Pathol Veg Entomol Agric Fr 23:101–122

Beamont A (1953) Cyclamen diseases. Gard Chronicle 133:240

Bellardi MG, Vicchi V (1998) Impatiens necrotic spot tospovirus in cyclamen in Veneto (Northern-East Italy) [*Cyclamen persicum* Mill.]. Colture Protette, Italy

Bertaccini A (1990) Cyclamen: a new host of mycoplasma-like organisms. Phytopathol Mediterr 29:213–214

Bongini V (1930) Le macchie fogliari del Ciclamino (*Phyllosticta cyclaminis* Brun.). Difesa delle Piante 7:4–9

Bongini V (1940) Note fitopatologiche. Boll Lab Sper R Osserv Fitopatalog Torino 18:1–34

Bonifacio A (1960) A cyclamen wilt. Rivista della Ortoflorofrutticoltura Italiana 44:165–171

Brielmaier-Liebetanz U, Bohmer B (1988) *Cryptocline cyclaminis* – investigations on the susceptibility of Cyclamen cultivars and on the range of host plants. Gesunde Pflanzen 40:253–256 (Germany, FR)

Butcher RW (1934) Cyclamen wilt disease. Tenth annual report Cheshunt experimental research station, Hertfordshire 1924, pp 69–72

Carta C (1993) *Erwinia rhapontici* (Millard) Burkholder: a new report from cyclamen (*Cyclamen persicum* Mill.). Phytopathol Mediterr 32:257–260

Chandrashekar M, Diriwaechter G (1984) Soft rot of cyclamen in Australia caused by *Erwinia carotovora* ssp. *carotovora*. Australas Pl Path 12:60–62

Chase A (2009) Effect of fungicides on anthracnose on cyclamen caused by *Colletotrichum*. Chase Horticultural Research Inc. http://www.chasehorticulturalresearch.com/pdfs/chasebase/229.pdf

Daughtrey M, Tobiasz M (2005) Suppression of Fusarium wilt of cyclamen with bioantagonists and fungicides. Phytopathology 95:S128

Daughtrey ML, Wick RL, Peterson JL (1995) Compendium of flowering potted plant diseases. American Phytopathological Society, St. Paul

Daughtrey ML, Jones RK, Moyer JW, Daub ME, Baker JR (1997) Tospoviruses strike the greenhouse industry. Plant Dis 81:1221

Davis LH (1950) *Ramularia cvclaminicola* Trel. the cause of cyclamen stunt disease. Phytopathology 40:027–1034

de O Resende R, Pozzer L, Nagata T, Bezerra IC, Lima MI, de Brito Giordano L, Kitajima EW, de Ávila AC (1996) New tospoviruses found in Brazil. Acta Hortic 431:78–89. doi:10.17660/ActaHortic.1996.431.7

Donahoo RS, Lamour KH (2008) Interspecific hybridization and apomixis between *Phytophthora capsici* and *Phytophthora tropicalis*. Mycologia 100:911–920

Elliott DR, Lebas BSM, Ochoa-Corona FM, Tang J, Alexander BJR (2009) Investigation of Impatiens necrotic spot virus outbreaks in New Zealand. Australas Pl Pathol 38:490–495

Elmer WH (2001) The influence of nonpathogenic *Fusarium* species and *Pseudomonas fluorescens* on Fusarium wilt of cyclamen, 2000. Biol Cult Tests Control Plant Dis 16:O40

Elmer WH (2002a) Efficacy of microbial inoculants as preventative fungicides for suppression of Fusarium wilt of cyclamen. Biol Cult Tests Control Plant Dis 17:O07

Elmer WH (2002b) Influence of *Fusarium oxysporum* f. sp. *cyclaminis* and sodium chloride on growth of cyclamen and Fusarium wilt. Plant Dis 86:389–393

Elmer WH (2006) Efficacy of acibenzolar-S-methyl in the management of Fusarium wilt of cyclamen. Crop Prot 25:671–676

Elmer WH, Daughtrey ML (2012) Fusarium wilt of cyclamen. In: Garibaldi A, Katan J, Gullino ML (eds) Fusarium wilts of greenhouse vegetable and ornamental crops. APS Press, St. Paul, pp 213–219

Elmer WH, McGovern RJ (2004) Efficacy of integrating biologicals with fungicides for the suppression of Fusarium wilt of cyclamen. Crop Prot 23:909–914

Elmer WH, Gent MPN, McAvoy R (2011) Influence of partial saturation in ebb and flow irrigation on flooded floors to suppress Pythium root rot of ornamentals. Crop Prot 33:29–33

Ewald A, Orlicz-Luthardt A, Winkelmann T, Schwenkel H-G (2000) Interspecific hybrids of *Cyclamen persicum* Mill. × *Cyclamen purpurascens* Mill.: propagation, somaclonal variation, resistance to *Fusarium* wilt and suitability as an outdoor crop. Acta Hortic 508:309–310

Gallitelli D (2000) The ecology of Cucumber mosaic virus and sustainable agriculture. Virus Res 71:9–21

Garibaldi A (1988) Research on substrates suppressive to *Fusarium oxysporum* and *Rhizoctonia solani*. Acta Hortic 221:271–277

Gerlach W (1954) Untersuchungen über die Welkekrankheit des Alpenveilchens. (Erreger: *Fusarium oxysporum* Schlecht. f. sp. *cyclaminis* n. f.). Phytopath 22:125–176

Gerlach WPP, Schubert R (2001) A new wilt of cyclamen caused by *Phytophthora tropicalis* in Germany and the Netherlands. Plant Dis 85:334–334

Gillespie DR, Menzies JG (1993) Fungus gnats vector *Fusarium oxysporum* f. sp. *radicis-lycopersici*. Ann Appl Biol 123:539–544

Gladstone LA, Moorman GW (1989) Pythium root rot of seedling geraniums associated with various concentrations of nitrogen, phosphorus, and sodium chloride. Plant Dis 73:733–736

Gladstone LA, Moorman GW (1990) Pythium root rot of seedling geraniums associated with high levels of nutrients. HortScience 25:982

Goto T, Sazarash H, Nozawa H, Nakayama K, Natsuaki T (2001) Necrotic spot of cyclamen and exacum caused by impatiens necrotic spot virus (INSV)(Abstract in Japanese). Jpn J Phytopathol 67:173

Gullino ML, Minuto A, Gilardi G, Garibaldi A (2002) Efficacy of azoxystrobin and other strobilurins against Fusarium wilts of carnation, cyclamen and Paris daisy. Crop Prot 21:57–61

Harrison UJ, Shew HD (2001) Effects of soil pH and nitrogen fertility on the population dynamics of Thielaviopsis basicola. Plant Soil 228:147–155

Hong CX, Richardson PA, Kong P, Jeffers SN, Oak SW (2006) *Phytophthora tropicalis* isolated from diseased leaves of *Pieris japonica* and *Rhododendron catawbiense* and found in irrigation water and soil in Virginia. Plant Dis 90:525

Iyozumi H, Hirayae K, Komagata T, Tsuchiya K, Hibi T, Akutsu K (1996) Biocontrol of cyclamen gray mould (*Botrytis cinerea*) by *Serratia marcescens* B2. Ann Phytopathol Soc Jpn 62:559–565

Kamińska M (1975) Susceptibility of cyclamen to certain viruses. Prace Instytutu Sadownictwa w Skierniewicach B 1:67–172

Kessel GJT, de Haas BH, Lombaers-van der Plas CH, Meijer EMJ, Dewey FM, Goudriaan J, van der Werf W, Köhl J (1999) Quantification of mycelium of *Botrytis* spp. and the antagonist *Ulocladium atrum* in necrotic leaf tissue of cyclamen and lily by fluorescence microscopy and image analysis. Phytopath 89:868–876

Khristova E (1958) New diseases of decorative plants in Bulgaria and their control. Ovorshtarst Gradinarst 1:38–41

Köhl J, Gerlagh M, Grit G (2000) Biocontrol of *Botrytis cinerea* by *Ulocladium atrum* in different production systems of cyclamen. Plant Dis 84:569–573

Korbin M, Kaminńska M (1998) Characterization of Cucumber mosaic cucumovirus isolates. Phytopathol Pol 16:71–78

Krebs E-K (1985) Erst bei hoher Sporendichte birgt *Fusarium* Gefahr. Gaertnerboerse und Gartenwelt 48:1791–1793

Krebs E-K (1986) Brennflecken Kranheit ist *Cyptocline cyclaminis*. Gaertnerboerse und Gartenwelt 86:1868–1869

Krebs E-K (1987) Brennflecken-Kranheit ist sicher bekampfbar. Gaertnerboerse und Gartenwelt 87:1731–1733

Kuter GA, Hoitink HAJ, Chen W (1988) Effect of municipal sludge compost curing time on suppression of Pythium and Rhizoctonia diseases of ornamental plants. Plant Dis 72:751–756

Lemattre M (1973) Problème bactérien en culture de cyclamen. In: ‘Le cyclamen’. Journée d’étude organisée sous l’égide du Cercle National des Jeunes Horticulteurs par le Service Technique de la Fédération Nationale des Producteurs de l’Horticulture et des Pépinières (F.N.P.H.P.). Paris, 22 Feb 1973, pp 63–70

Leroux P (2007) Chemical control of Botrytis and its resistance to chemical fungicides. In: Elad Y, Willimans B, Tudzynski P, Delen N (eds) Botrytis: biology, pathology and control. Springer, Amsterdam, pp 195–222

Liu B, Munster M, Johnson C, Louws FJ (2011) First report of anthracnose caused by *Colletotrichum fragariae* on cyclamen in North Carolina. Plant Dis 95:1480

MacKenzie SJ, Mertely JC, Seijo TE, Peres NA (2008) *Colletotrichum fragariae* is a pathogen on hosts other than strawberry. Plant Dis 92:1432–1438

Marque JM, Kobori RF, Kato SM (2006) Ocorrência de Colletotrichum gloeosporioides em ciclamem cultivado em vaso sob estufa no Estado de São Paulo. Fitopatol Bras 31:3

Massey LM, Tilford PE (1932) Cyclamen stunt. Phytopathology 22:19

Maya MA, Matsubara Y-I (2013) Tolerance to Fusarium wilt and anthracnose diseases and changes of antioxidative activity in mycorrhizal cyclamen. Crop Prot 47:41–48

Maya MA, Ito M, Matsubara Y (2014) Tolerance to heat stress and anthracnose in mycorrhizal cyclamen. Acta Hortic 1025:143–148

Migheli Q, Garibaldi A (1995) Evaluation of antagonistic strains of *Fusarium* spp. in the biological and integrated control of Fusarium wilt of cyclamen. Crop Prot 14:221–226

Minuto A, Migheli Q, Garibaldi A (1995) Evaluation of antagonistic strains of *Fusarium* spp. in the biological and integrated control Fusarium wilt of cyclamen. Crop Prot 14:221–226

Minuto G, Minuto A, Tinivella F, Gullino ML, Garibaldi A (2004) Disease control on organically grown cyclamen. Manage Plant Dis Arthropod Pests BCAs IOB/wprs Bul 27:89–93

Minuto A, Ciuffo M, Dani E, Turina M, Minuto G (2011) Uncommon symptoms caused by infection of *Cucumber mosaic virus* on potted cyclamen. Colture Protette 40:68–71

Mirkova E (1995) Sensitivity of Phytophthora and Pythium to metalaxyl. Rasteniev”dni Nauki 32:120–121

Moore WC (1947) Report of fungus, bacterial, and other diseases of crops in England and Wales for the years 1943–1946. G B Minist Agric Fish Food Bull 139:78–79

Moorman GW, Lease RJ (1992) Benzimidazole- and dicarboximide-resistant *Botrytis cinerea* from Pennsylvania greenhouses. Plant Dis 76:477–480

Moorman GW, Kang S, Geiser DM, Kim SH (2002) Identification and characterization of *Pythium* species associated with greenhouse floral crops in Pennsylvania. Plant Dis 86:1227–1231

Olsen H, Benson CM (2010) First report of crown rot caused by *Phytophthora tropicalis* on gloxinia in North Carolina. Pl Health Prog. doi:10.1094/PHP-2010-0708-03-BR

Orlicz-Luthardt A (1998) Studies on the resistance of cyclamen to Fusarium wilt. Beitr Züchtungsforsch 4:48–49

Oudemans P, Coffey MD (1991) A revised systematics of twelve papillate Phytophthora species based on isozyme analysis. Mycol Res 95:1025–1046

Panagopoulos CG, Psallida PG (1970) A bacterial soft rot of cyclamen. Annales de l'Institut Phytopathologique Benaki 9:83–89

Pitta GPB, Teranishi J (1979) Ocorrencia da murcha (*Fusarium oxysporum* Schl. f. *cyclaminis* n.f.) do ciclamen (*Cyclamen persicum* Mill.). Biologica 45:213–215

Raabe RD, Hurlimann JH (1981) Control of Pythium root rot of cyclamen. Calif Pl Path 53:3–4

Rattink H (1986) Some aspects of the etiology and epidemiology of Fusarium wilt on cyclamen. Meded Fac Landbouww Univ Gent 51(2b)

Rattink H (1990) Epidemiology of Fusarium wilt in cyclamen in an ebb and flow system. Neth J Plant Pathol 96:171–177

Reimherr P (1985) Atlas of diseases and pests of cyclamen. Gb + Gw 85(48):1780–1790

Romero AM, Rivera MC (2005) Cyclamen soft rot caused by Erwinia chrysanthemi in Argentina. Australas Pl Pathol 34:279–280

Rouxel F, Grouet D (1974) Présence en France de la fusariose vasculaire du Cyclamen. Ann Phytopathol 6:475–478

Seglie L, Spadaro D, Devecchi M, Larcher F, Gullino ML (2009) Use of 1-methylcyclopropene for the control of *Botrytis cinerea* on cut flowers. Phytopathol Mediterr 48:253–261

Someya N, Kataoka N, Komagata T, Hirayae K, Hibi T, Akutsu K (2000) Biological control of cyclamen soilborne diseases by *Serratia marcescens* strain B2. Plant Dis 84:334–340

Spengler G (1979) A new fungicide for the control of root and stem rots. Experiments on the control of soil-borne oomycetes in the cultivation of ornamental plants with Fongarid 50 WP. Deutscher Gartenbau 33:989–990

Tompkins CM, Snyder WC (1972) Cyclamen wilt in California and its control. Plant Dis Rep 56:493–497

Trelease W (1916) Two leaf-fungi of cyclamen. Trans Ill State Acad Sci 9:143–146

Vozelj N, Petrovič N, Novak MP, Tušek M, Mavrič I, Ravnikar M (2003) The most frequent viruses on selected ornamental plants and vegetables in Slovenia. In: Proceeding 6th Slovenian conference on plant protection, Zreče, 4–6 March 2003

Weintraub PG, Zeidan M, Spiegel S, Gera A (2007) Diversity of the known phytoplasmas in Israel. Bull Insectol 60(2):143–144

Wollenweber HW, Reinking OA (1935) Die fusarien: ihre beschreibung, schadwirkung und bekämpfung. P. Parey, Berlin

Woudt LP, Neuvel A, Sikkema A, van Grinsven MQJM, de Milliano WAJ, Campbell CL, Leslie JF (1995) Genetic variation in *Fusarium oxysporum* from cyclamen. Phytopathol 85:1348–1355

Wright ER, Rivera MC, Mascarini A, Nuñez LS, Gentile CM (2006) Florist's cyclamen anthracnose caused by *Colletotrichum gloeosporioides* in Argentina. Australas Pl Dis Notes 1:1–2

Yunis H, Elad Y (1989) Survival of dicarboximide-resistant strains of *Botrytis cinerea* in plant debris during summer in Israel. Phytoparasitica 17:13–21