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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Keywords

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Contents

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.

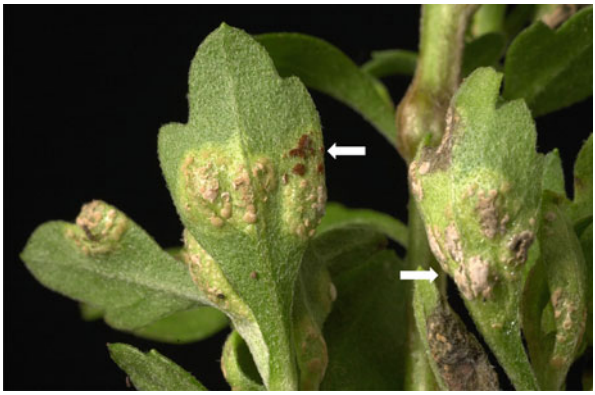

Fig. 5 Rust symptoms and signs: Brown rust (brown pustules, *left*) white rust (white pustules, *right*) (Tracey Olson © 2017. All Rights Reserved.)

- ***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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