FIGURE 6.2 Aecial cups of *P. helianthi* under Argentina conditions. (Courtesy of Dr. Tom Gulya, USDA-ARS, Northern Crops Research Lab, Fargo, ND.)

lower temperature (10°C) begin to germinate about 15 days after their formation, but those formed at higher temperature do not germinate (Hennessy and Sackston 1970).

c. *Basidiospores* (sporidia): These are monokaryotic, nonrepeating spores produced on promycelium.
d. *Pycniospores*: These are haploid gametes; are small, oval, and hyaline; appear shining and viscous in mass; and represent the sexual spores (spermatia) produced in flask-shaped (1 mm in diam.) pycnia (sexual stage) formed on young seedlings on the cotyledons or on true leaves, primarily on the first leaf in about 8–14 days following infection resulting from inoculation with sporidia (Bailey 1923). Insects transfer nectar containing haploid pycniospores of one mating type (+) to the receptive hyphae (–) of the opposite mating type, thereby affecting cross-fertilization.
e. *Aeciospores*: These are unicellular, dikaryotic, nonrepeating spores produced in aecia. Aecia develop on the abaxial surface of the leaf under the fertilized pycnia and discharge dikaryotic aeciospores (Figure 6.2). Aeciospore infection results in the formation of uredinia that produce prodigious numbers of dikaryotic urediniospores. The urediniospores are disseminated by wind and become airborne to cause infection to nearby neighboring or distant sunflower plants.

Variability, Host Specificity, and Pathotypes

Variability and differences in pathogenicity among isolates of *P. helianthi* have been clearly demonstrated as reviewed by Kolte (1985) and Pandey et al. (2005). Though *P. helianthi* is common on cultivated sunflower (*H. annuus*), it also attacks a number of other *Helianthus* species such as *H. decapetalus*, *H. petiolaris*, *H. subcanescens*, and *H. tuberosus* (Parmelee 1977, Shopov 1980). The occurrence of distinct physiologic races of *P. helianthi* on the cultivated sunflower was established first by Sackston (1962) using well-defined 0–4 infection types on a set of host differentials and provided a basis for the explanation of pathogenic differences observed by earlier workers on a variety of *Helianthus* species. A modified Sackston's (1962) numerical rating system described by Yang et al. (1989) can be currently used for rust evaluation as follows: 0 = immune, no uredia or

hypersensitive flecks; 1 = high resistance, presence of hypersensitive flecks or lesions, or pustules smaller than 0.2 mm in diameter with or without chlorotic halo; 2 = resistant, pustules smaller than 0.4 mm; 3 = susceptible, pustules 0.4–0.6 mm in diameter; and 4 = highly susceptible, pustules larger than 0.6 mm in diameter. Reactions 0, 1, and 2 are classed as resistant, while reactions 3 and 4 are rated as susceptible. Rust reaction can be thus rated visually on the basis of both pustule size (infection type) and leaf area covered by pustules (severity). Recent advances allow every isolate to be characterized by its virulence and avirulence toward all known resistance genes in the host. Differentiation of such pathotypes is by reference to their interaction with sunflower cultivars carrying specific genes/genetic factors R1, R2, R3, R4, R5, and R6 conditioning the response (Putt and Sackston 1963, Limpert et al. 1994, Qi et al. 2011). This high-resolution technique obviates the need for detailed race description. New races or, in modern terms, new virulence combinations (*pathotypes*) appear frequently in response to the selection pressure extended by cultivars resistant at the time of introduction. Surveys of virulence, that is, of pathotypes (races) in the late 1990s and in the 2000s, have been carried out in Argentina (Huguet et al. 2008, Moreno et al. 2011), Australia (Kong et al. 1999, Sendall et al. 2006), Canada (Rashid 2004, Gulya and Markell 2009), South Africa (Los et al. 1995, Anonymous 2010), Turkey (Tan 1994, 2010), and the United States (Qi et al. 2011).

In North America

In North America (NA) particularly in Canada and the United States, four NA races (1, 2, 3, and 4) of *P. helianthi* were identified by Sackston (1962) using three standard Canadian sunflower rust differential lines, following a maximum of 2^3 races to be differentiated. Later, the sequential numbering system of race identification was changed to a coded triplet system to produce a virulence formula using a set of nine differentials, which allows theoretical 2^9 races to be identified, assuming no duplication of genes between lines (Gulya et al. 1990a, Gulya and Markell 2009). An international ad hoc committee approved the use of these lines and triplet code for rust race nomenclature (Gulya and Masirevic 1988). To compare the older race classification system and the triplet coding system, the previous NA race 1 corresponds to race 100 of the coded triplet system, NA race 2 to race 500, NA race 3 to race 300, and NA race 4 to race 700. The differentials used in this system include inbred lines S37-388; Canadian lines MC 90, MC 29, and P-386; and lines HA-R1 through HA-R5. S37-388 is universally susceptible to all races, and others have different reaction patterns and are all derived from diverse pedigrees (Gulya and Masirevic 1996, Rashid 2006).

In Australia

Kong et al. (1999) have given a chronological record of the appearance of major pathotypes in Australia from 1978 to 1997, and 23 pathotypes have been recognized, mostly from commercial crops. Frequent shifts in virulence have occurred since the first change was determined in 1983. These have resulted in successive *boom and bust* cycles where commercial sunflower hybrids with resistance to the prevalent pathotypes became susceptible during the rapid shifts in virulence. Almost all pathotypes identified since 1986 trace to a common progenitor, Aus 4. Results of virulence surveys (avirulence and virulence) data accumulated over 25 years revealed that diverse pathotypes of *P. helianthi* evolve in wild sunflower populations providing a continuum of genetically heterogeneous hosts on which *P. helianthi* can potentially complete its sexual cycle. This results in sexual recombination in the causal fungus in seasons that favor completion of the sexual cycle and subsequent selection of recombinant pathotypes and that mutation too contributes steadily to the development of new virulence genes in the population of *P. helianthi* (Gulya 2006, Sendall et al. 2006). Many new pathotypes have been identified due to an increase in the number and availability of differential hosts. At present, 21 differential hosts are routinely used for pathotype identification, whereas only 4 were available in 1983. Many of these differential hosts have not been characterized genetically, but based on their rust reaction, many are presumed to contain more than a single gene for rust resistance.

In Other Countries: Argentina, Turkey, and India

The occurrence of different races of *P. helianthi* in Argentina was first recognized in 1957. Races 100, 300, and 500 were discovered during the 1960 decade and the race 700 during the 1970s. There has been a significant change in the composition of the races of *P. helianthi* in Argentina since 1985. Interestingly, presently (in the 2000s), no isolate of *P. helianthi* belongs to the group of races 100, 300, or 500. In fact all the collected isolates belong to the group of race 700, the first predominant race being 700 followed by race 740. Other variants of race 700, namely, 701, 704, 720, 744, and 760, have also been reported (Huguet et al. 2008, Moreno et al. 2011). This indicates that the deployment of several rust resistance genes (viz., R1 and R2) in sunflower commercial hybrids during the last 30 years determined a selection pressure over rust populations and an associated drift in the frequency of virulence genes. In Turkey, Tan (2010) accomplished race identification of *P. helianthi* under field conditions where seedlings of 23 differential genotypes were naturally infected in the main sunflower production area and concluded the prevalence of rust race 1 (= newly designated as race 100) and race 3 (= race 300) in Turkey, and races 2 (= race 500) and 4 (= race 700) being nonprevalent in that country. Pathogenic variability in *P. helianthi* in India has been studied, but the reports about variability for this pathosystem appear to be scarce (Patil et al. 1998, 2002).

Epidemiology and Disease Cycle

Sunflower rust can occur at any time during the growing season, but disease onset is dependent on the environment and inoculum source. When the disease occurs early, it is usually the result of primary infection originating from primary inoculum sources such as sporidia from germination of surviving teliospores on previous sunflower crop or wild sunflowers, or from aeciospores on volunteer seedlings or from urediniospores formed on volunteer seedlings in high-altitude areas and carried through air currents. The secondary infection occurs usually through repeatedly produced urediniospores in a crop season, and late-season epidemics are generally a result of urediniospores blown in from distant fields (Kolte 1985).

When teliospores act as primary sources, they germinate early in the spring by producing a promycelium from each cell bearing four haploid sporidia also referred to as basidia. The sporidia are of (+) and (–) mating groups, which results through meiosis, while the teliospores undergo the germination process (Kolte 1985, Pandey et al. 2005). Under favorable conditions, when a sporidium comes in contact with the surface of the cotyledon, leaf petiole, or hypocotyl of a sunflower, it produces a germ tube that penetrates directly and establishes the infection, resulting in the development of flask-shaped pycnium producing pycniospores. The (+)- and (–)-type sporidial infections result in the development of their respective types of pycnia and pycniospores where receptive hyphae (–), the female, and spermogonia (+), the male, cross-fertilize, and mating between these two opposite types occurs through insects or rainwater to produce a dikaryotic thallus, which subsequently forms aecia with binucleate aeciospores in about 8–10 days. Aeciospores become airborne and infect sunflower foliage usually near where they are produced. They germinate at 6°C–25°C with an optimum temperature of 16°C for 1 h. But the establishment of infection requires 10 h. Aeciospores, like urediniospores, usually germinate by producing a single germ tube from one of the germ pores, within 4 h after inoculation if the free moisture is present. The germ tube forms an irregularly shaped appressorium over stomata 6–8 h after inoculation. The infection peg is then formed from the lower surface of the appressorium and penetrates the substomatal vesicle, from which two or more infection hyphae arise. When the infection hypha contacts a cell, a septum is formed and a haustorial mother cell is produced, from which knob-shaped to elongated numerous haustoria are formed in the host cell establishing a nutritional relationship with it (Sood and Sackston 1972). The invading hypha grows rapidly in susceptible varieties and culminates in the aggregation of hyphae under the epidermis, resulting in the formation of uredosori containing dikaryotic urediniospores, the economically important stage of the disease cycle. Urediniospores can be disseminated to long distances by wind and infect most of plant tissues. The process of infection through urediniospores

is similar to the one described for aeciospores. In favorable conditions of free moisture (dew) and warm temperatures (12°C–29°C), the uredial stage repeats its cycle every 10–14 days. Because infection is favored by free moisture, infection may be most severe in leaf depressions, on leaf veins, where moisture persists. When temperature falls beyond the favorable range for infection and disease development, the repeating cycle (uredinia) slows and stops. Late-season cold temperatures or host maturity will initiate the changes from the uredinial stage into the overwintering telial stage. Once the telia occur, the disease cycle for that growing season ceases. In the spring, teliospores germinate and produce sporidia, which are visible by microscopic observation only. Sporidia will infect leaves, leading to the formation of pycnium, and the cycle repeats.

Factors Affecting Infection and Disease Development

Kolte (1985) reviewed factors affecting sunflower rust infection and disease development. A day temperature range of 25°C–30.5°C with relative humidity of 86%–92% promotes greater rust intensity, and the relative humidity is positively correlated with the severity of rust. Water-congested sunflower plants are more susceptible to *P. helianthi*. A day temperature of 25°C and a night temperature of 18°C have been found more conducive for the development of the disease under Canadian conditions. Temperature also affects the incubation period. The incubation period following infection through the uredospores is reported as 5, 8, and 7 h at temperatures of 18°C, 14°C, and 22°C, respectively.

Light intensity in the range of 1200–2000 fc influences the maximum production of pustules. Darkness at the time of inoculation and throughout the early stages of infection tends to diminish the intensity of symptoms.

The severity of rust is reported to be less on 15-day-old plants and increases with age, the maximum being on 75-day-old plants. Susceptibility during senescence is directly related to reduced protein synthesis and not to changes in protein content of leaves.

Excess nitrates in the soil or in solution encourage rust infection and defoliation by the rust, but boron and other micronutrients applied to soil reduce its incidence.

Disease Management

Host Resistance

Among the various strategies for rust control, the deployment of diverse resources of resistance in commercial hybrids and varieties remains the most effective approach. Cultivated sunflower originated from the genus *Helianthus* that consists of 51 wild species, 14 annual and 37 perennial, and all are native to the Americas. A large amount of genetic variation in terms of host resistance exists in the wild species, providing genetic diversity for improvement. The origin of most rust resistance genes present in the cultivated sunflower can be traced to wild species mainly *H. annuus*, *H. argophyllus*, and *H. petiolaris* (Hennessy and Sackston 1970, Zimmer and Rehder 1976, Jan et al. 1991, Quresh et al. 1991, 1993, Quresh and Jan 1993, Gulya et al. 2000). One line (PS 1089) derived from *H. argophyllus* × cultivated sunflower and two lines (PS 2011 and PS 2032) derived from *H. petiolaris* × cultivar crosses are reported to be immune to the prevalent races in India (Sujatha et al. 2003). Thus, several sources of rust resistance are known, and the R1, R2, R3, R4, R5, and R6 genes have been characterized and used widely to develop rust-resistant commercial hybrids and varieties (Seiler 1992, Rashid 2006, Sendall et al. 2006, Gulya and Markell 2009, Lawson et al. 2010, Qi et al. 2011, 2012). Genome localization of sunflower rust resistance genes has been documented (Bulos et al. 2012, 2013).

The rust resistance genes R1 and R2 were the first to be discovered in sunflowers and originated from the wild sunflower. Gene R1 present in the inbred lines MC 69 and MC 90 conferred resistance to rust races 100 and 500 (old races 1 and 2). A sequence characterized amplified region (SCAR) marker SCTO6 (950) was found that cosegregates with rust resistance gene R1 and mapped to linkage group (LG) 8 (Lawson et al. 1998, Yu et al. 2003). However, R1 gene is no longer effective

against current virulent races (Qi et al. 2011). In contrast, the gene R2 present in inbred line MC 29, an old Canadian line, showed resistance to 90% of 300 rust isolates tested in the United States in the years 2007 and 2008 including race 336, the predominant race in North America. However, MC 29 is moderately susceptible to race 777, the most virulent race currently known in North America (Qi et al. 2011). R2 has been used in Australian sunflower breeding program and provides resistance to all known Australian races (Sendall et al. 2006, Lawson et al. 2010).

Rust resistance gene R3 identified in the line PhRR3 conferred resistance to two Australian rust races (Goulter 1990). Selecting from Argentinean open-pollinated varieties, five multirace resistant lines, HA-R1 to HA-R5, were released in 1985 (Gulya 1985). The R4 locus is located on LG 13 in *H. annuus* (Sendall et al. 2006). The rust resistance gene R4 present in the germplasm line HA-R3 was derived from an Argentinean interspecific pool with Russian open-pollinated varieties crossed with *H. annuus*, *H. argophyllus*, and *H. petiolaris* (Gulya 1985, de Romano and Vazquez 2003). These lines HA-R1, HA-R4, and HA-R5 were also reported to carry alleles of the R4 locus, whereas the line HA-R2 had a different gene, R5 (Miller et al. 1988). HA-R2 was a selection from the Argentinean open-pollinated cultivar Impira INTA. This cultivar was developed from the interspecific cross between *H. argophyllus* and *H. annuus* cultivar Saratov selection Pergamino (de Romano and Vazquez 2003). Therefore, it is believed that the R5 gene in HA-R2 originated from *H. argophyllus*. Gene R5 conferred resistance to 86% of 300 rust isolates tested in the United States in the years 2007 and 2008, including the predominant race 336, but conferred susceptibility to race 777. Sunflower rust-resistant lines previously released by the USDA were evaluated for their reaction to current virulent races. This evaluation identified nine germplasm lines, HA-R6, HA-R8, RHA 397, RHA 464, PH3, PH4, PH5, TX16 R, and RFANN-1742, that were resistant to both races 336 and 777 (Miller and Gulya 2001, Jan et al. 2004, Jan and Gulya 2006, Hulke et al. 2010, Qi et al. 2011). Similarly germplasm lines in sunflower have been screened for rust resistance in India (Velazhahan et al. 1991).

It is thus revealed from the previous text that incorporating effective resistance genes into sunflower inbred lines and commercial hybrids should mitigate the threat posed by current virulent rust races not only in the North American situation but also in rest of the sunflower-growing countries in the world. The potential to reduce losses due to rust stimulates genetic efforts to develop molecular markers linked to effective rust resistance genes in order to facilitate marker-assisted selection (MAS) (Tang et al. 2002, 2003, Knapp 2003). Molecular markers have been identified for a number of sunflower rust-resistant R genes. These markers have been used to detect resistance genes in breeding lines and wild sunflower. For example, two SCAR markers, SCT06950, which is associated with the rust resistance gene R1, and SCX20 (600), which is linked to the Radv gene, mapped to linkage groups (LGs) 8 and 13, respectively (Yu et al. 2003, Qi et al. 2011). Lawson et al. (2010) reported mapping of the R2 gene to sunflower LG 9. Qi et al. (2011) mapped the R4 gene to a large nucleotide-binding site and leucine-rich repeat (NBS-LRR) cluster on LG13. Molecular mapping of the gene R5 has not been reported in the literature. However, this gene is reported to be associated with two simple sequence repeats (SSRs) as reported by Sendall et al. (2006). A germplasm line HA-R2 carrying the rust resistance gene R6 was released as a multirace rust-resistant line in 1985 but has not been widely used in commercial hybrid production. R6 remains effective against the prevalent rust races of sunflower in North America. Molecular marker analysis demonstrated by Qi et al. (2012) revealed that the LG2 markers showed association with rust resistance. Genotyping of the 94 F2 individuals (progenies derived from the crosses HA 89 with HA-R2) with 23 polymorphic SSR markers from LG2 confirmed the R6 location on LG2, flanked by two SSR markers, ORS1197-2 and ORS653a, at 3.3 and 1.8 cM of genetic distance, respectively. The markers for R6 developed by Qi et al. (2012) will provide a useful tool for speeding up deployment of the R6 gene in commercial sunflower hybrid production.

The future of the development of sunflower inbred lines or hybrids with high levels of durable resistance will depend on the ability to select genotypes that have combinations of effective resistance genes. Knowledge of virulence evolution of the pathogen population and available DNA

markers closely linked to host R genes is a prerequisite for successful gene pyramids. MAS is the choice. To improve the efficiency of MAS, it is important that the recombination frequency between the target gene and the marker be as low as possible. Developing a molecular marker that is located within the rust resistance gene (gene-specific marker) will eventually solve the problem, and the durable genetic resistance through gene pyramiding will be effective for the management of rust.

Chemical Control

Though fungicides may be considered as a last alternative in controlling the rust disease, lack of genetic resistance to some races of causal fungus, *P. helianthi*, necessitates the use of effective fungicides to reduce the impact of rust disease on sunflower yield and quality of seed. Earlier fungicides like dithiocarbamates (maneb, zineb, mancozeb), elemental sulfur, Bordeaux mixture, benodanil, and oxycarboxin were established to be effective for the control of the disease (Kolte 1985). Now newer fungicides, tebuconazole 39 (Folicur) at 0.125 kg/ha, pyraclostrobin 25 (Headline) at 0.15 kg/ha, prothioconazole 48 (Proline) at 0.2 kg/ha, boscalid 25 (Lance) at 0.25 kg/ha, and propiconazole 12.5 + trifloxystrobin 12.5 (Stratego) at 0.18 kg/ha, are reported to be highly effective in managing the sunflower rust (Gulya 1991, Shtienberg 1995, Markell 2008, NDSU 2009). The action threshold for fungicide application is 3% uredopustule coverage on upper leaves (Shtienberg 1995). This, therefore, means fungicidal management of rust should be considered when rust is found on the upper leaves and the plant is in the range of vegetative stages up to the R6 growth stage, or when rust pustules cover 5% of the lower leaves at or before flowering. Recommendations from fungicide research trials indicate that when rust has infected the upper four leaves at 1% or less, then fungicides like Headline and Quadris can be used. If the infection of the upper four leaves is 3% or greater, then Folicur may be used. All effective fungicides (Proline, Folicur, Headline, and Stratego) can reduce the rust incidence and severity as expressed in area under the disease progress curve (AUDPC). Triazoles (Proline and Folicur) tend to have lower AUDPC values than strobilurins (boscalid). The AUDPC due to the triazole group of fungicide spray is reduced by 50%, consequently increasing the crop yield by 10%–20%. Confection sunflower, with their higher value and greater susceptibility, would more likely pay back the cost of fungicide application. The effectiveness of early or late applications or both may vary between years depending on the earliness of the rust infection and disease development. An infection on the upper leaves at the growth stage of R6 or later will not likely have a negative yield effect (Shtienberg 1995, NDSU 2009), and hence, fungicide spray may not be necessary.

In the United States, no fungicides have the federal label or use against sunflower rust. A *specific exemption* under any emergency may be granted by the Environmental Protection Agency (EPA) in some years for use of a specific fungicide for that single year (NDSU 2009). Interestingly, the Colorado State in that country supported the request for the recommendation of use of Folicur 3.6 F when rust epidemics threaten the crop.

Cultural Control

Cultural management practices include plowing under or early-season management of volunteer sunflower plants carrying infection and all crop remnants by removal and destruction by fire. Growing of the sunflower crop 500 m away from the site of the previous years' plot is useful in minimizing the incidence and severity of the sunflower rust (Mitov 1957). Sunflowers should not be planted 2 years in a row in the same field. If possible, avoid planting next to a field that had sunflower last year. If rust occurs on volunteer plants in the vicinity of a planted field, they should be destroyed as soon as possible to prevent the spores from blowing into the planted field. Depending on the occurrence of disease in a particular locality, the choice of planting dates may be used to advantage to avoid the disease (Kolte 1985). Early planting and short-season hybrids will generally have less rust. Avoid dense plant stand and high-nitrogen fertilization (Perez et al. 2002).

Controlling wild sunflowers is a very important step in the management of sunflower rust. All 51 species of *Helianthus* found in North America are hosts to the rust pathogen. All spore stages

readily occur on wild sunflowers, which increase the sunflower rust problem in two ways. First, when the early spore stages appear on wild and volunteer sunflowers, the onset of uredinia is earlier. This allows more infection cycles to take place, which creates a greater yield loss potential. Second, sexual recombination occurs when the pathogen completes its sexual cycle. This may result in new races that overcome available resistance. Therefore, removal of wild sunflower populations around fields is desirable and strongly recommended.

DOWNY MILDEW

Symptoms

Disease symptoms of various kinds depending on the age of tissue, level of inoculum, environmental conditions (moisture and temperature), and cultivar reaction become evident as seedling damping-off, systemic symptoms, local foliar lesions, and basal root or stem galls.

Damping-Off

When susceptible sunflower plants are subject to subterranean infection by the downy mildew fungus, damping-off in the seedling stage occurs, particularly under cool (12°C–13°C) and very wet soil conditions. Seedlings are killed before or soon after emergence, resulting in reduced plant stands under field conditions. Affected plants dry and become windblown.

Systemic Symptoms

Sunflower plants carrying systemic infection are severely stunted. Close correlation has been found between fungal growth and height of the seedlings following inoculation, depending on the susceptibility and direction of spread of the fungus. In a susceptible variety, the pathogen tends to colonize the whole plant. Leaves of affected plants bear abnormally thick, downward-curled leaves that show prominent yellow and green epiphyllous mottling (Figure 6.3). A hypophyllous downy growth of

FIGURE 6.3 Downy mildew of sunflower. (Courtesy of Dr. Chander Rao and Dr. Varaprasad, DOR, Hyderabad, India.)

the fungus, consisting of the conidiophores and conidia, develops and covers large areas that are concurrent with the epiphyllous yellow spot (Cvjetkovic 2008). The stem becomes brittle. The systemically infected sunflower plants show loss of phototropic and negative geotropic responses. Such plants also show pronounced reduction of the development of secondary rootlets. Flower heads of the affected plants remain sterile and produce no seeds, or only occasionally the seeds are produced on such heads. When the older plants are infected, the symptom expression may be delayed until flowering without visible chlorotic symptoms on leaves.

Local Foliar Lesions

Small, angular greenish-yellow spots appear on leaves as a result of secondary infection through zoospores liberated from wind-borne zoosporangia. The spots may enlarge and coalesce to infect a larger part of the leaf. Plants are susceptible to such infection for a longer period than to systemic infection. The fungal growth becomes visible at the lower surface of the diseased area and persists for some time under humid conditions. Such local foliar symptoms usually do not result in systemic symptoms and are, therefore, considered to be of less economic importance.

Basal Root or Stem Galls

Development of basal gall symptoms occurs independently of the infection that results in systemic symptoms. The root infection may result in the formation of galls at the base of the plants on primary roots. Such roots are discolored, scurfy, and hypertrophied; the number of fibrous secondary roots is reduced, and the plants become susceptible to drought. The percentage of plants with basal gall symptoms seldom exceeds 3% in a particular field. Such plants are less vigorous and subject to lodging. Lodging of plants with basal gall symptoms results in fracturing directly through the galled area, thus causing total loss of a particular plant.

Geographical Distribution and Losses

Downy mildew of sunflower generally is found in more temperate regions where emerging seedlings are exposed to low temperature and abundant precipitation. Like sunflower, its major host, the causal fungus *Plasmopara halstedii* (Farl.) Berl. and de Toni, is considered to be of North American, Western Hemisphere, origin. The disease was first described in the northeastern United States in 1882, and in 1892, it was found on *H. tuberosus* in Russia (Kolte 1985). As the sunflower expanded to other countries, the disease has followed it closely, especially after World War II. The fungus has been distributed by seed trade rapidly and is reported to occur in various sunflower-growing countries all over the world except in Australia and New Zealand, though downy mildew on *Arctotheca* and *Arctotis* in Australia and New Zealand has been attributed to *P. halstedii* (Constantinescu and Thines 2010). It takes first place on its economic importance for sunflower production in the United States, Canada, and European countries. Epidemic outbreak of the disease in 2007 and 2008 caused 85% losses in sunflower yield in Turkey (Göre 2009). The overall loss directly attributed to downy mildew was estimated to be a half million dollars in the Red River Valley in 1970. When long periods of precipitation and cool weather follow the planting, losses to the extent of 80% have been reported from major sunflower-producing areas of eastern Europe. In France, where the sunflower is grown continuously for 2 years, about 70%–80% of plants have been reported to show incidence of the disease. In certain fields, about 90% incidence of the disease has been reported from Yugoslavia. Systemic infections are most destructive, occasionally causing 50%–95% yield reduction (Rahim 2001). Yield losses may be due to the total loss of seedlings, resulting from damping-off of the seedlings induced by the disease. Yield losses may also become more noticeable and serious when large field areas such as low spots are affected. Yield losses are generally additive, the combination of plant mortality, lighter, and fewer seeds produced by surviving plants, and lower oil content.

Pathogen: *Plasmopara halstedii* (Farl.) Berl. and de Toni

Classification

Domain: Eukaryota
Kingdom: Chromista
Phylum: Oomycota
Class: Oomycetes
Order: Peronosporales
Family: Peronosporaceae
Genus: *Plasmopara*
Species: *halstedii*

The pathogen *P. halstedii* (synonym *P. helianthi*) is an obligate parasite conveniently used for a group of closely related pathogens that cause downy mildew on sunflowers and many other genera and species of the subfamilies Asteroidae and Cichorioideae of the family Compositae.

The sporangiophores are slender, monopodially branched at nearly right angles with three sterigmata at the very end bearing ovoid to ellipsoid zoosporangia singly at the tips of branches. It is interesting that entirely new types of sporangia are formed on sunflower roots differing from those produced on leaves. The sporangiophores emerge through stomata on leaves. The size of sporangia is variable as is the number of biflagellate zoospores released by one single sporangium. The zoosporangia germinate by the formation of biflagellate zoospores or by germ tubes. The sporangia germinate in 2% sucrose in tap or distilled water. The temperature range for the germination of zoosporangia is 5°C–28°C, with an optimum temperature range being 16°C–18°C. The zoosporangia formed at low temperature (8°C) may show low germination (1%–6%), whereas those formed at high temperature (27°C) are reported to show high germination (86%–95%). The vegetative thallus is composed of intercellular hyphae that produce globular haustoria that penetrate into the host cells allowing the obligate biotrophic fungus to absorb nutrients.

Sexual reproduction is by means of oogamy resulting in the formation of thick-walled oospores in the intercellular spaces of roots, stems, and seeds that act as surviving structures. The oospores are brown with a slightly paler wall and measure about 27–32 μm in diameter.

Physiological Races

The fungus completes the sexual cycle annually, affording maximum opportunity for the recombination of virulence genes and the development of new races, which is evident from reports of work done by several workers from different parts of the world. The identification and nomenclature of these races are based on the reaction of a set of differential lines (Sackston et al. 1990, Tourvieille de Labrouhe et al. 2000). Physiological races of *P. halstedii* were first reported by Zimmer (1974) and distribution of races appeared to be geographically separated. For example, in 1991, a total eight races of the fungus were reported. Races 1, 4, and 6 were confined in Europe and races 2, 3, and 4 in Asia. Race 5 was confined to greenhouse; race 7 was reported in Argentina, and race 8 was reported in North Dakota in the United States (Gulya et al. 1991). Currently worldwide, 36 races have been identified controlled by 15 dominant resistance genes, and such a set of 15 differential host lines is RHA 265, RHA 274, RHA 464, DM 2, PM 17, 803, HAR-4, HAR-5, HA 335, HA 337, RHA 340, HA 419, HA 428, HA 458, and TX 16; among them about 6–7 are the world's dominant races, the four races (DM 700, DM 710, DM 730, DM 770) being the prominent ones (Gulya 2007a, Gulya et al. 2011, Viranyi and Spring 2011). In the United States, 11 races have been identified (2000–2008), but no isolate of *P. halstedii* from that country could overcome the PI (6) gene (HA 335) since it was released in 1988 until 2009 when the first *hot* race (DM 734) attacking the PI (6) gene was identified, and in 2010, it was further detected to be prevalent in North Central Dakota and Minnesota in the United States. Four more *hot races* (DM 314, DM 704, DM 714, DM 774) that are able to overcome the PI (6) gene (HA 335, HA 336) and PI 7 gene (HA 337, HA 338, HA 339) have been identified to be prevalent mostly in North Dakota and also in Minnesota in the United States.

In France, race 100 was first identified in 1965 and was well controlled by two resistance-specific genes PI (1) and PI (2). Zimmer and Fick (1974) found that the gene PI (1) provides resistance against race 100 and PI (2) against races 100 and 300. These two genes controlled the downy mildew population in Europe until 1998 when new races emerged (710 and 703) in France (Tourviellie de Labrouhe et al. 2000, Delmotte et al. 2008, Jocic' et al. 2012). Later research showed that these races were introduced from the United States via infected seeds (Roeckel-Drevet et al. 2003). Since then, a monitoring network that includes breeders and extension partners has been conducted by the French Ministry of Agriculture allowing to follow the evolution of the pathogen. Thus, 15 races more could be identified, especially race DM 304 (the first race in France to overcome the PI (6) gene) since 2000 and in 8 years (2000–2008). Six more *hot races* (DM 307, DM 314, DM 334, DM 704, DM 707, DM 714) have been identified in France (Sakr et al. 2009, Sakr 2010, Tourvieille de Labrouhe et al. 2010, Ahmed et al. 2012).

In Bulgaria, during 1988–2000, over a period of 12 years, only two downy mildew races were known. Now there are five races 300, 330, 700, 721, and 731; race 700 is dominant in the largest area (Shindrova 2013). In Romania, for approximately 35 years, there existed only two races, but in the last decade, five races of the downy mildew pathogen have been reported (Teodorescu et al. 2013). Races 100, 300, 310, 330, 710, 703, 730, and 770 have been identified in Spain. Race 703 is of high virulence in the northeast, while Race 310 seems to occur over the south, the main sunflower-growing region of the country (Molinero-Ruiz et al. 2003). In Hungary, five races (100, 700, 730, 710, 330) are prevalent (Kinga et al. 2011). In Russia, seven races of the pathogen could be identified in the Krasnodar region of the Russian Federation, and it is determined that against a background of dominant race 330, races 710 and 730 are also economically significant. A conclusion has been made about the necessity of separate testing on resistance of sunflower to these races and extraction of a material with complex resistance to them (Antonova et al. 2010). In Italy, HA 335 containing the efficient genes for resistance to *P. halstedii* never shows any symptoms under varied favorable climatic conditions (Raranciuc and Pacureanu-Joita 2006). In Serbia, race 100 was the race until 1990. In 1991, the presence of race 730 was confirmed in that country (Lac'ok 2008). The most predominant single race in sunflower-growing Karnataka, Andhra Pradesh, and Maharashtra states of India appears to be race 100 (Kulkarni et al. 2009).

In the last decade, advanced tools of biotechnology have enabled discernment of intraspecific groups of *Plasmopara* on the molecular level and led to the shift from a morphological to a phylogenetic species concept (Spring and Thines 2004, Viranyi and Spring 2011). With molecular markers based on the partial sequence of the nuclear internal transcribed spacer (ITS) regions, Spring et al. (2006) and Thines et al. (2005) detected polymorphism between profiles of races 100, 310, and 330, as well as between groups of populations representing races 700, 701, 703, 710, and 730. Giresse et al. (2007) found high genetic variability between isolates from France and Russia using single nucleotide polymorphism (SNP) markers, whereas Sakr (2010) utilized expressed sequence tags (EST)-derived markers to determine the genetic relationship between races. Evidence for asexual genetic recombination in *P. halstedii* is also reported (Spring and Zipper 2006).

Disease Cycle and Epidemiology

Pathogen survives through oospores in the residue of the preceding sunflower crop in soil or through oospores on seeds from the systemically infected plants. Some oospores have been reported to remain dormant up to 14 years. Overwintering oospores in plant residues in soil or seed germinate mostly under wet conditions the following spring. Primary infection is effected during seed germination in the soil and the emergence of sunflower seedlings. It may be caused by fungus mycelium or oospores present on infected seeds, or by oospores present in infected soil into which healthy seeds are sown. Starting from a single oospore that germinates and gives rise to a single sporangium, zoospore differentiation and release follow. In the presence of free water, the zoospore swarms rapidly and, if a host tissue (root, root hair, stem, or less commonly leaf) is available, settles on an infection site where encystment and subsequent germination take place. Penetration of the host is direct through

the epidermis. Once established, the fungus grows intercellularly, and in a compatible host/pathogen combination, it starts with systemic colonization toward the plant apex. Systemic mycelium may be present in all plant tissues except meristems. When conditions are favorable, asexual sporulation takes place by means of sporangiophores arising primarily through stomata or other openings on the invaded tissue. Oospores are also produced in infected plant parts, primarily in roots and stem.

The number of diseased plants depend on the amount of inoculum on seeds and in soil. No matter if primary infection starts from seeds or soil, the course of disease development in infected plants is identical. The fungus develops in unison with the development of young plants. It penetrates the root, stem, and cotyledons and reaches the meristematic tissue at the top of young plants. The fungus develops inside the infected plants intercellularly, in all plant parts, invading the young tissues and depriving the infected plants of assimilates and water. This is why infected plants lag behind healthy ones in growth and development. This way of fungus expansion inside the plant tissues is called a systemic infection. It begins with the infection of the germ and ends with the infection of the head and seeds. The fungus penetrates all parts of the seed (husk, endosperm, and germ), which then produces a new infected seedling. In that way, conditions are created for the occurrence of the disease in the subsequent sunflower-growing season.

For the development of the downy mildew of sunflower, rain is the critical factor during the first fortnight of growth, because only then are the seedlings susceptible to systemic infection. The period of maximum susceptibility to systemic infection is as short as 5 days at 22°C–25°C under greenhouse conditions. Under field conditions where mean air temperature during emergence is 13.2°C, plants remain susceptible for at least 15 days, provided enough rain during this period becomes available to provide soil water for only a few hours (Raranciuc and Pacureanu-Joita 2006). The percentage of infected plants is increased with depth of sowing.

The age of the sunflower seedlings is also an important factor in the development of systemic symptoms of downy mildew. Susceptibility of the seedlings decreases as the age advances; 3-day-old seedlings are the most susceptible to systemic infection. Therefore, any environmental factor that favors rapid seedling development shortens the interval of maximum susceptibility. Although seedling development is directly proportional to soil temperature, the range of soil temperature that normally prevails during the spring planting season is not a factor-limiting infection by downy mildew, particularly in the Red River Valley area of the United States and Canada (Kolte 1985).

Spread of the disease in relation to soil type has been studied. The heavy clay soils and flat topography of the Red River Valley area result in poor drainage, which favors downy mildew. The spread of the disease under field conditions preferentially follows the line of slope. Besides, tillage and running water are likely to be important factors in the spread of the disease. Sunflower plants suffering from boron deficiency become more susceptible to downy mildew.

Disease Management

Host Plant Resistance

Host plant resistance using race-specific genes designated as Pl, of which 22 have been described, is the most effective (Gulya 2007). Genes that confer resistance to downy mildew are dominant and often form clusters (As-Sadi et al. 2012, Vincourt et al. 2012). A number of Pl genes have been reported (Pl (1) to PI (15), Plv, Plw, Plx-z, Mw, Mx, Plarg, Pl HA-R4), and the position of 11 genes has been determined on the SSR genetic map (Mulpuri et al. 2009, Jocić et al. 2012, Liu et al. 2012). Sunflower and *P. halstedii* have a typical *gene-for-gene relationship* for each virulence gene presented in the pathogen exists a corresponding resistance gene in the host plant. If the plant has an effective resistance gene that will counteract the virulence gene in the pathogen, the infection will be stopped near the penetration site expressing a hypersensitive reaction (HR) that is manifested as an extensive cell death in the infected tissue. The constant evolution of new physiological races, due to pathogenic variability and selection pressure resulting from the use of resistant hybrids and seed

treatment fungicides, continuously challenges breeders to identify and introduce new resistance genes or gene clusters. Wild sunflower species have been a plentiful source of genes for downy mildew resistance. Downy mildew can be controlled by single, race-specific major dominant genes. Multirace resistant germplasms from wild sunflower species have been developed. The multitude of genes from the wild species for downy mildew resistance is supported by the number of germplasm releases that incorporate protection against ever-evolving pathotypes of downy mildew that infect cultivated sunflower.

It is, however, not advisable to use only one resistance gene in developing new cultivars. Rather, several different resistance genes should be employed, either by growing different hybrids carrying the different resistance genes or by pyramiding such genes. This strategy may extend the life cycle of each gene by keeping the selection pressure less effective against all known races minimizing the development of a new race. For most of these resistance genes, sequence-specific markers have been developed, which facilitate their detection and make the selection process faster and more reliable. A considerable number of sunflower hybrids that are genetically resistant to downy mildew have been released for commercial cultivation from time to time (Shirshikar 2008). Some of these hybrids are Sungene-85, MSFH-47, Pro-009, Prosun-09, SH-416, DRSF-108, PCSH-243, PRO-011, SCH-35 or Maruti, NSH-23, Sunbred-2073, NSSH-303, K-678, and MISF-93. Most commercial hybrids marketed as *downy mildew resistant* become susceptible to the new races; a few hybrids, however, can be bred that show resistance to major prevailing races in a specified sunflower-growing region (Raranciuc and Pacureanu-Joita 2006, Seiler 2010).

By combining the parial resistance provided by minor genes with specific resistance genes, durable resistance could be achieved (Labrouhe et al. 2008, Tourvieille de Labrouhe et al. 2008, Vear et al. 2008) or by introducing genes from different clusters with different origins in a single genotype (Jocić et al. 2010). Defeated hypostatic genes may be resistant to such new races. Hence, the combination of these defeated genes with novel genes, to which the pathogen has not been exposed, will extend the useful life of the defeated genes and will provide more durable resistance (Lawson et al. 1998). A great number of researchers have contributed to better understanding of the mechanisms involved in downy mildew resistance. The developments in biotechnology resulted in molecular markers for detecting PI genes and provided means for MAS. Candidate resistance genes have been proposed. For example, a marker derived from a bacterial artificial chromosome (BAC) clone has been found to be very tightly linked to the gene conferring resistance to race 300, and the corresponding BAC clone has been sequenced and annotated. It contains several putative genes including three toll-interleukin receptor–nucleotide-binding site–leucine-rich repeat (TIR–NBS–LRR) genes. However, only one TIR–NBS–LRR appeared to be expressed and thus constitutes a candidate gene for resistance to *P. halstedii* race 300 (Franchel et al. 2012). Resistance to *P. halstedii* can be of two types: Type I resistance can restrict the growth of the pathogen as in the case of" PI ARG gene "controlled by TIR–NBS–LRR genes and Type II resistance cannot restrict the growth of the pathogen, allowing the pathogen to invade, and subsequently an HR occurs as in the case of the "PI 14" gene controlled by coiled-coil CC–NBS–LRR genes (Radwan et al. 2011). MAS could be used for detecting not only major but also minor genes and would bring researchers a step closer to achieving sustainable resistance to downy mildew (Jocić et al. 2012).

Chemical Control

A wide range of commercial fungicides are available in the market with different modes of action for the management of downy mildew (Gisi 2002, Gisi and Sierotzki 2008). Coating of seeds with metalaxyl derivatives is most frequently used, as it provides protection at the time of primary infection, that is, at early stages of development of sunflower especially if a new pathogenic race occurs. Seed treatment with the aforementioned chemical at the rate of 3–6 g/kg seed is reported to give complete check of downy mildew of sunflower. In certain situations, the plants may remain completely protected throughout the growing period by following the aforementioned treatment. Strobilurins (especially trifloxystrobin) exhibit high activity against *P. halstedii* and is a promising group of fungicides

for controlling sunflower downy by seed treatment and foliar spray (Sudisha et al. 2010), though other fungicides are not considered effective for the control of the foliar portion of this disease and are not generally recommended. Concerning issues related to the use of chemical management include emergence of pathogen races resistant to fungicides as reported from several countries, notably from France, Germany, Turkey, and Hungary (Viranyi and Spring 2011); negative environmental effects of fungicides; and the economic feasibility of the disease management measures.

Seed treatment, combined with the use of a downy mildew–resistant hybrid or cultivar, offers the best promise for the management of the disease.

Induced Host Resistance

Besides the traditional management strategies, alternative or supplementary methods are reported to be effective in providing protection against sunflower downy mildew. One such possible solution is the use of systemic acquired resistance (SAR), that is, activation of the defense system of the plants. Commercially available immunoactivator Bion 50 W (benzo(1,2,3)-thiadiazole-7-carbothioic acid *S*-methyl ester) at 320 mg/L has been found to reduce the infection of sunflower by *P. halstedii* (Tosi et al. 1999, Korosi et al. 2009, 2011). Seed treatment with beta-aminobutyric acid (BABA) at the concentration of 50 mM also induces resistance to *P. halstedii* in sunflower (Nandeshkumar et al. 2009). Chitosan-induced resistance is also found to be effective against downy mildew in sunflower (Nandeshkumar et al. 2008). Induction of resistance by culture filtrate of *Trichoderma harzianum* against the disease is also reported (Nagaraju et al. 2012). This method of using specific chemical compounds for triggering plant defense mechanisms proves to be effective in diminishing the severity of infection of downy mildew in genotypes without genetic resistance (Gisi 2002, Ba'n et al. 2004).

Cultural Control

The emergence of pathogen strains resistant to chemicals and the occurrence of new races able to overcome specific resistances have led to include cultural practices for a more sustainable management of downy mildew. The choice of planting sites and optimum sowing time should be such that seedlings emerge rapidly and such that it reduces chances of free soil water during the period of susceptibility. For example, sunflower hybrid planting seed is almost exclusively produced in California. Due to the lack of summer rains and furrow irrigation, California-produced seed is relatively disease free, and thus, it regularly meets phytosanitary restrictions imposed by many countries (Gulya et al. 2012). Fields should be selected such that these are at least 500 m away from a field on which sunflower had been grown the previous year (Jocic' et al. 2012). Seed meant for sowing should be clean, and the seed should be obtained from a disease-free area; crop rotation is possible but not feasible, since the pathogen persists in soil and plant residues for 5 or more years, but proper crop rotation, that is, maintaining intervals of 4–5 years between two sunflower crops in the same field with other nonsusceptible crops, appears to be a quite desirable practice. The incidence of downy mildew increased from 42% in the second year to 100% in the fourth without crop rotation in Spain. While in plots where wheat and sunflower are grown alternately, the incidence has been reported to go only up to 15%–16%. Since wild and volunteer sunflowers and weed are hosts for the pathogen, eliminating these plants will help reduce overall inoculum built-up in the fields. Sowing should be performed at optimum time with avoidance of late planting (Covarelli and Tosi 2006, Jocic' et al. 2010).

Biological Control Hypovirulence in *P. halstedii*

Plasmopara halstedii virus (PhV) is an isometric virus found in the oomycete *P. halstedii* (Gulya et al. 1990a, 1992, Mayhew et al. 1992). The fully sequenced virus genome consists of two ss(+) RNA strands encoding for the virus polymerase and the coat protein (CP), respectively. Most of the field isolates of *P. halstedii* from different countries show morphologically and biochemically indistinguishable virions (Heller-Dohmen et al. 2008). The virions are isometric and measure 37 nm in diameter with one polypeptide of 36 kDa capsid protein and two segments of ssRNA (3.00 and 1.6 kb) that have been found to harbor PhV. The complete nucleotide sequence of PhV has been

established and it shows similarities to the *Sclerophthora macrospora* virus (SmV) and viruses within the *Tombusviridae* family as well as *Nodaviridae* (Heller-Dohmen et al. 2011). The presence of PhV leads to hypovirulence effects by weakening the aggressiveness of *P. halstedii* (Grasse et al. 2013). The PhV thus offers a great promise for obtaining a biological control of downy mildew disease of sunflower though practical utility of such an effect is yet to be investigated.

Regulatory Control

P. halstedii is listed as a plant quarantine pathogen (Ioos et al. 2012). Plants grown from infected seed, although they show no visible systemic symptoms, have been reported to produce infected seed and disseminate the pathogen. In addition, as seen earlier, there exists a physiological specialization within the *P. halstedii*, suggesting the importance of prevention of chance introduction of the more prevalent and widely virulent North American race of *P. halstedii* into areas where it does not occur. In Australia and perhaps in South Africa and in India, strict quarantine regulations have precluded its introduction.

ALTERNARIASTER BLIGHT

Symptoms

Symptoms of the disease are characterized by the development of dark brown to black, circular-to-oval spots, varying from 0.2 to 5.0 mm in diameter. The spots are surrounded by a necrotic chlorotic zone with a gray-white necrotic center marked with concentric rings (Figure 6.4). Initially the spots are small and they gradually increase in size, making their first appearance on the lower leaves. As the plant grows, the spots subsequently are developed on middle and upper leaves. At the later stages, elongated spots are formed on petioles, stem, and ray florets. Under high humidity conditions, the spots enlarge in size and coalesce resulting in blighting of leaves and sometimes rotting of flower heads.

Geographical Distribution and Losses

Alternariaster blight (formerly termed as *Alternaria* blight) of sunflower was first described in Uganda in 1943 and has since been recognized as a potentially destructive disease in most of the

FIGURE 6.4 *Alternaria* leaf spot of sunflower. (Courtesy of Dr. Tom Gulya, USDA-ARS, Northern Crops Research Lab, Fargo, ND.)

sunflower-growing areas of the world. It is reported from Argentina, Australia, Brazil, Bulgaria, India, Japan, Romania, Tanzania, Yugoslavia, South Africa, and the United States (Amabile et al. 2002, Calvet et al. 2005, Berglund 2007, Singh and Ferrin 2012).

In subtropical sunflower-growing areas, *Alternariaster* blight is considered as a major disease and can cause yield losses from 15% to 90% (Berglund 2007). The disease has been reported to reduce the seed and oil yields by 27%–80% and 17%–33%, respectively, in India. A negative correlation between increase in disease intensity (25%–96%) and yield components and oil content has been established (Kolte 1985, Chattopadhyay 1999). The most affected components due to the disease are the number of seeds per head, followed by the seed yield per plant. The disease also affects the quality of the sunflower seeds by adversely affecting the seed germination and vigor of the seedlings (Amaresh and Nargund 2004, Wagan et al. 2006). The loss in seed germination varies from 23% to 32% (Ahamad et al. 2000, Pandey and Saharan 2005). The nature of yield reduction is determined to some extent by the stage of plant growth when the disease epidemic develops. For example, the relationship between severity and yield in the R3 (second phase of inflorescence elongation) growth stage has proved that plants with disease severity higher than 10% show yield lower than 500 kg/ha regardless of the sowing dates. This value can therefore be used as a damage threshold for the disease (Leite et al. 2006).

Pathogen: *Alternariaster helianthi* (Hansf.) Simmons (= *Alternaria helianthi* (Hansf.) Tubaki and Nishihara)

Classification
Kingdom: Fungi
Division: Ascomycota
Class: Dothideomycetes
Order: Pleosporales
Family: Leptosphaeriaceae
Genus: *Alternariaster*
Species: *helianthi*

The pathogen has been first described as a member of the genus *Alternariaster* by Simmons (2007) and has been renamed *Alternariaster helianthi* (Hansford) Simmons (formerly *Alternaria helianthi* and *Helminthosporium helianthi*) as type and has hitherto been monotypic based on the absence of conspicuous internal pigmented, circumhilar ring found commonly in conidia and conidiophores of the *Alternaria* fungus. The phylogenetic analysis made by Alves et al. (2013) confirms the segregation of *Alternariaster* from *Alternaria* by showing that *Alternariaster* is a well-delimited taxon belonging to the Leptosphaeriaceae instead of the Pleosporaceae to which *Alternaria* belongs (Schoch et al. 2009). The mycelium is a septate, rarely branched, brown, and 2.5–5.0 μm in width. The conidiophores are hypophyllous, solitary or in small groups, straight to slightly sinuous, 100–225 × 7.5–10 μm, simple 3–6 septate, pale to chestnut brown, smooth, conidiogenous cells tretic, integrated, and terminal to intercalary and sympodial (Alves et al. 2013). The conidia are dry, solitary, and cylindrical to subcylindrical, occasionally with cells of different sizes, 60–115 × 11–29 μm, with rounded apex and base, transversally 5–9 septate (1–2 longitudinal or oblique septa), often deeply constricted at septa, eguttulate, subhyaline to pale brown, smooth, and thickened and darkened hilum (Figure 6.5). Germ tubes are oriented particularly to the main axis of the conidium and also polar (Alves et al. 2013). The conidia are not produced in chains, but 2–3 conidia in short chains may be observed in the culture as well as on the diseased host bits, on incubation in moist chambers. Genetic variability in isolates of *A. helianthi* has been assessed by random amplified polymorphic DNA (RAPD) analyses, which reveal the presence of six genetically distinct groups in India. The isolates Ah-1, Ah-7, and Ah-14 are reported to be genetically distinct (Prasad et al. 2009). In general, potato-dextrose agar (PDA) has been used for isolation by several workers, and it appears that the fungus produces very scanty mycelial growth and moderate to

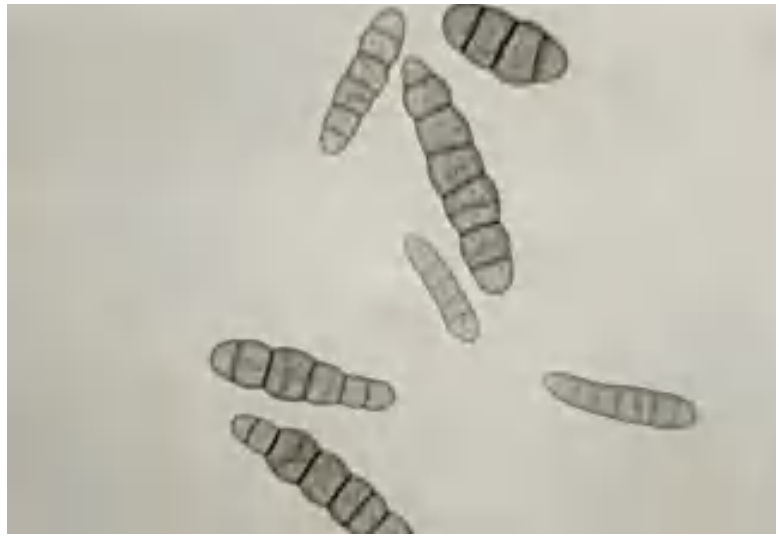

FIGURE 6.5 *A. helianthi* spores. (Courtesy of Dr. Tom Gulya, USDA-ARS, Northern Crops Research Lab, Fargo, ND.)

abundant sporulation on PDA. Comparatively good mycelial growth could be obtained on sunflower leaf extract agar medium (SLEAM) and Richards agar medium. Autoclaved carrot discs have been proved excellent for sporulation and luxuriant growth of *A. helianthi*. SLEAM with 2% sucrose and sterilized carrot disc supports maximum sporulation (Sujatha et al. 1997). On potato–carrot agar, the colony is raised centrally, with aerial mycelium felted, while having a wide periphery of flat sparse olivaceous buff to greenish glaucous mycelium with irregular margins (Alves et al. 2013). The estimated minimum temperatures for mycelial growth rate and for conidium germination are 5.5°C and 7.9°C, respectively, while the maximum temperatures are 32.9°C and 40.0°C, respectively (Leite and Amorim 2002). The optimum temperature for growth of the fungus in culture is 26°C, and it sporulates at the temperature range of 5°C and 35°C–40°C, with an optimum sporulation temperature of 20°C. Sporulation and mycelial development of the fungus occur best at pH 5.3–5.9 (Kolte 1985).

Epidemiology and Disease Cycle

The pathogen is seed borne and can, therefore, be introduced into new areas from infected seed (Salustiano et al. 2006, Micheli et al. 2007). Udayashankar et al. (2012) have developed the species-specific PCR-based diagnostic technique that provides a quick, simple, powerful, reliable alternative to conventional method in the detection and identification of *A. helianthi*. Locally, however, infested stubbles and crop debris left on the top of the soil from one growing season to the next is the most important source of inoculum from which primary infections are established. On such plants, the fungus overwinters as mycelium. Since sunflower can be grown throughout the year in all crop seasons, volunteer plants of infected sunflower or overwintering sunflower may also be an important primary source of inoculum. The exact process of penetration and infection at the host tissue level is through the cuticle and cell wall, and junctions between epidermal cells are the most frequent sites of appressoria formation (Romero and Subero 2003). The pathogen produces a specific toxin in culture and produces typical symptoms of the disease when inoculated on leaves (Kalamesh et al. 2012). The toxin inhibits seed germination as well as root and shoot growth under in vitro conditions (Madhavi et al. 2005a).

Relative lesion density and severity are influenced by temperature and leaf wetness duration. The disease appears to be more severe at a temperature of 25°C. The minimum temperature for disease

development, estimated by generalized beta function, is 13.0°C, and the maximum is 35.8°C. Relative lesion density increases with increasing periods of leaf wetness, as described by a logistic model (Leite and Amorim 2002). Positive significant correlation with relative humidity and a negative significant correlation with air temperatures and sunshine hours have been observed for disease development (Das et al. 1998, Amaresh and Nargund 2004). Thus, hot weather and frequent rain during milk and wax stages of sunflower plant development favor *Alternariaster* blight infection.

Disease Management

Host Plant Resistance

Attempts to identify the sources of resistance to the disease have been made by several workers (Madhavi et al. 2005b, Murthy et al. 2005, Gopalkrishnan et al. 2010). Wild species such as *H. tuberosus*, *H. occidentalis*, *H. resinosus*, and *H. argophyllus* are highly resistant to the disease and can be used in breeding for disease resistance (Madhavi et al. 2005b, Sujatha and Prabakaran, 2006, Prasad et al. 2009). The pathogen has been found to be restricted to epidermal cells in resistant wild sunflower as well as increase accumulation of phenols (Madhavi et al. 2005a). Sources of resistance to the disease have also been located in several germplasm accessions and hybrids. These are HPM-15R, HPM-116, and HPM-140 (Amaresh and Nargund 2000); LC-985, Performer, Select, Lc1029, and LC1093 (Raranciuc and Pacureanu 2002); 135, 1171, P-1019, 347, 446, 1039, 1210, and 1483 (Mesta et al. 2005); PEH-K04 hybrids 43, 50, 60, 77, 80, 81, 84, 92, and 98 (Nagaraju et al. 2005); RHA 587 and ARG × RHA 587 (Reddy et al. 2006); EC 68414 (Dawar and Jain 2010); sunflower hybrid parental lines CMS7-1A, DRS 9, DRS 63, and DRS 34; and four hybrids CMS7-1A × DRS 22, CMS7-1A × DRS 9, DCMS 15 × DRS 9, and DCMS 15 × DRS 63 (Sujatha et al. 2008). Higher peroxidase activities are recorded in sunflower genotypes with high threshold levels of resistance and lesser in susceptible genotype indicating strong evidence for the important role of peroxidase enzymes in the central defense system against necrotrophic pathogen *A. helianthi*, which could be used as a reliable biomarker for assessing resistance (Anjana et al. 2007, 2008). A number of sunflower genotypes are reported to possess partial resistance to the disease. The gametophytic selection combined with the conventional sporophytic selection can be considered as an effective tool in population improvement program to achieve a high level of resistance in a relatively short time (Chikkodi and Ravikumar 2000, Shobana Rani and Ravikumar 2006). Selection for resistant pollen on the stigmatic surface results in a corresponding increase in progeny resistance and successive pollen selection to further improve disease resistance of progeny. Repeated cycles of selection are required to achieve a useful level of resistance in sunflower, since resistance to *Alternariaster* in sunflower is polygenetically controlled (Chikkodi and Ravikumar 2000). Resistance to *Alternariaster* blight can be inducted or improved in the progenies derived through mutagenic treatment when seeds of sunflower genotypes are treated with 20 and 30 Kr of gamma rays (Oliveria et al. 2004, Patil and Ravikumar 2010, Shobharani and Ravikumar 2010). SAR in sunflower against *Alternariaster* blight can be inducted due to foliar application of salicylic acid at the concentration of 20 mM and Bion (acibenzolar at 0.05–5.0 mM). A lag period of 3–7 days is required for the induction of SAR (Ratnam et al. 2004a,b).

Chemical Control

Chemical management with protective (nonsystemic) fungicides such as iprodione, chlorothalonil, and mancozeb each at 0.2% spray as well as with therapeutic (systemic) fungicides such as hexaconazole, carbendazim, and propiconazole each at 0.1% spray has been found effective against the disease. But systemic fungicides are more effective than the nonsystemic ones (Amaresh and Nargund 2000, 2002, Amaresh et al. 2000, 2004, Singh 2000). Seed treatment with a mixture of carbendazim + iprodione in a 1:1 ratio at 0.3% followed by foliar spray; systemic fungicide hexaconazole (0.1%) gives best management of the disease with high yield (Rao et al. 2007, 2009). Combination of carbendazim + mancozeb in the ratio of 1:1 at 0.2% spray can be used most effectively in the

management of the disease avoiding the possibility of fungicide-resistant strains of the pathogen (Singh 2002, Mathivanan and Prabavathy 2007).

Cultural Control

Sanitation measures like selecting pathogen-free healthy seed and destruction of crop residues from previously affected crop help to reduce the initial inoculum intensity that can delay the onset of the disease by 11 days (Leite et al. 2005, Jurkovic et al. 2008). Occurrence and severity of the disease depend on the season and planting dates. This can be selectively used in disease management. For example, late August to mid-September planting of sunflower in most sunflower-growing states in India remains free from most major diseases with only traces of *Alternariaster* infection (Singh 2002, Amaresh et al. 2003, Mesta et al. 2009, Gadhave et al. 2011). Such a planting date is recommended for raising disease-free seed crop of sunflower.

Biological Control

Efficacy of *Pseudomonas fluorescens* as seed dresser can be enhanced by biopriming the sunflower seed for the effective and eco-friendly management of *Alternariaster* blight of sunflower (Rao et al. 2009). Antagonistic fungi *Gliocladium virens* (Anitha and Murugesan 2001) and *Trichoderma virens* (Mathivanam et al. 2000) are reported to be effective in managing the infection of *A. helianthi* in sunflower. Antibiosis is indicated as the mechanism of antagonistic effect of *G. virens* on the pathogen. Prior infection of sunflower plants with sunflower mosaic virus reduces the severity of *Alternariaster* blight of sunflower (Bhardwaj and Mohan 2005).

Sclerotinia WILT AND STEM ROT

Symptoms

Symptoms of the disease appear in three different phases on the sunflower—(a) basal stalk rot and wilt, (b) midstalk rot, and (c) head rot—and they are rather considered as three distinct diseases caused by the same pathogen.

Basal Stalk Rot and Wilt

Basal stalk rot and wilt are triggered through root infection from the fungus present in the soil and can appear in sunflower seedlings, but usually they appear during anthesis and seed development stages when the plants attain a height of about 5–6 ft (Figure 6.6). At first, wilted plants are scattered in the field, but later they are commonly found in series within rows. This disease usually appears in patches within the field. The incipient *Sclerotinia* wilt in sunflower is characterized by rotting through the taproot or through the hypocotyl axis (Darvishzadeh et al. 2012). Water-soaked lesions occur on the taproot at the soil line and on some fibrous root. If moisture conditions remain conducive, lesions on the stem below the soil level get covered with dense white growth of the fungus, which can be seen with loosely attached black sclerotial bodies that are irregular in size and shape. Similar bodies are found in stem and root piths, the occurrence of which is a reliable diagnostic feature of the disease.

Midstalk Rot

Plants infected later in the season may not wilt, and the only exterior symptoms may be a small brown lesion at the stem base or at any part of the stem, often in the upper half, which often results in breakage of the stem at the point of infection. Stems of severely diseased plants shred into vascular strands, becoming straw colored as they dry (Figure 6.7). Such stems are weak, and the plants lodge easily. Symptoms of stem rot in the upper half of the stem are usually due to airborne ascospores. Infection may start initially in leaf axils before progressing down the petiole to the stalk. The rotted part of the stem may or may not show the presence of sclerotia.

FIGURE 6.6 Basal stalk rot of sunflower caused by *S. sclerotiorum*. Note the fungal growth and sclerotia on the affected stem. (Courtesy of Dr. Tom Gulya, USDA-ARS, Northern Crops Research Lab, Fargo, ND.)

FIGURE 6.7 Midstalk rot of sunflower caused by *S. sclerotiorum*. (Courtesy of Dr. Tom Gulya, USDA-ARS, Northern Crops Research Lab, Fargo, ND.)

FIGURE 6.8 Head rot of sunflower caused by *S. sclerotiorum*. (Courtesy of Dr. Tom Gulya, USDA-ARS, Northern Crops Research Lab, Fargo, ND.)

Head Rot

Sometimes, the plants may remain healthy until the flower heads are produced. The flower heads, once formed, during the long period of their formation from budding to seed maturity stage, may be attacked by the fungus. The symptoms may become visible in any part of the receptacle. The affected flower portions may show the presence of a conspicuous white mycelial growth of the fungus, making evident the spread of the rot throughout the flower head. The head may ultimately be shredded resembling a broom-like appearance, and most of the tissue of the flower head is converted into a continuous mat of sclerotial tissue (Figure 6.8). Severely affected heads show incomplete filling of the head with seed. The head rot may be partial or complete. Seeds formed on partially affected heads may show the presence of sclerotia on their surfaces.

Geographical Distribution and Losses

Sclerotinia disease (Basal stalk rot/wilt/head rot) is one of the most damaging diseases of oilseed sunflower distributed all over the world in temperate regions and under cool tropical conditions, often at intermediate altitude. It has become economically important in all sunflower-growing areas in North American countries (Canada, the United States, and Mexico), East Europe, and other countries like Argentina (Fusari et al. 2012), Croatia (Ćosić and Postic 2008), Serbia (Maširević and Jasnic 2006b), Turkey (Tozlu and Demirci 2011), Egypt (El-Deeb et al. 2000), Iran (Bolton et al. 2006, Davar et al. 2010), Tanzania and South Africa (Anonymous 2010), and South Asia and China (Pandey and Saharan 2005). Yield loss depends on the sunflower development stage in which the disease occurs. If infection occurs in the early sunflower development stage, the yield loss will be approximately equal to the disease occurrence percentage. Disease occurrence percentage and yield losses caused by *Sclerotinia* wilt can even reach 100% because it causes whole plant devastation (Lamey et al. 2000, Saharan and Mehta 2008). Sunflower plants infected at the beginning of the flowering stage can lose up to 98% of their potential yield, while plants infected 8 weeks after

flowering can lose only 12% of their potential yield (Maširević and Gulya 1992). In the United States, annual losses on all crops caused by *Sclerotinia sclerotiorum* exceed $200 million, while in 1999, *Sclerotinia* head rot epidemic on sunflower caused crop loss valued at $100 million (Bolton et al. 2006). In Serbia, *Sclerotinia* wilt is the most common form of *Sclerotinia* disease and appears in sunflower crop more frequently than the other two forms. Its average frequency in Serbia is about 15%–20%, but in some years, the frequency can reach even around 50% (Tančić et al. 2011). Seed quality, as measured by test weight, oil, and protein content, is also adversely affected by the disease in partially infected plants at the near maturity stage of the crop with increase in shell percentage resulting in reduction in economic value (Eva and Andrej 2000, Zandoki and Turoczi 2004). The presence of sclerotia in seed can reduce the grade and market value of the crop. No toxins are produced by *Sclerotinia* in sunflower seed, but heavy contamination with sclerotia is considered unacceptable for human or animal consumption.

Pathogen(s): *Sclerotinia sclerotiorum* (Lib.) de Bary, *Sclerotinia trifoliorum* Fuckel, *Sclerotinia minor* Jagger

Classification
Kingdom: Fungi
Phylum: Ascomycotina
Class: Leotiomycetes
Subclass: Leotiomycetidae
Order: Helotiales
Family: Sclerotiniaceae
Genus: *Sclerotinia*
Species, *S. minor* Jagger, *S. sclerotiorum* (Lib.) de Bary

S. sclerotiorum was first recognized as a sunflower pathogen in 1861 in the Unites States (Kolte 1985). *S. minor* is another species reported from South America, Australia, Canada, and California (United States) causing root rot and wilt on sunflower, but is much less commonly found than *S. sclerotiorum*. *S. trifoliorum* has also been reported to be associated with the disease in Chile and Russia (formerly Soviet Union). They produce a fluffy white mycelium on and in infected plant parts. This mycelium aggregates itself into sclerotia, which are the structures that allow *Sclerotinia* species to survive in soil in the absence of a plant host. *S. minor* has uniformly round sclerotia measuring 0.5–2 mm, while those of *S. sclerotiorum* produce larger and irregular sclerotia, some measuring 1–5 cm. Sclerotia produced by *S. sclerotiorum* in heads are very similar in size and shape to sunflower seeds. Sclerotia exhibit either myceliogenic (eruptive) or carpogenic germination, the former giving rise to white vegetative hyphae that extend from sclerotia that have been stimulated to germinate by host plant exudates and the latter to apothecia as described in detail by several researchers (Bolton et al. 2006, Saharan and Mehta 2008). *S. minor* sclerotia rarely form apothecia, germinating instead by the direct emergence of hyphae (*myceliogenic* germination); *S. sclerotiorum usually* germinates carpogenically; and only occasionally, it germinates myceliogenically. At soil depths of up to 2 cm, apothecia can extend from the sclerotia of *S. sclerotiorum/S. trifoliorum* to reach the soil surface. A single sclerotium can produce as many as eight apothecia. Apothecia are tan to light brown, flesh-colored discs averaged about 2–8 mm in diameter and may be difficult to see. The asci are cylindrical hyaline and are produced in tightly packed masses at the upper surface of the apothecium. The asci measure 66–136 μm in length and 6–10 μm in width. The ascospores, only visible with a microscope, are monostichous, ellipsoid, one celled, thin walled, clear, or nonpigmented numbering eight per ascus and measuring 7.4–11 × 3.7–4.6 μm in size. The paraphyses are filiform. The ascospore morphology may be somewhat differing between *S. sclerotiorum* and *S. trifoliorum*.

Variability in the Pathogen

Isolates of *S. sclerotiorum* differ significantly in aggressiveness (Ekins et al. 2005, 2007, Zandoki et al. 2006), variation in oxalic acid production (Durman et al. 2005), and mycelial compatibility groups (MCGs) (Durman et al. 2003, 2005, Zandoki et al. 2006). Aggressiveness is positively correlated to colony radial growth, percent large sclerotia, and dry weight per sclerotium (Durman et al. 2003). However, there appears to be no correlation between genetic diversity among isolates and virulence differentiation (Li et al. 2005). The population structure of *S. sclerotiorum* on sunflower in Australia shows that the sclerotia, all eight ascospores within an ascus, are of only one genotype as revealed through multicopy restriction fragment length polymorphisms (RFLPs), MCGs, and RAPDs. Single and multicopy RFLP analyses have shown that majority of sunflower plants are infected by only one genotype (Ekins et al. 2011). Interestingly, isolates of *S. sclerotiorum* from the United Kingdom are reported to form a population that is significantly different from other populations (Li et al. 2009). Two very distinct sclerotia-producing strains of *S. sclerotiorum*, one as a normal strain (normal black sclerotia with white medulla) and the other as an aberrant strain (tan sclerotia with brown medulla), are known to be prevalent in Russia (formerly the Soviet Union) and Canada (Huang and Yeung 2002). The tan sclerotia produced by the aberrant strain have been found to have no dormancy and more than 85% of the sclerotia germinate myceliogenically on moist sand at 16°C–20°C with or without chilling treatment. Serotonin (5-hydroxytryptamine) is present in large amount in normal black sclerotia but absent or present in small quantity in abnormal sclerotia. Abnormal sclerotia instead contain a large amount of 5-hydroxyindole acetic acid (Kolte 1985, Huang and Yeung 2002).

Epidemiology and Disease Cycle

The pathogen is a facultative parasite and attacks over 400 plant species of 75 botanical families ensuring all time possibility of alternative sources of primary infection (Lazar et al. 2011). Sclerotia are the most important means of perennation. The survival time in soil is very variable, but 5–6 years is thought to be an upper limit. Survival of mycelium in seeds may also occur, but epidemiologically, it is of little consequence. Under most conditions, myceliogenic germination is of limited importance because only limited saprophytic spread occurs in natural nonsterile field soils. However, where sclerotia and susceptible plants are in close proximity, devastating stem base infections or root rot may result. Most sclerotial germination occurs at optimum of 24% soil moisture when the sclerotia are embedded at lower depths of soil up to 5 cm where the average temperature of soil (5–10 cm in depth) during the growing season of sunflower in rain-fed condition remains to be 30°C (Irany et al. 2001). A prerequisite for carpogenic germination is a period of chilling to break dormancy followed by rising temperatures and a high humidity. In temperate latitudes, apothecia typically mature during spring and early summer, although there are many reports relating to other seasons of the year. Conditions suitable for carpogenic germination of *S. minor* probably occur in southern regions in Australia, and carpogenic germination is probably a rare event in northern regions, and if it does occur, it probably does not coincide with first flower bud and anthesis stages in sunflower crops (Ekins et al. 2011). The apothecial stripes elongate in response to light and the ascospores are wind dispersed. Ascospores landing on potential hosts such as sunflower need water for germination, a requirement of 16–24 h being typical. Germination is possible throughout the range of 0°C–25°C, with an optimum at 15°C–20°C, and the pathogen is unable to cause infection at 30°C–35°C (Raj and Saharan 2001, Vuong et al. 2004). Continuous wetness on leaves within the canopy or on flowers on sunflower for a period of 42–72 h is needed for ascospore infection of the capitulum, and symptoms appear about 5 weeks later. This threshold can be used to define regions at risk. Head rot due to *S. sclerotiorum*, however, is best developed at 80% relative humidity for shorter periods of 16–24 h (Raj and Saharan 2000a). Disease appearance significantly vary depending on the quantity of rainfall, high crop density, sowing dates, temperature over the vegetation,

and selection of sunflower hybrid for sowing (Alexandrov and Angelova 2004, Simic et al. 2008). Also, it seems that an exogenous nutrient base is required for infection. Wounded, dead, or senescent tissues are readily colonized and serve as a food base from which infection of healthy tissues can take place. Ascospores are thought to be discharged along with mucilage that can cement the spores to host tissue, more particularly the senescent petals and other flower parts that provide a major avenue of infection from the site where the flower parts lodge, the sunflowers being most susceptible during the first flower bud stage coincidence of flowering, and ascospore release becomes a major factor of epidemiological significance in the occurrence of airborne infection causing stem or head rot (Raj and Saharan 2000b). Germinated ascospores produce appressoria that can vary from simple lobed forms to complex multibranched cushion-like structures. Entry is usually by direct penetration through the cuticle assisted by extensive endopolygalacturonase pectolytic and cellulolytic enzymes during the early phase of colonization causing dissolution of the host cell structure resulting in the development of stem or head rot symptoms (Cotton et al. 2002). *S. sclerotiorum* secretes several acid proteases, and one of the genes, acp1, encoding an acid protease, has been cloned and sequenced. The acp1 gene is expressed in plant infection, which is low at the beginning of infection but increases suddenly at the stage of necrosis spreading, suggesting thereby that glucose and nitrogen starvation together with acidification can be considered as key factors controlling *S. sclerotiorum* gene expression during pathogenesis (Poussereau et al. 2001a). Similarly, another gene asps encoding aspartyl protease is expressed in the beginning of infection of *S. sclerotiorum* in sunflower (Poussereau et al. 2001b).

The toxic metabolite, oxalic acid, produced by the fungus also plays an important role in the development of wilt symptoms. A positive correlation has been found between oxalic acid and shikimate dehydrogenase activity during the infection process caused by *S. sclerotiorum* in sunflower (Enferadi et al. 2011). Oxalic acid has been shown to move systemically in the plant and accumulate to critical level, and this elicits the wilt syndrome. Metabolic profiles of sunflower genotypes with contrasting response to *S. sclerotiorum* infection have been studied (Peluffo et al. 2010). There is induction of glycerol synthesis in *S. sclerotiorum* that exerts a positive effect on osmotic protection of fungal cells that favors fungal growth in plant tissues (Jobic et al. 2007). Monoculture with high level of N fertilization and irrigation exacerbate the disease (Gergely et al. 2002). Sclerotia from such affected sunflower plants are returned to the soil as the host decomposes, or they may be distributed by cultural operations, harvesting, etc. In most regions, the absence of a conidial stage and the environmental requirements for apothecium formation restrict *S. sclerotiorum* to a single annual infection cycle, and the disease is referred to as a single-cycle disease.

Disease Management

Host Plant Resistance

In Cultivated and Wild Helianthus *Species Germplasm*

There have been great efforts in searching for tolerance to midstalk rot in both cultivated sunflower and wild sunflower species through artificial inoculation methods (Castano et al. 2001, Becelaere and van Miller 2004, Vasić et al. 2004, Giussani et al. 2008). Screening parental inbred lines for resistance to *S. sclerotiorum* is an important step in developing sunflower hybrids with improved resistance to the disease (Hahn 2002, Huang 2002). A number of sunflower lines and hybrids with various levels of tolerance have been reported (Ronicke et al. 2004, Binsfeld et al. 2005, Castaño and Giussani 2006, Reimonte and Castano 2008), but complete resistance has not yet been observed. The level of tolerance is not yet considered adequate for the control of the disease, which is polygenic and under additive control, so that breeding programs have to combine favorable genes from different sources (Castaño et al. 2001, Becelaere and van Miller 2004, Davar et al. 2010). This confirms the need to consider different isolates in the stem rot resistance breeding programs (Darvishzadeh 2012), and selection for resistance to the disease could start at the inbred line development stage.

Sunflower is an unusual host in that it is prone to both head rot and stalk rot, and since resistance to each phase is independent, this doubles the breeding efforts (Vear et al. 2007). The most resistant breeding lines and commercial hybrids exhibit as low as 10%–15% head rot or stalk rot compared to 90%–100% on susceptible material. Near immunity to stalk rot is observed in most perennial *Helianthus* species and less so in annual species (Gulya 2007b, Silva et al. 2007).

The genotypes that show broad partial resistance to the disease are restorer lines RHA 439 and RHA 440 and maintainer line HA 441 (Miller and Gulya 2006); inbred lines SWS-B-04 (Ronicke et al. 2005a,b), R-28 from *H. argophyllus* (Baldini et al. 2002, 2004, Verzea et al. 2004), HA 302 (Rodriguez et al. 2004), 765, KS 7 (Wang et al. 2010), and 3146 (Wang et al. 2010); maintainer lines HA 451 and HA 452 (Miller et al. 2006); restorer lines RHA 453, RHA 4555 (Miller et al. 2006), and TUB-5-3234 (Micic et al. 2005a); inbred line NDBLOS sel (Micic et al. 2004, 2005b); and two hybrids Pioneer 6480 and Pioneer 6479 (Mosa et al. 2000). Four sunflower hybrids have been developed possessing resistance to ascospore penetration and mycelia extension in the capitulum tissue and could, therefore, be recommended for cultivation in the province of Buenos Aires in Argentina without increasing the risk of *S. sclerotiorum* attack (Godoy et al. 2005).

Molecular Breeding and Transgenic Sunflower for Resistance to Sclerotinia *Diseases in Sunflower*

The SSR markers associated with partial resistance to different isolates could be used in pyramiding polygenes in sunflower disease breeding programs (Micic et al. 2005a,b, Darvishzadeh 2012). Utilization of molecular markers to aid breeders in selecting genotypes with desirable traits through MAS has proved to be very effective. For example, in numerous studies, DNA markers associated with different traits have been reported. Baldini et al. (2002, 2004) used single-marker regression and identified several amplified fragment length polymorphism (AFLP) and SSR markers associated with basal stem resistance to *S. sclerotiorum* in sunflower. Markers of introgressed zones of *H. argophyllus*, *H. debilis*, *H. praecox*, and *H. petiolaris* in the resistant lines are assumed to be good candidates to identify the segments carrying stalk rot–resistant quantitative trait loci (QTLs). The possibility of detecting *H. petiolaris* accessions with a high level of resistance to *S. sclerotiorum* than others is indicated (Caceres et al. 2006). Independent QTLs, other than that for stalk rot resistance, have been identified for head rot resistance (Ronicke et al. 2005a, Yue et al. 2008). However, the prospects of MAS for resistance to *S. sclerotiorum* are limited due to the complex genetic architecture of the trait. The MAS can be superior to classical phenotypic selection only with low marker costs and fast selection cycles (Micic et al. 2004). Attempts have been made to establish resistance against *S. sclerotiorum* by genetic engineering (Scleonge et al. 2000, Schnabl et al. 2002, Hu et al. 2003, Lu et al. 2003, Sawahel and Hagran 2006). These studies are based on a gene controlling the production of an enzyme oxalate oxidase (OXOX). Oxalate is a phytotoxin secreted by *S. sclerotiorum* (Vasic et al. 2002). It weakens the plant tissue and crops with natural resistance to *S. sclerotiorum* such as wheat, barley, maize, or rice, producing OXOX, which breaks down and detoxifies the phytotoxin produced by *S. sclerotiorum*. Contrary to such crops, sunflower has a very low OXOX activity. An OXOX gene from wheat has been isolated and inserted into sunflower plants via *Agrobacterium*-mediated transformation. The *Sclerotinia*-induced lesions in transgenic sunflower are found to be significantly smaller than those in the control leaves (Hu et al. 2003). Compared with the original line, this gene increased resistance, but in general, the level of resistance is not better than in lines obtained by conventional breeding. Therefore, it should be possible to combine the transgenic lines with natural resistance to provide a level of resistance higher than in the available commercial hybrids (Bazzalo et al. 2000). Transgenic sunflower plants constituently expressing OXOX gene exhibit enhanced resistance against the oxalic acid (OA) generating fungus *S. sclerotiorum* (Hu et al. 2003). It is, however, apprehended that OXOX transgene will more likely diffuse naturally after its escape from the host plants (Burke and Rieseberg 2003). Since OA plays a vital role in the establishment of pathogenicity, attempts made to degrade OA will enhance resistance against *S. sclerotiorum* by increasing the production of H_2O_2 mediated through oxidative

burst. Such genetically modified cultivars may become a major means of *Sclerotinia* stalk rot management in the future (Link and Johnson 2012). Accumulation of phenolic compounds, their deposition on cell walls and lignifications, is a well-characterized mechanism of disease resistance against *S. sclerotiorum* (Prats et al. 2003, Rodríguez et al. 2004). Conceivably, resistant plants also have higher associated levels of phenylalanine ammonia lyase (PAL), which facilitates the biosynthesis of important phenolic derivatives such as lignin, and shikimic acid and the related enzymatic activity of shikimic dehydrogenase (SKDH), which are useful in identifying a biochemical paradigm that provides a clear correlation to disease-resistant genotypes (Enferadi et al. 2011). Accumulation of scopoletin, one of the coumarins as phytoalexins, may well confer head rot resistance with minimal plant damage and might be one of the basis for resistance to *S. sclerotiorum* (Prats et al. 2006, 2007).

Chemical Control

Foliar infection from airborne ascospores and lack of genetic resistance to *Sclerotinia* head rot need to identify foliar fungicide applications to reduce the impact on sunflower yield and quality. Systemic (azoxystrobin, benomyl, topsin, boscalid, and penthiopyrad) and protectant (iprodione, procymidone, vinclozolin, and fluazinam) fungicides have been demonstrated to be successful and economical if properly timed to manage *Sclerotinia* diseases of sunflower particularly the head rot disease (Link and Johnson 2012). Results suggest that plant coverage rather than systemic movement of the chemical is important for good management. Fungicides, applied as protectants before infection, especially during the bloom period, are effective in inhibiting infection by ascospores in fields with a history of infestation with *S. sclerotiorum* (Rashid 2011). The number of fungicide applications required for disease management depends on the length of the crop season duration of the cultivar or hybrid and the period of time that *weak* tissues (flower petals) are available for colonization by ascospores. If only one application is made, the early application is more effective than the late application. Better results are obtained from a two-application system, one at flowering and another 15 days later (Dietz 2011). In order to be effective, it is necessary that fungicides penetrate deep into the canopy to adequately cover the flowers and the places on the plant where the senescing petals might adhere or become lodged. Among the previously mentioned fungicides, penthiopyrad (a new Group 7 active ingredient) has preventive, residual, and postinfection activity. The strength of this group of fungicide is coupled with the activity that is both translaminar and locally systemic. Penthiopyrad goes through the plant tissue to attack fungal pathogen. It penetrates internally from the upper sprayed leaf surface to the lower unsprayed surface and provides an extended period of control of *Sclerotinia* infection.

Cultural Control

Well-drained sunny field sites away from the previous year's infested plot should be preferred for sowing. Certified seeds should be used to ensure the purity of the seeds without any contamination of sclerotia. The type of tillage operations may affect disease incidence. There is evidence that minimum or reduced tillage that maintains sclerotia on or near the soil surface may promote microbial degradation of sclerotia, whereas deep burial of sclerotia promotes their survival. The number of apothecia, however, may be reduced by tillage practices that bury the sclerotia deep in the soil, such as with a moldboard plow. If sclerotia are buried by deep tillage, use shallow tillage in subsequent years to avoid bringing the sclerotia back near the soil surface. Tillage operations also redistribute sclerotia throughout the soil and can actually increase disease incidence by creating a more uniform distribution of sclerotia within a field (Nelson and Lamey 2000). Crop rotation with nonhost crops such as wheat, barley, beets, and flax reduces the number of sclerotia in the soil by loss of viability over time. In addition, sclerotia may germinate in the absence of a host crop, but without subsequent host infection, new sclerotia are not returned to the soil and numbers are gradually reduced. Crop rotation is most effective when initiated before the fungus becomes a serious problem in a field. If numbers of sclerotia in a field are low, rotations of 3–5 years with a nonhost crop may be sufficient (Rashid 2003). Once the pathogen is well established in a field, and the soil is highly infested with

sclerotia, crop rotation may be of less value because of the long survival time of these propagules. When a crop is irrigated, the goal is to manage irrigation events to reduce the frequencies of 12–24 h periods of leaf wetness, especially during the bloom period, when flower petals can become colonized by the ascospores of *S. sclerotiorum*. To reduce disease due to *S. minor*, hyphal germination of sclerotia can be reduced by allowing the soil surface to dry thoroughly between irrigation events. Each irrigation event must therefore provide sufficient water to allow for a prolonged dry period. Cropping practices that reduce the intensity and duration of a disease-favorable microclimate within the canopy can lessen the disease's severity. Factors that may influence the microclimate include row spacing and orientation, nitrogen fertilizers, and cultivar selection. Studies on row spacing in sunflower crops consistently show that *Sclerotinia* wilt/basal rot incidence is lower in crops with wide row widths than those planted in narrow rows. Consequently, the management goal is to space rows at the distance that will maintain plant densities for maximum yield while providing for adequate room to facilitate air movement to reduce high-moisture microclimates within the canopy. Because infection by ascospores of *S. sclerotiorum* and *S. trifoliorum* requires an extended period of free moisture, orienting rows parallel to the direction of the prevailing winds also may be of some value in quickly drying the canopy after a rain or irrigation event. In addition, to avoid dense crop canopies, applied nitrogen should not exceed the optimal rate for a particular crop. Lastly, when choices are available, cultivars that mature early and have a more upright, as opposed to a vining (prostrate), growth habit can provide *avoidance* or *escape* resistance, generally resulting in less disease (Rashid 2003, Turkington et al. 2011, Link and Johnson 2012). Deep burial of sclerotia prevents them from producing apothecia. One must avoid bringing these buried sclerotia to the surface in following seasons. Once they return to the soil surface and are still viable, they can again cause disease. Selected nonhost crops in rotation with maize will reduce inoculum.

Biological Control

Sclerotia of *S. sclerotiorum* are subject to attack by soil microorganisms such as *Coniothyrium minitans*, *Talaromyces flavus* (teleomorph of *Penicillium vermiculatum*), *Sporidesmium sclerotivorum*, *Trichoderma viride* (Ashofteh et al. 2009, Link and Johnson 2012, Tozlu and Demirci 2011), *T. harzianum* (Singh et al. 2004), *Bacillus* sp. (Yu et al. 2006), *P. fluorescens* (Behboudi et al. 2005), and certain isolates of *Actinomycetes* (Baniasadi et al. 2009). Among these antagonists, only *Coniothyrium minitans* and *Trichoderma* spp. have been practically used for biological control of the sunflower wilt caused by *S. sclerotiorum*. In the fields effectively, it appears that secretion of β-1, 3-glucanase from *C. minitans* degrades and lyses sclerotial tissues. *C. minitans* will produce hundreds of pycnidia on the surface of a colonized sclerotium giving it the aspect of a spiny, irregular surface. Usually, few hyphal threads will grow out of an infected sclerotium. This mycoparasite will spread as conidia in the soil. *C. minitans* has a good saprophytic ability and can grow on plant residues or be easily cultured on artificial media. *C. minitans* has been released as a commercial product for suppression of the wilt phase of the disease. In practice, dried spores of this antagonist are sprayed either onto pathogen-infested crop debris at the end of a season or onto the soil surface before planting, and the disease control is economical. Use of micronutrient zinc solely or in combination with molybdenum improves the biocontrol activity of *P. fluorescens* strain UPPF 61 (Ashofteh et al. 2009, Heidari-Tajabadi et al. 2011). Another biocontrol agent (Agate-25K) based on *Pseudomonas chlororaphis* is reported to be effective in the control of *Sclerotinia* disease of sunflower in Russia (Vinokurova 2000). The head rot phase of the disease has been successfully controlled by field testing of honeybee (*Apis mellifera*)–dispersed *Trichoderma* formulation (a mixture of six isolates of *Trichoderma* including *T. koningii*, *T. aureoviride*, and *T. longibrachiatum*) containing *Trichoderma* conidia and viable hyphal fragments, industrial talc, and milled corn kernels in Argentina (Escande et al. 2002). An isolate of *Epicoccum purpurascens* (*E. nigrum*) well adapted to the fluctuating conditions typical of natural environments could contribute to achieving an acceptable level of control of head rot (Pieckenstain et al. 2001). Interestingly under Argentina conditions, the microorganisms, particularly the fungal flora that colonize florets of *Sclerotinia*-tolerant

sunflower varieties, play a part in an indirect mechanism that protects flowers from ascospore germination and pathogen growth (Rodriguez et al. 2001). Spontaneously occurring hypovirulence in the tan sclerotial isolate S10 of *S. sclerotiorum* from sunflower in Manitoba, Canada, has been characterized, and the preliminary in vitro transmission test indicated that the hypovirulence in the hypovirulent isolate is transmissible, but double-stranded ribonucleic acids (dsRNAs) have not been detected in hypovirulent and virulent isolates derived from S10. The existence of dsRNA-free hypovirulence in S10 progenies suggests that another hypovirulence mechanism may exist in *S. sclerotiorum* (Li et al. 2003). There is, however, a great potential of making use of this typical phenomenon of hypovirulence in the biological control of *Sclerotinia* diseases of sunflower.

Antifungal protein, trypsin inhibitor (serin proteinases), is a potent antifungal compound associated with sunflower seeds, can completely inhibit the germination of *S. sclerotiorum* ascospores at a concentration of 14 μm/mL indicating the possibility of its use in disease management (Mendieta et al. 2004).

CHARCOAL ROT

Symptoms

The most obvious and common symptom of the disease, under field conditions, is the sudden wilting of plants, which usually appears after pollination, though such plants may have become infected very early in the season (Figure 6.9). Symptoms first observed in plants approaching physiological maturity consist of silvery gray lesions girdling the stem at the soil line, reduced head diameter, and premature plant death (Gulya et al. 2010, Mahmoud and Budak 2011). Pith in the lower stem is completely absent or compressed into horizontal layers. Black spherical microsclerotia are observed in the pith area of the lower stem, underneath the epidermis, and on the exterior of the taproot (Figure 6.10). The pathogen generally affects the fibrovascular system of the roots and basal internodes and impedes the transport of nutrients and water to the upper parts of plants. Progressive wilting, premature aging, loss of vigor, and reduced yield are characteristic features of *M. phaseolina* infection. The internal stem shows a shredded appearance. Later, the vascular bundles become covered with small black flecks or microsclerotia of the fungus.

FIGURE 6.9 Charcoal rot–affected sunflower plants under field conditions. (Courtesy of Dr. Chander Rao and Dr. Varaprasad, DOR, Hyderabad, India.)

FIGURE 6.10 Charcoal rot–affected root of sunflower (left) in comparison to healthy root (right). (Courtesy of Dr. Chander Rao and Dr. Varaprasad, DOR, Hyderabad, India.)

Geographical Distribution and Losses

The charcoal rot of sunflower is widely distributed throughout tropical, subtropical, and warm temperate regions. It is widespread throughout Latin America, Eastern and Southern Africa, Egypt, West Asia, Middle East including Iran and Turkey (Habib et al. 2007, Mahmoud and Budak 2011, Ijaj et al. 2012), and South Asia, more particularly in Pakistan (Khan 2007). With change in climate, the diseases are also reported to occur in the otherwise relatively cooler regions of the United States (Gulya et al. 2002, Ullah et al. 2011, Weems et al. 2011) and Europe (Sarova et al. 2003, Bokor 2007, Veverka et al. 2008, Csondes et al. 2012). Crop loss estimates are available to the extent of 64% in the Krasnodar region of Russia, 46% in India (Kolte 1985), and 90% in Pakistan (Khan 2007). Under favorable conditions, total failure of the crop in specific areas has been recorded (Khan 2007, Ijaz et al. 2013). The overall yield losses in all varieties at flowering, ripening, and sowing stages are reported to be in the range of 7%–45%, 6%–41%, and 5%–37%, respectively, in Pakistan (Wagan et al. 2004). It is thus evident that continuous increasing trend of charcoal rot is alarming for farmers and authorities in sunflower business not only in Pakistan (Khan et al. 2003) but also in neighboring Iran (Rafiei et al. 2013). Significant decrease in yield is reported with increasing population density of the pathogen. Decrease in seed yield is reported to be 41%, 62%, and 79% at low, moderate, and high pathogen densities, respectively, in Pakistan (Khan et al. 2005a). *M. phaseolina* grows well on sunflower seeds and has been shown to increase the content of oil and free fatty acids in the seeds, as well as discoloration of the oil.

Pathogen: *Macrophomina phaseolina* (Tassi) Goid

Classification
Kingdom: Fungi
Phylum: Ascomycota
Class, Dothideomycetes
Subclass: Incertae sedis
Order: Botryosphaeriales

Family: Botryosphaeriaceae
Genus: *Macrophomina*
Species: *phaseolina* (Tassi) Goid

The details of morphology and culture characteristics of the pathogen are described in Chapter 2 under peanut diseases. High levels of pathogenic variability and genetic diversity have been observed between *M. phaseolina* isolates from different geographical origins or even different hosts after characterization with different markers (RAPDs, RFLPs, and AFLPs) (Almeida et al. 2003, Tancic et al. 2012), although isolates from the same species and same location had related pathotypes. Chlorate-sensitive and chlorate-resistant types of isolates of *M. phaseolina* are known (Mohmmad et al. 2001, Aboshosha et al. 2007), and variation among isolates in pathogenicity is evident (Khan et al. 2005b, Csondes et al. 2010).

Genetic diversity of *M. phaseolina* from Hungary indicates the coexistence of different haplotypes in such country. There appears to be a geographical dominance of a given haplotype and closer genetic relationship might exist between spatially distinct haplotypes (Csondes et al. 2012). Significant pathogenic and genetic variability has been observed within the Iranian isolates obtained from sunflower (Rayatpanah et al. 2012a,b).

Epidemiology and Disease Cycle

In addition to its survival through sclerotia in soil or in the form of sclerotia carried in crop residues, it is also reported to be seed borne in sunflowers (Csondes 2011). However, the pathogen is less aggressive in the preemergence phase, but more serious in the postemergence phase and later stages of growth (Arafa et al. 2000).

M. phaseolina forms appressoria on the epidermis of sunflower. They may aid in both mechanical and chemical penetration, which is direct. Penetration of the adult stem is by mass action of hyphae, which is intra- and intercellular. Initially, infection is restricted to the root epidermal cells and cortical cells, cell configuration including organelles become distorted, and intercellular spaces are occupied by the hyphae, which appear amorphous, with intracellular invasion occurring later (Naz and Ashraf 2006). The incubation period appears to be 6–10 days in plants inoculated with sclerotial suspension and 3–5 days with pycnidiospores.

The role played by various pectolytic and some cellulolytic enzymes in the infection of sunflower plants by the fungus has been well studied. The possibility of production of a non-host-specific toxin by *M. phaseolina* is indicated. The development of necrotic spots on leaves due to *M. phaseolina* is attributed to this toxin. It is reported that the pathogen does not grow beyond the necrotic regions on the inoculated leaves, and the virulence of the different isolates has not been found to be correlated with toxin production in culture. Sunflower plants are most susceptible to charcoal rot at reproductive stage (Suriachandraselvan and Seetharaman 2003). Infection of sunflower seed by *M. phaseolina* takes place when anthesis in the outer quarter of the inflorescence radius is complete. The infection progresses during the seed development stages before the seed reaches maturity. Maximum seed infection occurs when the seed in the outer quarter remains soft. The incidence of the disease increases with increasing salinity level of irrigation water (Fayadh et al. 2011). Moisture stress and higher temperature and periods of drought also favor the development of the disease (Alexandrov and Koteva 2001). At lower temperature (20°C–25°C), seedling mortality due to the disease varies in the range of 8%–67%, whereas at 30°C–35°C, the mortality rate varies from 75% to 100% (Kolte 1985). Disease incidence increases with increase in plant density and sclerotial population in soil (Perez et al. 2002).

Disease Management

Host Plant Resistance

Little is known about the relative resistance of most sunflower varieties. It is demonstrated that sunflower varieties respond differently to artificial as well as to natural infections in the field. This indicates the possibility of control of charcoal rot by breeding for resistance. A number of sunflower genotypes,

SF-87, PTH-1, and SMT (Hafeez and Ahmad 2001), A-43, G-100, G-133, G-17, G-33, G-29, G-10, and G-78 (Khan et al. 2010), CMS 19× R 43, B line 1052/1, and CMS 350/1× R 43 (Dalili et al. 2009), and Giza 102 (Aboshosha et al. 2008, El-Hai et al. 2009), have been found to be resistant in various degrees to charcoal disease, and sunflower genotypes RF81-74*AF80-460/2/1, RF81-1/2*AF81-112, RF81-1/2*AF80-452/2/2, RF81-06/1*AF80-448/1/2, HYSUN33, and AZARGOL show significantly low incidence of charcoal rot (0.33%) under sick soil conditions in Iran (Rafiei et al. 2013). Protein analysis, peroxidase activity, and peroxidase isozyme pattern derived from the sunflower cultivar Giza 102 can be used as genetic markers for host resistance studies in sunflower to *M. phaseolina* (Aboshosha et al. 2008). Sunflower plants regenerated from tolerant callus from hypocotyl explants from a tissue culture medium exhibit more resistance against *M. phaseolina* (Ramadan et al. 2011).

Chemical Control

Seed treatment with carbendazim, thophanate methyl, and thiabendazole each at 2.5 g/kg of seed has been reported to be effective to manage sunflower crops from the seed-borne infection and increasing seed germination (Bhutta et al. 2001). Similarly, fenpropimorph (Corbel) is effective in managing the *M. phaseolina* infection as seed treatment (Piven' et al. 2002, 2004). Other measures of chemical control as used for charcoal rot of peanut may be useful for sunflower crop also, and phosphorus as calcium superphosphate has been reported to be effective in decreasing the incidence of the disease. Maximum control of *M. phaseolina* infection has been obtained when sunflower seeds are coated with Na alginate in combination with Ca carbonate and gum arabic followed by Na alginate in combination with Ca carbonate and carboxymethyl cellulose (Muhammad and Shanaz 2012). Spermine (SP) as seed soaking and/or foliar application of K and/or Zn is helpful in reducing the harmful effects of charcoal of sunflower (El-Metwally and Sakr 2010). Seed soaking or foliar spray of antioxidants (citric acid and salicylic acid at 10 mM) and micronutrients (manganese and zinc at 2 g/L) significantly reduces the incidence of charcoal rot (El-Hai et al. 2009).

Cultural Control

The use of clean seed, the application of organic matter, balanced NPK fertilizer application, long rotations with nonhost crops, avoidance of excessively dense plant populations, and sanitation including the burial of debris by hand or by plough have been suggested as cultural practices against charcoal rot (Aleksandrov 2000, Bistrichanov et al. 2000, Alexandrov and Koteva 2001). Soil amended with nursery fertilizers (urea, diammonium phosphate (DAP), and frutan at 0.1%) in combination with seed treatment with gamma rays (60 cobalt) emitting gamma rays for 2 min results in reduced charcoal rot infection (Naheed et al. 2011). There is a possibility of exploiting the allelopathic effect of *Chenopodium* species (*C. album*, *C. murale*, *C. ambro*) against *M. phaseolina* infecting sunflower (Muhammad and Javaid 2007).

Biological Control

Seed coating with antagonist *Trichoderma reesei* and cotton cake or with *T. harzianum* and mustard cake has proved to be effective in protecting sunflower plants from charcoal rot (Muhammad et al. 2010, Muhammad and Zaki 2010, Ullah et al. 2010). Application of *T. viride* (4 g/kg of seed), + 10 kg/ha soil + FYM (12 tons/ha), and neem cake has also been found effective in reducing charcoal rot incidence (Mani and Hepziba 2003, Mani et al. 2005, Sudha and Prabhu 2008, Suthinraj et al. 2008). The highest disease suppression (61%) has been reported with rice straw composted with cow manure and inoculation with *T. harzianum*. Amendment of compost with *T. harzianum* accelerates composting and can improve disease suppression effect (Morsy and El-Korany 2007). A urea, *Rhizobium*, and *T. harzianum* combination gives better biocontrol effect against the pathogen (Siddiqui et al. 2000). Combined use of *Pseudomonas aeruginosa* with sea weeds significantly decreases the infection caused by *M. phaseolina* (Shahnaz et al. 2007). VAM fungus *Scutellospora auriglobosa* is consistently associated with sunflower variety Helico 250 under Pakistan conditions, and this VAM fungus is found to increase the growth of sunflower with reduction in the incidence of charcoal rot (Jalaluddin et al. 2008).

Effect of Plant Extracts

Efficacy of some plant extracts (*Eucalyptus camaldulensis*, *Azadrichta indica*, *Allium sativum*, and *Datura alba*) and plant products against *M. phaseolina* has been experimentally demonstrated (Arshad et al. 2008, Ullah et al. 2007).

STEM NECROSIS DISEASE

Symptoms

The disease is observed at all growth stages starting from seedlings to mature plant. The characteristic field symptoms of the disease include mosaic on leaves that leads to extensive necrosis of leaf lamina, petiole, stem, and floral calyx and complete death of seedlings eventually (Figure 6.11). Early infection either kills the plant or causes severe stunting with malformed head filled with chaffy seeds (Ravi et al. 2001). Necrosis at the bud formation stage makes the capitulum bend and twist resulting into complete failure of seed setting and maturation (Figure 6.12).

Geographical Distribution and Losses

Sunflower necrosis disease (SND) is becoming a potential threat to sunflower cultivation in the Indian subcontinent. The disease was first recorded in parts of Karnataka state in 1997. Since then, the disease has become increasingly important in Andhra Pradesh, Karnataka, Maharashtra, and Tamil Nadu, the four major sunflower-growing states of India, and is a limiting factor in sunflower production; up to 80% of the plants of some open-pollinated varieties and hybrids were affected during the 1999 survey in sunflower-growing areas, and yield losses ranging from 30% to 100% have been reported (Shirshikar 2010). Early-infected plants remain stunted and develop malformed heads with poor or no seed setting, resulting in complete loss of the crop (Papaiah Sardaru et al. 2013). There has been a continuous threat to sunflower production in India due to tobacco streak virus (TSV) epidemics and reduction of over 40% in the yield since 1997, amounting to annual loss of Rs. 76 crores (Jain et al. 2003). The disease is also reported to occur in Australia and the Netherlands (Sharman et al. 2008) and in Iran (Hosseini et al. 2010, 2012).

FIGURE 6.11 Stem necrosis of sunflower caused by the sunflower stem necrosis virus. (Courtesy of Dr. R.K. Jain, IARI, New Delhi, India.)

Pathogen

The disease is caused by a strain related to TSV infecting sunflower: TSV-SF (genus, *Ilarvirus*; family, Bromoviridae). In electron microscopy, *Ilarvirus*-like particles can be detected in crude sap of SND-affected sunflower and *Chenopodium quinoa* plants inoculated with leaf extracts prepared from SND-affected sunflower plants. In addition to several other herbaceous virus indicator plants, groundnut, cowpea, and cotton, which are significant crops in India, become infected. Back transmission to healthy sunflower seedlings with leaf extracts of systemically infected indicator plants results in identical symptoms of SND, hence confirming the ilar-like virus as the causative agent of SND (Ravi et al. 2001, Prasada Rao et al. 2009). Thus, all the experimental data unequivocally prove that the virus causing SND in many sunflower varieties and in many different growing regions in India is a strain of TSV. The association of a tospovirus, antigenically related to groundnut bud necrosis (GBNV) and watermelon silver mottle (WSMV) viruses, with the disease has been reported earlier (Jain at al. 2000, Venkata Subbiah et al. 2000). TSV first described by Johnson (1936) is the type species of the genus *Ilarvirus*, of the family Bromoviridae that includes viruses having tripartite quasi isometric particles of size 27–35 nm. The virus has three nucleoprotein particles designated as RNA-l (3.4 kb), RNA-2 (3.1 kb), and RNA-3 (2.2 kb). RNAs 1–3 are genomic and encodes proteins la (119 kDa), 2a (91 kDa), and 3a (32 kDa), respectively, whereas RNA-4a (0.9 kb) and RNA-4 (1.0 kb) are subgenomic expressed from RNA-2 and RNA-3. RNA 4a encodes 2b (22 kDa) and CPs (28 kDa), respectively. The TSV genome is infectious only in the presence of its CP or RNA-4. None of the SND causing TSV-SF full genomes could be sequenced, but many researchers have sequenced and reported full-length RNA3 that hosts the movement protein and CP gene (Bag et al. 2008).

Transmission

Mechanical/Sap Transmission

The virus can be transmitted by mechanical or sap inoculation from sunflower to sunflower. Sap extracted in 0.05 M phosphate buffer with 0.075 thioglycerol as inhibitor is more efficient in transmitting the virus (Lokesh et al. 2008b, Pankaja et al. 2011). In general, *Ilarvirus* has a wide host range as they are efficiently sap transmissible to many of the host plants belonging to Amaranthaceae, Chenopodiaceae, and Fabaceae. A rapid and efficient sap inoculation method for tobacco streak virus (TSV-SF) has been developed for screening a large number of sunflower genotypes for resistance to the disease (Sundaresha et al. 2012).

Vector Transmission

The major mode of transmission of TSV-SF is by infected pollen, which can spread by wind or carried by thrips, which transport infected pollen on their bodies (Chander Rao and Shanta Laxmi Prasad 2009). The virus–vector specificity relationship is yet to be established for this virus. Pollen and thrips collected from TSV-infected *Parthenium* weed released together show 58.3% and 70% disease incidence at vegetative and flowering stages of the sunflower crop. *Thrips palmi* successfully transmits the virus to sunflower test plants on acquisition access period (AAP) of 2–3 days and inoculation access period (IAP) of 3–5 days (Lokesh et al. 2008b). A single thrip has been found enough to acquire and transmit the virus from an infected to healthy sunflower plant, and it is revealed that the vector *T. palmi* could acquire the virus with an AAP of 3 days from the cotyledonary leaves of an infected sunflower plant, with a resultant 16.67% transmission. Similarly, an IAP of 6 days is necessary for successful transmission of the virus with 13.33% transmission (Pankaja et al. 2010b, 2011).

Seed Transmission

Certain strains of TSV are known to be transmitted in the seed of a range of host species (Prasada Rao et al. 2009). However, transmission of the TSV isolate occurring in India is not spread through the seed (Papaiah Sardaru et al. 2013, Prasada Rao et al. 2009, Bhat et al., 2002a

Pankaja et al. 2010a). Even in the absence of seed transmission, primary inocula of the TSV are provided by secondary hosts and weed hosts prevalent in and around the sunflower fields by the thrip vector.

Diagnosis

Polyclonal antiserum against TSV-SF has been developed for the rapid diagnosis of TSV using the direct antigen coating-enzyme linked immunosobent assay (DAC-ELISA) method (Ramiah et al. 2001a,b). The serological electroblot immunoassay diagnosis method for CP of the sunflower necrosis virus (Bhat et al. 2002a) and an efficient reverse transcription-polymerase chain reaction (Bhat et al. 2002b, Srinivasan and Mathivanan 2011a) have been developed. Recently, Sarovar et al. (2010a) have reported a high-efficiency immunocapture reverse transcription-polymerase chain reaction (IC-RT-PCR) for RNA3 of TSV-SF, and they have also developed a serological and probe-based blotting technique for the detection of TSV-infected sunflower plants (Pankaja et al. 2010a, Sarovar et al. 2010b). The serological relationship has been confirmed by Western blot analysis and immunoelectron microscopy (IEM) decoration assays using sunflower necrosis virus (SNV) and TSV antisera in reciprocal tests. In RT-PCR, using oligonucleotide primers deduced from conserved sequences within TSV RNA 3 and flanking the entire CP region, an approximately 1000 bp dsDNA fragment could be amplified from SNV-infected sunflowers. A sequence analysis of cloned sunflower necrosis virus (SNV) PCR fragments revealed nucleotide identities of approximately 90% with TSV RNA 3 and a CP amino acid homology between SNV and TSV of more than 90%.

Epidemiology, Host Range, and Disease Cycle

The virus survives throughout the year on several weeds, namely, *Parthenium hysterophorus*, *Tridax procumbens*, *Phyllanthus* sp., *Euphorbia geniculata*, and *Digera arvensis*. A total of 12 weeds, namely, *D. arvensis*, *A. aspera*, *Lagasca mollis*, *P. hysterophorus*, *A. hispidum*, *A. conyzoides*, *C. bengalensis*, *E. geniculata*, *Phyllanthus niruri*, *Malvastrum coromandelianum*, *Abutilon indicum*, and *Physalis minima*, have been found to be infected with the natural infection of TSV. Of these, *Parthenium* is the most widely distributed and is a symptomless carrier of TSV and produces several flushes of flowers during its life cycle ensuring continuous supply of TSV-infected pollen. It hosts the virus as well as thrips and produces copious pollen throughout the season and acts as a primary source of inocula initiating and sustaining the TSV infection during a crop season. Besides, thrips colonizing flowers of these plants can become externally contaminated with pollen and movement of these thrips to new hosts results in introduction of the virus into fields. Windblown pollen of *Parthenium* contaminates the leaves and thrips arriving independently may well contribute to infection. Epidemiological studies on SND indicated the positive correlation between the thrips population and the weather parameters, namely, maximum and minimum temperature and sunshine and dry spells, whereas negative correlation was observed with rainfall and relative humidity. Disease incidence is positively correlated with thrips population and minimum temperature, relative humidity, and rainfall. However, negative correlation with maximum temperature can be observed (Upendhar et al. 2006, 2009). The disease incidence is higher in kharif and summer seasons, whereas it is low in rabi season. The sunflower cultivars sown during July and August show high necrosis incidence compared to postrainy season, that is, September onward (Shishikar 2003).

Disease Management

Host Plant Resistance

The most economical and convenient way to manage TSV is to grow resistant varieties. So far, complete resistant varieties/hybrids are not available in sunflower. The sap inoculation technique has been optimized for large-scale screening of sunflower genotypes against SND.

Systematic studies have been undertaken for the identification of reliable sources of resistance to SND in wild sunflowers (Sujatha 2006). Babu et al. (2007) screened 30 hybrids along with their parents against SND under natural conditions, using a 0–4 scale. Fourteen hybrids (CMS 378A × RHA 265, CMS 378A × DSI 218, CMS 378A × RHA 344, CMS 234A × RHA 265, CMS 234A × RHA 271, CMS 234A × RHA 344, CMS 234A × RHA 345, CMS 234A × RHA 346, CMS 7-1A × RHA 345, DCMS 41 × RHA 274, DCMS 41 × SF 216, DCMS 42 × RHS 273, DCMS 42 × RHA 859, and DCMS 43 × DSI 218) and two parents (CMS 378 A and CMS 234A) recorded resistant reaction. In general, hybrids indicated better tolerance than the populations and inbreds. Among the 96 genotypes screened, only 8 (RHA 284, RHA 5D-1, RHA 265, RHA 859, RHA 297, RHA 365, CR-1, and R-214-NBR) have not been found to be infected by the disease (Ajith Prasad 2004).

Transgenic Approach

Pradeep et al. (2012) amplified, cloned, and sequenced the CP gene of TSV from sunflower (*H. annuus* L.). In their study, a 421 bp fragment of the TSV CP gene could be amplified and gene constructs encoding the hairpin RNA (hpRNA) of the TSV CP sequence has been subcloned into the binary vector pART27. This gene construct was then mobilized into the *Agrobacterium tumefaciens* strain LBA4404 via triparental mating using pRK2013 as a helper. Sunflower (cv. Co 4) and tobacco (cv. Petit Havana) plants were transformed with *A. tumefaciens* strain LBA4404 harboring the hpRNA cassette, and in vitro selection was performed with kanamycin. The integration of the transgene into the genome of the transgenic lines was confirmed by PCR analysis. Infectivity assays with TSV by mechanical sap inoculation demonstrated that both the sunflower and tobacco transgenic lines exhibited resistance to TSV infection and accumulated lower levels of TSV compared with nontransformed controls (Papaiah Sardaru et al. 2013).

Cultural Control

Removal of virus sources especially weeds that germinated with early rains, in fallow lands, on road sides, and on field bunds helps in reducing secondary inoculum thereby reducing the TSV incidence. Moreover, sunflower and groundnut should not be grown side by side or at least synchronization of the flowering period of sunflower with groundnut crop should be avoided as sunflower crop provides infective pollen inoculum with TSV. Similarly, removal of early-infected sunflower will not reduce disease incidence as early-infected sunflower does not produce flowers. TSV-susceptible crops like marigold and chrysanthemum should not be grown adjacent to sunflower fields. Natural barriers such as tall grasses in the field protected the adjacent crops from the disease. The tall grasses might obstruct not only wind-borne-infected pollen from outside weeds but also wind-borne thrips. Sowing 7–11 rows of fast-growing cereals (pearl millet, sorghum, or maize) as border crop around fields that obstruct the movement of thrips from landing on crop plants were found to reduce disease incidence in sunflower (Chander Rao et al. 2002, Basappa et al. 2005, Lokesh et al. 2008c). Mesta et al. (2004) reported that the use of border crop-like sorghum reduced the incidence of SND from 18% to 37%. Bare patches in the field attract thrips landing. Optimum plant population discourages thrips landing on the sunflower crop indicating that maintenance of optimum plant population is one of the options for the management of TSV infection (Papaiah Sardaru et al. 2013). The date of sowing of crops mainly depends on rainfall pattern and distribution (Lokesh et al. 2008d). Shirshikar (2003) opined that the incidence of SND could be minimized if sunflower is sown in the postrainy season, that is, from September onward (Shirshikar 2003, Upendhar et al. 2006, 2009). Intercropping with red gram or castor is helpful in reducing disease intensity compared to monocropping of sunflower (Sreekanth et al. 2004).

Chemical Control

Seed treatment with imidacloprid at 5 g/kg seed and imidacloprid (0.5%) spray reduces disease incidence with higher yield compared with other treatments (Lokesh et al. 2008c). Management trial for SND at All India Coordinated Research Project (AICRP) on oilseeds revealed that seed treatment with either imidacloprid at 5 g/kg seed or thiomethoxam at 4 g/kg seed followed

FIGURE 6.12 Stem necrosis of sunflower: Necrotic streaks on stem. (Courtesy of Dr. Varaprasad and Chander Rao, DOR, Hyderabad, India.)

by two sprays at 30 and 45 days found to reduce necrosis disease and increase seed yield significantly over untreated control (Shirshikar et al. 2009, Shirshikar 2010).

Antiviral Compounds

The use of various antiviral materials such as *Prosopis*, goat milk, and *Bougainvillea* in combinations has been used to induce resistance in sunflower against TSV-SF (Lavanya et al. 2009). Among them, *Bougainvillea spectabilis* with goat milk, *Prosopis chilensis* with goat milk, *B.spectabilis* alone, and *P. chilensis* alone are found highly effective in inducing resistance in sunflower against SND. The combinations of treatments that involve plant products with goat milk are reported to be more effective than the individual ones. Significantly enhanced PR proteins like β-1,3-glucanase and oxidative enzymes like peroxidase, polyphenol oxidase, and PAL have been observed in sunflower using previously mentioned antiviral materials.

Biological Control

Plant growth–promoting microbial consortia (PGPMC)–mediated biological management of SND under field conditions has been experimented (Srinivasan et al. 2009, Srinivasan and Mathivanan 2011b). Powder and liquid formulations of two PGPMCs (PGPMC-1, consisting of *Bacillus licheniformis* strain ML2501 + *Bacillus* sp. strain MML2551 + *Pseudomonas aeruginosa* strain MML2212 + *Streptomyces fradiae* strain MML1042; PGPMC-2, consisting of *B. licheniformis* MML2501 + *Bacillus* sp. MML2551 + *P. aeruginosa* MML2212) when evaluated along with farmers' practice (imidacloprid + mancozeb) in farmers' fields, significant disease reduction, increase of seed germination, plant height, and yield parameters have been recorded with an additional seed yield of 840 kg/ha, an additional income of Rs. 10,920/ha with a benefit–cost ratio of 6:1.

OTHER SUNFLOWER DISEASES

Some other diseases of potential importance are briefly given in Table 6.1.

TABLE 6.1
Other Sunflower Diseases of Potential Importance

Disease	Pathogen/Causal Agent	Geographical Distribution	Significant Disease Description	References
Black stem	*Phoma macdonaldii* Boerem.	Argentina, Bulgaria, China	Attacks roots and collars of the plants, resulting in early death. Penetration of the fungus into the root occurs through natural fissures or through the epidermis and appears to be similar in both resistant and susceptible lines, but the colonization rate of the stele is reduced in partially resistant line. *P. macdonaldii* and *Phomopsis helianthi* (*Diaporthe helianthi*) coexist in Argentina in sunflower, and frequently on the same plant, causing overlaying spots on the petiole insertion, with mixed colors.	Al-Fadil et al. (2009), Bistrichanov et al. (2000), Falico de Alcaraz et al. (2000), Frei (2010), Wu et al. (2012)
Head rot	*Rhizopus stolonifer* (Ehren. Fr) Lind.	Russia, Turkey, United States	Mechanical or physical damage on the back of the sunflower head results in head rot disease and significantly reduces the seed yield.	Yldrm et al. (2010), Kolte (1985), Pandey and Saharan (2005)
Head rot	*Rhizopus oryzae* Went & Prinsen Gernling	Australia, Egypt, United States	This is the first report of *R. oryzae* causing head rot on sunflower in New Mexico. Heads were brown to dark brown with discoloration extending down the sepals and peduncles into the stems. The basal parts of the heads were shredded and had grayish, fluffy mycelial mats visible in the lumen, and kernels were mostly seedless.	Sanogo et al. (2010)
Stem canker or gray stem spot	*Diaporthe helianthi* Munt. (anamorph *Phomopsis helianthi* Munt.; Cverk et al.)	Bulgaria, Croatia, Serbia, Russia	Dangerous quarantine disease causes drastic reductions in yield and oil content in the sunflower (*Helianthus annuus* L.) crop. The use of disease-tolerant genotypes and fungicides is the basis of current disease control. However, there is considerable genetic variability of the pathogen that could lead to the occurrence of new strains, which could be more aggressive or more resistant to chemical control.	Bistrichanov et al. (2000), Vasyutin et al. (2003), Vrandecic and Jurkovic (2008), Debaeke and Estragnat (2003, 2009), Says-Lesage et al. (2002)
Gray spot	*Septoria helianthi* Ellis & Kellerman	Brazil, Bulgaria, Pakistan, Serbia	Dissemination of *S. helianthi* conidia is the occurrence of precipitation on the leaves, causing splashing leading conidia from the lower leaves to the upper leaves of the plants. There is a relationship of the severity of occurrence of septoria leaf spot with the occurrence of rainfall and supplementary irrigations in the sunflower crop.	Bistrichanov et al. (2000), Masirevic and Jasnic (2006), Hamid and Jalaluddin (2007), Loose et al. (2012)
Stem black spot	*Phoma oleracea* var. *helianthi-tuberosi* Sacc. (= *Leptosphaeria indquistii* Frezzi)	Argentina	At R6 and R7, nitrogen fertilization significantly increases disease incidence (31% and 12%, respectively) and stem spot number (43% and 22%, respectively). However, the interaction between genotype and nitrogen fertilization appears to be not significant.	Velazquez and Formento (2000)

(*Continued*)

TABLE 6.1 (*Continued*)
Other Sunflower Diseases of Potential Importance

Disease	Pathogen/Causal Agent	Geographical Distribution	Significant Disease Description	References
White blister rust	*Pustula obtusata* (syn. *Pustula tragopogonis*, *Albugo tragopogonis*, *Pustula helianthicola*)	Europe (southwest of France, Belgium) and other parts of the world including Australia, North and South America, South Africa, and China with losses as much as 70%–80%	Spraying the crop with of 64% Sandofan WP (oxadixyl) or metalaxyl fungicide is effective in controlling the disease.	Rost and Thines (2012), Chen et al. (2008), Crepel et al. (2006)
Gray mold	*Botrytis cinerea* Pers. Ex. Fr.	Bulgaria, Japan, Romania, Russia	Sumilex 50 WP (procymidone) gives better protection against *B. cinerea*, and systemic acquired resistance (SAR) is mediated in sunflower by abiotic inducers such as salicylic acid (SA), benzo-(1,2,3)-thiadiazole-7-carbothioic S-methyl ester (BTH), 2,6-dichloroisonicotinic acid (INA), or EDTA for the control of *Botrytis* infection.	Bistrichanov et al. (2000), Tomioka and Sato (2011), Eva (2004), Dmitriev et al. (2003)
Sclerotium wilt	*Sclerotium rolfsii* Sacc.	Most tropical countries and warmer regions in temperate zones	For root and stem rots, biocontrol is effective; *Bradyrhizobium* sp., *Rhizobium* sp., *Trichoderma harzianum*, *T. pseudokoningii*, *T. polysporum*, *T. virens*, and fluorescent *Pseudomonas* are the most potentially useful biocontrol agents.	Bhatia et al. (2005), Cilliers et al. (2002), Fouzia and Shahzad (2011)
Verticillium wilt	*Verticillium albo-atrum* Reinke & Berth, *V. dahliae* Kleb., *V. sulphurellum*	Argentina, Serbia, Canada, United States, China	The phenotypic changes induced by *V. dahliae*'s necrosis and ethylene-inducing proteins (VdNEP) indicate that this protein acts both as a defense elicitor and a pathogenicity factor in sunflower. Rotations especially where sunflower follows potato should be avoided in disease management.	Alkher et al. (2009), Creus et al. (2007), Fu et al. (2012), Gulya et al. (2012), Pandey and Saharan (2005)
Powdery mildew	*Erysiphe cichoracearum* (*Golovinomyces cichoracearum* var. *cichoracearum*; *Podosphaera xanthii* and *Leveillula taurica* are also the causes of powdery mildew in Taiwan)	Brazil, India, Serbia, Taiwan	In general, the disease is more severe at the full flowering and preflowering stages during winter. The highest percent disease control is obtained by two sprays of fungicides difenoconazole (88.64%), penconazole (87.59%), and propiconazole (85.91%).	Akhileshwari et al. (2012), Chen et al. (2008), Almeida et al. (2008)

(*Continued*)

TABLE 6.1 (*Continued*)
Other Sunflower Diseases of Potential Importance

Disease	Pathogen/Causal Agent	Geographical Distribution	Significant Disease Description	References
Root and collar rot	*Rhizoctonia solani* Kuhn AG-IV (*Thanatephorus cucumeris*)	India	Symptoms include leaf yellowing and wilting, root rot, and death of plants; mycelium can be observed on necrotic crowns. Reported to be seed borne also in sunflower. *Trichoderma harzianum*–mediated biocontrol may be related to alleviating *R. solani*–induced oxidative stress in sunflower.	Lakshmidevi et al. (2010), Raj et al. (2008), Singh et al. (2011), Srinivasan and Visalakchi (2010)
Crown gall	*Agrobacterium tumefaciens* (Smith & Town) Conn.	Almost all sunflower-growing regions	Though the disease appears to be important, it is less commonly observed under natural conditions. It can be easily produced on sunflower by artificial inoculations.	Binboga-Meral (2007)
Bacterial stalk and head rot	*Pectobacterium atrosepticum*	Turkey	Dark and water-soaked necrotic areas develop on stems and heads, and bacteria appear as droplets and ooze from the diseased tissues.	Bastas et al. (2009)
Bacterial stalk rot	*Erwinia chrysanthemi* and *E. carotovora* subsp. *carotovora*	Taiwan	The disease mainly appears on the stem and cause water-soaked symptoms. In some severe cases, the stem pith disintegrates and shows hollow stem symptoms. "Moon bright" and "Sunbright" cultivars show better resistance when inoculated with Ech. Tetracycline has been proved to be the most effective in controlling the disease.	Hseu et al. (2004)
Angular leaf spot/bacterial blight	*Pseudomonas syringae* pv. *helianthi* (Kawamura) Young, Dye, & Wilkie	Brazil, Mexico, Serbia	The bacterium is disseminated and transmitted through the seeds from infected plants. Diseased plants are obtained from either naturally infected seed or artificially inoculated seed, the latter being more effective. Bacteria also inhibit seed germination by 50%. It is concluded that *P. syringae* pv. *helianthi* infects sunflower seed from infected plants and that this is one mechanism of the disease transmission.	Balaz and Popovic (2006), Borba Filho et al. (2007), Maselli et al. (2000, 2002)
Apical chlorosis	*Pseudomonas syringae* pv. *tagetis* (Hellmers) Young, Dye & Wilkie	Mexico, United States	Tagetitoxin-affected leaves are so severely chlorotic that they become white. Tagetitoxin-affected leaves have 99% less chlorophyll per unit fresh mass than healthy plant leaves. Electron microscopy of sections of chlorotic (toxin-affected) sunflower leaf cells reveals that leaf palisade and spongy parenchyma cells possess few chloroplasts and that those present contain disorganized thylakoids and grana and no apparent starch grains. Rates of photosynthetic CO_2 assimilation per unit leaf area in toxin-affected leaves are significantly lower than in healthy leaves.	Kong et al. (2004), Robinson et al. (2004)

(*Continued*)

TABLE 6.1 (*Continued*)
Other Sunflower Diseases of Potential Importance

Disease	Pathogen/Causal Agent	Geographical Distribution	Significant Disease Description	References
Sunflower mosaic	Sunflower mosaic virus (SuMV), a distinct species within the family Potyviridae	India, United States	Most of the seeds from the infected plants are grayish black, whereas those from healthy plants are black. The virus is transmitted by *Myzus persicae* and *Capitphorus elaegni* (*Capitophorus elaeagni*) and also is seed borne in at least one sunflower cultivar. Phylogenetic analysis of the coat protein amino acid sequence revealed that SuMV is most closely related to tobacco etch virus (TEV). There is a significant reduction in *Alternaria* infection when the virus is inoculated 20 days prior to fungal infection.	Gulya et al. (2002), Bharadwaj and Mohan (2005)
Chlorotic mottle	Sunflower chlorotic mosaic virus (SuCMoV)	Argentina	It causes systemic chlorotic mottling symptoms; growth reductions and severe yield losses. Chlorotic symptom development induced by SuCMoV infection is accompanied by changes in different redox-related metabolites and transcripts. Oxidative damage is expressed after symptom development in this host–pathogen combination.	Rodriguez et al. (2012), Lenardon (2008), Arias et al. (2005)
Root knot	*Meloidogyne incognita* (Kofoid & White) Chitwood	Egypt, India, Pakistan, United States	Sunflower plants show yellowing, stunting, and death of plants in the field resulting in 16.44% yield losses. The combined infection with *Meloidogyne incognita* plus any of the fungi such as *R. solani*, *M. phaseolina*, and *F. solani* on sunflower results in significant reduction in the number of root galls and nematode egg masses. Rugby 10 G (cadusafos) and Furadan (carbofuran) are the most effective nematicides useful in the root knot disease management. Soil amendments with certain organic plant materials and biological control agents can be useful in controlling *Meloidogyne incognita* infection.	Rehman et al. (2006), Mokbel et al. (2007), Prasad et al. (2001), Mohammad et al. (2001)
Reniform nematode	*Rotylenchulus reniformis* Linford & Oliveira	India, Pakistan	The minimum damaging threshold of *Rotylenchulus reniformis* on sunflower is 1000 nematodes/plant. Some botanicals (furfural, sugarcane by-product, and extracts of *Calotropis procera* leaf and root) have been found to be effective in minimizing the damage.	Prasad et al. (2001), Singh and Prasad (2010), Ismail and Mohamed (2007)

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Section V

Sesame

Sesame (*Sesamum indicum* L.; syn. *S. orientale* L.) variously named as *gingelly* or *til* belongs to the family Pedaliaceae. It is an annual, 1.0–1.5 m tall, herbaceous plant, maturing in 70–140 days. The basic chromosome number is 13 pairs ($2n = 26$). Molecular marker techniques such as amplified fragment length polymorphism, random amplified polymorphic DNA (RAPD), inter-simple sequence repeats (ISSR), and simple sequence repeats have been widely used in genetic diversity studies in sesame (Yadava et al. 2012, Pathak et al. 2014). The genome size of *S. indicum* is estimated to be about approximately in the range of 354–369 Mb (Ashi 2006, Wei et al. 2011).

The flowers are solitary, axillary, short-pediceled, and zygomorphic and are borne on the upper stem or branches. Self-pollination is the rule, but natural cross-pollination due to visiting bees may usually be seen to the extent of 5%. The fruit is a capsule and contains numerous small ovate seeds. Sesame seed contains high oil content 45%–52% (Hegde 2009) with 83%–90% unsaturated fatty acids, 20% proteins, and various minor nutrients such as vitamins and minerals and a large amount of characteristic lignans (methylenedioxyphenyl compounds) such as sesamin, sesamol, sesamolin, and tocopherols. Therefore, sesame seeds with high amounts of nutritional components are consumed as a traditional health food for its specific antihypertensive effect and anticarcinogenic, anti-inflammatory, and antioxidative activities. Sesame is thought to have originated in India, though its origin is sometimes traced to southern and southwestern Africa and also to the East Indies. The crop is mainly grown in the tropics and subtropics. Sesame grows on a variety of soils, but good yield is obtained on light, sandy loam, well-drained soils of moderate fertility. Principal sesame-producing countries are India, China, Korea, Iran, Turkey, Burma, and Pakistan in Asia; Egypt and Sudan in Africa; Greece in Europe; Venezuela, Argentina, and Colombia in South America; Nicaragua and El Salvador in Central America; and Mexico and the United States in North America. The largest producer and exporter of sesame seed in 2011 was Myanmar, secondly India, followed by China, Ethiopia, Nigeria, and Uganda. China is the world's largest consumer, and 70% of the world's sesame crop is grown in Asia, followed by Africa having a gross share of 26% in the world (FAOSTAT 2011).

About 65% of the annual sesame crop is processed into oil, and 35% is used in food. The food segment includes about 42% roasted sesame, 36% washed sesame, 12% ground sesame, and 10% roasted sesame seed. Because protein content and oil content are inversely proportional, seeds with an increased oil content have a decreased protein content. The oil quickly permeates and penetrates the skin, entering the blood stream through the capillaries. While in the blood stream, molecules of sesame seed oil maintain good cholesterol (HDL) and assist the body in removing bad cholesterol (LDL).

Different diseases of major economic importance affecting the crop are described in the following chapter.

7 Sesame Diseases

Phytophthora BLIGHT

Symptoms

The disease can attack plants of all ages after they attain 10 days of age. Symptoms appear on all aerial parts of the affected plants. The first symptom is the appearance of water-soaked brown spots on leaves and stems (Figure 7.1). The spots gradually extend in size. Under favorable weather conditions, the brownish discolored spots spread rapidly both upward and downward and also around the stem. The brownish area later turns deep brown and becomes black with the spread of the infection. The capsules are also affected. In humid weather, the white woolly growth of the fungus can be seen on the surface of affected capsules. Capsules on affected branches are poorly formed. The seeds remain shriveled in the case of severe attack.

Geographical Distribution and Losses

Phytophthora blight of sesame was first reported from India by Butler (1918). Widespread occurrence of the disease has now been reported from Argentina, Dominican Republic, Egypt, India, Iran, Sri Lanka, and Venezuela (Verma et al. 2005). The disease has been reported to be of economic importance in the states of Assam, Gujarat, Madhya Pradesh, and Rajasthan in India and in Sri Lanka (Kolte 1985, Pathirana 1992, Kalita et al. 2000, 2002). Since the disease generally kills the affected plants, it can be observed that the net loss is directly proportional to the incidence of the disease. The mortality of the plants due to the disease may be as high as 72%–79%. The disease is becoming increasingly more important in Assam in recent years where the losses in yield in sesame crop range between 51% and 53% (Kalita et al. 2002).

Besides causing blight, the pathogen is found to be associated with vivipary in immature seeds of sesame contained in greed pods of plants raised from naturally infected seeds. It is an unusual phenomenon that besides increasing the seed infection also renders poor-quality seeds. The host–pathogen interaction results in abnormal seedling emergence, which lacks vigor and further survival (Dubey et al. 2011).

Pathogen

The pathogen is *Phytophthora parasitica* (Dastur) var. *sesami* Prasad (*P. nicotianae* B. de Haan var. *parasitica* [Dastur] Waterh).

Mycelium of the fungus in young culture is coenocytic and profusely branched, but septa can be observed in 2-month-old cultures. The hyphae are hyaline and are 2–8 μ thick.

The fungus does not form sporangia on culture media, but abundant sporangia can be observed in nature on woolly mycelium growing on infected capsules. The sporangiophores are branched sympodially and bear ovate-to-spherical sporangia terminally. They have a prominent apical papilla and measure 25–50 × 20–35 μ in size.

The mycelium, when floated in tap water, forms zoosporangia readily in 48 h. The zoospores are formed inside, and they clearly get separated within the sporangium. The zoospores are liberated in water if the mycelium is flooded with water. The antheridium can be observed at the base, and attachment is typically amphigynous. The oospores are spherical, smooth, double walled, and hyaline.

FIGURE 7.1 Phytophthora blight of sesame. The first symptom is the appearance of water-soaked brown spots on leaves. (Courtesy of Dr. Anil Kotasthane, IGKV, Raipur, India.)

The fungus grows well on oatmeal/agar at an optimum temperature of 30°C. Culture of the fungus may show tendency to lose virulence if it is maintained on artificial medium for a long period. The fungus is reported to be thiamine deficient. Its growth becomes good when thiamine is incorporated (200 μg/L) in the medium. The fungus grows best at pH 6.5.

Epidemiology and Disease Cycle

The pathogen can survive in mycelial form up to 50°C temperature, and culture having chlamydospores may survive up to 52°C. Viability of the culture can be kept in a refrigerator for 1 year at 5°C. These studies suggest that the fungus can survive in soil during the summer and winter where temperature never rises beyond 50°C or drops below 5°C. The fungus survives in soil during the unfavorable period in the form of dormant mycelium and/or in the form of chlamydospores. In addition to soil, seed also appears to play an important role in the recurrence and spread of the disease. In seed, the mycelium has been located in the embryo. However, there are reports that the fungus reduces seed viability but it is not seed borne (Maiti et al. 1988). The mycelium in the host tissue is inter- or intracellular, but it does not form haustoria. The sporangiophores emerge in groups by rupturing the epidermis, but sometimes they emerge through stomata (Verma et al. 2005). The zoosporangia are formed abundantly if humid weather prevails for 2–3 days but soon stop formation if a dry spell appears. The secondary infection occurs through zoospores. *P. parasitica* var. *sesami* is restricted in its pathogenicity to sesame plants only. Sehgal and Prasad (1966) have shown variation in virulence among various isolates of *P. parasitica* var. *sesami*. Single-zoospore isolates show great variations, under similar conditions of infection, in virulence, which may range from nonpathogenic to highly pathogenic. A few isolates of *P. parasitica* var. *sesami* can lose virulence, but the loss in virulence is not permanent, since a few cultures can regain the loss of virulence after passage through the host. On repeated host passages, the culture can even become more virulent than the original ones (Sehgal and Prasad 1971).

Heavy rains for at least 2 weeks and high humidity (above 90%) for 3 weeks or more favor the development of the disease. When such favorable conditions persist for a longer time, the infection

appears quite fast. It is observed that the initial development of the disease is much earlier when the soil temperature is 28°C, while the initial appearance of the disease is delayed with an increase in the soil temperature up to 37°C. The pathogen is favored by 30°C, can tolerate 35°C, but fails to grow at 37°C. Hence, soil temperature of 28°C–30°C is necessary for disease development (Prasad et al. 1970). It is further reported that incidence of the *Phytophthora* blight of sesame shows a close parallelism to the growth of the fungus. Bright sunshine hours for 2–3 days are not favorable for disease development since zoospore formation is stopped under such conditions. The disease appears to become more severe in heavy soils (Verma 2002). The moderate nitrogenous fertilizer application leads to more incidence of *Phytophthora* blight of sesamum (Verma and Bajpai 2001).

Disease Management

Host Plant Resistance

Out of several strains and varieties of sesame (*Sesamum orientate* L.) and five other species, namely, *S. occidentalis* Heer and Regal, *S. indicum* L., *S. laciniatum* Willd., *S. prostratum* Retz., and *S. radiatum* Schum. and Thonn., tested for resistance to the disease, none is identified to be resistant to the disease (Kolte 1985, Choi et al. 1987). However, under All India Coordinated Research Project on Oilseeds, a number of sesame lines over several years of crop season testing have been found to be tolerant to *Phytophthora* blight. These lines are TC-25, JLSC-8, TKG-21, AT-60, AT-64, B-14, Chopra-1, Durga (TKG-6), JLT-3, JLT-7, Lakhora-1, Phule till-1, RT-46, T-12, and T-13. These lines/strains that have shown tolerance over longer duration can be grown to manage the adverse effect of the disease on yield (Verma et al. 2005). In Venezuela, three lines, 71-184-1, 79-129-2, and 71-145-3 (selected from B_4 of Ajinio Atar 55), are reported to be disease resistant. The National Institute of Crop Science in Korea has developed a new black-seeded variety *Kangheuk*, which is a high-yielding, high-lodging, and *Phytophthora* blight–resistant variety (Shim et al. 2012). Epiphytotic conditions and nonavailability of resistant germplasm had prompted the use of gamma ray–induced (450–600 Gy) mutation breeding for the development of *Phytophthora* blight–resistant sesame variety *ANK-S 2* in Sri Lanka (Pathirana 1992).

Chemical Control

Seed-borne infection can be controlled by treating the seed with thiram (0.3%). Secondary infection and further spread of the disease can be brought under control by three sprayings of Bordeaux mixture (3:3:50), each at an interval of 1 week after the appearance of the disease (Verma et al. 2005). Spray application of dithiocarbamate fungicides such as mancozeb (0.3%) or zineb (0.3%) and Fytolan (copper oxychloride) (0.3%) is reported to be effective in the control of the disease (Kalita et al. 2000, 2002).

Cultural Control

Sanitation and clean cultivation should be followed as additional measures to control the disease. Use of sowing date depending upon the prevailing local conditions and crop fields with light soil with proper drainage should be preferred to avoid heavy losses due to disease. The intercropping of sesame with soybean, castor, maize, sorghum, or pearl millet in the ratio of 1:3 or 3:1 shows a low incidence of the disease with higher yield. Application of farm yard manure (FYM) or neem cake with inorganic fertilizers N_{60}, P_{40}, and K_{20} reduces the disease incidence (Verma et al. 2005). Planting of sesame in 0.2 mm wide ridge in plots mulched with vinyl reduces the spread of the disease by at least 30% and increases the yield by 22% (Choi et al. 1984).

Biological Control

Species of *Pseudomonas*, *Bacillus*, and *Streptomyces*, which are most active at 25°C–27°C at field capacity moisture level, can be suppressive to *Phytophthora* species in soil (Erwin 1983). Antagonistic *Trichoderma* species, namely, *T. viride*, *T. harzianum*, and *Pseudomonas fluorescence*,

when used as seed treatment, not only reduce the disease significantly but substantially increase the sesame yield (Verma 2002). Application of phosphorus-solubilizing bacteria (PSB) along with neem cake or 50% NPK + FYM or 100% NPK + PSB reduces the disease (Verma and Bajpai 2001).

CHARCOAL ROT

Symptoms

Sesame plants may be attacked immediately after sowing. The germinating seeds may become brown and rot. In the seedling stage, the roots may become brown and rot, resulting in the death of the plants. If the plants survive, the older plants are affected at the base of the stem indicating the formation of lesion that later spreads to the middle portion of the stem and becomes ashy, causing drooping of leaves and top of the plants (Figure 7.2). Such plants make poor growth and remain stunted. The mycelium of the fungus progresses upward in the stem, and as the stem dries, pycnidia are formed as minute black dots. The stem may break off, and the blackening may extend upward on the stem. The capsules are also affected. Such capsules open prematurely, exposing shriveled and discolored seeds (Figure 7.3). Seeds may show the presence of sclerotia on the surface.

Geographical Distribution and Losses

Reports of occurrence of charcoal rot of sesame have been made from all over the sesame-growing areas in the world (Kolte 1985, Verma et al. 2005). The disease is particularly reported to be quite serious, limiting the production of the crop in Ismailia Governorate Region in Upper Egypt (Abdou et al. 2001, El-Bramawy and Wahid 2007); Southeastern Anatolia Region in Turkey (Sağır et al. 2009); in the Portuguesa state in Venezuela (Cardona and Rodriguez 2006, Martinez-Hilders et al. 2013); in the Chandrapur district of Vidarbha region of Maharashtra, the Gwalior Division of Madhya Pradesh, and in the states of Haryana and Chhattisgarh in India (Kolte 1985,

FIGURE 7.2 Severely affected sesame plant showing charcoal symptoms. (Courtesy of Dr. Anil Kotasthane, IGKV, Raipur, India.)

FIGURE 7.3 Charcoal rot–affected sesame plants. Note the ashy color of the stem with infected discolored capsules. (Courtesy of Dr. Anil Kotasthane, IGKV, Raipur, India.)

Deepthi et al. 2014); and in Pakistan (Akhtar et al. 2011). Seedling mortality due to seed-borne infection aggravates the disease problem by reducing the plant stand per unit area, resulting in low yield. About 5%–100% yield loss due to the disease is reported. An estimated yield loss of 57% at about 40% disease incidence is reported (Maiti et al. 1988). In Venezuela, losses in sesame due to charcoal rot have been evaluated resulting up to 65% of seed weight reduction for affected plants (Martinez-Hilders et al. 2013). The importance of the charcoal rot lies not only in affecting the yield and causing quantitative and qualitative losses (Sağır et al. 2009) but also in increasing soil infestation with the causal fungus. For example, sclerotia of *Macrophomina phaseolina* in Venezuelan soils of sesame production areas have been estimated to be up to 200 per gram of soil (Martinez-Hilders et al. 2013). If the disease appears simultaneously with *Phytophthora* blight or with *Fusarium* wilt, the losses in yield usually are very high.

Pathogen

The pathogen is *M. phaseolina* (Tassi) Goid. The morphological and physiological characteristics of the pathogen have been described under chapters on peanut and sunflower diseases. Molecular methods used for determining the level of genetic diversity and polymorphism among *M. phaseolina* populations affecting sesame include random amplified polymorphic DNA (RAPD), amplified fragment length polymorphism, and inter-simple sequence repeats (ISSR). It is revealed that *M. phaseolina* populations in all the major sesame production regions in China (Wang et al. 2011), Iran (Bakhshi et al. 2010, Mahdizadeh et al. 2012), Mexico (Munoz-Cabanas et al. 2005), and Venezuela (Martinez-Hilders and Laurentin 2012, Martinez-Hilders et al. 2013) are highly genetically diverse based on genomic data. High level of genotypic variability is likely due in part to the exposure of the pathogen to diverse environment and a wide host range within these countries. However, no clear association between geographical origin and host of each isolate has been found, though isolates from the same location show a tendency to belong to their respective closer groups indicating closer genetic relatedness (Bakshi et al. 2010).

Leaf, stem, and root extracts of urd and mung beans have been reported to show an inhibitory effect on sclerotial formation of the sesame isolate in vitro (Kolte and Shinde 1973). Ammonium chloride also has an inhibitory effect on sclerotial formation of the fungus (Kolte 1985). The sesame isolates are chlorate sensitive and grow normally with numerous dark microsclerotia production on the potassium chlorate–containing medium (Rayatpanah et al. 2012). Interestingly, the soybean and sunflower isolates are chlorate sensitive and divided into two classes. Class 1 includes the isolates that grow sparsely with a feathery-like pattern, and Class 2 includes the isolates that grow well with a nonfeathery pattern. Isolates with feathery-like pattern are more virulent on soybean and sunflower (Rayatpanah et al. 2012). Two distinct strains, namely, pycnidia-forming and sclerotia-forming strains, have been reported from Bangladesh. The pycnidial strain is reported to be more pathogenic on sesame than the sclerotial strain (Kolte 1985, Verma et al. 2005).

Epidemiology and Disease Cycle

The fungus survives as free sclerotia in soil or as mycelium and sclerotia carried in crop debris. It spreads by the movement of soil and crop debris (Al-Ahmad and Saidawai 1988) and through the sesame seeds. The sesame seed has been found to carry the fungus on and inside the testa as sclerotia or as stromatic mycelium. The standard blotter method, use of a selective medium and scanning electron microscopy, facilitates the detection of seed-borne infection in sesame (Verma et al. 2005, El-Wakil et al. 2011). There is a positive correlation between microsclerotia on sesame seed with percent plant infection (Gupta and Cheema 1990). It is reported that the germinating seed and seedlings stimulate normal sclerotial germination and attract developing mycelium to the host roots. Entry may occur directly through the cuticle and epidermis, infection cushions and appressoria are also reported to be formed on sesame plants prior to infection, and the pathogen produces cell-wall-degrading pectolytic and cellulolytic enzymes. The most aggressive isolate produces more cell-wall-degrading enzymes than the less aggressive isolates (Gabr et al. 1998).

A high soil temperature (35°C) and low osmotic potential reduce plant vigor and favor growth of the fungus and initiation of infection. Maximum temperature of 31.6°C, minimum temperature of 24°C, and relative humidity of 88% favor severe charcoal rot disease development (Deepthi et al. 2014). The response of the sesame crop to stress conditions has been found to be of significant importance in epidemiology, and irrigation reduces infection by reducing drought stress. Periods of drought between heavy rains favor the development of the disease in Africa. Strains of *M. phaseolina* are known for their wide host range and infect a large number of weeds and rotation crops, which function as a source of inoculum and survival of the pathogen (Simosa and Delgado 1991, Kolte 1997).

Disease Management

Host Plant Resistance

Differences in host resistance to charcoal rot through screening of genotypes and molecular marker techniques in sesame have been noted in trials in several Asian and South American countries (Chattopadhyay and Sastry 2000, Melean 2003, El-Fiki et al. 2004b, El-Bramawy and Shaban 2007, El-Bramawy et al. 2009, Gao et al. 2011, Zhang et al. 2012). The results reveal that there is high heritability for host resistance indicating additive gene nature of the resistance characters and consequently a high gain from selection. High resistance gives the lowest seed yield, and great care is taken during selection and pedigree selection for resistance breeding program to *M. phaseolina* (Mahdy et al. 2005b, El-Bramawy and Wahid 2006, 2007, El-Shakhees and Khalifa 2007). In Egypt, sesame line P5 (NM 59) and the F6-derived lines C6.3, C1.10, and C3.8 are the most valuable sources of resistance to charcoal rot disease. The P5 line is resistant to both charcoal rot and *Fusarium* wilt, whereas the F6-derived line C6.3 is the most resistant and top-yielding one against combined infection of charcoal rot and *Fusarium* wilt diseases (Shabana et al. 2014).

Different sesame germplasm lines and cultivars that have been found tolerant or less susceptible to charcoal rot are ORM 7, ORM 14, and ORM 17 (Dinakaran and Mohammed 2001, Subrahmaniyan et al. 2001, Thiyagu et al. 2007); TLC-246, TL6-279, and TLCCCCC-281 (John et al. 2005); ZZM0565, ZZM0570, Xiangcheng dazibai, Xincai Xuankang, Shangshui farm species, and KKU 3 (Zhao et al. 2012); mutants NS 13 P1, NS 163-1, NS 270 P1, and NS 26004 (Akhtar et al. 2011); UBQ5, UF 4A, and alpha-tubulin (Liu et al. 2012); UCLA-1, EXP-1, and DV-9 (Melean 2003); and Aceteru-M, Adnan (5/91), Taka 2, B 35, and mutation 48 (El-Fiki et al. 2004b).

The sources of resistance appear to differ in the mechanism of resistance. Factors such as morphological traits like single stem (Li et al. 1991); medium branch numbers (El-Bramawy 2008, El-Bramawy et al. 2009); creamy or white seed color (El-Bramawy et al. 2009); antifungal nutritional components such as phytin, trypsin inhibitor, and tannins (El-Bramawy and Embaby 2011); certain biochemical factors as faster rate of activity of polyphenol oxidase enzymes in Chinese sesame cultivar *Yuzhi 11* (Liu et al. 2012); and different isoenzyme band patterns (Zhang et al. 2001) have been linked with resistance to charcoal rot in sesame. The mature plant reaction, through hybridization studies, indicated that susceptibility in the mature plant is dominant over tolerance, and it is controlled by 1, 2, or 3 pairs of genes (Kolte 1985).

Chemical Control

Seed treatment with carbendazim (0.1%–0.3%) gives complete control of seed-borne infection of *M. phaseolina* in sesame when used as seed treatment fungicide (Choudhary et al. 2004, Rajpurohit 2004b, Shah et al. 2005, John et al. 2010). Other seed treatment fungicides are thiophanate methyl (John et al. 2010), Benlate or Rizolex T at 3 g/kg seed (El-Deeb et al. 1985, El-Fiki et al. 2004a), mancozeb (Mudingotto et al. 2002), thiram, captan, and carboxin (Verma et al. 2005). Soil treatment with fungicides is effective but impracticable. The integration of fungicide carbendazim seed treatment (0.1%) with carbendazim-tolerant strain of *T. viride* (Tv-Mut) as induced by mutating the native strain of the fungus by UV irradiation and soil supplemented with 20 kg P and 15 kg K/ha show the highest reduction (91.7%) in sesame stem–root rot incidence caused by *M. phaseolina* (Chattopadhyay and Sastry 2002). Aminobutylic acid and potassium salicylate can effectively control charcoal rot in sesame by induction of host resistance against *M. phaseolina* and increasing plant height, indole acetic acid (IAA) content, and peroxidase (PO) activity (Shalaby et al. 2001). Soaking sesame seeds in indole butyric acid at 100 ppm or salicylic acid at 4 mM produce healthy stand of plants.

Cultural Control

The average charcoal rot incidence can be lowered down by choice of sowing date and levels and time of irrigation depending on the local conditions in a particular geographical area. Early sowing by June 10 in Egypt and following *hills-over-furrows* method of sowing and giving only one irrigation during the whole growing season to a crop fertilized with N at 65 kg, P at 200 kg, and K at 50 kg/feddan (0.42 ha) result in significant reduction in charcoal rot incidence (Shalaby and Bakeer 2000). Similar results are evident under early sowing and frequency of irrigation in Turkey (Sağır et al. 2010). Lowering concentration of Ca, Na, Mg, and Fe and increasing concentration of K, Cu, and Zn in the soil by applying chemical fertilizers and organic manure may reduce very much the charcoal root rot incidence (Narayanaswamy and Gokulakumar 2010). Sesame crop grown as mixed or intercropped with green gram in 1:1 is useful in the management of the charcoal rot and results in higher sesame yield in the arid region (Rajpurohit 2002, Ahuja et al. 2009). It is noteworthy to note that green gram (mung) and black gram plant extracts are inhibitory to the growth of *M. phaseolina* (Kolte and Shinde 1973). Six weeks of soil solarization of infested crop field sites in the summer months result in good sesame seed germination and better disease management under Indian conditions (Chattopadhyay and Sastry 2001).

Biological Control

Effect of antagonistic fungi and bacteria isolated from the rhizosphere of sesame is reported to be efficiently more effective in controlling the root rot and stem rot of sesame caused by *M. phaseolina* (El-Bramawy and El-Sarag 2012). Sesame seed treatment with (a) *T. viride* at 4 g/kg of seed (Rajpurohit 2004a, Hafedh et al. 2005, Rani et al. 2009, Zeidan et al. 2011), (b) *T. harzianum* (Pineda 2001, Cardona and Rodriguez 2002, 2006, El-Fiki et al. 2004b, Nair et al. 2006, Sattar et al. 2006, Moi and Bhattachrya 2008), (c) *P. fluorescens* (Jayshree et al. 2000, Moi and Bhattachrya 2008), and (d) *Bacillus subtilis* (Nair et al. 2006, Elewa et al. 2011) has been found effective in the control of charcoal rot disease. Green manure of *Crotolaria* amended with *Trichoderma* constitutes a viable alternative for the control of charcoal rot of sesame (Cardona 2008).

A combination of seed treatment and soil application of the antagonists through the application of clay granules impregnated with *T. harzianum* or *P. fluorescens* at sowing time appears to be much more effective in the control of the charcoal rot (Pineda 2001, Cardona and Rodriguez 2002). Application of vesicular–arbuscular mycorrhizae (VAM), namely, *Glomus* spp., together with biocontrol agents *T. viride* or *B. subtilis* significantly helps in efficient control of root rot (*M. phaseolina*) and *Fusarium* wilt diseases of sesame than individual application of either VAM or antagonists (Elewa et al. 2011, Zeidan et al. 2011). Soil solarization in combination with fungal antagonists *T. pseudokoningii* and *Emericella nidulans* singly or in mixed inocula reduces charcoal rot incidence in sesame significantly (Ibrahim and Abdel-Azeem 2007). Seed treatment with *Azotobacter chrococum* and seed + soil treatment with *Azospirillum* also reduce the disease by about 30% (Verma et al. 2005, Maheshwari et al. 2012).

Effect of Plant Extracts

Extracts of *Thevetia neriifolia* (Bayounis and Al-Sunaidi 2008a), *Azadirachta indica*, *Datura stramonium*, *Nerium oleander*, *Eucalyptus camaldulensis* (Bayounis and Al-Sunaidi 2008b), and *Helichrysum* flower (Shalaby et al. 2001) show inhibitory effect on the growth of *M. phaseolina*, indicating their potential use in the control of the disease. The extracts of *Eucalyptus* (*Eucalyptus rostrata*, *E. camaldulensis*), peppermint (*Mentha piperita*), and thyme (*Thymus serpyllum*), when used in sand culture or under in vitro conditions in growth media and inoculated with *M. phaseolina*, have been found to show increase in sesame seed germination despite the presence of *M. phaseolina* in the culture, indicating potential usefulness of these extracts (Sidawi et al. 2010).

Fusarium WILT

Symptoms

Plants get infected at any stage of the crop development including the damping-off phase in the seedling stage (Fallahpori et al. 2010, 2013). During later stages of the plant, yellowing of the leaves is the first noticeable symptom of the wilt in the field. Leaves become yellowish, droop, and dessicate. Sometimes such leaves show inward rolling of the edges and eventually dry up. The terminal portion dries up and becomes shrunken and bent over. In a severe infection, the entire plant becomes defoliated and dry. In a less severe infection or when mature plants are infected, only one side of the plant may develop symptoms, resulting in partial wilting, and a half stem rot symptom has been reported (Cho and Choi 1987). A blackish discoloration in the form of streaks appears on infected plants. Discoloration of the vascular system is conspicuous in the roots. Roots in the later stages show rotting, wholly or partially corresponding with that side of the plant showing disease symptoms. Numerous pink pinhead-sized sporodochia (containing macroconidia of the fungus) may be seen scattered over the entire dried stem. The capsules of wilted plants also show numerous sporodochia.

Geographical Distribution and Losses

Fusarium wilt of sesame was reported for the first time from North America in 1950 (Armstrong and Armstrong 1950). Since then, the disease is reported to occur in Egypt, Colombia, Greece, India, Iran, Israel, Japan, Korea, Malawi (formerly Nyasaland), former Soviet Union, the United States, and Venezuela. Similar disease has been reported from Pakistan, Peru, Puerto Rico, and Turkey (Kolte 1985, Verma et al. 2005). The disease can be devastating on susceptible varieties of sesame, but many local varieties have been found to have some degree of resistance to local races of the fungus. Epiphytotic occurrence of the disease was reported in 1961 and 1964 in the United States and in 1959 in Venezuela.

Pathogen

The pathogen is *Fusarium oxysporum* (Schelt.) f. *sesami* Jacz. Isolation of the causal fungus could be obtained more easily from the infected dry sample (dry sample isolation) compared to conventional direct isolation technique from freshly infected sesame plants (Su et al. 2012).

The fungus produces profuse mycelial growth on potato dextrose agar. The mycelium is arid, hyaline, septate, and richly branched, turning light pink when old. The microconidia are formed abundantly. They are hyaline, ovoid to ellipsoid, unicellular, and measure 8.5 × 3.25 μ in size. In the old culture, the macroconidia are formed sparsely. They are 3–5, septate, and measure in the range of 35–49 × 4–5 μ in size.

The macroconidia are produced abundantly in sporodochia as they develop on affected plants. The chlamydospores are globose to subglobose, smooth, or wrinkled and measure 7–16 μ in diameter. Physiological studies on the pathogen have been made. The fungus grows best on Richard's medium. It grows at the temperature range of 10°C–30°C with an optimum temperature of 25°C. Nitrate nitrogen and pH 5.6–8 support the maximum growth of the fungus. Illumination inhibits spore germination (Liu et al. 2010).

Epidemiology and Disease Cycle

The fungus is restricted in its host range to sesame. Morphological differences and similarities have been reported in different isolates of *F. oxysporum* f. *sesami*. Three strains have been reported in Venezuela on the basis of morphological differences, but these strains are reported to show a similar degree of pathogenicity. It is revealed that there is a relationship between vegetative compatibility groups of the pathogen and geographic origin of the isolates collected from the different sesame-growing regions (Basirnia and Banihashemi 2005). The pathogenic variation and molecular characterization of *Fusarium* species isolated from wilted sesame have been studied (Li et al. 2012).

The pathogen is reported to be seed and soil borne, and it may persist for many years in the soil. The amount of seed transmission of the pathogen varies in the range of 1%–14% depending on the severity of systemically infected sesame plants (Basirnia and Banihashemi 2006). It appears that the fungus penetrates the host through root hairs and causes trichomycosis. The most virulent isolates produce more cell-wall-degrading enzymes than the less virulent ones (Gabr et al. 1998). The culture filtrate of *F. oxysporum* f. *sesami* has been reported to have an inhibitory effect on sesame. Shoot and root growth is also inhibited by culture filtrate of the fungus indicating the production of toxic substances by the pathogen. Some elements like vanadium, zinc, boron, molybdenum, and manganese are highly inhibitory to *F. oxysporum* f. *sesami* (Gabr et al. 1998).

High soil temperature to a depth of 5–10 cm and 17%–27% water-holding capacity during dry periods is favorable for the development of the disease. Drought stress in the sesame plants predisposes the plants to infection and development of wilt and influences the host genotype reaction to the disease (El-Shemey et al. 2005, Kavak and Boydak 2011). The *Fusarium* wilt of sesame is reported to be associated with nematode attack in South America and with *M. phaseolina* in Egypt, India, and Uganda (Kolte 1985, 1997). The density of the fungus becomes higher in soil under continuous cropping (Paik et al. 1988).

Disease Management

Host Plant Resistance

Sesame *Fusarium* wilt–resistant accessions/genotypes have been identified from different sesame-growing countries. These are NSKMS 260, 261, and 267 and TMV 3 (Badri et al. 2011, Jyothi et al. 2011); S-5, S-4, H-9 (El-Bramawy and Wahid 2007); S2, H4, mutant 8, UNA 130, H1, S1 (El-Bramawy and Wahid 2009); somaclonal strain GZO 25 (Abd-El Moneem et al. 1996); Camdibi, WS-143, WS-313, Birkan (Silme and Cagrgan 2010); and Sanliurfa-63189 (Kavak and Boydak 2006). A considerable degree of variability in differences in resistance to *Fusarium* wilt among sesame genotypes has been reported, and the breeding methodology in sesame improvement (i.e., the selection, pedigree method, and hybridization) depends upon the nature and magnitude of the gene action in controlling the genetic behavior of the disease resistance trait (El-Bramawy et al. 2001, Zhang et al. 2001, El-Bramawy 2003, Ammar et al. 2004, Mahdy et al. 2005b, Kavak and Boydak 2006, El-Shakhess and Khalifa 2007, El-Hamid and El-Bramawy 2010). Parental line P5 and F6-derived lines C1.6, C1.10, C3.8, C6.3, C6.5, and C9.15 are reported to be the most resistant sesame lines to the *Fusarium* wilt disease (Shabana et al. 2014). Higher resistance of a germplasm line, in general, is reported to be lower yielder (El-Hamid and El-Bramawy 2010). However, high-yielding multiple disease-resistant sesame cultivar *Yuzhi 11* has been developed in China through breeding (Wei et al. 1999).

Satisfactory sources of *Fusarium* wilt resistance in sesame have been developed by mutagenesis and mutation breeding techniques (Uzun and Cagrgan 2001, Soner Slme and Cagrgan 2010). For example, *Birkan* is a high-yielding sesame mutant cultivar derived from 400 Gy gamma radiation in Egypt (Silme and Cagrgan 2010).

Sesame accessions with medium branch number and creamy or white seed color are the only covariate that significantly correlates with the infection caused by *F. oxysporum* f. sp. *sesami*, and these traits can be directly used for direct selection of sesame accessions that are resistant to *Fusarium* wilt and charcoal rot diseases (El-Bramawy et al. 2009). Depending on the genotypes, gene action for resistance to *Fusarium* wilt has been found to be additive with high heritability (El-Bramawy 2006, Bayoumi and El-Bramawy 2007, El-Bramawy and Wahid 2007), nonadditive (El-Bramawy and Shaban 2007, 2008, El-Shakhess and Khalifa 2007), and with epistatic effects (Bakheit et al. 2000).

Besides the genetic factors, some wilt-resistant genotypes possess the high value of the pathogen antinutritional factors such as phytic acid, trypsin inhibitor, and tannins (El-Bramawy and Embaby 2011). Many other *Fusarium* wilt–resistant sesame genotypes show significant differences in yield and yield components, total phenol contents, polyphenol oxidase, and PO enzyme activity indicating the importance of biochemical constituents in the expression of mechanism of resistance/tolerance to *Fusarium* wilt in sesame (Ghallab and Bakeer 2001).

Chemical Control

Seed treatment with benomyl or carboxin at 0.2% or with carbendazim (0.25%) or thiram (0.3%) results in significant control of the disease up to about 45 days after seed germination (Ahmed et al. 1989, Shalaby 1997). Sesame seeds soaked in ascorbic acid and salicylic acid (5 mM) for 24 h and sown and then treated with ascorbic acid and salicylic acid 15 days after sowing give best control of the disease through induced host resistance (Abdou et al. 2001).

Cultural Control

Balanced fertilization and insect pest control ensure good growth of the crop and help in the reduction of the disease. Trace elements such as copper, manganese, and zinc decrease the incidence of wilt of sesame (Abd-El-Moneem 1996). In heavily infested soil, at least 5 years should elapse between two sesame crops. Cultivation of sesame in rotation with onion or wheat is helpful in the reduction of the *Fusarium* wilt in sesame (El-Kasim et al. 1991). Sanitation and clean cultivation and choice of sowing dates depending on the known prevailing local conditions are taken into practical

use in disease management (Verma et al. 2005). For example, sowing the sesame crop around June 10 through *hills-over-furrow* and fertilizing the crop with NPK (65, 200, and 50 kg/feddan, respectively, in 0.42 ha) and giving one irrigation during the growing season in Egypt are a very useful cultural practice package for the management of the disease in sesame (Shalaby and Bakeer 2000).

Biological Control

Several microbial antagonists such as *T. viride*, *Gliocladim virens* (Kang and Kim 1989, Wuike et al. 1998, Sangle and Bambawale 2004, El-Bramawy and El-Sarag 2012), *Bacillus polymyxa* (Hyun et al. 1999), *B. subtilis* (El-Sayeed et al. 2011), *Enterobacter cloacae* (Abdel-Salam et al. 2007), *Pseudomonas aeruginosa* (Abdel-Salam et al. 2007), *P. putida* and *P. fluorescens* (Farhan et al. 2010), *Streptomyces bikiniensis*, and *S. echinoruber* (Chung and Hong 1991, Chung and Ser 1992) are inhibitory to the growth of *F. oxysporum* f. sp. *sesami* and show high potential for their use in the management of *Fusarium* wilt of sesame. *Trichoderma* species grown on cow dung slurry and cow dung are the most effective in the control of the wilt disease of sesame (Sangle and Bambawale 2004).

Fusarium wilt of sesame can be controlled with application of plant-growth-promoting rhizobacteria, and this practice offers a potential nonchemical means for disease management. A combination of *Azospirillum brasilense*–based Cerialin and *Bacillus megaterium*–based Phosphoren biofertilizers plus Topsin (100 ppm) has been found to give significant reduction of *Fusarium* wilt incidence, with increased morphological characteristics and plant yield (Ziedan et al. 2012). Similarly, a mixture of *P. putida* 2 plus *P. fluorescens* 3 treatment together (Fusant) as biocide and biofertilizer gives better control of the wilt disease with higher sesame crop yield (Farhan et al. 2010). Fertilizer-adaptive variant tetracycline-resistant strain *TRA2* of *Azotobacter chroococcum*, an isolate of wheat rhizosphere, has been found to show plant-growth-promoting attributes and strong antagonistic effect against sesame wilt and charcoal rot pathogens. Seed bacterization with the strain TRA2 results in significant decrease in *Fusarium* wilt disease incidence and increase in vegetative growth of sesame plants (Maheshwari et al. 2012).

Glomus spp. (VAM) protect the sesame plants by colonizing the root system and consequently reduce colonization of fungal pathogens in sesame rhizosphere by stimulation of bacteria belonging to the *Bacillus* group. These bacteria show high antagonistic potential, and this significantly reduces *Fusarium* wilt incidence in sesame (El-Sayeed Ziedan et al. 2011).

Effect of Plant Extracts

Extracts of leaves of thyme, eucalyptus, and garlic reduce the incidence of *Fusarium* wilt disease of sesame. Extract of peppermint (*M. piperita*) leaves not only reduces the wilt incidence but also increases the yield of sesame plant (Sidawi et al. 2010).

Alternaria LEAF SPOT

Symptoms

Symptoms of the disease appear mainly on leaf blades as small, brown, round-to-irregular spots, varying from 1 to 8 mm in diameter. The spots later become larger and darker with concentric zonations demarcated with brown lines inside the spots on the upper surface (Figure 7.4). On the lower surface, the spots are lighter brown in color. Such spots often coalesce and may involve large portions of the blade, which become dry and are shed. Dark brown, spreading, water-soaked lesions can be seen on the entire length of the stem. The lesions also occur on the midrib and even on veins of leaves. In very severe attacks, plants may be killed within a very short period after symptoms are first noted, while milder attacks cause defoliation. Occasionally, seedlings and young plants are killed exhibiting pre- and postemergence damping-off.

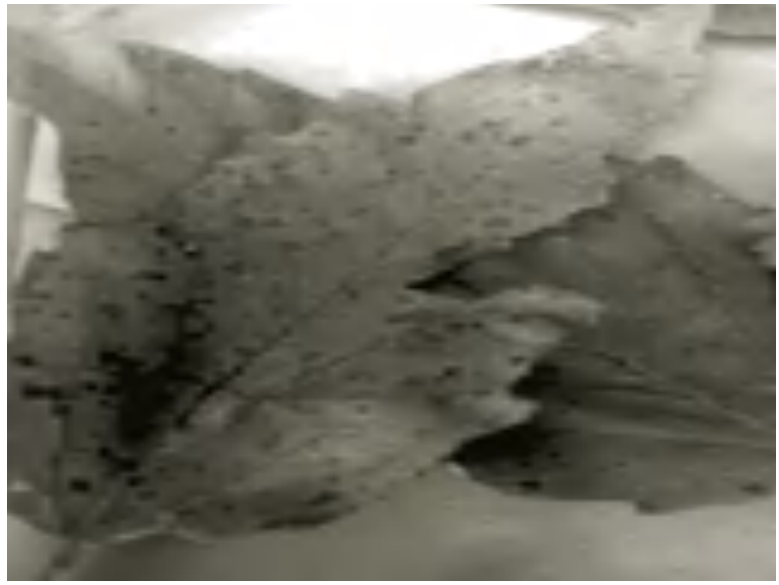

FIGURE 7.4 *Alternaria* leaf spot of sesame. (Courtesy of Dr. B.A. Tunwari, Federal University, Wukari, Nigeria, and H. Nahunnaro, Modibbo Adama University of Technology, Yola, Nigeria.)

Geographical Distribution and Losses

Alternaria leaf spot of sesame was first described by Kvashnina (1928) from the North Caucasus region in the former Soviet Union. Kawamura in Japan studied a similar leaf spot pathogen on sesame and named it as *Macrosporium sesami* Kawamura. Mohanty and Behera (1958) from India reported *Alternaria* blight of sesame and found the causal organism to be closely resembling *M. sesami*. However, it differed from *M. sesami* in that some of the spores were catenulate. On the basis of the catenulation, the fungus was placed in *Alternaria* and renamed as *A. sesami* (Kawamura) Mohanty and Behera. In India and in the United States, it was earlier referred only by the name *Alternaria* sp. The first identification of *A. sesami* in the United States was probably made by Leppik and Sowell in 1958.

The *Alternaria* leaf spot is now reported to occur in most of the tropical and subtropical areas of the world. Epiphytotic occurrence of the disease has been reported from the Stoneville area in Mississippi in 1962, the Tallahassee area in Florida in 1958, and the coastal area of Orissa in 1957 and Maharashtra in India in 1975 (Kolte 1985). It is now reported to be of more economic significance in Egypt (El-Bramawy and Shaban 2007, 2008), India (Naik et al. 2007), Kenya (Ojiambo et al. 2000a,b), Nigeria (Enikuomehin et al. 2011), Pakistan (Marri et al. 2012), and Uganda (Mudingotto et al. 2002).

The amount of damage to the sesame plant is dependent on the stage of growth and environmental conditions. Disease severity is negatively correlated with the seed yield, 1000-seed weight, and seeds/capsule (Ojiambo et al. 2000b). The disease causes 20%–40% loss in sesame crop in the state of Uttar Pradesh in India (Kumar and Mishra 1992). It is, however, reported that about 0.1–5.7 g seeds/100 fruits are lost due to the disease under Karnataka conditions in India (Kolte 1985).

Pathogen

The pathogen is *Alternaria sesami* (Kawamura) Mohanty and Behera. The conidiophores of the fungus are pale brown, cylindrical, simple, erect, 0–3 septate, and not rigid, arise singly and measure 30–54 × 4–7 μ, and produce conidia at the apex. The conidia are produced singly or in chains of two. They are straight or slightly curved, obclavate, and yellowish brown to dark or olivaceous brown in color and measure 30–120 × 9–30 μ (excluding the beak). The conidia have 4–12 transverse

septa and 0–6 longitudinal septa at which they are slightly constricted and terminate in a long hyaline beak measuring 24–210 × 2–4 μ. The beak may be simple or branched.

The optimal temperature for the growth of the fungus is in the range of 20°C–30°C, and the optimum pH for growth is 4.5. Maximum growth of the fungus is reported on mannitol followed by lactose as carbon sources, and the ammonium form of nitrogen is superior to the nitrate form.

Epidemiology and Disease Cycle

A. sesami mainly survives through seed up to 11 months, and it can also perpetuate in infected debris for nearly 11 months under field conditions (Agarwal et al. 2006, Naik et al. 2007). From infected capsules, *A. sesami* can penetrate into the seed coat, where it remains viable until germination of seed. The spores of the fungus attached to the seeds or capsule may serve to carry and disseminate the pathogen. The disease becomes most severe on plants established from seeds with 8% infection, and the disease severity increases with increased seed infection level (Ojiambo et al. 2000a, 2003, 2008). Though the infection process appears to be similar to other *Alternaria* species, culture filtrate from *A. sesami* reveals the presence of toxin, the tenuazonic acid (Rao and Vijayalakshmi 2000).

Seed infection is observed to be highest in plants inoculated between 8 and 10 weeks of age and least at 1, 6, and 12 weeks of age (Ojiambo et al. 2008). Excessive rainfall favors the development of the disease. The fungus is restricted to sesame in its pathogenicity. Distinct physiological races have not been identified, although differential virulence among isolates of *A. sesami* has been described from India and the United States.

Disease Management

Host Plant Resistance

Development and use of resistant sesame varieties is the best option. Hairy plants on the whole are reported to be free from attack due to *A. sesami*. The disease-resistant genotypes are *S. occidentalis* cvs. Heer, Regel; *S. radiatum* cvs Schum. and Thonn. and *S. malabaricum* (Shekharappa and Patil 2001b); and S-122 (Marri et al. 2012) and RT 273 (Eswarappa et al. 2011). Single dominant allele and 10 kb RAPD marker have been identified for resistance to *Alternaria* leaf spot of sesame (Eswarappa et al. 2011).

The old sesame lines *SI 948* (Kulithalai), *SI-1561*, *1683*, *1737*, *2177*, and *2381*, and Rio are reported to be resistant to the disease. Sesame varieties *Sirogoma* and *Venezuela 51*, NO 4, E-8, JT-7, JT-63-117, A-6-5, JT-66-276, Anand-9, JT-62-10, VT-43, and Anand-74 are also reported to be moderately resistant to the disease (Kolte 1985). Some other sesame genotypes that are moderately resistant to the *Alternaria* leaf spot are Navile-1, 351888, 899, 908, TC28, Madhavi, Co-1-12, Co-1-16, TC-25, and Tarikere (Basavaraj et al. 2007) and MT-15, DORS-102, DS-14, and DS-10, which show multiple-disease resistance including to *Alternaria* leaf spot disease (Jahagirdar et al. 2003). Biparental mating or diallel selective mating and heterosis breeding have been suggested for the development of *Alternaria*-resistant cultivars (El-Bramawy and Shaban 2007, 2008).

Induced Host Resistance

Resistance-inducing chemicals like salicylic acid at 1% conc. and boiagents *T. viride* and *P. fluorescens* induce systemic resistance in sesame against *A. sesami* and result in higher plant vigor index (Savitha et al. 2011, 2012). Aqueous leaf extract of neem (*A. indica*) provides the control of the *Alternaria* leaf spot disease without adversely affecting spore germination of *A. sesami*, and protection of sesame plants against *A. sesami* by neem extract is due to the stimulation of plant natural defense response as the treated sesame plants exhibit significantly higher level of enzymes like phenylalanine ammonia lyase, PO, and contents of phenolic compounds (Guleria and Kumar 2006). Similarly, it is noteworthy to observe that extract of another plant, *Mikania scandens*, when treated on inoculated sesame plants induces host resistance to the delay of the development of the *Alternaria* leaf spot (Lubaina and Murugan 2013a,b).

Chemical Control

Two sprays of mancozeb at 0.25% (Mudingotto et al. 2002, Rajpurohit 2003) or a combination of mancozeb at 0.25% plus methyldemeton at 1 mL/L (Rajpurohit 2004b) or mancozeb at 0.25% plus streptocycline at 0.025% (Shekharappa and Patil 2001a) have been found to be effective in the management of *Alternaria* leaf spot of sesame with increase in yield of sesame crop.

Cultural Control

Salt density at 2%–5% concentration can be used to sort out the infected seed from the seed lots to maintain healthy nucleus seed after further washing and drying the seed (Enikuomehin 2010). Seeds floated at 2% and 5% salt conc. are characteristically discolored, malformed, infected, and lightweight.

Experimental evidence has been presented in Nigeria that intercropping sesame with maize in a single alternate row (1:1) arrangement can be useful in reducing the severity of *Alternaria* leaf blight of sesame (Enikuomehin et al. 2010, 2011).

WHITE LEAF SPOT OR *Cercospora* LEAF SPOT

Symptoms

Small circular spots are scattered on both leaf surfaces. At first, they are minute, and later they increase in size to become 5 mm in diameter with whitish center (white spot) surrounded by a blackish purple margin (Figure 7.5). The spots may enlarge rapidly, coalesce into irregular blotches that often become about 4 cm in diameter, and are concentrically zoned. Under humid conditions, the disease becomes severe involving premature defoliation. The disease causes defoliation particularly in early maturing varieties. On petioles, the spots are elongated. Capsules show more or less circular, brown-to-black lesions (1–7 mm).

Geographical Distribution and Losses

The white spot of sesame is reported from Australia, Brazil, China, Colombia, the Dominican Republic, India, Nicaragua, Sri Lanka, Suriname, the United States, and Venezuela (Kolte 1985, Shivas et al. 1996, Verma et al. 2005).

FIGURE 7.5 *Cercospora* leaf spot of sesame. (Courtesy of Dr. Anil Kotasthane, IGKV, Raipur, India.)

The disease is endemic in most of the sesame-growing areas of Takum, Donga, Wuakeri, Bali, Kurmi, and Karim-Lamido in Taraba state and major sesame-growing regions of Nigeria, which has assumed more serious occurrence in the forest/Savannah transition zone of southwest Nigeria to which the crop has been recently introduced. It is widely prevalent in other countries of Africa (Uwala 1998, Einkuomehin 2005). The losses due to the disease in Nigeria range from 22% to 53% (Einkuomehin et al. 2002). It is reported that the disease severity in India can be as high as 53%–96% resulting in an average yield loss of 20% (Mohanty 1958, Patil et al. 2001).

Pathogen

The pathogen is *Cercospora sesami* Zimmerman (*Mycosphaerella sesamicola*). Stromata are slight to none. Conidiophores are olivaceous, septate, usually single or in fascicles of up to 10, epiphyllous, nodulose, and thickened toward the tip and measure 40–60 × 4 µ. Conidia are hyaline, cylindric, toothed upward, and commonly 7–10 septate and measure 90–135 × 3–4 µ. The pathogen is reported to sporulate well on carrot leaf decoction agar medium. *C. sesami* perpetuates through infected seed and also through plant residues in soil.

Disease Management

Some of the sesame genotypes, namely, IS 4, 15, 21, 29, 41, 41A, 41B, 128, and 128B, FS 150 (H 60-18) from Morocco, ES 234 from Mexico, and ES 242 (Precoz) from Venezuela (Kolte 1985); 65b-58, 60/2/3-1-8B, 69B-392,73a-96B from Nigeria (Poswal and Misari 1994, Nyanapah et al. 1995); and BIC-7-2, Sidhi 54, Rewa 114, and Seoni Malwa from India (Tripathi et al. 1996), have been reported to be resistant to the disease. Two sesame cultivars, E 8 and NCRTBEN-01 from Nigeria, show better stand establishment with certain degree of tolerance to the disease (Nahunnaro and Tunwari 2012a).

Many synthetic fungicides had shown promise in the management of sesame diseases (Shokalu et al. 2002). However, the high cost of such chemicals forbids their use by ordinary farmers. Seed treatment with systemic fungicides like carbendazim (0.15%) or Bayleton (0.15%) is reported to be effective in the control of the seed-borne inoculum. Sesame crop sprayed with carbendazim at 0.1% or Quintal at 0.2 gives best degree of disease management with increase in seed yield by 31.28% (Hoque et al. 2009, Palakshappa et al. 2012). Two sprays of a mixture of mancozeb at 0.2% plus endosulfan 35 EC at 1 mL/L, first spray being given at flower initiation stage and the second at pod formation stage, result in good control of insect pests and *Cercospora* leaf spot disease (Ali and Singh 2003).

Hot-water treatment of seeds at a temperature of 53°C for 30 min gives good control of the disease. Aqueous leaf extract of plants *Aspilia africana*, *Chromolaena odorata*, *A. indica*, and *Allium sativum*, when sprayed once every week, gives significant reduction in disease severity (Enikuomehin 2005, Nahunnaro and Tunwari 2012b). The plant extracts of garlic, *Ocimum*, and *Chromolaena* are comparable to synthetic fungicide (benlate) in reducing the amount of *Cercospora* leaf spot on sesame (Enikuomehin and Peters 2002, Tunwari and Nahunnaro 2014).

Plant debris should be burned after threshing and before plowing. Early-sown crop in the middle of June to first week of July is less affected due to *Cercospora* leaf spot, and these sowing dates are preferred for sowing sesame in wider row spacing of 20–30 cm in India and Nigeria (Tripathi et al. 1998a, Enikuomehin et al. 2002, Verma et al. 2005). Intercropping-induced microclimatic effects influence foliar disease severity including that of *Cercospora* leaf spot of sesame. Grain yield, weight of 1000 seeds, number of capsules/plant, and weight of seed/plant have been observed to be significantly higher in the 1:1 row arrangement than the sole crop or other row arrangements. The study made by Enikuomehin et al. (2008) demonstrates that intercropping sesame with maize in a single alternate row (1:1) arrangement can be used to reduce white leaf spot severity of sesame.

PHYLLODY DISEASE

Symptoms

Affected sesame plants express symptoms, depending on the stage of crop growth and time of infection. A plant infected in its early growth remains stunted to about two-thirds of a normal plant, and the entire plant may be affected. The entire inflorescence is replaced by a growth consisting of short,

FIGURE 7.6 Phyllody of sesame. Note the transformation of flower parts into green leaflike structures. (Courtesy of Dr. Anil Kotasthane, IGKV, Raipur, India.)

twisted leaves closely arranged on a stem with very short internodes. However, when infection takes place at later stages, normal capsules are formed on the lower portion of the plants, and phylloid flowers are present on the tops of the main branches and on the new shoots that are produced from the lower portions.

The most characteristic symptom of the disease is transformation of flower parts into green leaflike structures followed by abundant vein clearing in different flower parts (Figure 7.6). The calyx becomes polysepalous and shows multicostate venation compared to its gamosepalous nature in healthy flowers. The sepals become leaf like but remain smaller in size. The phylloid flowers become actinomorphic in symmetry, and the corolla becomes polypetalous. The corolla may become deep green, depending upon the stage of infection. The veins of the flowers become thick and quite conspicuous. The stamens retain their normal shape, but they may become green in color. Sometimes, the filaments may, however, become flattened, showing its tendency to become leaf like. The anthers become green and contain abnormal pollen grains. In a normal flower, there are only four stamens, but a phylloid flower bears five stamens. The carpels are transformed into a leaf outgrowth, which forms a pseudosyncarpous ovary by their fusion at the margins. This false ovary becomes very enlarged and crop. In Sudan, red varieties of sesame have been found to be affected to the extent of 100%.

Inside the ovary, instead of ovules, there are small petiole-like outgrowths, which later grow and burst through the wall of the false ovary producing small shoots. These shoots continue to grow and produce more leaves and phylloid flowers. The stalk of the phylloid flowers is generally elongated, whereas the normal flowers have very short pedicels. Increased IAA content appears to be responsible for proliferation of ovules and shoots. Sometimes, these symptoms are found to be accompanied with yellowing, cracking of seed capsule, germination of seeds in capsules, and formation of dark exudates on the foliage (Akhtar et al. 2009, Pathak et al. 2012).

Normal-shaped flowers may be produced on the symptomless areas of the plants, but such flowers are usually dropped before capsule formation, or the capsules are dropped later leaving the stalk completely bared.

Geographical Distribution and Losses

Prevalence of the sesamum phyllody erroneously named *leaf curl* is traced since 1908 in Mirpur Khas area of India (now in Pakistan), as cited by Vasudeva and Sahambi (1955), and a detailed historical account of the occurrence and causal agent of the disease has been reviewed earlier by Kolte (1985). It has been reported from India, Iran, Iraq, Israel, Myanmar, Sudan, Nigeria, Tanzania, Pakistan, Ethiopia, Thailand, Turkey, Uganda, Upper Volta, Venezuela, and Mexico (Kolte 1985, Salehi and Izadpanah 1992, Esmailzadeh-Hosseini et al. 2007, Akhtar et al. 2009). The first evidence of association of mycoplasma-like organism (now known as phytoplasma) with the disease was obtained in Upper Volta by Cousin et al. (1971).

Affected plants remain partially or completely sterile, resulting in total loss in yield. As much as 10%–100% incidence of the disease has been recorded in the sesame crop in India. The yield loss due to phyllody in India is estimated to about 39%–74%. The losses in plant yield, germination, and oil content of sesame seeds may be as high as 93.66%, 37.77%, and 25.92%, respectively. It is estimated that a 1% increase in phyllody incidence decreases the sesame yield by 8.4 kg under Coimbatore conditions in India. Robertson (1928) from Burma reported up to 90% incidence of the disease in the Sagaing and Lower Chin districts. A survey conducted in Thailand during 1969 and 1970 indicated that the phyllody was so severe in northeastern Thailand that farmers decreased the acreage for the sesame. Phyllody is a very serious disease, which can inflict up to 80% yield loss with a disease intensity of 1%–80% (Kumar and Mishra 1992, Salehi and Izadpanah 1992). The average phyllody incidence is reported to be about 20% with yield losses in sesame seed yield due to phyllody ranges to be 7%–28% in Pakistan (Sarwar and Haq 2006, Sarwar and Akhtar 2009).

Pathogen: Certain Strains of 16 Sr Taxonomic Group of Phytoplasma

The pathogen is now investigated to be phytoplasma (formerly referred to as mycoplasma-like organism—wall-less bacteria belonging to the class Mollicutes). Light microscopy of hand-cut sections treated with Dienes stain shows blue areas in the phloem region of phyllody-infected sesame plants (Al-Rawi et al. 2001, Akhtar et al. 2009). The phytoplasma pleomorphic bodies are reported to be present in phloem sieve tubes of affected sesame plants. Electron microscopy has revealed that the big pleomorphic bodies, ranging from 100 nm diameter to 625 nm diameter, are present in the sieve tubes. Generally, the phytoplasmas are round, but some may be 1500 nm long and 200 nm wide. Bodies with beaded structures can also be noticed. The phytoplasmas are bounded by a single unit membrane as is typical for the Mollicutes and show ribosome-like structure and DNA-like strands within. Phytoplasma cells contain one circular double-stranded DNA chromosome with a low G + C contents (up to only 23%), which is thought to be the threshold for a viable genome (Bertaccini and Duduk 2009, Weintraub and Jones 2010). They also contain extrachromosomal DNA such as plasmids. Since phytoplasmas cannot be grown in axenic culture, advances in their study are mainly achieved by molecular techniques. Molecular data on sesame phytoplasmas have provided considerable insight into their molecular diversity and genetic interrelationships, which has in turn served as a basis for sesame phytoplasma phylogeny and taxonomy. Classification of phyllody phytoplasma associated with sesame has been attributed to at least three distinct strains worldwide including aster yellows, peanut witches' broom, and clover proliferation group (Al-Sakeiti et al. 2005, Khan et al. 2007).

Based on restriction fragment length polymorphism (RFLP) analysis of polymerase chain reaction–amplified 16S rDNA, sesame phyllody phytoplasma infecting sesame in Myanmar (termed as SP-MYAN) belongs to the group 16SrI and subgroup 16SrI-B. Sequence analysis has confirmed that SP-MYAN is a member of *Candidatus Phytoplasma asteris* and it is closely related to that of sesame phyllody phytoplasma from India (DQ 431843) with 99.6% similarity (Khan et al. 2007, Win et al. 2010). RFLP profiling and sequencing reveal that phytoplasma associated with sesame phyllody in Pakistan has the greatest homology to 16SrII-D group phytoplasmas (Akhtar et al. 2009), whereas in a separate study from the same country (i.e., in Pakistan), molecular evidence

of the cause of the sesame phyllody has been found to be phytoplasma belonging to subgroup 16SrII and its sequence is essentially reported to be identical to that of the phytoplasma causing sesame phyllody in Oman (Akhtar et al. 2008). Similarly, phytoplasma causing sesame phyllody in Yazd Province of Iran belongs to the 16SrII group, which is peanut witches' broom phytoplasma (Esmailzadeh-Hosseini et al. 2007). Interestingly, in the neighboring Turkey, phytoplasma associated with sesame phyllody belongs to 16S rDNA group closely related to clover proliferation group 16SrVI-A (Sertkaya et al. 2007).

Witches' broom symptom in sesame resembling sesame phyllody in Oman is caused by the phytoplasma strains (SIL, SIF) clustered with Omani Lucerne witches' broom forming a distinct lineage separate from groundnut witches' broom and sesame phyllody (Thailand) phytoplasma strains (Nakashima et al. 1995, 1999, Al-Sakeiti et al. 2005, Khan et al. 2007).

Transmission

The pathogen is transmitted by the leafhopper vectors (order: Homoptera). In India, Thailand, and Upper Volta, sesame phyllody is transmitted by *Orosius orientalis* (Matsumura) (*O. albicinctus*), whereas in Turkey and Iran, sesame phyllody is transmitted by *Circulifer haematoceps* (Mulsant and Rey) (Dehghani et al. 2009). However, Esmailzadeh-Hosseini et al. (2007) first reported transmission of a phytoplasma associated with sesame phyllody in Iran by *O. albicinctus.* The pathogen has also been experimentally transmitted to the cotton plant by the vector *O. cellulosus* (Lindberg). Attempts to transmit the pathogen through sap in Iran and through seed in Thailand have given negative results (Tan 2010).

Epidemiology and Disease Cycle

The pathogen has a wide host range and survives on alternate hosts like *Brassica campestris* var. *toria*, *B. rapa,* and *Cicer arietinum*, which serve as source of inoculum. The pathogen is transmitted by the leafhopper, *O. albicinctus*, in most sesame-growing areas in the world as discussed earlier. Most optimum acquisition period of vector is 3–4 days, and inoculation feeding period is 30 min. The incubation period of the pathogen in leafhoppers may be 15–63 days and 13–61 days in sesame. Nymphs are incapable of transmitting the phytoplasma. Vector population is more during summer and less during cooler months.

There is a significant positive correlation between phyllody incidence with maximum and minimum temperature and negative correlation with maximum relative humidity and rainy days, which could be then consequently related to increase or decrease in vector population in the respective environmental conditions (Choudhary and Prasad 2007).

The incubation period is considerably increased during winter months (October–January) due to low temperature. Among the weather factors, the night temperature (minimum temperature) prevailing from the 30th to the 60th day after sowing is found to have a greater increase of disease incidence. The minimum acquisition feeding period has been observed to be 8 h, while the minimum infection feeding period is 30 min during May and June. Both male and female insects are equally efficient in transmitting the pathogen. The nymphs of the insect are capable of acquiring the pathogen, but they are unable to transmit it, as by the time the incubation period is completed, they reach the adult stage. Once the leafhoppers have picked up the pathogen and become infective, the adult leafhoppers remain so throughout the remainder of their lives without replenishment of the pathogen from infected plants.

Even a single leafhopper may be able to cause infection. It is interesting that leafhoppers show a marked preference for the diseased plants over healthy ones. The diseased plants have been reported to harbor an insect population about two to six times the population on healthy plants—due to higher moisture, higher nitrogen, and lower calcium and potassium contents of the diseased plants. Lower content of calcium and potassium in the diseased plants is suspected to be the factor vulnerable for easy stylet and ovipositor penetration. Higher incidence of phyllody occurs when sesame crop is

fertilized with phosphorus without nitrogen (Borkar and Krishna 2000); there also exists a positive correlation between days to maturity of sesame crop and phyllody incidence (Gopal et al. 2005).

Disease Management

Host Plant Resistance

Selections of disease-resistant sesame lines, which would flower within 40–50 days after sowing, appear to be desirable and important from the yield viewpoint under Indian conditions (Kolte 1985, Selvanarayanan and Selvamuthukumaran 2000). From India, a considerable number of genotypes such as RJS 78, RJS 147, KMR 14, KMR 29, Pragati, IC 43063 and IC 43236 (Singh et al. 2007), SVPR-1 (Saravanan and Nadarajan 2005), AVTS-2001-26 (Anandh and Sevanarayanan 2005), Swetta-3, RT-127, No. 171 (Dandnaik et al. 2002), TH-6 (Anwar et al. 2013), and three wild species, that is, *S. alatum*, *S. malabaricum*, and *S. yanaimalaiensis*, are resistant to phyllody with mean incidence below 5%, which can be utilized as donor parents in resistance breeding to phyllody disease (Saravanan and Nadarajan 2005, Singh et al. 2007). A single recessive gene governs resistance in cultivated varieties (KMR 14 and Pragati), whereas wild species possess a single dominant gene conferring resistance to phyllody (Singh et al. 2007). Phyllody resistance in a land race of sesame is reported to be under the control of two dominant genes with complementary (9:7) gene action (Shindhe et al. 2011). Some genotypes in India have not been observed to show phyllody symptoms. Such genotypes are Ny-9, Sirur, Local, NKD-1037, K-50, TC-25, RT-15H, OCP-1827, No. 5, No. 16, No. 17, No. 18, No. 21, No. 23, and No. 24 (Dandnaik et al. 2002). Interspecific hybrids between *S. alatum* and *S. indicum* are, however, moderately resistant to phyllody (Rajeshwari et al. 2010). Advanced phyllody disease–resistant sesame mutant lines with earliness, more capsules, and high harvest index have been developed in Pakistan under the series NS11-2, NS11 P2, NS100 P2, NS 103-1, and NS240 P1 and phyllody disease–resistant sesame. These mutant lines can be of great potential use in breeding for disease resistance (Sarwar and Akhtar 2009).

Some other sesame lines as JT-7, JT-276, and N-32, though not resistant to the disease, have been found useful to escape the disease (Kolte 1985).

Chemical Control

Insect vector management is the method of choice for limiting the outbreaks of phytoplasmas in sesame. At the time of sowing, soil may be treated with Thimet® 10 G at the rate of 10 kg/ha or with Phorate 10 G at the rate of 11 kg/ha or with Temik® 10 G at the rate of 25 kg/ha to get the management of the disease through vector control (Nagaraju and Muniyappa 2005). An effective degree of management is obtained if the aforementioned treatment is combined with spraying of the crop with Metasystox® (0.1%) or with any other effective chemical (Misra 2003, Rajpurohit 2004b).

Tetracycline sprays at 500 ppm concentration at the flower initiation stage have proved to be effective against phyllody, but recovery is temporary.

A possibility of biochemical control by spraying manganese chloride has been indicated. It appears that manganese chloride oxidizes the phenol and protects or inhibits the enzymes, brining the auxin level to normal. Once hyperauxin is oxidized, the plant can gain its normal conditions (Purohit and Arya 1980).

Cultural Control

An appropriate sowing date may be useful in avoiding severe occurrence of the disease. The incidence of the disease is reported to be reduced considerably by sowing the crop in early August under Indian conditions. The reduced population of the vector in the growth period of sesame plants is perhaps important in keeping the disease under check (Mathur and Verma 1972, Nagaraju and Muniyappa 2005).

OTHER SESAME DISEASES

Other diseases of sesame are given in Table 7.1.

TABLE 7.1
Other Diseases of Sesame

Disease	Pathogen/Causal Agent	Geographical Distribution	Disease Description	References
Powdery mildew	*Oidium erysiphoides* (Fr.) *O. sesami* (Sreenivasulu et al.) *Sphaerotheca fuliginea* (Schlecht) Pollacci *Leveillula taurica* (Lav.) Trnaud *Erysiphe cichoracearum* DC	Ethiopia, Australia, India, Iraq, Japan, Tanzania, and Uganda Malawi, India, Sudan India, Sicily, Venezuela China, Thailand	Symptoms of the disease start as small whitish spots on the upper surface of the leaves. The spots coalesce to form a single spot, finally covering the entire leaf surface with dirty white fungal growth. Generally, the mildew is confined to the upper surface of the leaves. The perithecial stage may or may not be observed on sesame leaves. The disease causes a yield loss of 42%. For every 1% increase in disease severity, there is a yield loss of 5.63 kg/ha. There is a significant negative correlation between days to maturity and powdery mildew occurrence and severity. Use of host resistance appears to be the most promising method for the control of the powdery mildews of sesame. Several genotypes from India, namely, BB 3-8, TKG-22, NSKMS-260, G-55, Si 3315/11, hybrid Si 3315/11xSVPR, Co1, hybrids Co1 x S13216 and Co1 x YLM123, are resistant to powdery mildew. Inheritance of resistance/tolerance to powdery mildew reveals that susceptibility is dominant over tolerance and is controlled by two independent recessive genes with complementary epistasis. The crop can be protected from the powdery mildews by spray application of wettable sulfur (0.2%) fungicides or by dusting sulfur dust at the rate of 20 kg/ha or Karathane spray (1%).	Kolte (1985), Rao and Rao (2001), Kumaresan and Nadarajan (2002), Sarvanan and Nadarajan (2004), Gopal et al. (2005), Biju et al. (2011), Sravani et al. (2012), Rao et al. (2011, 2012)
Corynespora blight	*Corynespora cassiicola* (Berk. and Curt.) Wei.	Colombia, India, Tanzania, the United States, Venezuela	Dark, irregularly shaped spots appear on leaves and stems. They enlarge, become brown with light centers, and coalesce forming a blotchy configuration. Extensive defoliation occurs and the affected plants die. Affected stems are bent irregularly on the lesions. Cankers of various sizes also appear on the stem. In mature plants, the infected stem cracks lengthwise and breadthwise. The pathogen perpetuates through plant debris and infected seeds. It is, however, inactivated when infected seeds are stored at 26°C–28°C with 50% relative humidity. Despite having evidence of genetic diversity among isolates of *C. cassiicola*, there are no correlations between the morphological characteristics or rDNA-ITS region sequences and their host or geographical origin, ISSR markers being useful for intraspecies population studies in the pathogen. Genotypes IC-205292, IC-205 561, IC-205633, and Krisna are moderately resistant to the disease. Seed treatment with carbendazim at 0.1% and spraying the crop with mancozeb can be effective in disease management.	Kolte (1985), Verma et al. (2005), Choudhary et al. (2006), Qi et al. (2011), Singh et al. (2000)

(*Continued*)

TABLE 7.1 (*Continued*)
Other Diseases of Sesame

Disease	Pathogen/Causal Agent	Geographical Distribution	Disease Description	References
Brown angular leaf spot	*Cylindrosporium sesami* Hansford.	Nigeria, Saudi Arabia, Sudan, the United States, Venezuela; epiphytotic occurrence reported in the United States and Venezuela	Spots on the leaves are water soaked, brown, and limited to veinal areas and assume an angular shape. They are 2–20 mm in diameter and may enlarge rapidly to coalesce into extensive necrotic areas. In the ease of severe infection, defoliation occurs. The upper surface of the spot on the leaves shows the presence of dark subepidermal fungal acervuli. The affected leaves frequently show the association of spots caused by *A. sesami* and/or *C. sesami.* The fungus survives through the seed. Selection V-16 is reported to be resistant to the disease. Seed treatment with commonly used fungicides (1–3 g/kg seed) can be effective for the control of the disease.	Kolte (1985), Verma et al. (2005)
Angular leaf spot	*Cercospora sesamicola* Mohanty	Nigeria, Nicaragua, India, Panama	Leaf spots are angularly limited by leaf veinlets measuring 1–8 mm in size. Initially, the spots are minute and become visible as chlorotic lesions on the upper surface of the leaves; later, when the affected tissues become necrotic, the color of the spots changes to dark brown, whereas on the corresponding lower surface of the leaves, the color of the spots remains olivaceous brown. The fruiting bodies of the fungus might become visible on both surfaces of the leaves but chiefly on the lower surface. *C. sesamicola* perpetuates only through viable sclerotia in crop debris and possibly through infected seeds.	Kolte (1985), Verma et al. (2005)
Aerial stem rot	*Helminthosporium sesami* Miyake	Philippines, China, Japan, the United States	Leaf lesions vary from small brown spots 1 mm in diameter to large elongated lesions of about 2–20 mm. Lesions of the stem range from small flecks 1 mm in diameter to large, sunken, dark-brown spots 10 × 40 mm in size. High humidity favors spread of the disease, and young plants are much more susceptible than mature ones. Plants less than 21 days old are more susceptible than mature ones. Nitrogen increases the susceptibility. Phosphorus or potash alone or phosphorus and calcium show decrease in the severity of infection.	Kolte (1985)
Stem blight	*Alternaria alternata* (Fr.) Keissier	India	Blighting of stem is the major symptom. Seed treatment with captan at 3 g/kg seed and foliar spray with copper oxychloride (0.3%) at 20, 40, and 60 days after sowing.	Rao and Rao (2002)

(*Continued*)

TABLE 7.1 (*Continued*)
Other Diseases of Sesame

Disease	Pathogen/Causal Agent	Geographical Distribution	Disease Description	References
Bacterial blight	*Xanthomonas campestris* (Pammel) Dowson pv. *sesami* (Sabet and Dowson) dye	Sudan, India, Pakistan, the United States, Venezuela	Small, water-soaked, light-brown lesions develop on the margin of the cotyledonary leaf about 10–12 days after sowing. The lesions may spread, rapidly covering the entire cotyledons, which consequently become dry. About 4% mortality due to the disease in 4–6-week-old seedlings has been reported. If the seedling survives, dark brown, water-soaked spots also appear on the true leaves. In a severe infection, the lesions extend to the stem through the petiole, leading to the formation of brown discoloration, resulting in systemic invasion and death of the plant. The disease is known in Sudan as *Marad ed Dum*, meaning thereby the blood disease, due to the red color of infected plant tissue. Seed can carry the pathogen up to a period of 16 months. A weed plant, *Acanthospermum hispidum*, is reported to be susceptible to *X. campestris* pv. *sesami*. This host acts as a source of survival of the bacterium in its dried leaves from year to year. The bacterium does not survive in sesame plant debris in soil from year to year. The bacterium enters the host primarily through stomata and quickly becomes vascular. The secondary spread is by spattering rains. High temperature and humidity favor the disease. Seedling infection of sesame is most severe at soil temperature of 20°C. Infection does not take place when soil temperature is 40°C. The disease also becomes severe when the soil moisture is 30%–40% and relative humidity is 75%–87%. Seedling infection can be used as a valid test for determining the resistance of sesame to this disease. Three sesame genotypes SG-34, SG-22, and Sg-55 are resistant to bacterial blight. Chemical or antibiotic seed treatment or hot-water treatment of seed and antibiotic sprays to check secondary spread are the same as described for bacterial leaf spot. Streptocycline seed treatment for 2 h followed by three sprays at 10-day interval of streptocycline plus copper oxychloride effectively controls the disease.	Kolte (1985), Rai and Srivastava (2003), Samina et al. (2007), Isakeit et al. (2012), Naqvi et al. (2012)
Bacterial wilt	*Ralstonia solanacearum* biovar III	Andaman and Nicobar Islands and Assam State of India, Iran, Japan, South China	Soil treatment with combinations of bleaching powder, streptocycline, and mustard cake significantly controls the disease. The variety Pb Til No1 is less affected by this bacterium.	Dubey et al. (1996), Hazarika and Das (1999), Hua et al. (2012)

(*Continued*)

TABLE 7.1 (*Continued*)
Other Diseases of Sesame

Disease	Pathogen/Causal Agent	Geographical Distribution	Disease Description	References
Leaf curl	Leaf curl virus	India, Nigeria, Pakistan, Sierra Leone, Tanzania, Uganda, Zaire	Symptoms of the disease are characterized by curling of the leaves and marked thickening of the veins on the underside of the leaf, combined with a reduction in leaf size. Leaves may also become leathery, possessing dark-green color. Severely affected plants remain stunted and bear few flowers and capsules. The disease is considered to be a serious one causing considerable reduction in yield, especially when the infection takes place at the early stage of crop growth. The incidence of the disease in certain years is reported to be to the extent of 60% in India. The sesamum lines NP-6, T-13-3-2, 65-1/11, 67-13-1/2-1, Entebbe ex Uganda, and sesame mutant lines NS 11-P2, NS100-P2, NS103-1, and NS 240 P1 (developed in Pakistan) have shown a high degree of resistance to leaf curl. Inheritance of resistance to leaf curl from hybridization between resistant *S. radiatum* and *S. indicum* reveals that resistance to leaf curl and lodging is controlled by two independently assorting genes with both dominant alleles F and *L*, producing plants that are resistant to leaf curl and lodging. Two foliar sprays of the insecticide methyl demeton at 1 ml/L reduce leaf curl incidence.	Kolte (1985), Sarwar and Akhtar (2009), Rajpurohit (2004b), Falusi and Salako (2003)
Mosaic disease	Sesame mosaic virus	Czechoslovakia, India	Conspicuous chlorotic areas of irregular shape appear on the leaf lamina. The basal portion of the leaf may remain green, while the top portion and margins of the leaves become yellow, interveinal areas are usually yellow, and the young leaves may become completely yellow. As the disease progresses, top leaves become gradually smaller in size, and plants may not grow further. The disease is caused by the virus that is transmitted through sap and grafting.	Kolte (1985)
Cowpea aphid–borne mosaic virus disease	Cowpea aphid–borne mosaic virus	Paraguay	Yellowing and curling down of sesame leaves and shortening of the internodes.	Gonzalez-Segnana et al. (2011)

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Section VI

Safflower

Safflower (*Carthamus tinctorius* L.) is a highly branched, herbaceous, self-compatible, thistlelike annual plant. It varies greatly in height ranging from 0.3 to 1.5 m. It belongs to the family Compositae with 12 pairs ($2n = 24$) of basic chromosome number. Its haploid genome is approximately 1.4 Gb (Garnatje et al. 2006). Li et al. (2011) found that there are at least 236 known micro-RNAs (mRNAs) expressed in the safflower, 100 of which are conserved across the plants. The safflower genome is large and complex and it has not been fully sequenced, and relatively little is known about its encoded genes. As of October 2011, only 567 nucleotide sequences, 41,588 expressed sequences tags (ESTs), 162 proteins, and 0 genes from *C. tinctorius* have been deposited in the National Center for Biotechnology Information (NCBI)'s GenBank database. Interestingly, abundant genomic data for *C. tinctorius* and comprehensive sequence resources for studying the safflower transcriptome datasets have been generated that will serve as an important platform to accelerate studies of the safflower genome (Huang et al. 2012). The plant has many branches each terminating in a flower. The inflorescence is a dense capitulum of numerous regular flowers. The flowers are self-pollinated, but cross-pollination to the extent of 16% may occur under natural conditions. Each branch usually has 1–5 flower heads containing 15–20 seeds per head. The seed is ovate, having a flat top with longitudinal ribs, and represents the Cypsela type of fruit. The cultivated forms of safflower are supposed to have originated either from *Carthamus lanatus* Linn or from *C. oxyacantha* Bieb, evidently in two primary centers of origin—the mountainous regions of Afghanistan and of Ethiopia. Safflower cultivation has now extended over many parts of the world, both in the tropics and in the subtropics in more than 60 countries worldwide commercially producing about 600,000 tons seed yield annually. India (producing over half of the world produce), the United States, and Mexico are the three leading producers, with Ethiopia, Kazakhstan, China, the Arab world, Argentina, and Australia accounting for most of the remainder (Yadava et al. 2012). Safflowers have long taproots that facilitate water uptake in even the driest environments enabling these crops to be grown on marginal lands where moisture would otherwise be limited. Thus, safflower is a drought- and salt-tolerant crop. It can be grown in a range of soil types, but well-drained medium to heavy textured soils are best suited for its growth. Earlier, this crop had been grown for its flowers that can be used for dyes as well as in teas and as food additive. Extracts from the florets have been used to reduce

hypertension and blood cholesterol levels. Currently, safflower is preferred for its high-quality seed oil that is rich in polyunsaturated fatty acids. Prof. Paulden Knowles, the *father of California safflower*, has accumulated and documented a large collection of safflower germplasm that is currently being maintained by the United States Department of Agriculture (USDA) for further use in research towards improvement of safflower crop. Such a collection is also being maintained at National Bureau of Plant Genetic Resources (NBPGR), New Delhi, and Indian Institute of Oilseeds (ICAR), Hyderabad, India. Safflower diseases are described in this chapter.

8 Safflower Diseases

Alternaria BLIGHT

Symptoms

Seedlings from severely affected plants show lesions on the hypocotyls and/or cotyledons. Dark necrotic lesions measuring up to 5 mm in diameter may be formed on hypocotyls. Both hypocotyl and cotyledonary symptoms are commonly observed in the same plants. In some instances, the hypocotyl infection results in damping-off of the seedlings. In mature plants, small brown to dark-brown concentric spots of 1–2 cm diameter appear on leaves. The center of the mature spots is usually lighter in color. The spots frequently coalesce into large irregular lesions bearing the spores of the fungus. The fully mature spots tend to develop shot holes, and in severe infections, irregular cracking of the leaf blade occurs. The stems and petioles suffer less severe damage with elongated spots. On flower heads, the fungus first attacks the base of the calyx and later spreads to other parts of the flower. Infected flower buds shrivel without opening, and seeds obtained from a severely infected crop may show a dark sunken lesion on the testa.

Geographical Distribution and Losses

Alternaria leaf blight of safflower was first reported from India by Chowdhury (1944). The disease is now reported from all over the world from safflower-growing countries such as Argentina, Australia, Ethiopia, Israel, Italy, Kenya, Pakistan, Portugal, Russia, Spain Tanzania, the United States, and Zambia. The effect of the disease is reported to be quite serious in the northern Great Plains area of the United States (Bergman and Jacobsen 2005) and in the states of Bihar and Madhya Pradesh of India. Reports of severe damage of experimental safflower crops due to the disease have been made from Kenya and Tanzania in East Africa. Yields of safflower infected by the disease may be reduced in highly susceptible varieties by 50%–90% when a week of humid weather occurs following flowering but before maturity. A significant negative correlation has been established between disease severity and yield (Chattopadhyay 2001). Seeds of affected plants become discolored, showing reduced oil content with significant increase in the level of free fatty acids in the seeds that adversely affect the seed germination.

Pathogen

The pathogen is *Alternaria carthami* Chowdhury.

The mycelium is septate inter- and intracellularly with slight constrictions at the septa. It is subhyaline when young but becomes dark colored on maturity. The conidiophores are stout; erect; rigid; unbranched; septate; straight or flexuous, sometimes geniculate; and brown or olivaceous brown, paler near the apex, and arise singly or in clusters through the epidermis or stomata. The conidiophores are sometimes swollen at the base and measure 15–85 µm in length and 6–10 µm in width.

The conidia are borne on conidiophores and are solitary or in very short chains. They are smooth, straight or curved, obclavate, and light brown and translucent in shade and possess a long beak. The conidia sometimes show constrictions at the septa. They measure 36–171 µm (with beak) and 36–99 µm (without beak) in length and 12–28 µm in width. The spores have 3–11 transverse septa and up to 7 longitudinal or oblique septa. The beak of the spores is 25–160 µm long and 4–6 µm thick at the base—tapering to 2–3 µm with up to 5 transverse septa. The beak is almost hyaline at the apex

and light brown near the base. Some spores may be seen without beaks. Conidial beaks may form chlamydospores in culture. The optimum temperature at which the fungus grows is in the range of 25°C–30°C. It also tolerates a wide pH range, though maximum growth occurs at pH 6.0.

Epidemiology and Disease Cycle

The pathogen survives through seed as well as viable conidia of *A. carthami* on debris of naturally infected susceptible safflower varieties (Prasad et al. 2009, Gayathri and Madhuri 2014). *A. carthami* is readily isolated from seeds using relatively simple techniques. Isolation methods used in conjunction with the planting of seed to assess seedling health would appear to offer the most reliable means of detecting the presence of *A. carthami* in seed (Awadhiya 2000). The primary infection develops from infested seeds obtained from affected plants. Spines present on the leaf margin are the site of infection by the pathogen (Borkar 1997). An opening diameter of 120 µm at the apex of individual spines is a prerequisite for infection through spines. Spine apex openings vary with the location of the spine on the leaf margin and the relationship between the position of the spines on the leaf margin, and infection is governed by the diameter of the openings at the spine apex. Spores produced on lesions developing in plants grown from infected seeds become secondary sources of inoculum, and the pathogen occurs on the crop throughout the growing season. The macrolide antibiotics brefeldin A (BFA) and 7-dehydrobrefeldin A (7-oxo-BFA) have been characterized as phytotoxins and pathogenicity factors from *A. carthami*; the toxins are known to inhibit the endoplasmic reticulum–Golgi flux and processing (Kneusel 1994, Driouich et al. 1997). Rains coupled with high relative humidity above 80% and temperature in the range of 21°C–32°C under irrigated conditions accompanied by heavy dew or frequent showers, cyclonic storms especially at seedling, and grain formation stages favor the disease (Sastry and Chattopadhyay 2005, Gud et al. 2008, Murumkar et al. 2008a).

Disease Management

Host Plant Resistance

There is a considerable variation in response to *A. carthami* infection by a range of safflower varieties (Muñoz-Valenzuela et al. 2007, Thomas et al. 2008). Some genotypes such as EC 32012, NS 133, CTS-7218, HUS 524, and CTV 251 (Desai 1998); GMV 1175, GMV-1199, and GMV-1585 (Indi et al. 2004); GMV-5097, GMV-5133, and GMV-7017 (Murumkar et al. 2009a); and Ellite Line 21–33 (Pawar et al. 2013) show high degree of tolerance to *A. carthami* under high disease pressure and are identified as the most promising genotypes to be used in breeding program for incorporation of resistance to the disease. Semispiny to nonspiny genotypes of safflower are known to show a variable degree of tolerance to *A. carthami* infection. It is possible to combine high yield with high degree of tolerance to the disease (Mundel and Chang 2003, Harish Babu et al. 2005). Four wild *Carthamus* species, namely, *C. palaestinus*, *C. lanatus*, *C. creticus*, and *C. turkestanicus*, are reported to be immune to *Alternaria* leaf spot in laboratory as well as under field screening. Twenty-four F1s derived from crosses between *C. tinctorius* × *C. creticus*, *C. tinctorius* × *C. oxyacantha*, *C. tinctorius* × *C. turkestanicus*, *C. tinctorius* × *C. lanatus* × *C. palaestinus*, and *C. oxyacantha* × *C. tinctorius* have been screened to show no infection (immunity) by *A. carthami*. These resistant lines would serve as base material in disease resistance breeding to tag the resistant genes at molecular level for marker-assisted selections in the field for *Alternaria* blight resistance (Prasad and Anjani 2008a).

Seedling resistance of safflower genotypes to *A. carthami* is reported to be monogenic recessive, whereas adult plant resistance is under the control of two duplicate loci where at least one locus at homozygous recessive conditions confers the adult plant resistance (Gadekar and Jambhale 2002a).

Production of plants resistant to *A. carthami* via organogenesis and somatic embryogenesis (Kumar et al. 2008) and molecular breeding has paved the way for possible transgenic safflower

plants that can be used to breed for *Alternaria* blight resistance in safflower. The cloned esterase gene degrading the BFA (phytotoxin and pathogenic factor) provides the basis for generation of transgenic safflower plants (Kneusel et al. 1994).

Chemical Control

Seed treatment with mefenoxam + thiram or with difenoconazole + mefenoxam is effective in reducing the primary infection (Jacobsen et al. 2008). Secondary infection can be controlled by spraying the crop with any of the foliar fungicides such as mancozeb at 0.25%, difenoconazole at 0.5%, AAF (carbendazim 12% + mancozeb 63%) at 0.2% (Sumitha and Nimbkar 2009), and fosetyl at 0.1% (Bramhankar et al. 2001). For effective and economic management of the disease, the first spray of carbendazim at 0.1% should be given immediately after disease appearance (generally at rosette stage, i.e., 25 days after sowing) followed by need-based second and third spray at 15 days after the first spray and during flowering and seed setting stage, respectively (Murumkar et al. 2008a, 2009a). Fungicide application results in lower pathogen transmission from plants to seeds and from seeds to plants (Bramhankar et al. 2002). Two newer fungicides azoxystrobin (Quadris) and pyraclostrobin (Headline) have been registered as foliar fungicides for the control of *Alternaria* blight of safflower crop in Australia as reported by Bergman and Jacobsen (2005). These fungicides are also reported to be effective for the control of the *Alternaria* blight of safflower in the United States (Wunsch et al. 2013).

Cultural Control

The occurrence of the disease may be prevented by using disease-free seeds for sowing. Such seeds can be obtained from early-sown dry land crops rather than from irrigated areas. Alternatively, the infested seeds may be treated with fungicides as discussed earlier. Crop rotation and strict sanitation of crop debris effectively manage the disease. Basal soil application of KCl at 67 kg/ha significantly reduces the disease severity and increases the safflower seed yield (Chattopadhyay 2001). This practice can be integrated with spray application of effective fungicides and suitable sowing dates for better disease management.

Effect of Plant Extracts

Antifungal activities of extracts of various plants such as *Nerium*, *Datura*, garlic bulb, *Lantana*, *Eucalyptus*, neem, onion bulb, and *Ocimum* species have been demonstrated against *A. carthami*, which can be exploited further for practical disease management (Shinde et al. 2008, Ranaware et al. 2010, Taware et al. 2014).

Fusarium WILT

Symptoms

Symptoms of the disease are manifested at all stages of growth. In the seedling stage, cotyledonary leaves show small brown spots either scattered or arranged in a ring on the inner surface, and they may become shriveled and brittle and sometimes tend to become rolled and curved. The seedlings that survive the fungal attack regain vitality at the early stage of blossoming and again show symptoms of the disease at the time of seed setting. The symptoms become quite distinct when the plants are in the 6th to 10th leaf stage and about 15 cm in height. Four important characteristics of the symptoms may be helpful to identify the disease at this stage. These are (1) unilateral infection on branches and leaves, (2) golden-yellow discoloration of the leaf followed by wilting, (3) epinasty, and (4) vascular browning appearing only on one side of the root and stems of plants with unilateral top symptoms. The symptoms develop in acropetalous succession. The reddish-brown vascular discoloration of the root, stem, and petiole tissue of infected plants will vary considerably in intensity, depending on varietal reaction, severity of infection, and environmental conditions. On older plants,

the lateral branches on one side may be killed, while the remainder of the plant apparently remains free from the disease. Such plants may show partial recovery between bud formation and early blossoming, but the symptoms may reappear later. The severely infected plants produce small-sized flower heads that are partially blossomed. A large number of ovaries fail to develop seeds, or they may form blackish, small, distorted, chaffy, and abortive seed.

Geographical Distribution and Losses

Fusarium wilt of safflower was first observed in the Sacramento Valley of California, USA, in 1962 (Klisiewiez and Houston 1962) and in India in 1975 (Singh et al. 1975). The disease is also reported from Egypt (Zayed et al. 1980). Now, it is identified to be the most serious disease in all safflower-growing areas in India (Murumkar and Deshpande 2009). Plants grown from infected seed seldom survive beyond the seedling stage, thereby indicating that losses in stand of the crop may occur when infected seed is sown. The disease incidence in the United States is reported to be 10%–20% in most fields and as high as 50% in some fields. Yield losses may reach to 100% if susceptible varieties are grown in fields with a history of severe *Fusarium* wilt (Sastry and Chattopadhyay 2005). In India, it has appeared as a serious threat to safflower cultivation, destroying up to 25% of plants, amounting to considerable yield loss in the Gangetic valley. Fusarial mycotoxins, namely, diacetoxyscirpenol, T-2 toxin, and 12,13-epoxytrichothecene, have been reported to be produced in sufficient quantities on infested seeds of safflower in storage to be capable of causing mycotoxicosis.

Pathogen

The pathogen is *Fusarium oxysporum* Schlecht. f. sp. *carthami* Klisiewicz and Houston.

The fungus is readily isolated from diseased plant parts on potato dextrose agar (PDA). The mycelium is delicate pink en masse or white usually with a purple tinge, sparse to abundant, branched, and septate. Microconidia are borne on simple phialids arising laterally on the hypha or on short sparsely branched conidiophores, abundant, oval to elliptical, one celled, and slightly curved and measure 5–16 × 2.2–3.5 µm. The macroconidia are hyaline, may be up to 5 septate but are mostly 3 septate, are constricted at septa, are borne in sporodochia, are straight or curved, are often pointed at the tip with rounded base, and measure 10–36 × 3–6 µm mostly 28 × 4–5 µm. Chlamydospores are one celled, smooth, and faintly colored and measure 5–13 × 10 µm in size. They are formed abundantly and are both terminal and intercalary, usually solitary but occasionally could be formed in chains (Sastry and Chattopadhyay 2005).

Epidemiology and Disease Cycle

The fungus perpetuates through seed as well as through soil. Mycelium and spores contaminate the seed surface, but hyphae are also reported to be present in the parenchymatous cells of the seed coat of infected seed. The survival of the fungus through soil is mainly through chlamydospores in plant debris. Penetration by the pathogen, which makes its entry into host cells by mechanical means, is easier when the plants are in the seedling stage and tissues are soft. Shriveling of cortical cells is noted in the case of infected plants. It appears that the infection is facilitated by production of polygalacturonase, pectin methyl esterase, cellulase, and protease enzymes. Mycotoxins—diacetoxyscirpenol and T-2—have been detected in diseased safflower plants. The pathogen is also reported to secrete diacetoxyscirpenol, T-2 toxin, fusaric acid, and lycomarasmin in culture filtrate. But the exact role of either enzymes or toxins, as earlier, is little worked out or not known. It is, however, reported that the virulence of *F. oxysporum* f. sp. *carthami* is directly correlated with the amount of fusaric acid it produces. It is also reported that the virulence is lost if fusaric acid production is prevented. The amount of seed infection per head may be limited by the extent of fungus spread in vascular

tissue in the stem or lateral branch and seed head. The fungus apparently invades the seed through the vascular strands that extend into the seed through the pericarp–receptacle junction. Fungus spread in the tissue of pericarp and seed coat is intra- and intercellular. Isolates of *F. oxysporum* f. sp. *carthami* collected from different geographical areas have been found to show variation in morphology, culture characteristics, and pathogenicity (Sastry and Chattopadhyay 2003, Prameela et al. 2005, Murumkar and Deshpande 2009, Raghuwanshi and Dake 2009, Somwanshi et al. 2009). The fungus is specific in its pathogenicity on safflower and six other species of *Carthamus*. Four physiological races have had been distinctly identified by differential reactions of safflower varieties (Gila, Nebraska 6, UC-31 and US Biggs) in the United States to 14 isolates of *F. oxysporum* f. sp. *carthami* (Kilsiewicz 1975). *In India, also four distinct races have been identified based on differential reactions of 54 pathogen isolates of the pathogen using four (96-508-2-90, A1, DSF-4, and DSF-6) differential safflower lines. Molecular analysis of genetic variability using random amplified polymorphic DNA (RAPD), microsatellite, and ITS-RLFP markers has revealed three distinct groups among 54 isolates of F. oxysporum* f. sp. *carthami* (Prasad et al. 2004, 2007). *Thus a variety that exhibits resistance in one area may show susceptibility in another area.* The disease has been found associated with soils having a pH 4.3–5.0. In India, the disease is more prevalent along the Ganges River in acidic soils. The disease is also favored by high nitrogen and warm, moist weather. The wilt is reported to be more severe in fallow land and less severe where paddy or millets are cultivated before safflower. In uplands where the soil is neutral to alkaline and clay in texture, the disease incidence is reported to be low (Kolte 1985). Disease severity is favored by high temperature stress, poor drainage, and soil compaction. Any factor that contributes to reduced rate of root growth increases the plant's susceptibility to *Fusarium* wilt. High plant population also increases plant stress and favors infection. The effect of *F. oxysporum* f. sp. *carthami* is most apparent during flowering when plants and its productivity are more sensitive to stress. The disease severity decreases with lowering of temperature (from 21°C to 15°C) during the end of December to the first week of February, but it can increase as the temperature increases (23.6°C) under Indian conditions. Seedlings show less susceptibility to the disease with increase in age, and it differs with respect to different varieties and inoculum density (Sastry and Chattopadhyay 1999a).

Disease Management

Host Plant Resistance

Water culture technique using pathogen culture filtrate at 3.5% is proved to be very useful in screening for resistance of large number of genotypes of safflower (Shinde and Hallale 2009, Waghmare and Datar 2010). Thus, sources of resistance to *Fusarium* wilt disease in wild and cultivated *Carthamus* species have been identified. Wild safflower species like *C. oxyacantha*, *C. lanatus*, *C. glaucus*, *C. creticus*, and *C. turkestanicus* are immune to wilt. Resistant plants have been obtained by selection and reselection from advanced breeding lines derived from crosses of *C. oxyacantha* × *C. tinctorius* and *C. tinctorius* × *C. turkestanicus*. Some of the most promising safflower genotypes that are highly resistant to wilt are GMU-1553 (Gadekar and Jambhale 2002b); 86-93-36A, 237550, VI-92-4-2, and II-13-2A (Sastry and Chattopadhyay 2003); GMU-1702, GMU-1706, and GMU-1818 (Chavan et al. 2004); 96-508-2-90 (Anjani et al. 2005); HUS 305 (Sastry and Chattopadhyay 2003, Raghuwanshi et al. 2008, Singh et al. 2008b); WR-11-4-6, WR-8-24-12, WR8-19-10, WR-4-6-5, WR-5-20-10, and WR-8-17-9 (Singh et al. 2008b); released hybrids DSH-129, NARI-NH-1, and NARI-H-15; and released cultivars A-1, PBNS-40, and NARI-6 (Murumkar et al. 2008b, 2009b, Prasad and Suresh 2012). Information has been generated on the use of molecular markers for genotyping safflower cultivars (Sehgal and Raina 2005) as well as for characterization of safflower germplasm (Johnson et al. 2007).

C. lanatus ($2n = 22$) and the alloploid, produced after colchicine treatment of seedlings from a cross *C. lanatus* × *C. tinctorius* ($2n = 24$), are highly resistant to the disease. The disease resistance in the alloploid appears to be governed by dominant genes contributed by the

C. lanatus genome. Accumulation of antifungal compound, carthamidin (4. 5, 7. 8-tetrahydoxy flavone), in infected plants has been found responsible for resistance of the plants to infection. Resistance to *F. oxysporum* f. sp. *carthami* in some genotypes is governed by two dominant genes with complementary type of gene action, whereas in others, it is governed by inhibitory type of gene action (Shivani et al. 2011) and in still others seedling resistance is reported to be simple monogenic dominant, whereas adult plant resistance is found to be under the control of epistatic nonallelic interactions (Gadekar and Jambhale 2002b). The development of long-term wilt-resistant varieties may, however, be impeded, if additional races evolve in the natural population of *F. oxysporum* f. sp. *carthami* (Kolte 1985).

Chemical Control

Seed treatment with fungicides such as captan; carboxin, thiram, or a mixture of carboxin + thiram; benomyl; and carbendazim + mancozeb at 0.1% or 0.2% can reduce surface contamination by *F. oxysporum* f. sp. *carthami* and becomes effective in eliminating the pathogen from the seed, but all these are more effective when combined with wilt-tolerant varieties or cultural practices (Sastry and Jayashree 1993, Govindappa et al. 2011b).

Cultural Control

Nonhost crops such as chickpea, lentil, pea, and wheat, usually grown with safflower as a mixed crop or crop succession of safflower with these crops in India, have been found to increase safflower yield with a decrease in the wilt incidence by the secretion of compounds inhibitory to the growth of the pathogen (Kolte 1985, Sastry and Chattopadhyay 1999a, Sastry et al. 1993). Additionally, chickpea and wheat in its rhizosphere increase the population of antagonistic microflora that subsequently check the growth of the pathogen significantly. Exudates and extractives of the roots of *Ruellia tuberosa* L. show significant protective and curative action against the safflower wilt. The root extractive shows the potentiality of a foliar fungicide. The inhibitory effect of *R. tuberosa* on *F. oxysporum* f. sp. *carthami* is attributed to the 2,6-dimethoxy quinone, acacetin, and C16-quinone contents of root exudates and extractives. It is reported that wilt of safflower can be controlled by planting *R. tuberosa* (a local weed found in India) in the safflower field (Kolte 1985). Another method to control the disease is topping of the plants at the seedling stage to encourage vegetative growth: safflower produces a chemical imparting resistance to the plant at the flowering stage that increases when there is more vegetative growth. Soil solarization involving the method of covering the deep ploughed and irrigated fields with transparent polyethylene sheets for 6 weeks during the peak summer month is useful in causing the temperature in the soil beneath to rise to 40°C–53°C that sufficiently kills *F. oxysporum* f. sp. *carthami*. Soil solarization also stimulates the population of antagonistic microflora against the pathogen and reduces inoculum density of the pathogen (Sastry and Chattopadhyay 1999b, 2001).

Biological Control

Trichoderma harzianum, *Bacillus subtilis*, and *Pseudomonas fluorescens* (Gaikwad and Behere 2011b, Govindappa et al. 2011b), *Trichoderma viride* (Patibanda and Prasad 2004, Singh Saroj et al. 2006), and *Aspergillus fumigatus* (Gaikwad and Behere 2001) have been found to be antagonistic to the growth of *F. oxysporum* f. sp. *carthami* indicating their potential usefulness for the control of the disease. Local isolates of *Trichoderma* species show more promising results (Waghmare and Kurundkar 2011). Integrated disease management using different methods of disease control has been always useful (Sastry et al. 2002). For example, integrating the seed treatment with *T. harzianum* or *T. viride* at 4–10 g/kg seed with moderately susceptible safflower variety A-1 (Prasad and Anjani 2008b) or with the spray application of NSKE at 5% results in significant control of the disease with an increase in safflower yield (Singh Saroj et al. 2006).

Effect of Plant Extracts

Leaf extracts of *Parthenium hysterophorus*, *Leucaena leucocephala*, *Vinca rosea*, *Gliricidia maculata*, *Ocimum basilicum*, *Eucalyptus globulus*, *Azardica indica*, *Datura metel*, and *Bougainvillea spectabilis* have been found to inhibit the mycelial growth of *F. oxysporum* f. sp. *carthami* and also to reduce the percent wilt incidence. All the leaf extracts tested, however, are inferior to Thiram in reducing the percent wilt incidence of safflower (Kolase et al. 2000).

Phytophthora ROOT ROT

Symptoms

Phytophthora root rot can occur from preemergence to near-maturity stages of the safflower crop. On succulent plants of 2–3 weeks of age, the first visible symptom is water soaking and collapse of cortical tissue of the lower stems. The softening of the stem weakens the young plants that they fall over, shrivel, and die. On older plants near the bloom stage, black necrotic lesions encompass the roots and sometimes extend 2–5 cm above the ground on the lower part of the stems. There is a high negative correlation between lesion length on roots and percentage of live seedlings (Nasehi et al. 2013). The cortex of the affected roots ranges in color from dark brown to greenish black. In advanced stages, the vascular tissue and pith also become necrotic and dark colored. Leaves of such plants sometimes turn yellow and the entire plant then wilts. The wilting is the common symptom of *Phytophthora* root rot, and most of the infected plants do not recover from wilting. The taproot and lateral roots of affected plants totally rot.

Dead plants can occur individually or in patches. Irrigated plants killed by *Phytophthora* are most evident 4–5 days after watering and can easily be identified by a bleached-green color. Symptoms develop similarly in susceptible plants regardless of the varieties.

Geographical Distribution and Losses

Phytophthora root rot of safflower was first observed in 1947 in Nebraska, USA (Classen et al. 1949). It is also reported to occur in Afghanistan, Argentina, Australia, Dominican Republic, India, Iran, Mexico, and Venezuela (Kolte 1985, Nasehi et al. 2013).

Most of the irrigated safflower crops in the western parts of the northern Great Plains in California, USA, were reported to have been damaged due to *Phytophthora* root rot in 1950–1951. This subsequently resulted in forceful limitation of the safflower crop in the Imperial Valley of California, and it was limited to dry land and subirrigated land. Average losses in yield due to *Phytophthora* root rot in the United States have been reported to be about 3%. In the Isfahan Province of Iran, the incidence of the disease has been observed to be about 30% during 2005–2007 crop seasons (Nasehi et al. 2013). In a few instances in certain years, 80% of the plants in a crop have been reported to be killed.

Phytophthora root rot has also limited the development of safflower as a major commercial crop in the New South Wales area in Australia. Although the disease occurs in many other countries in which safflower is indigenous, its economic importance has not been realized, probably because of resistance of local varieties to the local races of the pathogen and also because of the fact that in most of those countries, the crop is grown usually on dry land or on the border of irrigated fields. The disease has not attained serious proportions during the recent past since late 1990s in India (Prasad and Suresh 2012).

Pathogen

Multiple species of *Phytophthora* are known to cause infection in safflower including *Phytophthora drechsleri* Tucker (*P. cryptogea* Pethyb. and Laff.). Early literature established that *P. drechsleri*

has the potential to be devastating in safflower production (Banithashemi 2004, Banithashemi and Mirtalebi 2008). The main pathogen is, therefore, *P. drechsleri* Tucker (*P. cryptogea* Pethyb. and Laff.).

The mycelium is hyaline, nonseptate, and branched, having uniform width of 4.5 μm. The sporangiophores are narrower than the hyphae, and the sporangia are hyaline to faint color, thin walled, nonpapillate, and pyriform to ovate and measure 24–38 × 15–24 μm. The zoospores measure 10–20 μm in diameter. Approximately 75% germination of washed zoospores occurs during a period of 3 h in water.

The fungus does not form chlamydospores. The oospores develop singly in the oogonia, and fully formed oospores are spherical, smooth, thick walled, yellow to bright brown, and granular and measure 16–45 μm in diameter. Compatible mating strains of *P. drechsleri* produce oospores abundantly in paired cultures on seed extract media and on safflower plant material.

The oospores germinate at temperatures between 15°C and 30°C, the optimum germination being at 24°C. The germ tube of the germinating oospore usually penetrates an oogonial wall or may pass through the oogonial stalk, terminating the formation of a sporangium and release of a variable number of motile zoospores. The germination percentage is increased with the advancing age of oospores (Kolte 1985, Sastry and Chattopadhyay 2005).

Epidemiology and Disease Cycle

Assuming that compatible fungal strains are present enabling the pathogen to form oospores, it is possible that the fungus survives through the oospores. Chlamydospores are not formed by the fungus in culture, but the presence of the chlamydospores of *P. drechsleri* in roots of artificially inoculated plants of common weed species has been reported. But the exact role of the chlamydospore is not known. Saffron thistle (*C. lanatus*) is an important alternative host of *P. drechsleri*. Other weed species may also act as alternative hosts and produce further means for the survival of the pathogen in soil.

Safflower plants exude substances into the soil that stimulate the growth of germinating zoospores that probably influence root infection. Zoospores of *P. drechsleri* encyst and begin to germinate within 1 h, and germination reaches to 100% within 3 h on hypocotyls of susceptible host. The germ tube may show a positive growth response toward the wound in contrast to random growth on uninjured epidermis. The infection hyphae penetrate directly. Intercellular penetration is accomplished at junctions of epidermal cell walls at the onset of spore germination or after further growth of the germ tubes. Penetration is sometimes preceded by a spindle-shaped enlargement of hyphal tips. Intracellular invasion occurs after initial penetration at cell wall junctions is accomplished. Infection sites become visible in stained epidermal cell layers as small pores between cell walls or by deposits are found on the inside of cell walls in direct contact with the invading hyphae. The presence of the deposits at infection sites is evident only in resistant Biggs safflower. Massive intercellular and intracellular spread of the hyphae occurs in tissues of the cortex and vascular regions of susceptible hypocotyls within 48 h and causes cell collapse and disruption of cell wall. The possibility of production of pectolytic enzymes by the pathogen has been speculated in the infection process.

It has been found that resistant varieties in one locality may be susceptible in another indicating prevalence of physiological races of the pathogen. Such pathogenic races have been reported to exist in the United States. Isolates of *P. drechsleri* designated as isolate nos. 201, 5811, and 45116 have been reported as distinct races. Of these races, two are capable of rapid growth on agar media at a temperature of 35°C, and the other shows little, if any, growth at 35°C. *P. drechsleri* isolates show variation in virulence, and certain isolates appear to be more virulent on stem than on roots.

Induction of water stress predisposes safflower plants to infection by *P. drechsleri* (Duniway 1977). The effect of such water stress conditions can actually be seen under natural conditions when drought condition prevails before irrigating the crop, followed by the severe development of the

disease after irrigation (Sastry and Chattopadhyay 2005). Wet soil conditions and flooding of the fields are expected to be important factors when production, release, and movement of zoospores are the factors limiting the disease development. Increased flooding time after infection of intact plants results in a greater percentage of affected plants.

Soil temperature is an important factor influencing the pathogenicity of *P. drechsleri* to safflower. The optimum temperature for disease development is 25°C–30°C, correlated closely with the most favorable temperature (30°C) for radial growth of the pathogen in culture. Soil temperature of 17°C is unfavorable for the development of root rot. At least a portion of the temperature effect is directly on the host. The pathogen is favored in vitro by high temperature (27°C–30°C), and it is also pathogenic under higher temperature. Pronounced increase in plant death occurs when the plants are exposed to temperature of 27°C and low light intensity. Reductions in the resistance of safflower by these factors, however, do not appear to be as great or as likely to occur in the field as does the predisposing influence of water stress. Safflower plants show reduced susceptibility to *P. cryptogea* after prior adaptation of roots to hypoxic (low oxygen) condition due to the formation of root aerenchyma and phytoalexin synthesis (Atwell and Heritage 1994).

Disease Management

Host Plant Resistance

Safflower varieties "Gila" and "US 10" (both developed by crossing "Nebraska 10" with "WO 14"), "Frio," "Vte," and "VFR-1" are reported to be moderately resistant against only one pathogenic race, and the resistance in these varieties is governed by a single dominant gene (Sastry and Chattopadhyay 2005). These varieties are suitable to irrigated culture, provided they are subirrigated or grown on beds with furrow-irrigated systems for recommended durations. The VFR-1 possesses more resistance than either Gila or US 10. The resistant reaction of VFR-1 cotyledons to *P. drechsleri* appears to be indicative of its root resistance, which also is conditioned by a single dominant gene. The "VFR-1" hypocotyl is, however, susceptible to *P. drechsleri* indicating that the cotyledonary reaction is not indicative of hypocotyl reaction. The cotyledonary reactions of Gila and "US 10" are not indicative of their root resistance, which appears to be conditioned by a dominant gene.

The moderately resistant varieties, as previous, fail to show resistance to *Phytophthora* root rot under heavy flood–irrigated or water-logged conditions of soils. The highest level of resistance (lower-stem and root rot resistance) is reported in a safflower introduction selected at Biggs, California. This selection was then named Biggs safflower, possessing resistance to all races of *P. drechsleri*. The hypocotyl resistance of Biggs safflower is conditioned by a single recessive factor. The Big safflower is also reported to be resistant under heavy flood–irrigated conditions. Commercial varieties with this level of resistance are not available. The Biggs safflower is not suitable for commercial production because of late maturity and low oil content. However, it can be a very useful source of resistance in a breeding program. Several safflower germplasm lines from all over the world have been screened, and genotypes in UC 150 and UC 164 series have been reported to be resistant (Kolte 1985). The most resistant genotypes under Iran conditions are KW 9, KW 12, and KW 15 (Nasehi et al. 2013). It may further be added that breeding safflower for resistance to *P. drechsleri* is complicated by the existence of different factors conditioning resistance in either the root, hypocotyl, or cotyledons.

It has been made clear that the host-resistant mechanism in this host–pathogen combination is activated upon penetration of the epidermis. Safynol (*trans-trans*-3, 1 l-tridecadiene-5-7. 9-triyne-l, 2-diol) and dehydro safynol (*trans*-1 l-tridecene-3, 5. 7. 9-tetrayne-1, 2 diol) antifungal polyacetylene compounds have been indicated as disease-resistant factors in stem rot of safflower incited by *P. drechsleri* (Johnson 1970, Allen and Thomas 1971).

Chemical Control

Seed treatment with captan and soil drenching at 0.2% is useful in reducing the preemergence mortality due to seedling blight (Prasad and Suresh 2012).

Cultural Control

Recommendations to commercial growers that can assist in reducing damage from this disease include growing safflower in beds, not permitting water to stand in the field after irrigating and not growing the crop successively on the same land. Rotation with nonsusceptible crops may also be desirable (Kolte 1985, Prasad and Suresh 2012).

RUST

Symptoms

Safflower rust has two pathological phases that become visible as (1) root and foot disease in the seedling phase expressing the rust symptoms on cotyledons, hypocotyls, etc., and (2) foliage phase disease, at later stages of plant growth, expressing the rust symptoms on leaves, flowers, fruits, etc.

The rust in the seedling phase mainly develops because of the infection of emerging seedlings due to basidiospores resulting from germination of seed- or soilborne teliospores. Initially, orange-yellow spots representing pycnia appear on cotyledons; this may be accompanied by drooping and wilting of the seedlings. The color of such spots later changes due to the development of uredinoid aecidia called primary uredia. Many such uredia develop as pustules, and adjacent pustules coalesce to form large rust pustules. The presence of the rust pustules has also been reported on the underground part, for example, taproot and lateral roots. According to Schuster and Christiansen (1952), longitudinal cracking of epidermal and cortical tissue of the infected area is seen frequently. Some of the cracking is mainly due to the adventitious roots that are sent out at the points of infection. These roots may provide means of survival for wilted plants. The stem of 8–10-week-old seedlings can be infected, and formation of orange-yellow pycnia can also be noticed on them. On relatively older plants, girdling of the invaded area due to collapse of the tissue is a very characteristic symptom. Such plants remain erect due to the stiff stem, but the leaves of such plants are generally in a wilted condition. Due to windstorm or rains, these plants often break at the girdled area. The foliar phase of the disease is characterized by the appearance of uredial pustules on leaves, flowers, and fruits. The uredia remain scattered, crumpent on leaves, and these have a chestnut-brown color. The teleutospores are formed in the uredopustules when the safflower plant matures, giving a dark-brownish color to the rust-affected plant parts.

Geographical Distribution and Losses

The most important disease of safflower is the rust caused by *Puccinia carthami* Cda. It was first described by Corda attacking *C. tinctorius* L. in Bohemia in 1840 (Arthur and Mains 1922). Occurrence of this disease is reported in all areas of commercial production of safflower and is endemic over wide areas of safflower's natural range (Kolte 1985). It has been more recently reported to occur in Oman (Deadman et al. 2005), on snow lotus (*Saussurea involucrata* (Kar. & Kir.) in China (Zhao et al. 2007), and in Romania–Bulgaria cross-border regional areas (Anonymous 2014). The disease is more serious in countries where the crop is grown year after year. This precludes the monoculture of the safflower. Severe epiphytotics of this rust were reported in Nebraska in 1949 and 1950, after the introduction of safflower crop there (Schuster and Christiansen 1952). Currently, it is rarely a problem in the Great Plains of the United States

because it occurs late enough in the season that yields are not affected (Lyon et al. 2007). Under Indian conditions, though the disease appears to have caused severe yield losses before 1990, now the disease has not been recorded to be a significant factor in limiting safflower production for the last 10–15 years (Prasad et al. 2006, Singh and Prasad 2007). However, the seed and seedling infection is further considered to be of economic importance as it provides the source of inoculum for initiating foliage infection. Additionally, severely contaminated seeds will not germinate well if saved for future plantings (Lyon et al. 2007). For foliage rust to cause significant reduction in yield, heavier infection must occur on the upper and lower leaves before the full-bloom period. When near-isogenic varieties, one resistant to rust (WO-14) and the other susceptible (N-8), are grown under conditions of a rust epidemic, the yield of the susceptible variety is reported to be 65% of the resistant, whereas under rust-free conditions, the relationship is found to be 95%. The average annual loss due to safflower rust in the United States has been estimated to be about 5%, costing about one million dollars (Kolte 1985). The major loss from safflower rust is the reduction in stand from planting untreated teliospore-infested seed or in planting where viable soilborne teliospores exist. Using artificially infested seed, the stand loss is recorded to be 98%, but only about 20% stand loss has been reported from the use of naturally rust-infested seed. Field trials with rust-resistant and rust-susceptible safflower varieties have shown that rust-infected but rust-resistant varieties exhibit stand loss of 26%. But the surviving plants of such resistant varieties have growth compensation ability, and loss in yield remains nonsignificant as compared to the stand loss of 55%–97% in susceptible varieties with a significantly reduced yield.

Pathogen

The pathogen is *P. carthami* (Hutz.) Corda. *P. carthami* is an obligate pathogen with an autoecious life cycle on *Carthamus* spp. It is a macrocyclic rust, and since true aeciospores are naturally omitted from the life cycle, the rust is reported to be of the *brachy-form* type. The uredosori are found scattered on both sides of the leaves usually near the pycnia. In some cases, uredia are formed in between two very closely situated pycnia. Uredosori contain numerous globoid or broadly ellipsoid uredospores measuring 21–27 × 21–24 µm in size. The wall of the spore is 1.5–2.0 µm thick. The uredospores have 3–4 equatorial germ pores, and they are light chestnut brown and echinulate. Teleutosori are formed in uredosori. The teliospores are bicelled, ellipsoid, 36–44 × 24–30 µm, slightly or not constricted at septa, chestnut-brown, rounded or somewhat obtuse at both ends, finely verrucose, 2.5–3.5 µm thick at the side with the spores usually depressed from the apical position. The teliospores are hyaline, fragile, and mostly deciduous with 10 lx-long pedicel. Pycnia, usually formed in groups, are subepidermal and flask shaped or spherical and measure 80–100 µm in diameter. A large number of flexuous hyphae are found protruding, and numerous pycniospores are seen oozing out through the ostiole. Normally, as described earlier, the true aeciospores are not formed in *P. carthami*. But in some of the cases, where single sporidial infections are kept undisturbed for 20–30 days after the formation of pycnia, aeciospore-like spores are produced. Such spores are termed primary uredospores or uredinoid aeciospore because of their position in the life cycle of the rust and morphological resemblance to aeciospores. The uredinoid aecia are amphigenous and chestnut brown and measure up to 0.4 µm in diameter, associated with pycnia in clusters.

Epidemiology and Disease Cycle

P. carthami is mainly perpetuated through teleutospores that remain dormant on the seed or on the buried debris of the previous crop throughout the uncropped season. Two types of teliospores have been reported. One of the two types is known to have the ability to germinate shortly after their formation, whereas the other type shows a dormancy period of 5–6 weeks.

Under field conditions, teliospores (showing dormancy) survive for 12 months, but not for 21 months. Affected safflower straw stored at 5°C has been reported to contain viable teliospores even after 45 months of storage at such conditions. Under natural conditions, the uredospores do not survive. However, they have been reported to remain viable for over a year under dry storage conditions at 8°C–10°C. At room temperature, the uredospores lose their viability within 3 weeks. On infected plants, the uredospores remain viable for 3 weeks at 30°C–31°C and for 3 days at 52°C–55°C. It is interesting that above 40°C, the rust tends to form teliospores directly (Kolte 1985). Some of the wild *Carthamus* species act as collateral hosts in the survival of *P. carthami* (Sastry and Chattopadhyay 2005). In India, this rust is commonly seen on wild safflower *C. oxyacantha*, and it appears that this host gets infected a month earlier than the cultivated safflower. Besides, viable teliospores have been observed on this wild safflower during the off-season, suggesting a potential source of survival of the pathogen. Other *Carthamus* species, for example, *C. glaucus* M B, *C. lanatus* L., *C. syriacus* (Boiss) Dinsm., and *C. tenuis* (Boiss) Bornm., also appear to act as collateral hosts to *P. carthami*. Out of the two types of teliospores as reported by Prasada and Chothia (1950), it is the resting teliospore that oversummers and remains viable to bring about the fresh primary infection in the following season. However, the primary infection in the safflower crop may also be initiated by teliospores formed on wild safflower species, especially by those teliospores that do not require a dormant period after their formation. These may infect the safflower crop directly or may attack the wild species first, and the uredospores then formed on wild species may be blown to initiate infection in the cultivated safflower. It is reported that the volatile substances, especially the polyacetylenes, from safflower crop debris stimulate the germination of teliospores. The optimum temperature for the germination of teliospore is 12°C–18°C. The teliospores germinate normally by producing four-celled promycelium with a cell bearing short sterigma and a kidney-shaped sporidium. Such a gametophytic generation, as it becomes visible through formation of sporidia, causes root and foot infection by direct penetration of epidermis or cortex of seedlings while it is underground during the seed germination process and before plant emergence. A higher percentage of seedlings showing the root and foot phase of the disease is favored by a lower temperature range of 5°C–15°C, whereas such an infection is hindered by a temperature of 30°C and 35°C. Soil moisture variation in the range of 35%–80% of water-holding capacity has not been found to influence the seedling infection due to rust. One of the important characteristics of seedling infection due to *P. carthami* is the elongation and hypertrophy of the affected seedlings. A week after primary infection by sporidia, orange spots consisting of spermogonia appear on cotyledons, and after 2 or 3 days, primary uredosori develop around them. These infect the first leaves, thus setting up the first foci of infection. Late in the crop season, secondary uredospores, the sporophytic generation of the fungus, cause foliage infection. The uredospores germinate by giving rise to a germ tube over a temperature range of 8°C–35°C, but the optimum temperature is between 18°C and 20°C. The germ tube forms the appressorium in the substomatal vesicle, facilitating the penetration of leaf tissues through stomata. Cool temperature and high relative humidity favor the infection. The incubation period is reported to be 10–14 days depending on the temperature. At optimum 18°C–20°C temperature, the incubation period is 10 days, whereas at 35°C, the rust uredospores germinate only in traces, and the infection may not occur. Further, at 40°C, the uredospores do not germinate at all. Artificial inoculation of leaves with uredospores has been found to give only uredo- and teliospores of *P. carthami*, and uredospore inoculation on seed does not cause seedling infection. Since *P. carthami* is an autoecious macrocyclic rust that rapidly completes the sexual cycle, maximum opportunity exists for the recombination. Different races of the rust have been identified in the United States. Different rust differential hosts to identify the races have been established in the United States (Kolte 1985).

Disease Management

Host Plant Resistance

Reaction of infected hypocotyls to rust has been used as a measure of resistance of safflower to *P. carthami*. The highly susceptible seedlings show abundant sporulation on the hypocotyl, and they do not survive, but the resistant seedlings do not show hypocotyl elongation; sporulation occurs only on cotyledons and the seedlings are not killed. Seedling rust resistance appears in most cases to be both physiologically and genetically related to foliage rust resistance. Lines with resistance to the foliage phase are also resistant to the seedling phase of the disease. Seedlings with a high level of resistance to the foliage phase exhibit less than 5% seedling death due to the seedling phase of rust. A close correlation has been found with the seedling rust resistance test as an efficient method for screening for foliage rust resistance. It is, therefore, concluded that foliage rust resistance may be effectively screened by the seedling test. The microliter drop (with a known number of teliospores suspended in a 1 mL) method has potential usefulness in host range and screening for resistance of large number of genotypes (Bruckart 1999). Reaction of several safflower introduction and selections for resistance to rust have been studied (Zimmer and Leininger 1965, Kalafat et al. 2009). Some safflower lines have been reported to be resistant to foliage as well as seedling phases of the disease. These are PI 170274-100, 193764-66, 199882-82, 220647-98, 220647-55, 250601-109, 250721-93, 253759-62, 253911-25, 253912-9, 253913-5-72, 253914-5-108, 253914-7-9, and 257291-68. Other genotypes such as No. 30 and No. 26 in Turkey (Kalafat et al. 2009) and No. 1 and Tayan No. 1 in China are resistant to rust (Liu et al. 2009). The safflower line N-l-1-5 is moderately susceptible to the foliage phase of the disease, but it has been found outstandingly resistant to the seedling phase of the rust. Other such lines possessing a high degree of seedling resistance are PCA, PI 195895, and 6458-5. The seedling resistance of N-l-1-5 is governed by a single dominant gene (N). In certain situations, this source should be given a prime consideration in breeding for seedling rust resistance. Theoretically, utilization of seedling rust resistance may have the same influence on the development of races of *P. carthami* as elimination of alternate host would have on the development of races in a heteroecious species of the same genus. Because the major source of primary infection for the foliage phase is seedling infection, the utilization of seedling rust-resistant varieties would reduce the amount of primary inoculum and would consequently reduce the opportunity for new pathogenic strains to arise from the vegetative recombination.

Noncultivated species with chromosome numbers $2n = 20$, 40, or 64 have been found resistant to the foliage phase of the rust. Resistance available in *C. oxyacantha* is governed by single dominant gene (OYOY), and this has been successfully transferred to the cultivated safflower. Safflower lines with A, M, or N genes have sufficient resistance to rust. The rust resistance present in certain varieties is not linked or suppressed by the gene controlling an economically important thin-hull character of safflower. Virulence of *P. carthami* is reported to be inherited in a recessive manner.

Several commercial varieties of safflower have been developed utilizing the different sources of resistance in a breeding program. However, the development of the new races, as seen earlier, has rendered the resistance ineffective depending upon the prevalence of races in a particular area. Induced resistance in safflower by exogenous chemicals such as salicylic acid (SA), oxalic acid, and vitamin K3 and by spray application of certain nutrients at certain concentrations is typically a systemic acquired resistance (SAR) characterized by systemicity and durability (Dordas 2008, Chen 2009). When the first and second leaves are sprayed with 4 mmol/L of SA, the activity of the defense enzymes (polyphenol oxidase, peroxidases, phenylalanine ammonia lyase [PAL], etc.) increases on the third and fourth leaves with decrease in rust disease index (Chen 2009).

Chemical Control

Seed dressing with fungicides such as maneb, mancozeb, captafol, and thiram (each at 0.2%–0.3% concentration) has been reported to check the seedling infection of safflower rust. The use of systemic fungicides such as oxycarboxin seed treatment has been found most effective in inhibiting spore germination and in the management of the disease when 24–48 oz of the fungicide is used for 100 lb of seeds. Two sprays of systemic fungicides like calixin at 0.05% at an interval of 15 days are useful in the management of the foliar phase of the rust on safflower (Prasad and Suresh 2012, Varaprasad 2012).

Cultural Control

Cultural practices such as avoiding growing safflower in low-lying areas, avoiding monocrop culture of safflower, and avoiding delay of irrigation until the crop exhibits moisture stress symptoms are effective in the management of safflower rust (Varaprasad 2012).

BROWN LEAF SPOT OR FALSE MILDEW

Symptoms

Grayish-chestnut to brown spots of 2–10 mm in diameter appear on the lower leaves. The undersurface of the spot may show the presence of white growth of the fungus, owing to the emergence of tufts of conidiophores bearing conidia (Minz et al. 1961, Rathaiah and Pavgi 1977). The disease is sometimes termed as *false mildew*. The spots may coalesce to cause withering of large area of the leaf. The capitulum may also be affected. The primary symptomatic differences between *Alternaria* and *Ramularia* are that *Alternaria* spots have a shotgun pattern with different colors to the leaf as the disease progresses while *Ramularia* spots are uniformly brown and the underside of the leaf has a white appearance due to the presence of fungal bodies on the underside of the leaf. The differences are apparent with training and experience but are otherwise difficult to distinguish to a casual observer.

Geographical Distribution and Losses

Brown leaf spot of safflower was first observed in 1924 in Siberia in the former Soviet Union. The disease was then reported to occur in several other countries, for example, Ethiopia, France, India, Israel, and Pakistan (Kolte 1985). The most important disease problem of safflower in northwest Mexico (particularly in the Yaqui Valley in the state of Sonora) is reported to be false mildew since the 2000 and has been common most years since then in that country causing losses in crop yield in the range of 6%–90% (Montoya 2005, 2008, Muñoz-Valenzuela et al. 2007). The disease has also been reported to occur in California, USA, and in Argentina, South America (Hostert et al. 2006). The disease is reported to cause a sharp decrease in yield and quality of the seed in the former Soviet Union and adversely affect growth of the plant. Epiphytotic occurrence of the disease was reported at Phaltan in the Maharashtra state of India in the 1981–1982 and in the 1988–1989 crop seasons (Sastry and Chatopadhyay 2005).

Pathogen

The pathogen is *Ramularia carthami* Zaprometov.

The hyphae are hyaline and septate and measure 2–3 μm in diameter. Prior to formation of sclerotia, the hyphae become dark brown, thick walled, and closely septate, increasing to 7 μm in diameter. The sclerotia are formed by continued multiplication of cells of a single hyphal branch just below the epidermis. The mature sclerotia are chestnut brown, spherical to globose, and markedly raised above the level of epidermis. They measure 40–80 × 50–70 μm in size.

The conidiophores are hyaline and unbranched and measure 15–81 × 3–5 µm. The conidia are one or two celled, rarely three celled, hyaline, and cylindrical with rounded apices and measure 14–25 × 4.5–6 µm. The spermogonia may develop within the old conidial stomata. Mature spermogonia are ovate to globose; dark brown to black; and at first embedded subepidermally in the leaf tissue, later becoming erumpent and ostiolate and measure 45–110 × 40–150 µm. The spermatia measure 3.5–4 µm. The possibility of *Mycosphaerella* Johanson as the perfect stage of *Ramularia carthami* is suspected.

Epidemiology and Disease Cycle

The conidial germ tubes enter the leaf by penetration of the stomata. On entering the substomatal chamber, the hyphae begin to spread intercellularly. The hyphae are never seen to penetrate the living host cell, but they penetrate after collapse and death of host cells. The pathogen is mainly airborne, spreading by means of conidia. The infection develops successfully at a temperature of >28°C coupled with high humidity (Patil and Hegde 1988). The disease becomes severe under irrigated conditions favoring the epiphytotic occurrence of the disease. The disease does not occur on a rainfed safflower crop.

Disease Management

Host Plant Resistance

The reaction of wild *Carthamus* sp. has been studied. *C. oxyacantha* and *C. flavescens* are resistant to *R. carthami*. In India, the safflower lines NS 133, HOE, 999, and 1021 (Kolte 1985) and IG FRI-116 (Kumar and Joshi 1995) are reported to be moderately resistant to brown leaf spot. Resistance to brown spot can be found in selections from the original brown spot–resistant GPB4 selection and from directed crosses of GPB4 onto varieties or experimental lines of safflower. Such improved brown spot–resistant safflower lines/varieties are S-746, S-334, S-336, and S-736 (Weisker and Musa 2013) and the two most resistant lines being 04-787 and 04-765, which should be used as a source of resistance to breed improved varieties of safflower (Muñoz-Valenzuela et al. 2007). A new linoleic variety "CLANO-LIN" tolerant to false mildew has been released in Mexico (Borbon-Gracia et al. 2011).

Chemical Control

Spraying the crop with copper oxychloride (0.3%) or mancozeb (0.25%) has been found to manage the disease. Aureofungin has also been found effective in the management of the disease. Three sprays of mancozeb (0.2%) or carbendazim (0.05%) at 15-day intervals starting at 55 days after sowing are also effective in the management of the disease (Patil and Hegde 1989, Prasad and Suresh 2012).

Cercospora LEAF SPOT

Symptoms

Safflower plants are affected a few weeks after planting or when plants are in the flowering stage. Symptoms on leaves are characterized by the formation of circular to irregular brown sunken spots measuring 3–10 mm in diameter. The lower leaves show the symptoms first, and gradually, the middle and upper leaves are also affected. The spots have a yellowish tinge at the border and they are sometimes zonate. As the disease progresses, the leaves turn brown and show internal necrosis, and the entire leaves may be distorted. Under moist conditions, the spots have a velvety grayish-white appearance caused by sporulation of the fungus. Minute

black fructification of the pathogen may be seen on both upper and lower sides of the spots of affected leaves. Stems and nodes may also be affected. In case the disease becomes quite severe, the bracts are also affected and show the presence of reddish-brown spots. Affected flower buds turn brown and die. The entire capitulum may also be affected without formation of seeds.

Geographical Distribution and Losses

The *Cercospora* leaf spot of safflower is worldwide in occurrence, particularly when safflower is grown in a large area as a pure crop. It is reported to occur in Ethiopia, India, Iran, Israel, Kenya, the Philippines, the former Soviet Union, and the western Great Plains and Northern Plains area in the United States (Mündel and Huang 2003). Epiphytotic occurrence of the disease was reported in the Coimbatore area in the southern part of India in 1921, 1924, and 1925. However, information on estimates of losses caused by the disease is not available. Observations made in 2006–2007 in Montana, USA, have demonstrated that safflower is an additional host for the sugar beet pathogen, *Cercospora beticola*. This creates new potential disease problems for both crops if grown within 4 years of each other (Lyon et al. 2007). This provides further evidence that safflower is an alternative host of *C. beticola*. This is of significant importance since irrigated safflower is increasingly being evaluated for rotation with sugar beet in Montana, USA, and two crops are occasionally grown adjacent to each other (Lartey et al. 2005, 2007).

Pathogen

The pathogen is *Cercospora carthami* (H. and P. sydow) Sundararaman and Ramakrishnan.

The mycelium is hyaline, smoky brown, septate, and branched and collects in the stomatal areas where stromata are formed. The conidiophores emerge separately or in fascicles (tufts of 12–20 conidiophores) on both leaf surfaces. Under wet conditions, they emerge directly from the epidermis (Kolte 1985). The conidiophores are simple, septate, occasionally branched, erect, and variable, measuring 104.74–209.6 × 4.6 μm in size. The conidia are hyaline, linear, with 2–20 septate, and borne on the conidiophores acrogeneously. They are broad at the base and taper toward the end in a whiplike manner, measuring 2.5–5 × 50–300 μm. The length of the conidia and number of septa vary according to prevalent environmental conditions. The conidia germinate readily in water, giving germ tubes from both ends as well as from the sides. Each cell is capable of giving out a germ tube (Sastry and Chattopadhyay 2005).

Epidemiology and Disease Cycle

C. carthami is reported to have a restricted host range, and it does not infect other plants except *Carthamus* sp. The pathogen perpetuates through a vegetative saprobic mycelium and through viable stromata embedded in crop debris. Stromata of the pathogen appear as small black dots in concentric rings on diseased leaves. The disease cycle is initiated when wind-blown or water-splashed conidia land on safflower and germinate in the presence of free moisture. The fungus infects plant parts through natural openings or wounds or through direct penetration. Heavy and continuous early morning dew or other free moisture is essential for infection, and the disease is most severe during warm, moist weather. The *Cercospora* leaf spot pathogen is disseminated by wind, water splashing, and movement of infested plant material (Lyon et al. 2007).

Disease Management

Though high degree of host plant resistance sources are known, five genotypes, namely, 8-12-1, SSF-650, 2-10-2, 4-13-1, and 2-11-2, are tolerant to both *Cercospora* leaf spot and aphid attack (Akashe et al. 2004). The disease can be managed by spray application of 1% Bordeaux mixture. Dithiocarbamate fungicides (0.25%) or copper oxychloride (0.3%) might also be effective in the management of the disease (Prasad and Suresh 2012).

Seed treatment with thiram 3 g/kg and spraying of mancozeb 2.5 g or carbendazim 1 g/L of water may be useful in the disease management. Four strains of rhizobacteria (GBO-3, INR937a, INR937b, and IPC11), when micromobilized with the safflower seed, have been found to be inducers of systemic resistance in safflower preventing infection caused by *C. carthami* (Govindappa et al. 2013). Few specific cultural control strategies have been developed for *Cercospora* leaf spot. Crop rotations of 3 years or longer to nonhosts (small grains or corn), through incorporation of crop debris, and avoidance of overhead and excessive irrigation will likely reduce the incidence and severity of *Cercospora* leaf spot (Lyon et al. 2007).

Macrophomina (*Rhizoctonia*) ROOT ROT

Symptoms

Initially, dark-brown to black lesions are formed on the roots. Later, infected plants may show a characteristic silvery discoloration of the epidermal and subepidermal layer of the stem base and the root (ashy stem and root). The fungus spreads up to the vascular and pith tissues of the stem, finally forming numerous small sclerotia, like finely powdered charcoal (charcoal rot) giving the infected tissues a grayish-black color. Sclerotia are found along the vascular elements and bordering the pith cavity. Affected plants are stunted and ripen prematurely. A new type of distinct stem-split symptom is reported more recently to occur on 30-day-old safflower plants as minute cracks 2–3 cm above the soil surface that extend to both upward and downward directions resulting in the formation of wide split. The split portion becomes hollow and brown with white to gray mycelia mat of the fungus on the inner surface (Govindappa et al. 2005). Such plants fail to withstand.

Geographical Distribution and Losses

In general, the disease is considered to be of less importance in normal crop–growing season in winter months, but because of changing climate in recent years, the disease has assumed wide prevalence in warm temperate and tropical regions of the world. It causes serious yield losses especially in dry seasons in Iran (Mahdizadeh et al. 2011, Lotfalinezhad et al. 2013). The *Rhizoctonia* phase of the disease is sporadic that regularly causes 1%–10% yield losses all over India (Prasad and Suresh 2012). The incidence of the disease is negatively correlated with yield and height of the crop (Chattopadhyay et al. 2003).

Pathogen

Macrophomina phaseolina (Tassi) Goid is the pathogen, which is the pycnidial stage of *Rhizoctonia bataticola* (Taub) Butler. The details of the characteristics of the pathogen and disease cycle have been described under peanut and sunflower diseases. Genetic diversity analysis using RAPD markers and UPGMA cluster analysis could distinguish isolates prevalent in safflower-growing areas into two major groups. Dendrograph generated by cluster analysis reveals varied levels of genetic similarity, and it ranges from 50% to 55% (Prasad et al. 2011, Navgire et al. 2014).

Disease Management

Host Plant Resistance

Seed germination using towel paper and infested soil cup techniques has been developed at the Indian Institute of Oilseeds, Hyderabad, India, for screening safflower germplasm lines for resistance to the disease (Prasad and Navneetha 2010). However, resistance sources have not been detected either in cultivated or in wild safflower. Diameter of lower stem (DLS) of safflower has been found to have positive and significant correlation with length and width of the necrotic lesion on the stem of safflower; hence, DLS trait should be used as an index for indirect selection of resistant genotypes in safflower (Pahlavani et al. 2007). Some of the disease-tolerant genotypes are IUT-k 115, GUA-va 16, CW-74, AC-Stirling (Pahlavani et al. 2007), AKS-152 and AKS-68 (Ingle et al. 2004), and NARI-6, SSF-658, A-2, PBNS 12, and PBNS 40 (Prasad and Suresh 2012). Four genotypes, namely, GMU-3259, GMU-3262, GMU-3306, and GMU-3316, are identified to be highly resistant with no seedling infection, whereas three genotypes GMU-3265, GMU-3285, and GMU-3297 are found to be resistant with only up to 1%–10% seedling mortality (Salunkhe 2014). These can be used in breeding program to improve resistance in safflower to charcoal rot and root rot caused by *M. phaseolina*.

Chemical Control

No practically useful economic chemical method is recommended for the control of the disease. However, the seed-borne inoculum of the pathogen can be minimized by treating the safflower seed with thiram or carbendazim (Subeej25 DS) at 2 g/kg seed for the control of the disease and for better plant stand establishment in the field (Prashanti et al. 2000a, Prasad and Suresh 2012).

Cultural Control

The use of clean seed, the application of organic matter, long rotations with nonhost crops, and avoidance of excessively dense plant populations and sanitation, including the burial of debris by hand or by plough during summer, have each been suggested as cultural practices (Prasad and Suresh 2012) in the disease management.

Biological Control

Biocontrol agents such as *Trichoderma harzianum*, fluorescent Pseudomonads (*P. fluorescens*), and *Bacillus subtilis* obtained from the rhizosphere soil of safflower and finally prepared as talc-based formulations are used as seed treatment; these biocontrol agents at 10 g/kg prove to be effective in the control of the disease and in triggering defense-related enzymes involved in phenylpropanoid pathways and phenols that induce systemic resistance. High activity of peroxidase, PAL, chitinase, polyphenol oxidase, and beta-1,3-glucanase could be observed in *P. fluorescens*– and *T. harzianum*–treated safflower plants after challenge inoculation with *M. phaseolina* (Prashanti et al. 2000b, Kaswate et al. 2003, Singh et al. 2008a, Govindappa et al. 2010, 2011a). Soil amendment with saw dust + soil in the ratio of 1:10 when combined with seed treatment with *T. harzianum* at 4 g/kg seed shows lowest preemergence mortality due to *M. phaseolina* (Deshmukh et al. 2003).

OTHER DISEASES OF SAFFLOWER

The other diseases of safflower are given in Table 8.1.

TABLE 8.1
Other Diseases of Safflower

Disease	Pathogen/Causal Agent	Geographical Distribution	Disease Description	References
Powdery mildew	*Erysiphe cichoracearum* f. sp. *carthami* (Milovtzova)	Afghanistan, France, India, Israel, the former Soviet Union, and the United States	Symptoms are characterized by the presence of white growth of the fungus on the safflower leaves. The spineless genotypes are more susceptible to the disease. The disease can be controlled by one or two sprays of wettable sulfur (0.2%) or Karathane (0.1%).	Kolte (1985), Prasad and Suresh (2012)
Powdery mildew	*Sphaerotheca fuliginea* (Schlecht.) Pollacci	South Korea	Powdery mildew on leaves.	Kwon et al. (2000)
Powdery mildew	*Leveillula taurica* (Lev) f. sp. *carthami* Arn.	Israel, the former Soviet Union, Sudan	Powdery mildew on leaves.	Sharifnabi and Saeidi (2004)
Fusarium root rot	*Fusarium solani* (Martius) Sacc.	Iran	Root rotting. There is sufficient genetic variation for resistance, and selection can be effective for the development of resistant germplasm to *F. solani*.	Nasehi et al. (2009)
Rhizoctonia blight	*Rhizoctonia solani* Kuhn, (= *Thanatephorus cucumeris* (Frank) Donk.)	Morocco, United States	Small circular, yellow lesions develop on the hypocotyls at the soil surface. The lesions rapidly increase in size and girdle the hypocotyls. Lateral root development is greatly reduced on severely infected seedlings.	Kolte (1985)
Sclerotinia wilt/rot	*Sclerotinia sclerotiorum* (Lib) de Bary	Argentina, Australia, Canada, India Israel, Russia, Turkey, the United States	Plants become yellowish, turn brown, wilt, and die. Large black sclerotia are formed on the crown, inside the stem, and on adjoining roots though roots themselves are not generally. Shredding of the cortical tissue of the lower stem takes place. Flower heads can fall from the affected plants, leaving the outer involucral bracts in situ. The severity of the disease is increased with increase in rainfall during the crop season. The details of morphology of the fungus, infection process, disease management, etc., are described under sunflower and peanut diseases. Interestingly, soil treatment with biocontrol fungal agent such as *Coniothyrium minitans* is the most effective method under the canopy of safflower for reducing carpogenic germination of sclerotia resulting into reduced production of ascospores, which is further useful in the reduction of the disease incidence.	Kolte (1985), Huang and Erickson (2004)

(*Continued*)

TABLE 8.1 (*Continued*)
Other Diseases of Safflower

Disease	Pathogen/Causal Agent	Geographical Distribution	Disease Description	References
Gray mold	*Botrytis cinerea* Persoon ex Fries		Unseasonal rains during the late stage or in-bloom stage of the crop favor flower infection. Infection of flowers is followed by invasion of the seed head, which causes the latter to become readily detached from the plant. The lower part of the plant provides substances that stimulate infection by *B. cinerea*. The spores of the pathogen become wind borne, and flower heads may be affected at any time from budding to postflowering. The disease is more abundantly seen in the coastal areas, which are subject to fog and in Sacramento Valley of the United States.	Kolte (1985)
Pythium root rots	*Pythium* species: *P. aphanidermatum*, *P. debaryanum*, *P. splendens*, *P. ultimum*	Argentina, Austria, Australia, Canada, Iran, India, the United States	Seedling of all ages shows mortality in patches. Infection begins at the collar region that becomes black and necrotic. Roots of affected plants are severely rotted. The disease becomes severe under irrigation but not under dry conditions. Seed treatment with thiram at 0.2%–0.3% or captan at 0.2%–0.3% is effective in the control of the disease. Resistance to *P. ultimum* is known in genotypes 34040, Arak 281, and Isfahan in Iran, and the resistance is under additive and dominance genetic effects. In Canada, microbial seed treatment with strains of *P. fluorescens*, *Bacillus cereus*, *B. megaterium*, and *Pantoea agglomerans* has been found to give effective control of the root rots/damping-off of safflower.	Kolte (1985), Bardin et al. (2003), Pahlevani et al. (2010), Ghaderi et al. (2011)
Verticillium wilt	*Verticillium albo-atrum* Reinke and Berth (*V. dahliae* Kleb.)	Australia, Iran, India, Morocco, Pakistan, the United States	Interveinal yellowing of the lower leaves with progressive upward discoloration, initially, may be on one side and later may extend to the entire plant. Plants are affected at any stage of the growth during cool weather, but are seldom killed unless the infection is severe. The pathogen survives through soil as well as through seeds. Microsclerotia are found on external and internal tissues of the pericarp and on the testa of some infected seed at harvest. Microsclerotia on the seed remain viable for 2 years. Clean crop cultivation including use of healthy microsclerotia-free seed and crop rotation with nonsusceptible crops should be preferred. Safflower should not follow cotton, sorghum, and peanut. Interrow or strip cropping with these crops should be avoided. The possibility of searching safflower lines possessing high degree of resistance exists in germplasm collection.	Kolte (1985), Koike et al. (2012)

(*Continued*)

TABLE 8.1 (*Continued*)
Other Diseases of Safflower

Disease	Pathogen/Causal Agent	Geographical Distribution	Disease Description	References
Anthracnose	*Colletotrichum simmondsii*, *C. carthami*, *C. gloeosporioides*	Austria, Czech Republic, Portugal	Development of circular spots on leaves and stem blight appears to be anthracnose-like symptoms. Affected plants show light brown elongated lesions on stem, dieback symptoms, and premature death. The date of sowing affects the disease development and degree of resistance of the genotypes to the disease.	Beldan (2012), Carneiro et al. (2012), Park et al. (2005), Uematsu et al. (2012), Vichova et al. (2011)
Downy mildew	*Bremia lactucae Regel* f. sp. *carthami*	Cyprus, Iran, Israel, the former Soviet Union	Mildew growth characterized by downy growth.	Kolte (1985)
Leaf spot	*Septoria carthami* Mourashkinsky	Australia, Bulgaria, Morocco, the former Soviet Union, Turkey	Spots are produced on leaves. The pathogen is likely to be seed borne and assume to be of plant quarantine significance.	Kolte (1985)
Leaf spot	*Ascochyta carthami* Kvashnina	Morocco, the former Soviet Union	Spots on the upper side of leaves show the presence of pycnidia measuring 76–224 μm in diameter.	Kolte (1985)
Bacterial blight or leaf spot	*Pseudomonas syringae* van Hall	India, Pakistan, United States	Necrotic spots and streaks with translucent center encircled with dark-brown to black margins appear on leaves and petioles. Later, the petioles rot, which extends through the stem into the roots killing the plants. The disease becomes severe under conditions of higher temperature, humidity, rains, sprinkler irrigation, and injury due to factors like frost.	Kolte (1985), Sastry and Chattopadhyay (2005)
Necrotic yellow disease	*Spiroplasma citri*	Fars Province of Iran	First report of *Spiroplasma* as the cause of diseases in safflower. Affected plants show stunting, yellowing, phloem discoloration, and local or general necrosis.	Khanchezar et al. (2012)
Safflower mosaic disease	Cucumber mosaic virus	India, Israel, Iran, Morocco, the United States	Safflower leaves show mosaic symptoms characterized by the development of irregular flecks of light green color interspersed with dark green areas. Some leaves become blistered and distorted. The virus is transmitted through sap and the aphids, *Myzus persicae* and *Uroleucon compositae*, but it is not transmitted through seed. Young plants are more susceptible and exhibit severe mosaic symptoms.	Kolte (1985), Kulkarni and Byadgi (2004a,b,c,d, 2005)

(*Continued*)

TABLE 8.1 (*Continued*)
Other Diseases of Safflower

Disease	Pathogen/Causal Agent	Geographical Distribution	Disease Description	References
Stem necrosis disease	Tobacco streak virus	Maharashtra (India)	The first report of the virus affecting safflower. Veinal and leaf necrosis, necrotic streaks on the stem, necrosis of the terminal bud, and plant death.	Rao et al. (2003)
Phyllody	Candidatus Phytoplasma trifolii–related strain (that shares close homology with 16SrVI group)	Afghanistan, Iraq, Iran, Israel	Affected plants show floral virescences, phyllody, proliferation of axillary buds along the stem and little leaf symptoms. The causal agent is closely related to *Phytoplasma* that causes brinjal little leaf and periwinkle little leaf.	Kolte (1985), Salehi et al. (2008, 2009)
Root knot	*Meloidogyne hapla, M. incognita, M. javanica*	India, South Africa, Southeast Asia	Root knot as such is least likely to be the major cause for lower yields of safflower crop. But growing safflower on root knot nematode–infested land may build up the population of nematodes, resulting in economic damage to safflower particularly under high soil temperature at 30°C.	Sastry and Chattopadhyay (2005)

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Section VII

Soybean

The soybean (United States) or soya bean (United Kingdom) (*Glycine max* L. (Merrill) is a species of legumes belonging to the plant family Papilionaceae, which is native to East Asia. The eastern half of North China is believed to be the primary center of origin, and Manchuria, the secondary center of origin. From there, it is believed to have spread to Korea and Japan, where it is widely grown for its edible bean, which has numerous uses (Yadava et al. 2012). Soybeans are now major crops in the United States, Brazil, Argentina, China, and India.

The cultivated soybean plant is an annual plant, generally exhibiting an erect, sparsely branched bush-type growth habit with pinnately trifoliate leaves. Purple or white flowers are borne on short axillary racemes on reduced peduncles. The pods are either straight or slightly curved, usually hirsute. The height of the soybean plant varies from less than 0.2 to 2.0 m. The inconspicuous, self-fertile flowers are borne in the axil of the leaf and are white, pink, or purple. The pods, stems, and leaves are covered with fine brown or gray hairs. There are one to three seeds per pod, and they are usually void to subspherical in shape. The seed coat ranges in color from light yellow to olive green and brown to reddish black. Like most other legumes, soybeans perform nitrogen fixation by establishing a symbiotic relationship with the bacterium, *Bradyrhizobium japonicum* (syn. *Rhizobium japonicum*). For best results, an inoculum of the correct strain of bacteria should be mixed with soybean seed before planting. Modern crop cultivars generally reach a height of around 1 m and take 80–120 days from sowing to harvesting.

The basic chromosome number is $2n = 40$. The cultivated soybean (*G. max*) genome size is estimated to be about 1.12 Gb DNA, and the wild soybean (*Glycine soja*) genome size is about 1.17 Gb DNA (Qi et al. 2014). The first draft sequence and gene models of *G. max* (domesticated soybean) as well as *G. soja* (wild soybean) have been known and available for use in research purposes since 2010 (Kim et al. 2010). The comparison between genome sequences of *G. max* and *G. soja* shows significant differences between genomic compositions of the two. Major traits of agricultural importance including yield and stress tolerance are polygenic, and the presence of these favorable alleles in *G. soja* help breeding program to improve beneficial traits into cultivated soybeans (Kim et al. 2010, Joshi et al. 2013).

In addition to high protein content (40%), the soybean seeds contain 18%–23% oil and thus add to the importance of the species as an edible-oil-yielding crop. Because of the terms of production and international trade and maximum share of about 57% of world's oilseed production, the plant is now classed as an edible oilseed rather than a pulse by the UN Food and Agricultural Organization. Soybean cultivation is successful in climates with hot summers, with optimum growing conditions

in mean temperatures of 20°C–30°C. Soybean can be grown in a wide range of soils with optimum growth in moist alluvial soils with a good organic content. The main producers of soybean are the United States (36%), Brazil (36%), Argentina (18%), China (5%), and India (4%). The three largest producers have recorded an average nationwide soybean crop yields of about 3 tons/ha. Analyzing the presently prevailing situation and the amount of available arable land and water resources in Brazil, it is expected to eventually become the number one soybean-producing nation in the world. Already, South America as a continent produces more soybeans than North America (combined U.S. and Canada production). In the past decade, large tracts of fertile land and low labor costs have fueled explosive growth in South America's soybean industry although poor road and rail infrastructure, as well as economic instability and environmental concerns, have been the primary checks to further expansion in South American countries. The introduction of this temperate crop to subtropical climatic conditions made it more vulnerable to problems like seed longevity, poor growth rate due to changed photoperiod, and various biotic and abiotic stresses (Hegde 2009, Yadava et al. 2012). Diseases of soybeans are described in Chapter 9 as follows.

9 Soybean Diseases

SEED ROT AND SEEDLING BLIGHT COMPLEX

Causal Fungi, Symptoms, and Environmental Relations and Economic Importance

Several species of fungi belonging to different genera, namely, *Aspergillus flavus*, *Aspergillus niger*, *Aspergillus fumigatus*, *Mucor mucedo*, *Penicillium chrysogenum*, *Fusarium oxysporum*, *Rhizopus stolonifer*, *Cephalosporium acremonium*, *Rhizopus leguminicola*, *Alternaria alternata*, *Colletotrichum dematium*, *Macrophomina phaseolina*, *Phoma* sp., *Sclerotium rolfsii*, and *Curvularia lunata* can be isolated from the seeds of soybean. A great number of fungi are observed on seed coat and cotyledon, followed by axis. These pathogenic seed-borne and/or soilborne fungi actually penetrate and colonize the seeds. Fungal hyphae are present intercellularly within the host tissues. Maceration, disintegration, and rupture of host cells are observed in infected seeds (Adekunle and Edun 2001, Ellis et al. 2013). *Fusarium solani* and *M. phaseolina* are recorded in the cotyledon and axis (Tariq et al. 2006). Though the frequencies of different microflora are often significantly different between the transgenic and conventional cultivars, the nature of microflora on these has been found to be similar (Villarroel et al. 2004). Germination of seed is directly related to the prevalence of fungi associated with the seed (Shovan et al. 2008) and infested soils (El-Hai et al. 2010).

Pythium species: This may be the first cause of seed rot and damping-off of soybean seedlings in a growing season worldwide causing enormous losses in yield due to lack of plant stand establishment. High-residue fields, and heavy or compacted soils, are at higher risk because of cooler, wetter conditions. Pathogen may attack seeds before or after germination and seeds killed before germination and emergence. On infected plants, the hypocotyls become narrow and are commonly *pinched off* by the disease. Emerged plants may be killed before the first true leaf stage. These plants have a rotted appearance. Diseased plants may easily be pulled from the soil because of rotted roots. The two species *Pythium ultimum* and *Pythium aphanidermatum* cause greater seed rot and damping-off than any other *Pythium* species under Canadian conditions. The interactions between temperature and *Pythium* spp. are more pronounced for *P. aphanidermatum*, which shows an increased percentage of seed rot with an increase in temperature (20°C–28°C), whereas *Pythium irregulare*, *Pythium macrosporum*, and *Pythium sylvaticum* show a decreased percentage of seed rot with an increase in temperature (Wei et al. 2011). Some differential disease responses can be detected between glyphosate-tolerant and glyphosate-sensitive cultivars following the application of certain category of such herbicides. However, glyphosate-tolerant and glyphosate-sensitive soybean cultivars react similarly to most herbicide treatments with respect to root rot and damping-off (Harikrishnan and Yang 2002).

Phytophthora species: This can attack and rot seeds prior to emergence and can cause pre- and postemergence damping-off. It produces tan-brown, soft, rotted tissue. At the primary leaf stage (V1), infected stems appear bruised and soft, secondary roots are rotted, the leaves turn yellow, and plants frequently wilt and die. *Phytophthora sojae* commonly infects at the seedling stage, causing pre- and postemergence damping-off. After emergence, infected plants will be clearly visible in low areas of fields but may also be hidden underneath the canopy of nearby plants within the row. *P. sojae* infection is favored by high soil moisture resulting from excessive rains, poor drainage, and heavy clay soil texture.

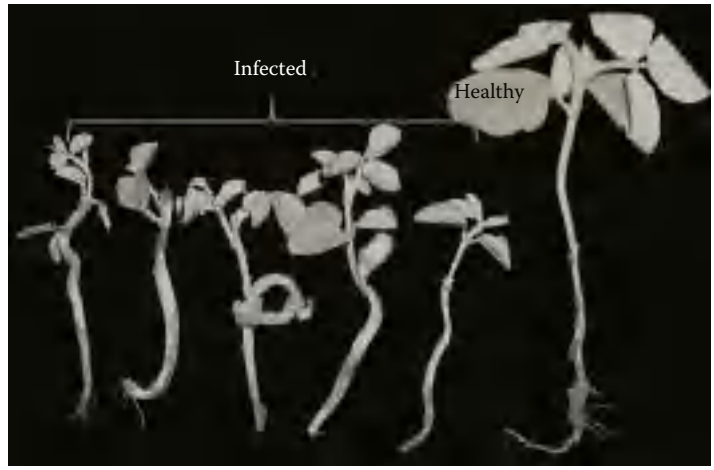


FIGURE 9.1 Seedling blight of soybean caused by *R. solani*. Note the RB lesion on soybean hypocotyls near the soil line. (Courtesy of Dr. Shrishail Navi, Iowa State University, Ames, IA.)

Rhizoctonia solani: This shows the highest ability to infect the seeds and decreases seed germination by 5.26%–15.8% with seedling mortality of 100% (Stephan et al. 2005, El-Hai et al. 2010). Trifluralin enhances the susceptibility of soybean radicles to *R. solani* (Montazeri and Hamdollah-Zadeh 2005). *Rhizoctonia* is more common in wet or moderately wet soils but not in saturated soils and its activity is most in warm soils (over 24°C), where soybean germination is slow or emergence is delayed. Infection may be superficial, causing no noticeable damage, or may girdle the stem and kill or stunt plants. *Reddish-brown (RB) lesion on soybean hypocotyl near the soil line is characteristic of Rhizoctonia infection* (Figure 9.1). It normally appears as the weather becomes warm (~27°C), is more often seen in late-planted soybean fields and causes loss of seedlings (damping-off) in small patches or within rows, and is usually restricted to the seedling stage. Stand loss is due to the soft and rotted seed with soil adhering to them. Plants may be killed by *damping-off* before or after *Rhizoctonia* infection.

Fusarium species: Seedling infection in soybean is caused by a complex of different *Fusarium* species that prefer different conditions; some prefer warm and dry soils, while others prefer cool and wet soils. Some species attack corn, wheat, and other host plants. They cause light- to dark-brown lesions on soybean roots that may spread over much of the root system, may attack the tap root and promote adventitious root growth near the soil surface, and may also degrade lateral roots, but usually do not cause seed rot. Less severe infections may degrade without resulting in plant death. Plant stand loss due to the *patchy* nature of *Fusarium* infection occurs in a specific area of the field. *F. oxysporum*, *Fusarium semitectum* (*Fusarium pallidoroseum*), *Fusarium graminearum*, and other fungal species such as *S. rolfsii*, *M. phaseolina*, and *A. flavus* appear to be dominant (Goulart et al. 2000, Goulart 2001, Pant and Mukhopadhyay 2002, Ellis et al. 2011). *Fusarium commune* as the cause of damping-off of soybean is the first report from the United States (Ellis et al. 2013). *F. oxysporum* f. sp. *glycines* reduces seed germination and seedling survivability by 40% and cause preemergence damping-off of soybean seedlings (Begum et al. 2007a).

For soybeans, the soilborne pathogens *P. sojae*, several *Pythium* species, and *R. solani* as described earlier are considered to be the most important seedling pathogens in the North Central states in the United States. A study conducted in Iowa concluded that these three organisms compose 90% of soybean-seedling diseases. Seed-borne fungi such as *Cercospora* species, *Phomopsis longicolla*, or *Fusarium* species can also play a role in seed and seedling disease, particularly if prevailing environmental conditions in the preceding crop season adversely affect the seed production.

Disease Management

Host Plant Resistance

Soybean genotypes showed differences in their reaction to seedling damping-off diseases caused by soilborne fungal pathogens such as *R. solani*, *M. phaseolina*, and *S. rolfsii*; for example, soybean cv Giza 21 shows the least incidence of preemergence damping-off followed by Giza 35 and Giza 83 but only Giza 35 showed the least incidence of postemergence damping-off (Amer 2005). Resistance to damping-off of seedlings caused by *P. aphanidermatum* in soybean cv Archer is governed by a single dominant gene *Rpa1*, which is independent of association of resistance governed by another gene *Rps1K* that confers resistance of Archer to *P. sojae* (Rosso et al. 2008). Many *P. sojae* races are found in South Dakota in the United States. Most of the genes that have been incorporated into soybean for resistance to *Phytophthora* are vulnerable to races found in South Dakota including Rps1k. Producers are required to keep a good history of their fields that are prone to *Phytophthora*, so that they may judge the effectiveness of resistance genes in their varieties. The best strategy would be to plant varieties with Rps1k, Rps3a, Rps6, or a combination thereof.

Resistance in Archer to *Pythium* damping-off and root rot is robust with its efficacy over a number of *Pythium* spp. covering *P. ultimum*, *P. irregulare*, *P. aphanidermatum*, *P. vexans*, and over a range of plant developmental stages of soybean (Bates et al. 2008). Though *Pythium* resistance in soybean cv Archer can withstand the adverse effect of flooding, the disease as such may account for a portion of the negative response of soybean to flooding (Kirkpatrick et al. 2006).

Chemical Control

Identification of seedling disease is essential in fixing the problems as different fungicides are effective in controlling different seedling diseases. Though there are limited choices in fungicide seed treatments for managing the seed rot and seedling diseases, the best seed germination and field emergence of soybean seedlings can be obtained by treating the seeds with thiram at 0.3% (Raj et al. 2002) or with a combination of carbendazim + thiram (Goulart et al. 2000, Sonavane et al. 2011) or with captan or fludioxonil or a combination of fludioxonil + metalaxyl-M (MAXIM XL) (Gally et al. 2004, Ellis et al. 2011) depending on the prevalence of pathogenic fungi. Seeds treated with 0.3% thiram maintain germination above the minimum seed certification standard up to 10 months of storage, after which seed germination can fall below the certification standard. Excellent seed germination with best degree of seedling blight control can still be achieved due to seed treatment with a mixture of thiram + carbendazim + antagonist *Trichoderma harzianum* or *T. hamatum* at 3 + 1 + 4 g/kg seed (El-Sayed et al. 2009, Khodke and Raut 2010). Treatment of soybean seeds with *R. japonicum* (*Rhizobia*) and Thiabendazole (Tecto) induces significant increase in seed germination of soybean in soils infested with *F. solani* and *M. phaseolina* (Al-Ani et al. 2011). Fungicide spray is recommended during the reproductive phase of soybean for disease control and for production of better quality soybean seeds in Brazil (Beal et al. 2007).

Soaking soybean seeds with plant growth chemicals such as ethrel, CCC, or IBA at 200 ppm has been found to be significantly effective in reducing preemergence and postemergence damping-off of soybean seedlings under salinity stress conditions of soils in Pakistan (El-Hai et al. 2010). Systemic resistance in soybean plants can be induced by prior soaking of seeds in a mixture of benzothiadiazole (0.25 g ai/L) and humic acid (4 g ai/L) for the control of damping-off and wilt diseases of soybean caused by *F. oxysporum* in Egypt (Abdel-Monaim et al. 2012, El-Baz et al. 2012).

Cultural Control

Cultural practices are also important. For example, soil drainage, delaying sowing after green manuring, and sowing at temperatures above the pathogen optimum; all precautions may be needed to prevent contamination of tools and irrigation water, in addition to the use of quality

seeds. Seed quality affects stand establishment and seedling rot in soybeans, particularly when seedlings are subjected to stress such as excessive moisture and low temperatures. In addition to quality seed, fungicide seed treatments are also highly recommended and often not optional. Fungicide seed treatments benefit stand establishment under adverse conditions such as cool, wet conditions and where pathogens are present. However, a fungicide seed treatment will not turn bad seed into good, and it will only provide a limited benefit under extreme weather and disease conditions. Certain organic soil amendments may have similar effects as an approach for indirect biological control through cultural practices. For example, dried powders of velvet bean and pine bark added to the soil at the rate of 50–100 g/kg of soil can reduce *R. solani*–induced damping-off and root and stem rot disease in soybean (Blum and Rodriguez-Kabana 2006a). Seed treatment with alum recorded maximum seed germination, root length, shoot length, and seedling vigor index (Chandrasekaran et al. 2000a,b).

Biological Control

Biological control has emerged as an alternative and promising means for the management of such type of diseases. Biological control agents like *Gliocladium virens* and *T. harzianum* antagonize pathogens by antibiosis, competition, mycoparasitism, or other forms of direct exploitation (Pant and Mukhopadhyay 2001). Damping-off of seedlings of soybean caused by *S. rolfsii* in the East of Java in Indonesia, Malaysia, Thailand, and the Philippines has been proved to be brought under control by *Actinomycetes* and VAM (Sastrahidayat et al. 2011). Antifungal activity of two bacteria obtained from the soybean rhizosphere as *Pseudomonas fluorescens* BNM296 and *Bacillus amyloliquefaciens* BNM340 has been shown to be antagonistic to *P. ultimum* causing damping-off and is able to increase seedling emergence rate under field conditions (Leon et al. 2009). Coating soybean seeds and roots with spores and mycelia of three antagonists (*Aspergillus sulphureus*, *Penicillium islandicum*, and *Paecilomyces variotii*) gives soybean germinating seeds and seedlings a very good protection from root rot and pre- and postemergence damping-off caused by *P. spinosum*. Applying these biocontrol agents (BCAs) to autoclaved and nonsterilized soil infested with *P. spinosum* provides an excellent way of protection (Al-Sheikh and Abdelzaher 2010).

T. harzianum isolate UPM40 and *Pseudomonas aeruginosa* isolate UPM13B8 have been proved to be the most effective candidates in inhibiting the mycelial growth of *F. oxysporum* f. sp. *glycines*, which causes soybean seed rot (Begum et al. 2007a). Certain bacterial isolates such as the ones belonging to *Bacillus* species (B3, B12, B80) and fluorescent *Pseudomonads* (FLPs) (B43, B51, B63, B64), when obtained from the soybean rhizosphere in Iran, have been found to show strong antagonistic effects against *P. sojae* causing damping-off soybean seedlings indicating their potential use in the management of soybean-seedling diseases (Tehrani et al. 2002, Zebarjad et al. 2006). Soybean seeds coated with a peat-bond formulation of biocontrol bacterial agent *Burkholderia ambifaria* isolate BC-F give significant disease (seedling blight) suppression with significantly greater plant stand over a considerable period of plant growth due to the ability of isolate BC-F to persist for long periods in association with roots of diverse crop plants in different soils and the production of a metabolite(s) with broad-spectrum antibiotic activity (Li et al. 2002).

Effect of Plant Extracts

Seed-borne fungi of soybean can be controlled by using leaf extract of medicinal plant and BCA. Soybean seeds when treated with leaf extracts of *Allium sativum* L. and *Azadirachta indica* A. Juss inhibit the growth of seed-borne pathogenic fungi resulting in control of the seed rot and damping-off of diseases ensuring better establishment of the stand of the plants under field conditions (Rathod and Pawar 2012). The proportion of pathogenic fungi has been found to be the lowest in soybean seeds treated with Biosept 33SL (from grapefruit seed and pulp extract) or Zaprawa Oxafun T (37.5% carboxin + 37.5% thiram) as studied by Patkowska (2006) from Poland.

ANTHRACNOSE

Symptoms

Plants may become infected at any stage of development and as a result exhibit a wide range of symptoms. The soybean is prone to be attacked by *Colletotrichum truncatum* at seed and seedling stages, resulting in pre- and postemergence damping-off (Begum et al. 2010). Seeds colonized with *C. truncatum* produce irregular gray spots with black specks. *C. truncatum* produces compact dark mycelium both intra- and intercellularly in the seed coat, cotyledon, and embryo. Mycelial growth is more abundant in the hourglass layer of the seed coat and hypodermis, where large intercellular spaces are present. Acervuli with setae and abundant hyaline sickle-shaped conidial masses are observed abundantly on the surface of infected seeds. Similar observations are found beneath the inner layers of the seed coat and upper surfaces of embryo and cotyledonary tissues. Brown conidial masses are produced during incubation and liberated in the form of ooze resulting in maceration and disintegration of the parenchyma tissues of the seed coat, cotyledon, and embryo (Begum et al. 2007b).

Infected seedlings that do not die early appear healthy until blossom, but chances of infection tend to increase with maturity and symptoms consist of appearance of brown, irregularly shaped spots on leaves, stem, pods, and petioles (Figure 9.2). The girdling of petioles by large lesions results in premature defoliation. When pods are infected, mycelium may completely fill the cavity and no seeds are produced (pod blanking) or fewer and/or smaller seed form. Seed that does form may appear brown, moldy, and shriveled or may look normal. Dark acervuli develop in lesions on all host tissues. Leaf infections, which generally develop as a result of secondary infection by conidia, may exhibit leaf rolling, necrosis of laminar veins, petiole cankers, and premature defoliation (Figure 9.3). In general, infected plants appear stunted and may have significant yield reduction. This disease is commonly observed at maturity (Galli et al. 2007). Additionally, the presence of pathogens in seeds may lead to significant reductions in seed germination, plant emergence and vigor, duration of seed storage, and crop yield.

Visible and near-infrared reflectance (Vis/NIR) spectroscopy technique has been applied to accurately detect the disease severity of soybean pod anthracnose in China (Feng et al. 2012a). According to the results, Vis/NIR spectroscopy is feasible for the identification of *C. truncatum* on soybean pods. There is a potential to establish an online field application of early plant disease detection based on visible and near-infrared spectroscopy (Feng et al. 2012b). The fungus infects seedlings, stems, petioles, leaves, and pods. Pathogen produces stria-like lesions in the pods of soybean variety "Tai 75" in China. Based on the pathogen isolated, morphological observation, rDNA ITS sequence analyses, and pathogenicity tests, it is demonstrated that the lesion is a type of infection caused by fungal pathogen *C. truncatum*. The same pathogen infects soybean pods resulting in two main types of symptom on various soybean varieties. The stria-like lesions appeared exclusively in the soybean variety "Tai 75," whereas the round blotches could be observed with the other soybean varieties. Pod disease incidence of stria-like lesions in the soybean variety "Tai 75" is observed to be 65.37%, whereas in the other soybean varieties, it could be in the range of 1.02%–12.25%. There is a clear host- and variety-dependent characteristics of infection of fungus *C. truncatum* (Lou et al. 2009).

Geographical Distribution and Losses

Anthracnose is an economically important disease widely distributed in almost all soybean-growing countries of the world. The causal fungus, *C. truncatum* (Schw.) Andrus and Moore, is present in almost all soybean-growing areas of the world (CMI map). It is generally more abundant in subtropical or tropical than temperate zones. It is reported to be the serious disease in Argentina (Daniel Ploper et al. 2001, Ramos et al. 2010), Austria (Zwatz et al. 2000),

FIGURE 9.2 Anthracnose leaf spots of soybean. (Courtesy of Dr. Anil Kotasthane, IGKV, Raipur, India.)

Brazil (Klingelfuss and Yorinori 2001), China (Feng et al. 2012a), India (Jagtap et al. 2012b), and Zambia (Mayonjo and Kapooria 2003). During the last decade, soybean yield losses have increased as the disease is associated with monocropping, no-till systems, and genetic uniformity of cultivars in the northern Pampeana region of Argentina (Ramos et al. 2010). This disease is severe in these areas, especially when precipitation and relative humidity is high. The combined attack of the disease with frogeye leaf spot (FLS) (*Cercospora sojina*) results in a yield loss of soybean yield in the range of 23.7%–32.5% in India (Mittal 2001). In addition to yield reduction, *C. truncatum* may affect seed quality. Seed-borne nature of the pathogen results in the shift in oil content reduction in the range of 18%–27%, beside the reduced seed germination up to 29.2% and viability by 26.9% coupled with lower seedling vigor (Galli et al. 2005, Nema et al. 2012). Moreover, seed infection can increase the electrolyte leakages compared to healthy seeds (Begum et al. 2008b).

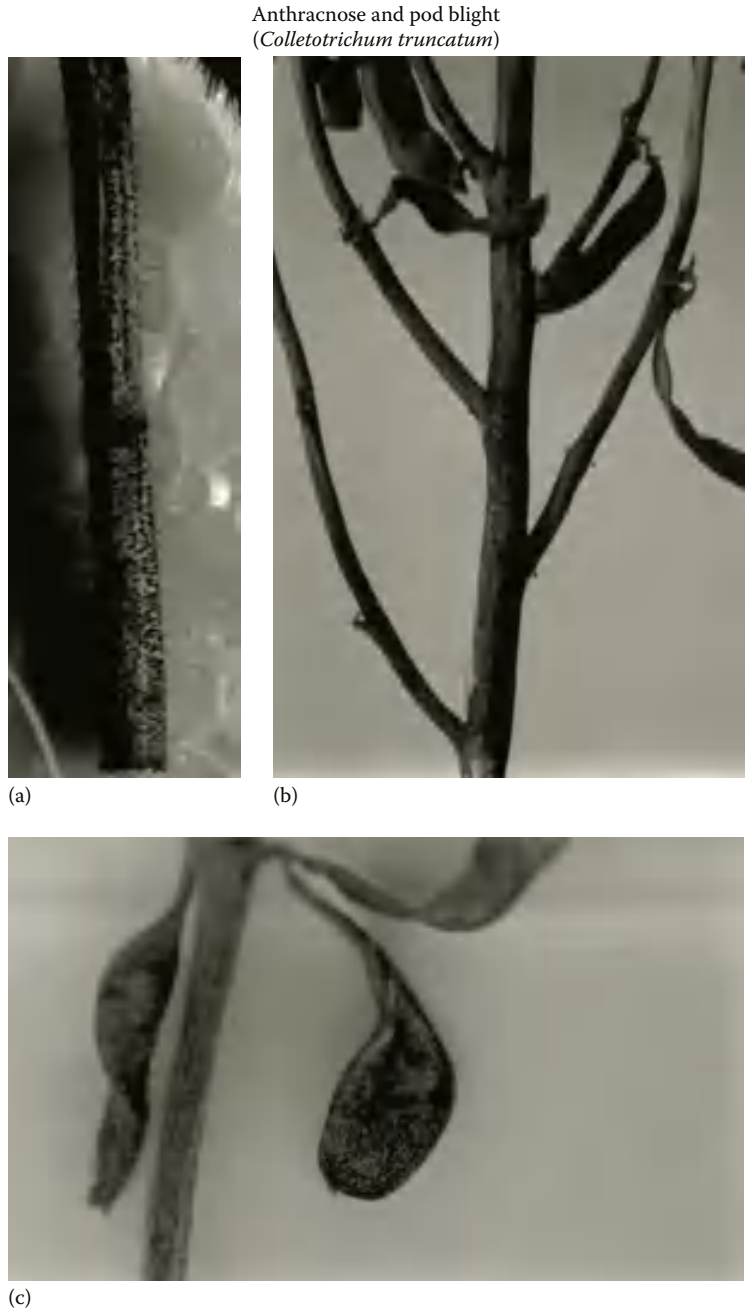


FIGURE 9.3 Anthracnose of soybean. Note the black dot-like acervuli on affected stem and pod of soybean. (a) Infected stem with acervuli of the pathogen. (b) Stem and pod blight symptoms. (c) Infected pods with acervuli of the pathogen. (Courtesy of Dr. G.K. Gupta, ICAR-Directorate of Soybean Research, Indore, India.)

Pathogen

The species most frequently associated with soybean anthracnose is *C. truncatum* (Schw.) Andrus and Moore (= *C. dematium* (Pers. ex Fr.) Grov (= *C. dematium* var. *truncatum* (Schw.)

V. Arx) (teleomorph *Glomerella truncata*). This is the imperfect fungus that belongs to the subdivision *Deuteromycotina* form-class *Deuteromycetes*, form-subclass *Coelomycetidae*, form-order *Melanconiales*, and form-family *Melanconiaceae*.

The morphology of both conidia and setae of *C. truncatum* isolates from soybean has been compared and found to be distinct. Curved conidial shape is more useful than size in isolate determination (Begum et al. 2010). Sucrose is proved to be the best carbon source for growth and sporulation of *C. truncatum* (Jagtap and Sontakke 2009). Growth (mycelial dry weight) is most pronounced at 28°C. Excellent sporulation is observed at 25°C–30°C and at pH 5.5, 6.0 (Singh and Singh 2001). It grows faster on soymilk dextrose agar than potato dextrose agar as soymilk used with agar or used alone as a broth has been found to be the best option for replacing the expensive processed culture media (Xiang et al. 2014). RAPD profiles generated by the random primers exhibit a high degree of variability among different isolates of *C. truncatum*. Infecting soybean and genetic relationships and molecular characterization of *Colletotrichum* species causing soybean anthracnose has been studied using AFLP method (Sharma 2009, Ramos et al. 2013). There appears to be a compatibility through perithecial formation among *Colletotrichum* spp. from chilli and soybean (Guldekar and Potdukhe 2011).

Epidemiology and Disease Cycle

The pathogen is seed borne, although diseased plant debris in the soil may also harbor the pathogen as primary source of inoculum. Percentage of seed-borne infection by *C. truncatum*, however, varies in soybean germplasms. For example, in total, 43 germplasms have been reported to be completely free from seed-borne infection in Bangladesh (Hossain et al. 2001). The production of acervuli of the fungus is obvious on the affected tissues under humid conditions. Consequently, rainy or wet weather facilitates the dispersal of the pathogen to become wind-borne and secondary spread takes place through conidia formed in acrevuli on stems, branches, and pods. Anthracnose disease severity becomes maximum when the average temperatures remain around 28.4°C with average relative humidity 76% and average rainfall 92.5 mm (Singh and Singh 2001). In the lowland situation in the soybean fields under the Chhattisgarh conditions in India, the average percent anthracnose severity is reported to be 76.36%, which is quite higher than the upland situation due to much more favorable environmental temperature (26°C–31.5°C), relative humidity (80%–99%), soil moisture (92%–97%), and soil temperature (23.5°C–28.2°C) in the lowland situation (Shukla et al. 2014). Infection of pods can occur even when they are green. But it remains quiescent until the pods start maturing. *C. truncatum* can thus establish latent infection without showing any visible symptom in all seed components (Begum et al. 2008b). Thus, there appears to be a prevalence of latent infection of *C. truncatum* in soybean at R5.2 growth stage under Brazil conditions (Klingelfuss and Yorinori 2001). Consequently, the development of anthracnose after harvest on apparently healthy pods is from the incipient (latent) infection of the pods in the field.

Virulence of the fungus varies with isolates indicating the existence of distinct strains of the pathogen. Soybean cultivars under natural conditions have been found to be affected by four isolates of *C. truncatum* (*Glomerella tucumanensis*) (Ct 1, Ct 2, Ct 3, and Ct 4). The frequency of isolate Ct 3 is reported to be higher on infected leaves and pods of cv. VLS 1 (Akhtar and Khalid 2008).

Diseases Management

Host Plant Resistance

Seedling test is useful to study host reaction to the pathogen (Costa et al. 2009). The inoculations at stage V1/V2 show differences in the reactions of cultivars when compared to the inoculations made at stage V5/V6. The high resistance at V5/V6 suggests a mechanism of *resistance of adult plant*

(Costa et al. 2006). There is evidence that the activity of phenolics and oxidizing enzymes increases in resistant soybean cultivars (Chandrasekaran et al. 2000d). A number of soybean genotypes such as Ceresia, Essor, Labrador, and Quito (Zwatz et al. 2000); Klaitur, PKV-1, MAUS 13, and Birsa (Gawade et al. 2009a); and Birsasoya-1 and JS (SH) 98-22 (Mahesha et al. 2009) have been shown to possess resistance reaction against the anthracnose disease. Among the aforementioned, Kalitur genotype appears to possess stable degree of resistance to anthracnose (Kumar and Dubey 2007). The most resistant soybean cultivars to damping phase of the disease are as follows: MSOY 8001, Conquista, MSOY 8400, Engopa, and Vencedora (Galli et al. 2007). The resistance in cultivars P30-1-1, Lee, and Himso 333 is governed by single dominant genes that are nonallelic (Kaushal and Sood 2002).

Chemical Control

Fungicide treatments significantly reduce infection on the average by 20%–40%. Folicur (2.5 g tebuconazole/L), with 43% efficacy, can give the best protection against *C. dematium* (Zwatz et al. 2000). In years with particularly high disease pressure, yield increases of up to 20% have been achieved. Considering incremental cost–benefit ratio (ICBR), the most economical treatment that results in giving the highest CBR is the fungicide carbendazim (CBR, 1:14.45) followed by a combination of carbendazim + mancozeb (CBR, 1:8.92) (Jagtap et al. 2012a). Similar results with the use of carbendazim (0.1% spray) have been obtained in a separate study made by Gawade et al. (2009b). Thiophanate methyl should be used as the first choice fungicide to control soybean pod anthracnose, then pyrimethanil, tebuconazole, etc. Applying fungicide two times at the stage of flower beginning, flower flourish, or seed filling of soybean, the control efficiency of soybean pods appears to be significantly higher than applying fungicide one time at the same stage. Applying fungicide one time at the stage of flower beginning and flower flourish or seed filling of soybean, respectively, the control efficiency reaches more than 95% (Wang et al. 2012b). Maximum grain yield (2425 kg/ha) has been obtained with propiconazole 0.05% treatment, which appears to be higher by 28.85% over unsprayed plants, and the differences on seed weight plant-1, 100 seed weight in healthy and infected plants, seem to be significant (Guldekar and Potdukhe 2010). Treatments with carbendazim (double application) and azoxystrobin (single application) have shown the lowest values of premature defoliation due to multiple foliar diseases including anthracnose, and the maximum yield increase could be obtained with single application of azoxystrobin (39.3%) and double application of carbendazim (32.4%) (Ploper et al. 2001). The best management of *C. truncatum* was obtained when the seeds were treated with fludioxonil + mefenoxam and thiabendazole + thiram. These treatments also contributed to improve the physiological performance of the seeds (Pereira et al. 2009). The application of thiophanate methyl resulted in the lowest incidence of pod blight caused by *C. truncatum* (Chaudhary et al. 2005). Propiconazole, Vitavax-200 (carboxin + thiram), at 100–400 ppm (Shovan et al. 2008), and azoxystrobin (Quadris) at 6–9 ounce (Padgett et al. 2003) are also effective in the management of the disease.

Systemic Acquired Resistance

Systemic acquired resistance (SAR) in soybean has been first reported following infection with *C. truncatum* that causes anthracnose disease (Sandhu et al. 2009). Pathogenesis-related (PR) gene GmPR1 is induced following the treatment of soybean plants with the SAR inducer, 2,6-dichloroisonicotinic acid (INA). Soybean GmNPR1-1 and GmNPR1-2 genes show high identities to *Arabidopsis* NPR1. Therefore, SAR pathway in soybean is most likely regulated by GmNPR1 genes (Sandhu et al. 2009). Both total and ortho-dihydroxy phenolics contents are greater in the resistant cultivar JS 89-37 (Chandrasekaran et al. 2000d). Alum at 5% reduced pod blight infection up to 90%. Dipotassium hydrogen phosphate and potassium chloride registered 75% and 65% reduction in pod blight infection over the control at 0.1%, respectively (Chandrasekaran et al. 2000a).

Biological Control

T. harzianum shows significantly variable antagonism ranging from 50.93% to 89.44% reduction of the radial growth of *C. dematium*. Among the promising antagonists, the T3 isolate of *T. harzianum* showed the highest (89.44%) inhibition of *C. dematium* radial growth (Shovan et al. 2008). Introduction of bacterized seeds carrying bacterial isolates with proven growth-promotion capabilities and antagonistic characteristics offer a valid alternative to chemical protectants. FLP strains GRP3, PEn-4, PRS1, and WRS-24 when studied in relation to natural occurrence of anthracnose caused by *C. dematium* also result in significant control of the disease (Tripathi et al. 2006). Soybean seed treatment with the tested bioagents and Rizolex-T also reduce damping-off and increase the survival of plants under field conditions. *Trichoderma lignorum* and *Trichoderma viride* are reported to be more effective than other treatments as they give results as good as Rizolex-T. The aforementioned treatments also increase fresh weight, dry weight, number of pods/plant, number of seeds/plant, and weight of seeds/plant (Saber et al. 2003). *T. harzianum* 5 inhibits the growth of *C. truncatum* (Chandrasekaran et al. 2000b). Bio-priming with *P. aeruginosa* or *T. harzianum* (by increased colony-forming units from 1.2×10^9 to 5.1×10^9 seed-1 after 12 h of bio-priming.) offers an effective biological seed treatment system and an alternative to the fungicide Benlate for the control of damping-off of soybean caused by *C. truncatum* of soybean (Begum et al. 2010). Two fungal BCAs, namely, *Trichoderma virens* isolate UPM23 and *T. harzianum* isolate UPM40, and a bacterial BCA, namely, *P. aeruginosa* isolate UPM13B8, strongly inhibit the growth of *C. truncatum* (Begum et al. 2008), though *T. viride* and *T. harzianum* are reported to be equally effective in reducing the disease (Guldekar and Potdukhe 2010).

Bacillus subtilis strains AP-3 and PRBS-1 isolated from soil samples of Paraná State, Brazil, have been found to be effective in inhibiting soybean seed–pathogenic fungi including *C. truncatum*, indicating their potential usefulness in the biological control of seed-borne infection of *C. truncatum* as well as in promoting soybean growth as the metabolites of AP-3 increase production of root hairs, while the metabolites of PRBS-1 stimulate the outgrowth of lateral roots in soybean (Araujo et al. 2005).

Effect of Plant Extracts

Aqueous leaf extract of garlic, *tulsi* and onion, ginger, and neem leaf extracts at about 20% concentration appears to be the best in inhibiting the radial growth and mycelial dry weight of the pathogen (Shovan et al. 2008, Jagtap et al. 2012a); the 10% leaf extract of *Lawsonia inermis* also reduces anthracnose disease incidence significantly (Chandrasekaran et al. 2000a). Combined application of leaf extract of *L. inermis* (5%) with alum at 1% and 0.1% can give 100% reduction in pod blight infection (Chandrasekaran and Rajappan 2002). Seed treatment with alum (0.1%) + a foliar spray with *L. inermis* leaf extract (1%) + alum (0.1%) reduce leaf anthracnose and pod blight incidences by 7.0% and 4.2%, respectively, with a grain yield of 2191 kg/ha (Chandrasekaran et al. 2000b). Dry hot water extract of *Berberis aristata*, *Boenninghausenia albiflora*, and *Lantana camara* has been shown to be highly potent against *C. truncatum* (Arora and Kaushik 2003). Among the other botanicals tested, minimum percent disease intensity (15.34%) could be recorded in *Trachyspermum ammi* seed extract (5%). The antifungal activity of essential oils from *Hortelã do Campo* (*Hyptis marrubioides*), *alfazema-do-Brasil* (*Aloysia gratissima*), and *erva-baleeira* (*Cordia verbenacea*) has potential as alternatives to synthetic fungicides in the control of anthracnose in soybean seeds (da Silva et al. 2012a).

Overall integrated disease management using all possible options is the best strategy in management of the anthracnose disease of soybean (Chandrasekaran et al. 2000c).

ASIAN SOYBEAN RUST

Symptoms

Two *Phakopsora* species are known to cause soybean rust. The more aggressive species is *Phakopsora pachyrhizi*, known as the Asian (or Australasian) soybean rust. *Phakopsora meibomiae*, the less virulent species, has only been found in areas in the Western hemisphere, and it is not

known to cause severe yield losses in soybean. The focus of the disease description in this section is therefore on Asian soybean rust (ASR) caused by *P. pachyrhizi*.

Early symptoms of ASR on the upper side of leaves consist of yellow spots that turn brown then become necrotic, surrounded by wide yellow areas, and chlorosis and brown flecking appear on the lower leaves in the canopy. The key diagnostic features of soybean rust are the cone-shaped angular lesions limited by leaf veins (Figure 9.4). Often, the first lesions appear toward the base of the leaflet near the petiole and leaf veins. This part of the leaflet probably retains dew longer, making conditions more favorable for infection. Lesions enlarge and, 5–8 days after initial infection, rust pustules (uredia, syn. uredinia) become visible. Uredia develop more frequently in lesions on the lower surface of the leaf than on the upper surface (Figure 9.4). Lesions are scattered within yellow areas that appear see-through (translucent) if the affected leaves are held up to the sun. Even though the lesions are small, each lesion often has several pustules. The anamorphic sori (uredo pustules)

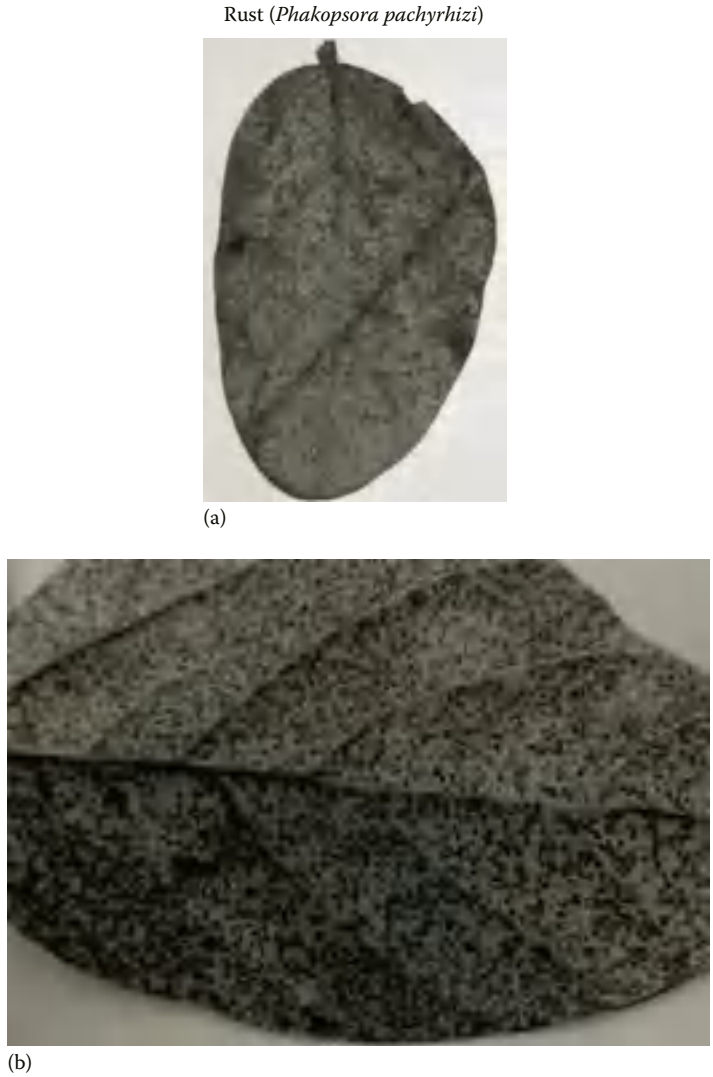


FIGURE 9.4 Asian soybean rust. Note the angular-shaped lesion-like uredo pustules on the undersurface of soybean leaf. (a) Symptoms of rust on leaf. (b) Urediniosori on lower surface of leaf. (Courtesy of Dr. G.K. Gupta, ICAR-Directorate of Soybean Research, Indore, India.)

are amphigenous, circular, minute (about the size of a pinhead), pulverulent, whitish becoming pale cinnamon brown, scattered or in groups on discolored spots, subepidermal becoming erumpent, and cone like, which can be confused with another disease, bacterial pustule. The uredia open with a round ostiole through which uredospores are released (Goellner et al. 2010). Besides leaves, uredo pustules can also appear on petioles, stems, and even cotyledons, but most rust pustules occur on leaves. Tan lesions on lower leaf surfaces contain small pustules surrounded by a small zone of slightly discolored necrotic tissue. The color of the lesion is dependent on lesion age and interaction with the host genotype. RB lesions with little sporulation indicate a semicompatible reaction, whereas tan lesions with much sporulation indicate a fully compatible reaction. It is used to compare virulence of *P. pachyrhizi* isolates from Asia (Bonde et al. 2006).

The symptoms may be observed at any time during the crop cycle but are more evident at or after flowering. The symptoms progress from lower to upper leaves. The symptoms develop further up the plant until all leaves were infected. As uredo pustules age, they may turn black because of the formation of a layer of teliospores in the pustules, turning pustules from uredinia into telia. Premature defoliation occur in infected plants.

Geographical Distribution and Losses

P. pachyrhizi is widespread in Asia and Oceania (but not in New Zealand). In the neotropics, another soybean rust fungus, *P. meibomiae*, occurs, which was once treated as synonymous with *P. pachyrhizi* but has now been taxonomically segregated (Ono et al. 1992).

The fungus that causes ASR, *P. pachyrhizi*, originally described in Japan in 1902, spread rapidly throughout Southeast Asia in the 1960s. It made a surprise appearance in Hawaii in 1994. It is thus apparent that ASR originated in tropical and subtropical regions of Asia and most likely spread to several African countries via wind currents. It appeared in Uganda in Africa in 1997, and in 2001, it was discovered in South America and moved north above the equator in 2004. Once it moved north of the equator, it moved to North America on wind currents. Now, soybean rust occurs in many countries throughout Africa including South Africa (Jarvie 2009), Asia including India (Hegde et al. 2002, Ramteke et al. 2003), and South and North America covering the United States (Goellner et al. 2010), Canada, and Mexico (Yanez Morales et al. 2009). After reports of its first occurrence in Brazil in 2001 and the continental United States in 2004, research on the disease and its pathogen has greatly increased (Vittal et al. 2012a). This disease destroys photosynthetic tissue and causes premature defoliation and, if untreated prior to the R6 growth stage, can result in severe yield reductions.

Yield losses as high as 20%–80% have been reported, but the amount of loss depends on when the disease begins and how rapidly it progresses. Yield loss equations for the ASR pathosystem using disease intensity at different phenological stages of the crop by manipulating sowing dates have been developed. The variables area under disease progress curve (AUDPC) present high correlation with yield, and variations in the severity of disease between crops affected the relationship AUDPC × yield (Hikishima et al. 2010). In April 2009, a severe rust outbreak in soybean developed at phenological stage R3 of the plants, leading to the complete defoliation (Pérez-Vicente et al. 2010). Yield losses due to the disease have been recorded to be in the range of 10%–90%, depending on the varieties used and local agroclimatic conditions (Sumartini 2010, Hegde and Mesta 2012). Predicting the time of rust appearance in a field is critical to determining the destructive potential of rusts. Mean rust-induced yield reductions have been estimated to be 67% when infection starts at R2 (full bloom) and 37% when infection takes place at R5 (beginning seed) growth stage (Kumudini et al. 2008). Yield loss increases during later sowing periods due to greater inoculum pressure hindering disease management and decreasing grain yield (Cruz et al. 2012, Akamatsu et al. 2013).

Soybean rust has been one of the most important problems in the agribusiness of the most important soybean-producing countries in South America, mainly in Brazil. Since its first detection in Paraguay and in the state of Paraná, Brazil, in 2001, the Asian rust has spread to all parts of Paraguay,

Bolivia, most of Brazil, and parts of Argentina. In the following years (2002 and 2003), it caused an estimated 4.011 million tons grain losses or the equivalent to U.S. $884.425 million (Yorinori 2004, Yorinori and Lazzarotto 2004). It is also reported to be of severe occurrence in Taiwan and Vietnam depending on the conducive environmental conditions for disease development (Tran et al. 2013). When untreated, soybean rust causes yield losses due to premature defoliation, fewer seeds per pod, and decreased number of filled pods per plant. For example, it reduces the weight of grains per plant in susceptible soybean TGx 1950-8F by 94.6% in Nigeria (Ittah et al. 2011).

The disease was reported at epidemic levels in Argentina (Pioli et al. 2005) and in Brazil in 2003/2004 (Nascimento et al. 2012, Roese et al. 2012) and in 2009–2010 (Garces Fiallos and Forcelin 2011). The main phytosanitary problem related to soybean in Brazil is ASR (do Nascimento et al. 2012).

The potential geographical distribution range of soybean rust may include most U.S. soybean production regions and that yield losses would be light in the north but moderate in the south if environmental conditions are conducive (Li and Yang 2009). ASR continues to spread across the southeast and midsouth regions of the United States (Luster et al. 2012). Immunodiagnostic assays using monoclonal antibodies have been developed to detect rust-infected soybeans and ASR spores from sentinel surveillance plots (Luster et al. 2012).

Pathogen

Soybean rust is caused by two species, *P. pachyrhizi* H. Sydow & P. Sydow and, less commonly, *P. meibomiae* (Arthur) Arthur. The latter species (*P. meibomiae*), commonly known as the cause of Latin American rust or legume rust, is found in the Western hemisphere and is not known to cause severe yield losses. In this section, most part of the subject matter is dealt with ASR caused by *P. pachyrhizi*.

Classification

Domain: Eukaryota
Kingdom: Fungi
Phylum: Basidiomycota
Subphylum: Pucciniomycotina
Class: Pucciniomycetes
Order: Pucciniales
Family: Phakopsoraceae
Genus: *Phakopsora*
Species: *pachyrhizi*

P. pachyrhizi is believed to have a microcyclic heteroecious life cycle, producing only uredinia and telia. Stage 0 = pycnial (spermitia) stage and Stage I = aecial stage (aecial spores) have not been found; Stage II = uredinial stage (uredinial spores) is quite common; Stage III = teleuto stage (teleutospores) can be observed but not common; and Stage IV = basidial sage (basidia or sporidia) is not identified.

The uredinia are pustular and open with a round ostiole through which uredospores are released (Goellner et al. 2010). Each pustule contains hundreds of spores. The urediniospores are almost sessile, obovoid to broadly ellipsoid, and 18–34 × 15–24 μm. The spore wall is uniformly ca 1 μm thick, minutely and densely echinulate, and colorless to pale yellowish brown. This coloration is different from many other rust pathogens whose spores are often reddish brown (rust colored). Four to eight (mostly 6, rarely 2–10) germ pores are equatorial or scattered on the equatorial zone or occasionally scattered on and above the equatorial zone of the spore wall. Germination of *P. pachyrhizi* urediniospores occurs through an equatorial (central) pore, producing a germ tube that ends in an appressorium, which the fungus uses to penetrate the host directly or through a stoma.

The pathogen is known almost exclusively by its uredinial stage, and there are only a few records, mainly from Argentina of the occurrence of telial stage. This is thought to be the first report of epidemiological and morphological characterization of ASR in Argentina and the first report of the telial stage of *P. pachyrhizi* on soybean in South America (Carmona et al. 2005). In case the telia are formed, telia are found on infected leaves intermixed with uredinia in old lesions. Teliospores measure 9 × 23.8 μm on average. Telia are hypophyllus, pulvinate and crustose, chestnut brown to chocolate brown, subepidermal in origin, and 2- to 7-spore layered. The teliospores are one celled, irregularly arranged, angularly subglobose, oblong to ellipsoid, and 15–26 × 6–12 μm. The wall is uniformly ca 1 μm thick, sometimes slightly thickened (up to 3 μm) apically in the uppermost spores, and colorless to pale yellowish brown and these have never been shown to germinate (Ono et al. 1992). In fact, the causal agents of soybean rust are two closely related fungi, *P. pachyrhizi* and *P. meibomiae*, which are differentiated based upon morphological characteristics of the telia. *P. pachyrhizi* originated in Asia–Australia, whereas the less aggressive *P. meibomiae* originated in Latin America (Goellner et al. 2010). Twenty-four simple sequence repeat (SSR) markers have been developed for *P. pachyrhizi* (Anderson et al. 2008). The molecular characterization of the pathogen is possible by PCR. Determination of the nucleotide sequence of the internal transcribed spacer (ITS) region reveals greater than 99% nucleotide sequence similarity among isolates of either *P. pachyrhizi* or *P. meibomiae*, but there is only 80% sequence similarity between the two species. Utilizing differences within the ITS region, four sets of polymerase chain reaction (PCR) primers have been designed specifically for *P. pachyrhizi* (Frederick et al. 2002).

Since sporidia, spermogonia, and aecia are not yet known and also if any alternate host is involved, the role of teleuto spores in the life cycle of the pathogen is not completely understood. It seems that urediniospores are the main, if not the only, means of dissemination and spread of the disease.

Considering the lack of a known sexual stage of *P. pachyrhizi*, hyphal anastomosis followed by the parasexual cycle may explain the genetic diversity in virulence among populations of *P. pachyrhizi* (Vittal et al. 2012b). This study establishes a baseline of pathogenic variation of *P. pachyrhizi* in the United States that can be further compared with variation reported in other regions of the world and in future studies that monitor *P. pachyrhizi* virulence with regard to deployment of rust resistance genes (Twizeyimana and Hartman 2012). Detailed information on the taxonomy and molecular biology of the pathogen has been reviewed (Goellner et al. 2010).

Epidemiology and Disease Cycle

Soybean rust pathogen is known to naturally infect 95 species from 42 genera of legumes, inclusive of important weed species like Kudzu vine (*Pueraria lobata*) and major crop species such as common bean (*Phaseolus vulgaris*). Such a broad host range is unusual among rust pathogens that normally have a narrow host range. The significance of the numerous alternative host possibilities for the soybean rust pathogen is that these may serve as an inoculum reservoir or a *green bridge* from one soybean planting season to the next (Jarvie 2009, Goellner et al. 2010). Alternative hosts are not to be confused with alternate host, which is a plant other than the principal host that is needed for a pathogen to complete its life cycle. In frost-free areas, such as South America, Central America, the Caribbean basin, southern Texas, and Florida, the inoculum source could be nearby on volunteer soybean plants, kudzu, or some other alternative host. In areas that experience frost, such as the Midwestern United States, inoculum must be blown in from overwintering sources that may be hundreds of miles away. The climatic and environmental factors are important in determining the development of ASR (Young et al. 2011). Temperature highs common to southeastern states are a factor in the delay or absence of soybean rust in much of the United States. For example, the highest numbers of urediniospores are produced when day temperatures peaked at 21°C or 25°C and night temperatures dipped to 8°C or 12°C (Bonde et al. 2012).

In warm regions, the host species particularly Kudzu vine (*P. lobata*) may harbor the fungus throughout the year or during seasons in which soybeans are not cultivated and may serve as the primary infection source. Kudzu (*Pueraria* spp.) is thus an accessory ASR (caused by *P. pachyrhizi*) that is widespread throughout the southeastern United States (Jordan et al. 2010).

In colder regions where aboveground parts of annual hosts die during winter, no source of new infections in the soybean-growing season has been identified. Soybean rust is sensitive to freezing temperatures and it will not survive anywhere that has adequate cold temperatures to kill off all vegetation. However, low temperature does not seem to be a limiting factor for the survival of *P. pachyrhizi* and that urediniospores could survive on volunteer plants until new soybean plants grow (Formento and de Souza 2006). ASR can only survive for extended periods of time on live host tissue. Therefore, it cannot overwinter anywhere above the freeze line (approximately Tampa Bay, Florida) since its primary hosts, kudzu and soybean, will be dead and defoliated. As such, it will have to blow into such areas each year to cause disease on soybean crops. Each year, inoculum (rust spores) must blow in from infected areas such as South Florida, Mexico, or South America to start the disease over again in the Southeastern United States. Over long distances, *P. pachyrhizi* is mainly spread by wind-borne spores (e.g., in the United States, it is considered that Hurricane Ivan transported it from South America to southern United States). Infections and sporulation by ASR are favored by cooler, wet weather. Hot dry weather will stop the spread of the fungus. *P. pachyrhizi* is unusual in that it penetrates from urediniospores directly through the leaf cuticle without entering stomata. This unusual mode of penetration suggests that disease resistance mechanisms might exist for soybean rust that does not exist for most rust diseases. *P. pachyrhizi* utilizes primarily mechanical force, perhaps with the aid of digestive enzymes, to penetrate the cuticle on the leaf surface. However, the lack of deformation lines in micrographs indicate that digestive enzymes, without mechanical force, are used by the penetration hypha to penetrate the outer and inner epidermal cell walls (Edwards and Bonde 2011). The germination of the uredospores on the soybean leaves occur after 2 h of wetness, with a maximum germination appearing after 4 h of wetness. Wetness interruption affects mainly the spores that initiate the germination (Furtado et al. 2011). Successful infection further is dependent on the availability of moisture on plant surfaces. At least 6 h of free moisture is needed for infection with maximum infections occurring with 10–12 h of free moisture. The development of the disease needs high humidity (>95%) and optimal temperature for infection process, that is, 15°C–28°C. This temperature range commonly occurred in the dry season; therefore, rust disease often attacked soybean in the dry season (Nunkumar et al. 2009, Sumartini 2010, Alves et al. 2011, Mesquini et al. 2011). Urediniospores of the pathogen remain viable during the 11 weeks of storage; the germination of the urediniospores and the severity of ASR are reduced after 3 weeks of storage, and the urediniospores stored at 20°C (plus or minus 2 degrees) for up to 11 weeks are able to cause disease in soybean plants (Beledelli et al. 2012).

This study indicates that extended periods of leaf wetness (18 h) increase disease severity and the rate of spread of the disease in the upper canopy. These results, in combination with spore monitoring, may be used to refine models of pathogen reproduction, prediction, and risk in certain regions (Narvaez et al. 2010).

Spore germination in the dark (40.7%) is found to be statistically different from spore germination in the light (28.5%). The same effect can be observed with appressorium formation, in the dark (24.7%) and in the light (12.8%) (Furtado et al. 2009). The dark incubation period of 8–16 h and light intensity of 600–400 lux (lx) are favorable for the infection of soybean rust urediospores. The infection of soybean rust can be reduced gradually with extended or shortened dark incubation period. The infection rate of the urediospores also decreases gradually with the light intensity increasing or decreasing. Higher light intensity (>3000 lx) or lower light intensity (<200 lx) is disadvantageous for the infection of the urediospores and it is advantageous for the infection of the urediospores with the light intensity changing to the favorable light intensity from the higher or lower light intensity (Mo et al. 2008).

Rain events are the most dominant cause of wetness in the lower canopy. It is revealed that a majority of the wet deposited *P. pachyrhizi* urediniospores would be removed from soybean leaf surfaces by subsequent rainfall, but sufficient percentages of spores (10%–25%) will likely remain on the leaf tissue long enough to germinate and infect during heavy summer rains lasting ≥30 min (Dufault et al. 2010b).

The uredinial stage is the repeating stage. This means that urediniospores can infect the same host on which they are produced (soybean) during the same season. The quantity of urediniospores over the crop fields is positively correlated to the disease severity and incidence as well as to cumulative rainfall and favorable days for *P. pachyrhizi* infection (Nascimento et al. 2012). Epidemics can develop quickly from only a few pustules because spore-producing pustules will develop within a week to 10 days after infection, and hundreds of spores are produced after about 3 weeks. For rust to be damaging, first infections will probably have to occur before the R3 stage of soybean development. Among other environmental factors, sunlight intensity negatively affects *P. pachyrhizi* biology with possible effects on disease epidemiology. Field observations suggest that higher disease severity occurs in shaded environments, such as on soybean leaves in the lower canopy and kudzu leaves under trees, compared with open ground. Soybean rust is more severe in the lower canopy and shaded (20% sunlight) areas, shade duration being at least 2 days (Dias et al. 2011).

Though row spacing and rainfall intensity do not show significant effect on the vertical distribution of uredinia throughout the soybean canopy, approximately half of the urediniospores can be retained within the upper portion of the soybean canopy, and the other half are distributed between the mid- and low-canopy sections (Dufault et al. 2010a).

On average, severe ASR epidemics develop when 18 cloudy days are observed after disease onset, and mild epidemics occur when only 8 cloudy days are observed. In four growing seasons in Brazil and two in the United States, the progress of ASR epidemics does not follow a wavelike pattern, and it results in an exponential distribution of distances to disease locations over time with variable monthly expansion rates. The disease front reach 500 km distance from major inoculum sources after 3 months similarly in both countries. Greater solar radiation intensity is associated with delays in epidemic onset and this knowledge may be useful to improve risk assessments for seasonal ASR epidemics. Variability in disease development across canopy heights in early-planted soybean may be attributed to the effects of solar radiation not only on urediniospore viability but also on plant height, leaf area index, and epicuticular wax, which influence disease development of SBR. These results provide an understanding of the effect solar radiation has on the progression of SBR within the soybean canopy (Young et al. 2012).

The studies made by Ponte et al. (2006) highlight the importance of rainfall in influencing soybean rust epidemics in Brazil, as well as its potential use to provide quantitative risk assessments and seasonal forecasts for soybean rust, especially for regions where temperature is not a limiting factor for disease development. Temperature variables show lower correlation with disease severity compared with rainfall and has minimal predictive value for final disease severity.

Factors that both increase and decrease the risk for ASR epidemics could be prevalent in the United States (Pivonia et al. 2005, Smith 2005), Brazil (Yorinori et al. 2005), Paraguay (Yorinori et al. 2005), and South Africa (Levy 2005). In the United States, soybean rust disease predictions are made on a daily basis for up to 7 days in advance using forecast data from the United States National Weather Service (Tao et al. 2009). Using microsatellite markers, genetic variability in *P. pachyrhizi* spore populations indicates that vertical genetic resistance, provided by single genes, is a risky strategy for soybean breeding programs that aim resistance to ASR (Tschurtschenthaler et al. 2012).

P. pachyrhizi, the soybean rust pathogen, overwinters on kudzu in the southern United States. However, even with severely affected kudzu adjacent to soybean fields, disease symptoms do not occur on soybeans until plants are in midreproductive stages of growth during mid- to late summer. These observations suggest that soybeans are exposed to airborne inoculum of the pathogen long before symptoms occur, and it is hypothesized that these plants may be latently infected (Ward et al. 2012). Soybeans can become infected by the rust pathogen during early stages of plant

growth, but symptoms often develop during the midreproductive stages. This extended latent infection period may be an optimum time for fungicide applications.

Surveys of virulence of pathogen population have been carried out in Asia, South America, and the United States for many years, and these studies have identified a wide range of races of *P. pachyrhizi*, by their interaction reaction on a set of differential lines of soybean having five specific genes Rpp1, Rpp2, Rpp3, Rpp4, and Rpp5 for rust resistance and two universal susceptible cultivars (Yamaoka et al. 2002, 2011, Pham et al. 2009). ASR resistance genes (Rpp1, Rpp2, Rpp3, Rpp4, Rpp5, and Rpp5) are genotyped with five single nucleotide polymorphism (SNP) markers (Monteros et al. 2010). Based on this study, a total of 16 soybean genotypes including cultivars and lines have been selected as a differential set to test the virulence of soybean rust populations from three South American countries, Argentina, Brazil, and Paraguay. Nine differentials are reported to carry resistance to *P. pachyrhizi* (Rpp) genes (Table 9.1).

Of the known Rpp1-4 sources of resistance, plant introduction (PI) 459025B (Rpp4) produces RB lesions in response to all of the *P. pachyrhizi* isolates, while PI 230970 (Rpp2) produces RB lesions to all isolates except one from Taiwan, in response to which it produces a susceptible tan (TAN) lesion. PI 200492 (Rpp1) and PI 462312 (Rpp3) produce TAN lesions in response to most *P. pachyrhizi* isolates (Pham et al. 2009).

This work will be useful in breeding and management of soybean rust by facilitating the identification of resistant genotypes and targeting cultivars with specific resistance to match prevailing *P. pachyrhizi* pathotypes in a given geographical zone (Twizeyimana et al. 2009).

The regional dynamics of soybean rust, caused by *P. pachyrhizi*, in six southeastern states (Florida, Georgia, Alabama, South Carolina, North Carolina, and Virginia) in 2005 and 2006 could be analyzed based on disease records collected as part of U.S. Department of Agriculture's soybean rust surveillance and monitoring program. Regional spread of soybean rust may be limited by the slow disease progress on kudzu during the first half of the year combined with the short period available for disease establishment on soybean during the vulnerable phase of host reproductive development, although low inoculum availability in 2005 and dry conditions in 2006 also may have reduced epidemic potential (Christiano and Scherm 2007).

TABLE 9.1
Asian Soybean Rust Differential Hosts

Differential		Resistance Genera	Origin
1.	PI 200492	Rpp1	Japan
2.	PI 368039	Rpp1	Taiwan
3.	PI230970	Rpp2	Japan
4.	PI417125	Rpp2	Japan
5.	PI462312	Rpp3	India
6.	PI459025	Rpp4	China
7.	Shiranui	Rpp5	Japan
8.	PI416764	ND	Japan
9.	PI587855	ND	China
10.	PI587880A	Rpp1	China
11.	PI587886	Rpp1	China
12.	PI587905	ND	China
13.	PI594767A	ND	China
14.	BRS 154	ND	Brazil
15.	TKS	ND	Taiwan
16.	Wayne	ND	United States

[a] Rpp1–Rpp5 have been mapped to different loci; ND, not determined.

Disease Management

Host Plant Resistance

High levels of ASR resistance are usually associated with one or a few dominant genes. Six dominant resistance genes (Rpp) as Rpp1 (in genotypes PI 200692, PI 200492, PI 3680390), Rpp2 (in genotypes PI 230970, PI 417125), Rpp3 (in genotype PI 462312 Ankur), Rpp4 (in genotype PI 459025 B), Rpp5 (in genotype Shiranui), and Rpp6 (in genotype PI 567102B) have been identified as capable of conferring ASR resistance in soybean and these have been mapped to different loci (Ivancovich 2008, Meyer et al. 2009, Schneider et al. 2011, Maphosa et al. 2012a, Morales et al. 2012). For example, genotypes PI 200492, PI 561356, PI 587886, and PI 587880A have been analyzed to identify SNP haplotypes within the region on soybean chromosome 18 where the single dominant ASR resistance gene Rpp1 maps, whereas ASR resistance in PI 594538A is governed by Rpp1-b gene (Monteros et al. 2010, Kim et al. 2012). Dominant alleles at three loci conditioning resistance to soybean rust races have been found in Nigeria and the symbols for the three loci controlling resistance to rust in soybean are designated as Rsbr1, Rsbr2, and Rsbr3 (resistance to soybean rust) (Iwo et al. 2012). Differential proteomic analysis of proteins involved in resistance to ASR has been done for understanding the host responses at the molecular level for effective control of the disease (Wang et al. 2012a).

While these dominant genes confer high levels of resistance and are relatively easy to incorporate into new soybean cultivars, they are not effective against all races of *P. pachyrhizi*. Deployment of varieties with new resistance genes is usually followed in a few years by the emergence of races of *P. pachyrhizi* that are virulent on them. This high degree of variability in the soybean rust pathogen is common in many rusts and requires the frequent discovery and incorporation of new sources of resistance. Currently, isolates of *P. pachyrhizi* exist that are virulent on each of the six known genes for resistance.

To select germplasm with levels of resistance to soybean rust, a differentiation must be made between the kinds of lesions on the leaves that are classified into three basic types: the resistant genotypes show RB nonsporulating pustules, whereas the moderately resistant genotypes show rectangular, RB sporulating pustules. The susceptible genotypes exhibit TAN-type gray sporulating pustules of light to medium density on all leaves, and premature defoliation is much common in these genotypes (Paul et al. 2011). The utility of detached-leaf assay for screening large number of genotypes soybean for rust resistance has been demonstrated (Twizeyimana et al. 2007) and that a determination of numbers and sizes of uredinia will detect both major gene and partial resistance to soybean rust (Bonde et al. 2006). Cell wall lignifications are markedly higher in inoculated resistant lines compared with inoculated susceptible lines, indicating a possible protective role of lignin in rust infection development in resistant soybean lines (Lygin et al. 2009, Schneider et al. 2011). Since the resistant genotype forms significantly lower lesion area, the reduced disease severity and the lack of sporulation in the resistant genotype will likely minimize the impact of the disease on canopy photosynthesis and yield (Kumudini et al. 2010).

The Asian Vegetable Research and Development Center (AVRDC) and several national agricultural research and educational institutions in Taiwan have conducted research to incorporate rust (*P. pachyrhizi*) resistance in soybean. Most races of rust in Taiwan produce TAN-type, profusely sporulating lesions and the predominant rust races are complex. Screening of germplasms initially resulted in three resistant lines, namely, PI 200492, PI 200490, and PI 200451. Consequently, PI 200492 has been used to develop three improved rust-resistant cultivars, namely, Tainung 3, Tainung 4, and Kaohsiung 3 in 1967, 1970, and 1971, respectively. Further screening of germplasms by AVRDC showed G 8586 (PI 230970), G 8587 (PI 230971), PI 459024, PI 459025, and *G. soja* (PI 339871) to be resistant. In subsequent years, all the aforementioned germplasms have been observed to be susceptible to rust, apparently due to new races of the pathogen. Recognizing the ineffectiveness of single gene resistance, AVRDC conducted research on rate-reducing and partial resistance and tolerance. A combination of genotypes combining the aforementioned three

strategies can withstand rust and give higher yield. The resistant and tolerant materials appear promising in India, Africa, and Latin America (Table 9.2). AVRDC's germplasms are available to any scientist who needs soybean rust-resistant/tolerant materials (Shanmugasundaram et al. 2004).

Soybean lines having resistant reactions to U.S., Brazil, and Paraguay isolates may be important sources for developing elite cultivars with broad resistance to ASR (Li 2009). Soybean major and minor rust-resistant genes showing predominantly additive effects are dispersed among parents and it is possible to select inbred lines superior to the best yielding parent from most crosses (Ribeiro et al. 2007).

The threat posed by soybean rust on soybean production is worsened by resistance breakdown associated with single gene resistance present in most cultivars. The marker gene pyramiding involving gene combination for three independent soybean rust resistance genes, Rpp2, Rpp3, and Rpp4 is feasible and can substantially increase resistance to soybean rust through reduced severity and reduced sporulating lesions (Maphosa et al. 2012a). Soybean genotype UG 5 as parental line has been proved to be the most outstanding one producing the greatest number of resistant populations underscoring the importance of additive gene effects in the control of soybean rust severity and sporulation rate (Maphosa et al. 2012b).

There are differences in virulence among Asian and Brazilian and the Japanese rust populations and should be considered in order to select and use resistant resources. The number of resistant varieties or resistance genes useful in these countries appear limited. Therefore, a resistant cultivar that is universally effective against soybean rust should be developed by pyramiding some major resistance genes and by introducing horizontal resistance (Yamanaka et al. 2010).

An-76, a line carrying two resistance genes (Rpp2 and Rpp4), and *Kinoshita*, a cultivar carrying Rpp5, could contribute differently to resistance to soybean rust and that genetic background plays an important role in Rpp2 activity. All three loci together work additively to increase resistance when they were pyramided in a single genotype indicating that the pyramiding strategy is one good breeding strategy to increase soybean rust resistance (Lemos et al. 2011, Kendrick et al. 2011).

The soybean cultivar Ankur (accession PI462312), which carries the Rpp3 resistance gene, when interacts with avirulent isolate Hawaii 94-1 of *P. pachyrhizi*, elicits hypersensitive cell death that limits the fungal growth on Ankur and results in an incompatible response (Schneider et al. 2011). Some soybean mutant lines obtained through seed irradiation using gamma rays (10–30 kR) and ethyl methanesulfonate (0.4%–0.8%) have been found to show improved rust resistance in India (Basavaraja et al. 2004).

Quantitative PCR (QPCR) assay of fungal DNA (FDNA) screening technique demonstrates its use to distinguish different types of resistance and could be used to facilitate the evaluation of soybean breeding populations, where precise quantification of incomplete and/or partial resistance is needed to identify and map quantitative trait loci (QTL) (Paul et al. 2011).

Molecular Breeding for Rust Resistance

A biotechnological approach may help to broaden resistance of soybean to this fungus. Molecular breeding is considered as a feasible method to improve soybean rust resistance and minimize the adverse effects from overuse fungicides. QPCR assay of FDNA screening technique demonstrates its use to distinguish different types of resistance and could be used to facilitate the evaluation of soybean breeding populations, where precise quantification of incomplete and/or partial resistance is needed to identify and map QTL (Paul et al. 2011).

Molecular markers in a backcross breeding program to introgress the Rpp5 gene of ASR resistance into HL203, an elite Vietnamese soybean variety, have been used (Tran et al. 2013), and a new chitinase-like xylanase inhibitor protein (XIP) from coffee (*Coffea arabica*) (CaclXIP) leaves has been cloned; CaclXIP belongs to a class of naturally inactive chitinases that have evolved to act in plant cell defense as xylanase inhibitors. Its role on inhibiting the germination of fungal spores makes it an eligible candidate gene for the control of Asian rust (Vasconcelos et al. 2011).

Two peptides, Sp2 and Sp39, have been identified that inhibit urediniospore germ tube development when displayed as fusions with the coat protein of M13 phage or as fusions with maize

cytokinin oxidase/dehydrogenase (ZmCKX1); when peptides Sp2 and Sp39 in either format are mixed with urediniospores and inoculated to soybean leaves with an 8 h wetness period, rust lesion development is reduced. Peptides Sp2 and Sp39, displayed on ZmCKX1, are found to interact with a 20 kDa protein derived from germinated urediniospores incorporating peptides that inhibit pathogen development and pathogenesis. Such molecular breeding programs may contribute to the development of soybean cultivars with improved, durable rust tolerance (Fang et al. 2010).

Chemical Control

Factors such as recent weather conditions, proximity to sources of ASR, cost of available products, and an estimate of a crops yield potential should be considered when choosing a fungicide program. An immunofluorescence technique in combination with propidium iodide (PI) staining–counterstaining has been developed to specifically detect viable *P. pachyrhizi* urediniospores. The method is rapid and reliable, with a potential for application in forecasting soybean rust based on the detection of viable urediniospores (Vittal et al. 2012b). This system of detection has been touted for use as a potential warning system to recommend early applications of fungicides.

The fungicides used to control ASR include the following: triazoles (cyproconazole, difenoconazole, epoxiconazole, tebuconazole) and strobilurins (azoxystrobin, pyraclostrobin, and trifloxystrobin). The treatments with these fungicides can control the disease showing severity average lower than 2, without difference among them (Soares et al. 2004, Gasparetto et al. 2011, Araujo et al. 2012, Debortoli et al. 2012, Doreto et al. 2012, Pogetto et al. 2012). Applications of triazole and triazole + strobilurin fungicides result in lower rust severity and higher yields compared with other fungicides. The strobilurin fungicides provide the highest yields in many locations; however, severity tends to be higher than that of the triazole fungicide. These fungicides are among the most effective for managing soybean rust and maintaining yield over most locations (Miles et al. 2007, Rezende and Juliatti 2010). The combination of azoxystrobin + cyproconazole or picoxystrobin + cyproconazole is reported to be the most efficient treatment when plants are foliar sprayed with the fungicide mixture at GS R3 and/or GS R5 resulting in the lowest AUDPC values and highest yields with a few exceptions (Mueller et al. 2009, Scherm et al. 2009).

Crop yield increase up to 26.9%, 33.3%, and 38.9% with the application of mancozeb, triforine, and tebuconazole, respectively, under the weekly, 2-weekly, and 3-weekly spray schedules, has been obtained with the highest economic return for mancozeb (Kawuki et al. 2002). Two sprays of triadimefon (Bayleton at 0.1%) are also very effective, as these can completely control the rust infection and increase the yield (32%) over the control. Tilt [propiconazole] when sprayed significantly results in rust control with increase in yield (Khot et al. 2007).

Substances added to the suspension or solutions of fungicides, such as adjuvants (NimbusReg), can influence the fungicide efficacy (Nascimento et al. 2012). The leaf area indices of soybean cultivars influence fungicide drop deposition and fungicide penetration into canopy resulting in the efficiency fungicide application for rust disease control, the fungicide applications being accomplished most successfully in R1 and R4 growth stages (Tormen et al. 2012).

A premix of 60 g azoxystrobin/ha + 24 g cyproconazole/ha when applied at R2 and R5 has been found to be the most efficient treatment in reducing rust severity and AUDPC and increasing yield by 50% (Godoy et al. 2009).

The demethylation inhibitor fungicide myclobutanil can be an effective component of spray programs designed to control the ASR. High degree of xylem systemicity is displayed by myclobutanil in soybean foliage and is a contributory factor toward its commercial effectiveness for the control of ASR (Kemmitt et al. 2008). Fungicides containing protective and curative properties like Silvacur Combi 30 SC and other triazole classes of fungicides could be applied at the first detection of soybean rust symptoms on lower trifoliate leaves. The disease seems to affect soybean after flowering, about 55 days after planting, and mainly during January to March when the weather is cool and moist (Sinha and Reyes 2009).

TABLE 9.2
Soybean Genotypes Resistant (R) or Moderately Resistant (MR) to Asian Soybean Rust (ASBR) as Reported from Different Countries in the World

Genotype	Country	R/MR/Genes	Reference(s)
DS 228 and DS 227	India	R	Khot et al. (2010)
PI 567099A	Paraguay	R (recessive at the Rpp3 locus)	Ray et al. (2011)
Hyuuga, PI 462312 (Ankur) (Rpp3) and PI 506764 (Hyuuga), PI 417089B (Kuro daizu	United States	R (Rpp3), (Rpp5)	Kendrick et al. (2011)
PI 567104B	United States	R (Rpp genes)	Walker et al. (2011)
PI 230970 (Rpp2), PI 459025B (Rpp4), PI 594538A (Rpp1b), PI 561356	United States	R/MR (Rpp2, Rpp4, Rpp1b)	Miles et al. (2011)
PI 594760B, TMG06_0011, TMG06_0012	United States	R	Garcia et al. (2011)
USP 97-08135	Brazil	MR	Araujo and Vello (2010)
PI 398998, PI 437323, and PI 549017, PI 230970 (Rpp2)	Vietnam	R	Pham et al. (2010)
(PI459025B)	United States	R (Rpp4C4)	Meyer et al. (2009)
BR01-18437 inbred line	Brazil	R (as parental line)	Ribeiro et al. (2009)
EC241778 and EC241780	India	R	Ammajamma and Patil (2009)
MNG 10.3 MNG 3.26	Uganda	R	Oloka et al. (2009)
Williams 82 (Rpp1)	United States	R	Paul and Hartman (2009)
GC00138-29, the cross GC00138-29 × Wondersoya	Uganda	R	Kiryowa et al. (2008)
PI 587886 and PI 587880A	United States	R	Ray et al. (2009)
PI 567102B, PI 200492 (Rpp1), PI 230970 (Rpp2), PI 462312 (Rpp3), and PI 459025B (Rpp4)	United States	R	Li (2009)
PI594538A, PI 200492	United States	R	Chakraborty et al. (2009)
EC 241778, EC 241780	India	R	Patil and Ammajamma (2006)
G 33, G 8527, G8586, G 8587, GC 60020-8-7-7-18, GC 87016-11-B-2, GC 87021-26-B-1, SRE-D-14A, SRE-D-14B, and SS 86045-23-2	Uganda	R/MR	Oloka et al. (2008)
PI 200456 and PI 224270	United States	R	Calvo et al. (2008)
PI567102B	United States	R	Li and Young (2009)
Cristalina and *IAC 100*	Brazil	MR (high partial R)	Martins et al. (2007)
Lu Pi Dou and *Hei Dou*	China	Leaf-yellowing prevention characteristic	Yamanaka et al. (2011)
Emgopa 313 and Monsoy 8211	Brazil	R	Azevedo et al. (2007)
Breeding lines: TGx 1835-10E, TGx 1895-50F, and TGx 1903-3F and Accessions (PI 594538A, PI 417089A, and UG-5)	Nigeria, United States, Uganda	R	Twizeyimana et al. (2008)

(*Continued*)

TABLE 9.2 (*Continued*)
Soybean Genotypes Resistant (R) or Moderately Resistant (MR) to Asian Soybean Rust (ASBR) as Reported from Different Countries in the World

Genotype	Country	R/MR/Genes	Reference(s)
PI 379618TC1, PI 417115, PI 423956, and the Shiranui and Kinoshita (PI 200487)	Brazil	R	Costamilan et al. (2008)
BRS 134, BRSMS Bacuri, CS 201, FT-17, FT-2, IACpl1, KIS 601, and OCEPAR 7	Brazil	R (RB-type lesion)	Arias et al. (2008)
PI 506863, PI 567341, and PI 567351B, PI 181456, PI 398288, PI 404134B, and PI 507305, PI 587886, PI 587880A, and PI 587880B, PI 587905 and PI 605779E, PI 594754, PI 605833, PI 576102B, and PI 567104B	Paraguay	R	Miles et al. (2008)
EC 241778 and EC 241780	United States	R	Patil et al. (2004)
MNG 7.13, MNG 8.10, and MNG 1.6	Sub-Saharan Africa and worldwide	R	Tukamuhabwa et al. (2012)
EC 325115, EC 251378, EC 389149, EC 432536, EC 241760, and EC 333917	United States	MR	Patil et al. (2004)
Ankur, PK 1029, TS 98-21, EC 389160, and EC 389165	India	R	Rahangdale and Raut (2004)
JS 19, RPSP-728 and PK 838	India	R	Verma et al. (2004)
Early TGx 1835-10E, late TGx 1838-5E	Uganda	MR	Kawuki et al. (2004)
EC 389160, *EC 393230*, and *TS 98-21*	India	R	Rahangdale and Raut (2003)
PI 567102B, PI 200492 (Rpp1), PI 230970 (Rpp2), PI 462312 (Rpp3), and PI 459025B (Rpp4)	Paraguay	R	Li (2009)
EC241780	India	R	Shivakumar et al. (2011)
TGx 1805-1F, TGx 1951-3F, TGx 1935-3F, and TGx 1972-1F	Nigeria	Highly R	Ittah et al. (2011)
TGx 1949-8F, TGx 1935-5F, TGx 1448-2F, TGx 1965-7F, and TGx 1936-2F	Nigeria	R	Ittah et al. (2011)
EC-241778 and EC-241780	India	R	Parameshwar et al. (2012)
EC 241780, EC 456573(A), EC 456580, EC 427283, EC 481454, EC 457172, EC 481441, and EC 457266(Ku)	India	R	Kurundkar et al. (2011)
TGx1987-62F, TGx1935-3F, TGx1951-3F, TGx1936-2F, TGx1987-10F, TGx1972-1F, and TGx1949-8F	Nigeria	Nigeria (IITA)	Iwo et al. (2012)

Three sprays of hexaconazole alone reduce rust disease severity considerably to higher level and result in significantly higher seed yield (24.79 q/ha), 100 seed weight (14.37), and the inclusion of nimbecidine in the spray schedule not only is more useful in reducing the cost of protection but also gives higher benefits in addition to giving insurance against resistance development by the fungus against hexaconazole (Hegde and Mesta 2012). Combined with the organosilicone adjuvant, Silwet L-77 plus fungicide pyraclostrobin + epoxiconazole contribute to improve soybean rust control increasing the productivity and weight of 1000 grains.

The efficacy of fungicides varies with the cultivars also. For example, three sprays of hexaconazole are sufficient to manage rust and produce high yields in JS-335, while two sprays of the same hexaconazole have been enough to lower disease severity and to obtain high yields for PK-1029 (Hegde et al. 2002). Similarly, the resistant line CB06-953/963 (Rpp4 gene) needs 13.3 days longer than the susceptible cultivar to reach the ETL; late-season fungicide applications reduce rust severity and increase the yield of the resistant cultivar (Koga et al. 2011). The cultivars M-Soy 8199RR and Emgopa 315RR that are less susceptible to disease and a control program termed *monitoring* (in which the appearance of new pustules of the pathogen is monitored to make the decision at each fungicide spray) have been found to be the most effective (da Silva et al. 2011).

The rate of 80 kg/ha K_2O associated with fungicide sprays with azoxystrobin + cyproconazole is promising to reduce the deleterious effects of ASR (Doreto et al. 2012). The glyphosate at rates between 0.84 and 1.68 kg/ha can delay the onset of ASR in soybeans (Feng et al. 2008).

Systemic Acquired Resistance

Silicon (Si) is recognized for its prophylactic role in alleviating diseases when absorbed by plants and has been proposed as a possible solution against soybean rust, caused by *P. pachyrhizi*. Soybean plants supplied with Si show reduction in ASR symptoms (Arsenault-Labrecque et al. 2012, da Cruz et al. 2012). Si can protect soybean plants against soybean rust through mediated resistance. Saccharin (3 mM) applied as a root drench at the second trifoliate (V3) and early reproductive (R1) stages has been found more effective than the foliar spray treatment at inducing SAR (Srivastava et al. 2011). The severity of the soybean rust (area under disease progress curve) is significantly reduced when the soybean plants are fertilized with the combination of 8 and 11 mmol/L of K and Ca, respectively (Pinheiro et al. 2011).

Soil applications of wollastonite ($CaSiO_3$) (Si 0.96–1.92 tons/ha) or foliar applications of potassium silicate (K_2SiO_3) (Si at 500–12,000 mg/kg) may lead to the development of SBR control practices that can benefit organic and conventional soybean production systems (Lemes et al. 2011). Silicon (Si) amendments have been studied as an alternative strategy to control SBR because this element is reported to suppress a number of plant diseases in other host. Potassium silicate (KSi) sprays (40 g/L) could reduce the intensity of soybean rust (Rodrigues et al. 2009). The foliar application of $MnSO_4$ (0.3%) records lower percentage of rust disease index (33.7) compared to the control (89.6) consequently increasing the yield. Considering the effect of $MnSO_4$, in terms of both yield and environmental advantages, it is suggested to replace traditional fungicide application with $MnSO_4$ (Morab et al. 2003).

Cultural Control

There are several cultural practices that may help manage soybean rust. In most areas of the United States where rust must be introduced each year for an epidemic to occur, changing planting and harvest dates may avoid disease. Planting date and soybean cultivar significantly affect disease severity, with severity being higher on soybean crops planted during the wet season than those planted in the dry season. This study suggests that selection of planting date could be a useful cultural practice for reducing soybean rust (Twizeyimana et al. 2011b). For example, early sowing (end of June) of the crop is less damaged (36.15%) when compared with crop sown in mid- and end of July in India (Shukla et al. 2005). For all of the sowing dates, the early-season cultivar, M-SOY 6101, shows a lower risk of being affected by the rust and consequently exhibits less yield loss exhibiting a lower

variance in yield, which represents more stability with regard to the inter-annual climate variability, that is, the farmers who use this cultivar will be able to recover more economic benefits (de Avila Rodrigues et al. 2012). Planting dates may also be delayed so that the vulnerable reproductive period occurs during dry conditions that do not favor rust.

In areas where the weather is marginal for rust development, wider row spacing along with lower plant populations may hasten canopy drying, thus reducing the dew period enough to prevent or at least slow disease development. Thus, the row spacing of 60 cm lowers AUDPC values and higher crop productivity (Madalosso et al. 2010). Cultural practices such as the use of reduced seed rates, increased row widths, and row orientation to the sun have been prescribed as environmental modifications that create a microclimate less conducive to foliar disease development. Therefore, it is important to determine the influence of different periods of leaf wetness and respective microenvironments on infection and rust development on soybean plants in a local geographical area in the field (Narvaez et al. 2010). The expression of partial resistance of both cultivars can be influenced due to variation of P and K levels. Lower doses of P and K induce a greater difference in the latent period of the pathogen. The association of genetic cultivar background to mineral nutrition might result in an integrated management disease program, along with evasion and chemical protection strategies (Balardin et al. 2006). The severity of soybean rust is higher in plants under crossed sown lines. The increase in the number of seeds from 15 to 30 per meter in the crossed sown lines reduces the severity of the disease only in some cultivars as in the case of M7211RR cultivar (Lima et al. 2012).

Biological Control

The fungus *Simplicillium lanosoniveum* can colonize *P. pachyrhizi* and significantly lower amounts of DNA of *P. pachyrhizi* and lower rust disease severity when soybean leaves are colonized with *S. lanosoniveum* indicating its potential use in biological control of soybean rust disease (Ward et al. 2012).

Effect of Plant Extracts

The essential oils of *H. marrubioides*, *A. gratissima*, and *C. verbenacea* are fungitoxic by inhibiting 100% of urediniospores of *P. pachyrhizi* and are effective at higher concentration only as preventive treatments in the control of the ASR. But these essential oils at these dosages are not as efficient as the pyraclostrobin + epoxiconazole–based fungicide (da Silva et al. 2012b). It is inferred that the essential oils from *Corymbia citriodora* (*Eucalyptus citriodora*), *Cymbopogon nardus*, *A. indica*, or *Thymus vulgaris* at concentrations of 1.0%, 0.5%, 1.0%, and 0.3% have the potential to reduce infection by *P. pachyrhizi*, agent of the ASR (Medic-Pap et al. 2007).

Integrated Control

An integrated management system must include intensive scouting for ASR during reproductive soybean growth stage; early disease diagnosis; use of moderately rust-resistant cultivars and use of fungicides from groups III and IV (strobilurins, triazoles, and mixture of both); alternative host elimination, including soybean volunteer plants; and early planting dates and diversification in planting dates and may be used in combination with appropriate cultural practices and fungicides when needed. Wider row spacing may also allow better fungicide application and penetration into the canopy, increasing the effectiveness of chemical control (Rupe and Sconyers 2008). Using a spray mixture of cow urine (10%) + plant extract of *Prosopis juliflora* (0.5%) or cow urine (10%) + neem oil (0.5%) has been found to be economically effective in rust disease management of soybean (Jahagirdar et al. 2012).

Spore trapping and aerobiological modeling are useful in maintaining the effectiveness of the Integrated Pest Management (IPM) Pest Information Platform for Extension and Education (ipmPIPE), increasing North American producers' profits by hundreds of millions of dollars each year. In the United States, control practices based on up-to-date maps of soybean rust observations and associated commentary from Extension Specialists delivered by the ipmPIPE may have

suppressed the number and strength of inoculum source areas in the southern states and retarded the northward progress of seasonal soybean rust incursions into continental North America (Isard et al. 2011).

SUDDEN DEATH SYNDROME

Symptoms

Sudden death syndrome (SDS), caused by *F. solani* f. sp. *glycines*, is a season-long root rot disease of soybean that results in severe foliar symptoms beginning in late vegetative and early reproductive stages of plant growth. Pattern of symptoms in the field ranges from distinct oval to circular patches to irregularly shaped bands or streaks across the field (Figure 9.5). In severe cases, a majority of field may show the symptoms. The SDS is characterized by root rot followed by the development of foliar symptoms. Root systems may show rotting and discoloration of lateral and tap roots. When split open, internal tissues of taproot and lower stem may show a light-gray to light-brown discoloration. Foliar symptoms begin as scattered yellow blotches in the interveinal leaf tissues. These yellow blotches increase in size and merge to affect larger areas of leaf tissues. Symptoms range from the development of chlorotic spots to severe interveinal chlorosis and necrosis (Figure 9.6). Veins typically stay green. The bright yellow blotches between the green veins give affected leaves a striking appearance. As the interveinal leaf tissue turns brown, it also dries out. Taproots of symptomatic plants are necrotic and stunted and stems exhibit a light tan discoloration, but never the dark-brown discoloration typical for brown stem rot (BSR); the pith of the SDS-affected stem remains white. This is a key symptom to differentiate SDS from BSR, a disease with similar foliar symptoms. The SDS is most recognized by the development of interveinal chlorosis and necrosis on leaves and premature defoliation (Leandro et al. 2012). In severe cases, the leaflets may drop off, leaving the petioles (leaf stalks) attached or they may curl upward and remain attached to the plant (Westphal

FIGURE 9.5 SDS of soybean at the seedling stage. (Courtesy of Dr. Shrishail Navi, Iowa State University, Ames, IA.)

FIGURE 9.6 SDS of soybean at the reproductive stage. (Courtesy of Dr. Shrishail Navi, Iowa State University, Ames, IA.)

et al. 2008). In other diseases that exhibit similar symptoms, the dead leaflets essentially tend to remain attached to the petiole. But these symptoms are not diagnostic by themselves. If the plants are uprooted when soil is moist, small, light-blue patches may be visible on the surface of the taproot near the soil line. These patches are blue spore masses of the fungi that cause SDS. As the root surface dries, the blue color fades, but these blue spore masses, seen in conjunction with the other symptoms mentioned earlier, are strong diagnostic indicators of SD.

Geographical Distribution and Losses

The SDS of soybean caused by *Fusarium virguliforme* was first discovered in Arkansas in 1971 in the United States and in South America in the early 1990s (Roy et al. 1997, Colletto et al. 2008). The disease has spread extensively since then and can be economically devastating depending on disease intensity and timing of disease onset in most soybean-growing regions of the North and South America and the world (Malvick and Bussey 2008, O'Donnell et al. 2010, Leandro et al. 2012, Mbofung et al. 2012). SDS is ranked in the top four on the list of diseases that suppressed soybean yield during 2003–2005 in the United States (Wrather et al. 2003, Aoki et al. 2005, Wrather and Koenning 2006). The extent of yield losses due to SDS depends on the severity and timing of disease expression relative to plant development in regard to yield components. If the disease develops early in the season, flowers and young pods tend to abort. When the disease develops later, the plants produce fewer seeds per pod or smaller seeds. SDS reduces soybean yields in four of the top eight soybean-producing countries in the world, Argentina, Brazil, Canada, and the United States. In the year 2006 alone, yield was reduced by 1.849 million metric tons worldwide (Wrather et al. 2010). Yield suppression of SDS in the United States increased from 3.7 million bushels in 1996 to 34.5 million bushels in 2009 (Wrather and Koenning 2009, Koenning and Wrather 2010). From 1996 to 2007, losses averaged U.S. $190 million a year in the Midwestern U.S. soybean-producing region (Robertson and Leandro 2010). Gibson et al. (1994) estimated yield reduction of 7–34 kg/ha per unit increase in SDS incidence, whereas others have reported total yield decreases of 12%–22% per unit increase in foliar symptom severity. The earlier severe disease develops, the more the yield is reduced. In Argentina, average yield loss is in the range of 192–3770 kg/ha (Mercedes Scandiani et al. 2012). Soybean cyst nematode (*Heterodera glycines*) and *F. virguliforme* causing SDS have a synergistic effect on yield when they occur jointly in the field (Gelin et al. 2006, Xing and Westphal 2009).

Pathogen

SDS is caused by soilborne fungi within a group (clade 2) of the *F. solani* species complex (Aoki et al. 2003, 2005). Phenotypic and multilocus molecular phylogenetic analyses, as well as pathogenicity experiments, have demonstrated that four morphologically and phylogenetically distinct fusaria can induce soybean SDS (O'Donnell et al. 2010). Among the four species of *Fusarium*, *Fusarium brasiliense*, *Fusarium cuneirostrum*, *Fusarium tucumaniae*, and *F. virguliforme*, only two, *F. virguliforme Akoi* (O'Donnell, Homma and Lattanzi) (syn. *F. solani* f. sp. *glycines*) and *F. tucumaniae*, are the main casual fungi in North and South America, respectively (Aoki et al. 2005). The fungus, *F. virguliforme* (syn. *F. solani* f. sp. *glycines*), is semibiotrophic, which grows slowly in culture and is difficult to isolate from diseased plants (Yuan et al. 2008). Once a pure culture is obtained, blue spores and other cultural characteristics distinguish the SDS pathogens from other *Fusarium* species that can infect soybean roots. In North America, the SDS pathogen is considered clonal and has been considered asexual; the pathogen, however, has never been isolated from diseased foliar tissues. Thus, one or more toxins produced by the pathogen have been considered to cause foliar SDS. One such toxin is the *F. virguliforme toxin* (FvTox1) that causes foliar SDS-like symptoms in soybean. This is a low-molecular-weight protein of approximately 13.5 kDa (FvTox1) purified from *F. virguliforme* culture filtrates. FvTox1 induces foliar SDS in soybean, most

likely through production of free radicals by interrupting photosynthesis (Brar et al. 2011). Of the four fusaria that have been shown to cause soybean SDS, field surveys indicate that *F. tucumaniae* is the most important and genetically diverse SDS pathogen in Argentina. The first report of sexual reproduction through perithecia formation by a soybean SDS pathogen, that is, *F. tucumaniae* that originated from Argentina, has been made by Scandiani et al. (2010). *F. tucumaniae* life cycle in South America includes a sexual reproductive mode, and thus, this species has greater potential for rapid evolution than the *F. virguliforme* population in the United States, which may be exclusively asexual (Covert et al. 2007). These findings support the hypothesis that the North America SDS pathogen is clonal and *F. virguliforme* in North America and *F. tucumaniae* in South America are the main casual fungi of SDS of soybean (Aoki et al. 2005, Westphal et al. 2008, Scandiani et al. 2011). A new TaqMan real-time PCR assay for the quantification of *F. virguliforme* in soil has been developed. The assay can be used as a diagnostic tool for rapid screens of field and greenhouse soil and for symptomatic and asymptomatic plants (Mbofung et al. 2012).

Epidemiology and Disease Cycle

The SDS pathogen survives between soybean crops as chlamydospores in crop residue or freely in the soil. As soil warms in the spring, chlamydospores near soybean roots are stimulated to germinate and then infect soybean roots (Westphal et al. 2008). Soybean seeds as the primary source of inoculum of SDS pathogen are also evident because the seeds contain the fungus mycelium after 12 months of storage and the fungus is transmissible after 12 months of storage (Balardin et al. 2005). The fungus also can survive in cysts of the soybean cyst nematode (SCN), *H. glycines*. The two pathogens *F. solani* f. sp. *glycines* (syn. *F. virguliforme*) × *H. glycines* act as a complex and the disease development is strongly dependent on high soil moisture (Xing and Westphal 2006).

Evidence for the existence of genetic variation in *F. virguliforme* has been provided and that the minor quantitative traits and environmental interactions are primarily responsible for the variation in aggressiveness found among isolates within the species (Mbofung et al. 2012). Variability of aggressiveness based on measurements of SDS foliar severity, shoot, root, and root lesion lengths; shoot and root dry weights; and total dry weights has been found among isolates (Li et al. 2009). Variability in carbon source utilization among *F. virguliforme* isolates is evident, but it is independent of geographic origin of the isolates (Tang et al. 2010). An international collection of *F. virguliforme* isolates has been established and maintained at the National Soybean Pathogen Collection Center, University of Illinois at Urbana-Champaign in the United States (Li et al. 2009). A real-time QPCR assay to compare the accumulation of genomic DNA among 30 *F. solani* f. sp. *glycines* (FSG) isolates in inoculated soybean roots has been developed. Isolates may differ significantly in their DNA accumulation on a susceptible soybean cultivar when detected and quantified using an FSG-specific probe/primers set derived from the sequences of the nuclear-encoded, mitochondrial small subunit ribosomal RNA gene (Li et al. 2008). Isolates of *F. virguliforme* from corn, wheat, ryegrass, pigweed, lambsquarters, canola, and sugar beet are the asymptomatic hosts of the pathogen (Malvick and Bussey 2008, Kolander et al. 2012). *F. virguliforme* may infect roots of soybean seedlings as early as 1 week after crop emergence. A protoplast-based fungal transformation system for *F. virguliforme* has been developed for the production of a green fluorescent protein (GFP)-expressing fungal transformant. The GFP-expressing fungus can be used to study fungal infection processes including fungal penetration, colonization, and spread, especially at the early stages of disease development (Mansouri et al. 2009). It is apparent that roots are most susceptible to infection during the first days after seed germination and that accelerated root growth in warmer temperatures reduces susceptibility to root infection conducive to foliar symptoms. However, soil temperature may not affect infections that occur as soon as seeds germinate (Gongora-Canul and Leandro 2011b). Cool temperatures are more favorable for root infection by *F. virguliforme* than warmer temperatures. Optimum soil temperature for root rot development is 15°C–17°C with root rot severity being lower at higher temperatures. Interestingly, in contrast to root infection, the

expression of foliar symptoms is favored by warmer temperatures of around 22°C–25°C. High soil moisture has been shown to favor SDS. Foliar symptoms are more severe in irrigated fields during wet season. The presence of continuous soil moisture throughout the growing season is most favorable for the development of the SDS. Rate of disease progress increases as inoculum densities increase for both root and foliar disease severities. The incubation period for root and foliar disease severity range from 9 to 18 and 15 to 25 days, respectively (Gongora-Canul et al. 2012).

The pathogen is capable of degrading lignin, which may be important in infection, colonization, and survival of the fungus (Lozovaya et al. 2006), but aboveground symptoms of SDS rarely appear until soybean plants have reached reproductive stages. The fungus produces toxins (FvTox1) in the roots that are translocated to the leaves (Brar and Bhattacharyya 2012). Often, symptoms first appear after heavy rains during reproductive stages; high soil moisture increases the disease severity (Xing and Westphal 2006). SDS is more severe when the SCN (*H. glycines*) is also present in a field and the cultivar is susceptible to both pathogens (Xing and Westphal 2006). The toxin requires light to initiate foliar SDS symptoms. Irrigation treatments during mid- to late reproductive growth stages result in significant increase in SDS foliar symptom development (de Farias Neto et al. 2006).

Both *F. virguliforme* and SCN are widespread. The close association of the pathogens is also apparent in the fact that the SDS pathogen can be isolated from cysts of SCN (Roy et al. 1997). The SCN, *H. glycines*, and the fungus *F. solani* f. sp. *glycines* that causes SDS of soybean frequently co-infest soybean fields. The infection of soybean roots by *H. glycines* does not affect root colonization by the fungus, as determined by real-time PCR. Although both pathogens reduce the growth of soybeans, *H. glycines* does not increase SDS foliar symptoms, and interactions between the two pathogens are seldom significant (Gao et al. 2006).

Although the pathogen may produce spores (macroconidia) on the surface of the taproot during the summer, these spores spread only short distances within a growing season. Over a period of years, flowing water and cultivation practices that move soil can move spores over longer distances within or between fields.

Disease Management

Host Plant Resistance

The use of resistant cultivars is the most effective method for controlling SDS in soybean. Although soybean cultivars that are less susceptible to SDS have been developed, no highly resistant cultivars are available (Njiti et al. 2002). Soybean genotypes with yellow seed coat show a relatively good field response to SDS and a moderate seed yield. Soybean cultivars show differences in their resistance to both the leaf scorch and root rot of SDS. Root susceptibility combined with reduced leaf scorch resistance has been associated with resistance to *H. glycines* (race 14) of the SCN (Kazi et al. 2008). These superior genotypes can be used as potential parents in soybean breeding programs (Gelin et al. 2006, Wen et al. 2014). Providing multiple resistance traits in the same variety is especially important to manage SDS, because both SDS tolerance and SCN resistance are frequently needed in the same variety (Butzen 2010). Research has led to the identification of soybean genotypes with 18 QTLs. However, it is possible that only 11 or 12 loci may contribute to host resistance as some of these loci may have multiple alleles. Some of these QTLs have been shown to be in close proximity to QTL that contribute to resistance to SCN with potential linkage between the two resistance QTLs (Leandro et al. 2012). Multigenic QTL present significant problems to analysis. Resistance to soybean SDS caused by *F. virguliforme* had been partly underlain by QRfs2 that could be clustered with, or pleiotropic to, the multigeneic rhg1 locus providing resistance to SCN (*H. glycines*) (Iqbal et al. 2009).

Soybean genotypes Ripley and PI 567374 both have partial resistance to SDS and the LG D2 QTL should be useful sources of SDS resistance (Farias Neto et al. 2007). The beneficial alleles of the QTL have been shown to be associated with resistance to either foliar disease severity or root rot severity or resistance to both foliar and root rot severity. QTL for resistance to *F. virguliforme*

are different from those that confer resistance to *F. tucumaniae*. The report that sexual reproduction occurs in nature in *F. tucumaniae* offers a greater challenge for disease management in regions where this species is found since host resistance to disease be easily overcome. As three other *Fusarium* species, as referred earlier, cause SDS in soybean in South America, it is important to use soybean varieties with broad resistance to the disease in this region (Leandro et al. 2012).

Molecular mechanisms underlying plant resistance and susceptibility to *F. virguliforme* have been studied using *Arabidopsis thaliana*. *A. thaliana* enabled a broad view of the functional relationships and molecular interactions among plant genes involved in *F. virguliforme* resistance. Dissection of the set functional orthologous genes between soybean and *A. thaliana* enabled a broad view of the functional relationships and molecular interactions among plant genes involved in *F. virguliforme* resistance (Yuan et al. 2008). Selection of seedlings in the greenhouse and marker-assisted selection (MAS) are faster and cheaper. DNA markers associated with loci contributing seedling resistance to *F. solani* in the southern and northern U.S. germplasm sources have been established. It is revealed that the SDS resistance can be a pleiotropic effect of shoot and root characters in partially resistant and relatively susceptible genotypes (Njiti and Lightfoot 2006). FvTox1 is an important pathogenicity factor for foliar SDS development, and expression of anti-FvTox1 single-chain variable-fragment (scFv) antibody in transgenic soybean can confer resistance to foliar SDS, and this could be a suitable biotechnological approach for protecting soybean crop plants from toxin-induced pathogen such as *F. virguliforme* (Brar and Bhattacharyya 2012). The fungal genome of *F. virguliforme* has been sequenced by conducting shotgun 454-sequencing. The genome sequence of *F. virguliforme* would become important public resource to a broad community of researchers engaged in developing tools to manage SDS (Srivastava et al. 2014).

Chemical Seed Treatment

Bayer CropScience has developed a chemical ILeVO for soybean seed treatment to provide protection for soybean seedlings from *F. virguliforme*, the fungus that causes SDS. ILeVO-seed treatment protects soybean from early-season infection and reduce late-season chlorosis and necrosis that leads to flower and pod abortion resulting in yield loss. The active ingredient in ILeVO is systemic and moves from the seed into the tissue of both stem and roots of soybean seedlings. The cotyledons and roots act as a sink for ILeVO, enabling the product to stay where it is needed to protect against early-season infection way in advance of SDS visual symptoms appearing in the field (Roden 2014). Since the fungus only colonizes the roots and base of the stem and it does not spread to the leaves and cannot be isolated from foliar portion, the foliar spray of the fungicides is not effective and hence foliar sprays of fungicides are not recommended.

Cultural Control

Soybean roots become less susceptible to xylem colonization and the subsequent development of foliar symptoms as plants mature. Therefore, practices aimed at protecting seed and seedling roots from infection may improve soybean sudden death management (Gongora-Canul and Leandro 2011a). In a regular growing season, an epidemic of SDS is highly correlated with the planting date and the disease tends to be more severe in earlier-planted soybeans in the United States (Navi and Yang 2008). Fields with a history of SDS should be planted later, rather than earlier in the spring. But planting may not be delayed to the point of compromising yield potential. A row spacing × infestation interaction indicated 7% greater yield in narrow rows (38 cm) than wide rows (76 cm) in uninfested plots, with no yield advantage to narrow rows in infested plots. In infested plots with greater SDS symptom expression, the yield advantage of narrow rows may be negated; therefore, cultivar selection is crucial when planting is done in narrow rows to maximize yield (Swoboda et al. 2011). Improving soil drainage, reducing compaction, evaluating tillage systems, and reducing other stresses on the crop if possible, in fields with recurring SDS problems, are useful SDS management practices. For example, a tillage system of disking or ridge till is effective in reducing the incidence of SDS as revealed from the studies done at the University of Missouri in the United States (Wrather et al. 1995).

The likely broad host range limits the efficacy of crop rotation and indicates that crops other than soybean can be damaged by *F. virguliforme* and can maintain or increase inoculum in soil crop rotation to have little impact on SDS incidence and severity (Xing and Westphal 2009, Kolander et al. 2012). Soil suppressiveness against the disease complex of the SCN and SDS of soybean is demonstrated (Westphal and Xing 2011). Chitosan is able to induce the level of chitinase antifungal enzymes to SDS pathogen in soybean resulting in the retardation of SDS development in soybean; it is thus helpful in partially protecting soybeans from *F. solani* f. sp. *glycines* infection (Prapagdee et al. 2007). SDS varies in severity from area to area and from field to field. This requires scouting fields when disease symptoms are present, ideally using GPS tools to map SDS-prone areas and then combination of crop management practices can help minimize the damage from SDS. By taking steps to manage *H. glycines* (resistant cultivars, nematicides), it is possible to help check SDS or at least manage its potential impact if not the disease itself.

CHARCOAL ROT

Symptoms

Symptom expression depends on the soybean plant's growth stage at the time of infection. Infected soybean seedlings show reddish discoloration of the hypocotyls appearing at soil level from root infection. Lesions become dark brown to black and infected seedlings may die under hot dry weather conditions. If wet and cool weather persists, infected seedlings survive but carry the latent infection and symptoms do not develop until plants reach reproductive stages, and only if heat and drought stress the plants. Hence, the disease is also known as *dry weather wilt and summer wilt* (Hartman et al. 1999). If the growing point is killed, a twin-stem plant may develop.

After flowering, the surface tissues (epidermis) of the lower stems of affected plants usually exhibit a light-gray or silvery discoloration and stems often have a shredded appearance. When the epidermis of lower stems and taproots is removed (by scraping with the thumbnail), extremely small, black fungal structures called microsclerotia are found embedded in the diseased tissue, which is the diagnostic feature of charcoal rot. Microsclerotia are tiny black masses of fungal tissue usually so numerous that they resemble charcoal dust, hence the name of the disease (Figure 9.7). The microsclerotia can be best seen with a good hand lens. Positive identification of the microsclerotia distinguishes charcoal rot from other similar diseases. The pycnidial stage is uncommon in soybean in contrast to formation of both microsclerotia and pycnidia on infected stem tissue of other host plants. Splitting the taproot often reveals dark-gray to blue-black streaks within. Later in the season, leaflets turn yellow, then die and shrivel, but remain attached to the plant. And finally, infected plants lose vigor and may die prematurely, and patches of such wilted and died plants are seen in the infested fields.

Geographical Distribution and Losses

Charcoal rot is a disease of economic significance throughout the world. It is widely distributed throughout tropical, subtropical, and warm temperate regions. Its effect is more pronounced in crops under biotic or abiotic stress. Changing global climatic conditions particularly occurrence of frequent drought or drought-like situations are making soybean more vulnerable to this disease. Charcoal rot is endemic in southern states in the United States and is a major problem in the central part of the Midwest, especially in Kansas and parts of Missouri. It is now occurring with greater frequency in the upper Midwest, with outbreaks reported in Illinois, Indiana, Iowa, Minnesota, North Dakota, and Wisconsin.

Root infection by germinating microsclerotia can occur very early in soybean plant development and about 80%–100% incidence of seedling infection can be observed within 3–4 weeks after planting. Based on estimates from 2006 to 2009, charcoal rot is listed as one of the 10 most

Charcoal rot (*Macrophomina phaseolina*)

(a) (b)

FIGURE 9.7 Charcoal rot of soybean-seedling mortality under field conditions and infected stems of soybean. (a) Seedling mortality by charcoal rot. (b) Infected lower stem and minute black sclerotia of pathogen in outer corticle and pith region. (Courtesy of Dr. G.K. Gupta, ICAR-Directorate of Soybean Research, Indore, India.)

yield-suppressing diseases in the United States (Koenning and Wrather 2010, Radwan et al. 2013). Charcoal rot ranks second among economically important diseases in the Midsouthern United States next to SCN. Estimated annual loss in soybean in the United States is about seven million bushels, whereas in Brazil, Argentina, and Bolivia, a loss of one million bushels could be attributed to charcoal rot in 1998. Interestingly, yield loss due to charcoal rot in soybean ranges from 6% to 33% even in irrigated environments in the United States (Mengistu et al. 2011). Infection with this pathogen reduces the number of pods per plant, seeds per pod, 100-seed weight, and seed yield. For example, infected plants may yield as low as 67.7 pods/plant, 1.5 seeds/pod, 6.5 g/100-seed and 6.4 g/plant compared to 205.8 pods/plant, 2.2 seeds/pod, 15.2 g/100 seed, and 69.7 g/plant of healthy plants. It is clear that soybean plants infected with charcoal rot have a reduced seed yield representing less than 10% of normal plant seed yield in Iraq (Abbas et al. 2003). Predominantly occurring in most of the soybean-growing states of India, charcoal disease causes 70% or more yield loss in soybean (Ansari 2010). Charcoal rot infection may alter seed composition and nitrogen fixation in soybean. The alteration in seed composition depends on cultivar susceptibility to charcoal rot and irrigation management (Bellaloui et al. 2008).

Pathogen

The disease is caused by the fungus *M. phaseolina* (Tassi) Goid. Its synonyms are *Macrophomina phaseoli* (Maubl.) Ashby, *Rhizoctonia bataticola* (Taub.) Butler, *Sclerotium bataticola* (Taub.) Butler., and *Botryodiplodia phaseoli* (Maubl.) Thir.

Classification
Kingdom: Fungi
Phylum: Ascomycota
Class: Dothideomycetes
Subclass: Incertae sedis
Order: Botryosphaeriales
Family: Botryosphaeriaceae
Genus: *Macrophomina*
Species: *phaseolina*

M. phaseolina is highly variable, differing in size of sclerotia and the presence or absence of pycnidia. *M. phaseolina* has a wide host range and geographic distribution, infecting more than 500 crop and weed species. The fungus is highly variable, with isolates differing in microsclerotial size and the ability to produce pycnidia. Microsclerotial morphology is a key taxonomic characteristic in the identification of this fungus. Cultural and morphological characteristics can vary as a result of continuous subculturing. The optimal temperature for growth in culture ranges from 28° to 35°C. The details of morphological characteristics of *M. phaseolina* have been described earlier under peanut and sunflower diseases chapters. The number of pycnidia that are produced by *M. phaseolina* isolates is dependent on induction medium; however, peanut butter extract–saturated filter paper placed over soy nut butter extract agar (PESEA) allows for greater pycnidia and conidia production than the other media. This conidia inoculum production method can facilitate soybean charcoal rot resistance screening evaluation with different soybean isolates (Ma et al. 2010).

Epidemiology and Disease Cycle

The fungus *M. phaseolina* is a causative agent of charcoal rot diseases in more than 500 plant species. The fungus is primarily soil inhabiting but is also seed borne in many crops including soybean. It survives in the soil mainly as microsclerotia. These are black, spherical to oblong in shape, and typically measure 0.002–0.008 in. in diameter. Microsclerotia produced in host tissues are released into soil as plant tissues decay. Corn, grain sorghum, and cotton generally support lower populations of microsclerotia in soil than does soybean. In dry soils, microsclerotia survive in soil or embedded in host residue for 2 or more years. In wet soils, microsclerotia cannot survive more than 7–8 weeks and mycelia no more than 7 days. Microsclerotia must germinate either on the surface of or in close proximity to roots for infection to occur. Pathogen growth and infection of soybean can occur at emergence and at the cotyledonary stage with 80%–100% of seedlings infected 2–3 weeks after planting. Phytotoxin, botryodiplodin, is suggested to be produced more abundantly by certain isolates of *M. phaseolina* facilitating infection in soybean (Ramezani et al. 2007).

Temperature optima for fungal growth and disease development are high (30°C–37°C). Considerable infection of soybean occurs at these temperatures. Seedling blight of soybean due to *M. phaseolina* is seen in tropical countries only where soil temperatures are at least 30°C at planting. Dry conditions, relatively low moisture and nutrients (NPK), and high temperature ranging from 25°C to 35°C are favorable for the disease at pod formation and filling stage (Ansari 2010). For example, August 2003 was the driest month recorded in Iowa, which may have contributed to the disease outbreaks in that crop season (Yang and Navi 2005). Similarly, in the humid tropics of southwestern Nigeria, areas with high soil moisture levels are unfavorable for the growth and pathogenicity of *M. phaseolina*, while areas with low soil moisture levels favor the growth and pathogenicity of the fungus (Wokocha 2000). Drought stress thus has been proved to increase *M. phaseolina* infections and reduces seedling dry weight in soybeans (Gill-Langarica et al. 2008). Low C:N ratio in the soil and high bulk density as well as high soil moisture content adversely affect the survival of microsclerotia. The scattered literature on these aspects has been reviewed (Gupta et al. 2012). The fungus is seed borne and invariably present in the seed coat of all the

infected seeds and moved into the cotyledons (including embryonal axis) of the 40% infected seeds (Tariq et al. 2006, Mengistu et al. 2012). The pathogen can remain viable for 15 months in seeds at room temperature and is transmitted to seedlings during germination by local contact (Kumar and Singh 2000). Although initial infections occur at the seedling stage, they usually remain latent until soybean plant approaches maturity (growth stages R5, R6 and R7). Plants infected after seedling stage generally show no aboveground symptoms until after midseason. There is a significant pathogenic and genetic variability within the soybean isolates of *M. phaseolina* from Iran, India, Italy, and Mexico (Munoz-Cabanas et al. 2005, Jana et al. 2005a,b, Rayatpanah et al. 2012a,b). Genetic variability studies among Brazilian isolates of *M. phaseolina* have revealed that one single root can harbor more than one haplotype. It is significant that *M. phaseolina* isolates from soybean are chlorate-sensitive isolates that grow sparsely with a feathery-like pattern and the isolates characterized by the feathery-like pattern are more virulent on soybean and sunflower (Rayatpanah et al. 2012a). Moreover, cultivation with crop rotation tends to induce less specialization of the pathogen isolates. Knowledge of this variation may be useful in screening soybean genotypes for resistance to charcoal rot (Almeida et al. 2003b). Genetic differentiation of *M. phaseolina* can be altered by crop rotation that *M. phaseolina* is a highly diverse species and also reveals a strong effect of the rotation system on genetic diversity (Almeida et al. 2008).

Cultivation with crop rotation probably tends to induce less diversity of the pathogen isolates (Rayatpanah et al. 2012b). The AFLP analysis has revealed great genetic diversity in *M. phaseolina* since more than 98% of amplified products appear to be polymorphic. But no clear association between geographical origin or host of each isolate and AFLP genotype has been found. A genetic dissimilarity greater than 10% is reported between a group of isolates from Mexico and Italy and isolates from other countries (Munoz-Cabanas et al. 2005).

Single primers of SSRs or microsatellite markers have been used for the characterization of genetic variability of different populations of *M. phaseolina* obtained from soybean and cotton grown in India and the United States. The variability found within closely related isolates of *M. phaseolina* indicated that such microsatellites are useful in population studies and represents a step toward identification of potential isolate diagnostic markers specific to soybean and cotton (Jana et al. 2005b). Universal rice primers (URPs) (primers derived from DNA repeat sequences in the rice genome) using PCR (URP-PCR) are sensitive and technically simple to use for assaying genetic variability in *M. phaseolina* populations (Jana et al. 2005a).

Disease Management

Host Plant Resistance

Strong resistance to charcoal rot does not exist among soybean cultivars. Six genotypes (one genotype in MG III, one in late MG IV, and four in MG V) have been identified as moderately resistant to *M. phaseolina* at levels equal to or greater than the standard DT97-4290, a moderately resistant high-yield potential cultivar (Paris et al. 2006, Ansari 2007). The genotypes identified as having moderate resistance across the 3 years could be useful as sources for developing resistant soybean cultivars (Mengistu et al. 2012). One such first report on soybean genotype with high levels of resistance to charcoal rot is *PI 567562A* and resistance in this genotype is greater than the standard *DT 97-4290* (Mengistu et al. 2012). Mexican lines H86-5030 and H98-1552, as well as Mexican cultivar Suaqui-86, are reported to be moderately resistant to *M. phaseolina* (Gill-Langarica et al. 2008).

Generally, the late maturity groups of soybeans are more tolerant to the disease. Lines B.P-692, J.K-695, and K.S-69035 show the highest tolerance to charcoal rot. Based on the results and the qualitative and quantitative agronomic characteristics, two lines (J.K-695 and B.P-692) have been selected as the suitable cultivars and are introduced as Sari and Telar, respectively, for cultivation in Mazandaran region in Iran (Rayatpanah et al. 2007). Early-maturing cultivars that do not have late reproductive growth stages might coincide with periods of drought stress and high temperatures

may help avoid severe damage to the disease during years with hot, dry summer weather conditions. Resistance to this pathogen in some genotypes is associated with drought tolerance. Some drought-tolerant soybean genotypes may resist root colonization by *M. phaseolina*, but this is not true for all drought-tolerant genotypes (Wrather et al. 2008). Ten genotypes (JS 335, G 213, Birsa Sova-1, GS 1, GC 175320, G 9, G-688, NRC 37, DSb 6-1, and RSC 14) have been identified as highly resistant (<1.0% morality) to *M. phaseolina* (Ansari 2007). Soybean cv Rawal is less susceptible to *M. phaseolina* (Ehteshamul-Haque et al. 2007). Planting earlier-maturing varieties in order to shorten the effect of a dry period at the end of the growing season is useful.

The cut-stem inoculation technique, which has several advantages over field tests, successfully distinguishes differences in aggressiveness among *M. phaseolina* isolates, and relative differences among soybean genotypes for resistance to *M. phaseolina* are comparable with results of field tests (Twizeyimana et al. 2012).

Induced Systemic Resistance

Some chemicals may play an important role in controlling the soybean charcoal rot disease, through induction of systemic resistance in soybean plants. The effect of two inducer chemicals, that is, riboflavin (B2) and thiamine (B1), on the induction of systemic resistance in soybean against charcoal rot disease and biochemical changes associated with these treatments in soybean plants have been investigated under greenhouse conditions. Riboflavin (0.1–15 mM) and thiamine (2.5–5 mM) are sufficient for maximum induction of resistance; higher concentration does not increase the effect (Abdel-Monaim 2011).

Plant growth–promoting rhizobacteria (PGPR), such as *B. japonicum* strain USDA 110, *Azotobacter chroococcum*, *Azospirillum brasilense*, *Bacillus megaterium*, *B. cereus*, and *P. fluorescens* when inoculated on soybean plants, result in inducing and enhancing the activity of PR proteins (chitinase and beta-1,3-glucanase), peroxidase, phenylalanine ammonia lyase (PAL), and phenolics and contribute to protect the soybean plants against *M. phaseolina* infection (Attia et al. 2011).

Chemical Control

Since *M. phaseolina* is also seed borne in soybean, seed treatment with effective fungicide can protect the seedlings from infection. Soybean seeds treated with thiophanate methyl applied as 0.1% or 0.2% dry seed treatment or as fungicide slurry with the addition of methyl cellulose result in the highest control of charcoal rot (Lakshmi et al. 2002). Seed treatment with carbendazim (as Bavistin 50 WP) (2.0 g/kg seed) and thiophanate methyl (as Topsin M) (1.0 g/kg seed) is also effective in eliminating the pathogen from infected seeds (Kumar and Singh 2000).

Cultural Control

Several disease management approaches involve the management of populations of microsclerotia using cultural practices to control charcoal rot. It may be a better alternative to suppress charcoal rot by using the no-tillage cropping system in comparison to conventional tillage (CT) system to conserve soil moisture and reduce disease progress (Almeida et al. 2003a, Mengistu et al. 2009a).

Farm practices that increase residue destruction immediately after harvest or those that enhance *Trichoderma* spp. populations may directly or indirectly lower the relative longevity of soilborne pathogens, including *M. phaseolina* (Baird et al. 2003). Water management can limit, but not prevent, colonization of soybean by *M. phaseolina*; excessively dense planting increases drought stress when water becomes limiting. Hence, avoiding excessive seeding rates is practiced so that plants do not compete for moisture, which increases disease risk during a dry season.

Macrophomina infection has been found to be lower in NPK treatment, and the lowest rate of disease development can be observed in the case of the highest NPK combination. By increasing the NK supply, the degree of infection is decreased (Csondes et al. 2008).

Soybean cultivars and other crop species in the host range differ in colonization, and these differences may affect soil densities of the fungus (Kendig et al. 2000). One-year corn–soybean rotation

is ineffective in managing charcoal rot since the fungus also causes corn stalk rot. However, the fungus is less damaging to corn than to soybean. Several years of corn or small grain crops rotations are necessary to reduce charcoal rot risk in severely infested fields. Although corn is a host, the microsclerotia numbers are still reduced under this crop. It requires at least 3 years without a soybean crop before microsclerotia levels are low enough to plant soybean again. Once the numbers of microsclerotia are low, a rotation of 1 year of soybean with 1 year of corn may keep microsclerotia numbers at low sustainable level (Kendig et al. 2000).

Microbial communities are more abundant and active in direct seeding (DS) than in CT in response to high nutrient content in soil (Perez-Brandan et al. 2012). Indeed, DS systems present higher soil OM and total N, K, and Ca than CT. Electrical conductivity and aggregate stability (AS) are also improved by DS. Soybean grown in high-quality soil is thus not affected by charcoal rot; however, under CT, disease incidence in soybean appears to have been 54%. These differences are correlated to the higher microbial abundance and activity under DS, the biological component being a key factor determining soil capacity to suppress the soilborne pathogen like *M. phaseolina* (Perez-Brandan et al. 2012).

Biological Control

Application of more than one antagonist of diverse origin is suggested as a reliable means of reducing the variability and increasing the reliability of biological control. *T. harzianum* and plant growth promontory rhizobacteria *P. fluorescens* when tested alone and in combinations for their relative biocontrol potential against *M. phaseolina* causing charcoal rot of soybean result in effective control of the disease (Mishra et al. 2011). *P. aeruginosa* strain Pa5 is a good candidate for use as BCAs against *M. phaseolina* on soybean cv Rawal (Ehteshamul-Haque et al. 2007). *P. fluorescens* isolates Pf-12 and Pf-63 inhibit the mycelial fungal growth of *M. phaseolina* through production of antibiotics as well as volatile metabolites, whereas *B. subtilis* isolates B-13, B-42, B-126, and B-84 do so through volatile and nonvolatile metabolite production. *P. fluorescens* isolates, however, also produce hydrogen cyanide. In greenhouse studies, the *B. subtilis* isolates B-13 and B-126 have been shown to be effective in reducing the intensity of charcoal rot of soybean by 59%–66%. The combinations of isolates B-13 and B-126 are also effective in reducing the intensity of disease (Sharifi-Tehrani et al. 2005). *Bacillus* sp. and *Trichoderma*-inoculated soybeans showed increased plant height, number of pods, vegetative growth, and aerial and radical weights (Cardona Gomez et al. 2000).

One strategy to control charcoal rot is the use of antagonistic, root-colonizing bacteria. *Rhizobacteria* A5F and FPT721 and *Pseudomonas* sp. strain GRP3 are characterized for their plant-growth-promotion activities against the pathogen. *Rhizobacterium* FPT721 exhibits higher antagonistic activity against *M. phaseolina* on dual plate assay compared to strain A5F and GRP3. FPT721 and GRP3 give decreased disease intensity. Lipoxygenase (LOX), PAL, and peroxidase (POD) activities have been detected in extracts of plants grown from seeds treated with rhizobacteria and inoculated with spore suspension of *M. phaseolina* (Choudhary 2011).

Another strategy to control charcoal rot is the use of antagonistic, root-colonizing PGPR. Effective biological control by the PGPR isolates indicates the possibility of application of *rhizobacteria* for control of soilborne diseases of soybean including that of charcoal rot in Pakistan and other countries (Inam-Ul-Haq et al. 2012). PGPR, such as *B. japonicum* strain USDA 110, *A. chroococcum*, *A. brasilense*, *B. megaterium*, *B. cereus*, and *P. fluorescens* when inoculated on soybean plants result in inducing and enhancing the activity of PR proteins (chitinase and beta-1,3-glucanase), peroxidase, PAL, and phenolics and contribute to protect the soybean plants against *M. phaseolina* infection (Al-Ani et al. 2011, 2012, Attia et al. 2011).

PGPR as mentioned earlier, phosphate-solubilizing bacteria (*B. megaterium* var. *phosphaticum*), and potassium-solubilizing bacteria (*B. cereus* and *P. fluorescens*) have been proven for their efficacy against *M. phaseolina* on soybean plants and for their influencing effect on percentage of healthy plant and growth. Data suggest the positive impact of PGPR in improving the stand and

vigor of soybean plants in *Macrophomina*-infested soil. In the field trial, results have shown that all tested PGPR significantly can decrease root rot and wilt disease incidence. *B. megaterium*–treated plots have been found to be the most effective treatment followed by the combination of *A. chroococcum*, *A. brasilense* and *B. megaterium*. The reduction in disease incidence reflected on plant growth and the apparent bacterial plant growth-promoting and bacterial BCAs could provide a means for reducing the incidence of root rot and wilt disease complex of soybean in addition to avoiding the use of fungicides. Such biocontrol approach should be employed as a part of IPM system (El-Barougy et al. 2009, Attia et al. 2011). For example, seed treatment with *B. japonicum* and *T. viride* and soil application of Zn with B and Fe reduce chaffy pods as well as the disease incidence up to 75%. Seed treatment with *Trichoderma* and irrigation at the time of moisture stress reduce the intensity of disease to about 50% (Ansari 2010).

YELLOW MOSAIC DISEASE

Symptoms

The diseased plants start appearing in the field when the crop is about a month old. Two types of symptoms—yellow mottle and necrotic mottle—are noticeable. The first visible sign of the disease is the appearance of yellow spots scattered on the lamina. They are mostly round in shape. In yellow mottle, the spots diffuse and expand rapidly. The leaves show yellow patches alternating with green areas and also later turn yellow. Such completely yellow leaves gradually change to a whitish shade and ultimately become necrotic. These color changes of affected plants are so conspicuous that the disease can be spotted in the field from a distance (Figure 9.8). In necrotic mottle, the center of yellow spots develops necrosis and the virus becomes systemic in the plant and all newly formed leaves show signs of mottle. There may be a reduction in size of leaves. Number and size of pods per plant and seeds per pod are generally reduced. The pods are deformed and contain shriveled undersized seeds.

FIGURE 9.8 Yellow mosaic of soybean at various stages of crop growth. (Courtesy of Dr. A.K. Tewari, GBPUA&T, Pantnagar, India.)

Geographical Distribution and Losses

At first, it was observed in North India in the early 1970s (Nene 1972) and since then, it has spread at alarming proportions. The disease is now endemic in South Asian countries (India, Pakistan, Bangladesh, Bhutan, Nepal, Sri Lanka). It is also reported to occur in the Philippines and Thailand. In the northern parts of India, the incidence of the disease may range from 20% to 80%. Soybean plants, if infected at prebloom stage, show 16%–73% losses in yield in different cultivars. Yield losses are of lower magnitude with infection of postbloom stage. In India, yield losses of 10%–88% had been reported due to YMD of soybean (Nene 1972, Bhattacharyya et al. 1999).

Pathogen: Mung bean yellow mosaic virus (MYMV) and mung bean yellow mosaic India virus (MYMIV).

Enzyme-linked immunosorbent assay, immunospecific electron microscopy, and whitefly transmission studies reveal that the etiological virus causing YMD in soybean is a begomovirus of the family Geminiviridae. Begomoviruses have characteristic icosahedral geminate particles that encapsidate the genome of circular single-stranded DNA. They infect dicots and are transmitted by the whitefly *Bemisia tabaci* Gennadius. Genomic components of the begomovirus that cause yellow mosaic disease (YMD) in soybean in Delhi, India, when cloned, sequenced, and evaluated for infectivity; nucleotide sequence analysis of the virus isolate revealed more than 89% identity with MYMIV; therefore, it is designated as a soybean isolate of MYMIV (MYMIV-Sb). Total nucleotide and predicted amino acid sequence analysis of MYMIV-Sb with other yellow mosaic virus isolates infecting legumes established dichotomy of the isolates into two species, namely, MYMIV and MYMV. The involvement of at least two distinct viruses in the etiology of soybean YMD in India is established (Usharani et al. 2004).

Yellow mosaic virus infecting soybean in northern India is distinct from the species-infecting soybean in southern and western India (Usharani et al. 2004). Girish and Usharani (2005) further determined the complete nucleotide sequences of two soybean-infecting begomoviruses from the central and southern parts of India, and the sequence analyses show that the isolate from Central India is a strain of MYMIV and the southern Indian isolate is a strain of MYMV. Thus, involvement of at least two distinct viruses in the etiology of soybean YMD in India is reported (Usharani et al. 2004). YMD of soybean is reported to be caused by soybean isolate of MYMIV (MYMIV-sb) (Radhakrishnan et al. 2008, Yadav et al. 2009). MYMIV-sb is similar to cowpea isolate of MYMIV (MYMIV-cp) in its ability to infect cowpea, but differing from blackgram (MYMIV-bg) and mung bean (MYMIV-mg), which do not infect cowpea (Usharani et al. 2005). Genomic analysis of DNA-A and DNA-B components of the MYMIV isolates shows characteristic differences in complete DNA-B nucleotide sequence correlating with host range differences (Usharani et al. 2005). Interestingly, MYMV virulent variant MYMV-Pp1 has been confirmed through nucleic acid spot hybridization using homologous probes to DNA-A and DNA-B of MYMV-Bg to cause infection in soybean (Biswas 2002).

They have a bipartite genome (two components, viz., DNA- 'A' and 'B'), which replicates via rolling circle replication (RCR) model with the help of few viral and several host factors. MYMIV is a representative of the genus *Begomovirus/Begomoviridae*, which is prevalent in the northern part of Indian subcontinent causing YMD. The most affected leguminous crops by MYMIV are *Cajanus cajan*, *G. max*, *Phaseolus aconitifolius*, *Phaseolus aureus*, *P. vulgaris* "French bean," and *Vigna mungo*. MYMIV possesses bipartite ssDNA genomes named as DNA-A and DNA-B, both being ~2.7 kb in size. Both components share a common region (CR) of about 200 bp containing the important *cis*-elements for viral DNA transcription and RCR.

Bipartite geminiviruses possess two movement proteins (NSP and MP), which mediate the intra- and intercellular movement. In order to accomplish the transport process, the MPs interact with viral nucleic acids in a sequence nonspecific manner (Radhakrishnan et al. 2008). Multiple DNA-B components could be detected with the soybean strain of MYMV species. The nucleotide sequence

similarity between the DNA-A components of the two isolates is higher (82%) than that between the corresponding DNA-B components (71%) (Girish and Usha 2005).

In bipartite begomoviruses, DNA-A encodes proteins required for replication, transcription, and encapsidation, whereas DNA-B encodes proteins required for movement functions. Phylogenetic analysis of complete DNA-A and amino acid sequence of various protein products of DNA-A clearly indicate the bifurcation of YMV isolates into two different species—MYMIV and MYMV. More number of isolates representing all geographical regions under soybean cultivation are required to be studied to find out if any recombinant between MYMIV and MYMV exists, as begomoviruses are known to show high frequency of recombinations. Phylogenetic study based on comparison of DNA-A nucleotide sequence of YMV isolates with other begomoviruses revealed a unique feature. Members of the genus *Begomovirus* are known to form clusters according to their geographical origin with distinct branches for viruses from America, Africa, and Asia.

Transmission

Female adults of the vector, *B. tabaci*, are more efficient vectors than males. Minimum acquisition feed time is 15 min and the same time is required for inoculation. Increasing feeding period up to 4 h increases transmission ability. Incubation period (latency) in the vector is at least 3 h, optimum being 5–6 h. Preacquisition starvation of the vector increases the efficiency to acquire the virus. In general, the vector is reported to acquire the virus 1–3 days before symptoms appear. A single viruliferous whitefly can transmit the virus but maximum infection is obtained with 10–20 whiteflies per plant. Neither female nor male adults can retain the virus throughout the life span. Normally, the female adults retain infectivity for 10 days and male adults for 3 days.

Epidemiology and Disease Cycle

Disease development is favored when maximum temperature and relative humidity prevail between 29.9°C–36.2°C and 62%–75%, respectively. The earliest YMD appearance of YMD is usually observed at 26–54 days after sowing (DAS). Disease spread becomes evident at 7–32 days after the initial disease appearance. The efficiency of whitefly (*B. tabaci*) as vector is affected by surrounding crops. YMD incidence is lower when soybean is alternated with mung bean. Cross inoculation tests revealed that YMD from mung bean or urd bean (*Vigna mungo*) is not directly transmitted to soybean, but YMD from soybean can be directly transmitted to French bean (*P. vulgaris*), *Alternanthera sessilis*, *Paracalyx scubiosus*, and *Sida rhombifolia* and vice versa. Disease development reaches its peak at 40–60 DAS then decreases thereafter. The distance-wise spread of YMD does not vary among high-, low-, and medium-risk fields. Epidemic development is observed at 60–70 DAS. At 50 DAS, disease development is positively associated with sunshine hours, relative humidity, cloudiness, temperature, and wind velocity (Gupta and Keshwal 2003).

Long-term surveillance study on disease flare-ups revealed that fields near irrigation canals, water points, low-lying areas and foot hills usually show high disease incidence when compared to unirrigated field plains in the state of Madhya Pradesh in India. Soybean cv. JS 81-335 and *Corchorus olitorius* have been found to act as bridge hosts in bringing inoculum of yellow mosaic virus from mung bean to soybean. Plant species such a*s P. scubiosus* is found to act as reservoir host of yellow mosaic virus inoculum. In addition, *A. sessilis* (*A. sessilis*) and *S. rhombifolia*, the weed hosts, have been found to help the multiplication and spread of inoculum. The study of weather parameter on yellow mosaic virus and whitefly population revealed that the rate of disease development is high when maximum temperature and relative humidity range between 31.0°C–36.2°C and 62%–75%, respectively (Gupta and Keshwal 2002).

Disease Management

Host Plant Resistance

Highly YMD-resistant soybean cultivars/genotypes such as SL 295, SL 328, SL 525, SL 603, UPSM 534, PK 1029, PK 1024, PK 416, and JS 9305 can be used as parents in crossing programs (Ramteke et al. 2007). Screening under controlled conditions with artificial inoculation with different isolates of the virus and pyramiding genes conferring resistance will help in breeding for durable resistance to MYMV in soybean (Lal et al. 2005, Ramteke and Gupta 2005). Soybean cultivars PK 1042, PK 1046, Pusa 20, and Pusa 40 are resistant to MYMV-Pp1. The resistant cultivars take longer time (16–29 days) to exhibit symptoms compared to susceptible cultivars (8–17 days) after inoculation (Biswas 2002). Soybean cultivar resistant to MYMIV infection induces viral RNA degradation earlier than the susceptible cultivar (Yadav et al. 2009, Yadav and Chattopadhyay 2014). More recently, out of 500 soybean germplasm lines collected from different parts of the world, only 48 genotypes have been detected to be resistant to YMD over 3 years (2007–2009) of consecutive hotspot screening (Kumar et al. 2014).

The inheritance of YMV resistance studied in two highly resistant varieties DS9712 and DS9814 indicated that the resistance is dominant and is controlled by single major gene (Talukdar et al. 2013). Similarly, the YMV resistance in wild accession, *G. soja*, is governed by a single dominant gene (Bhattacharyya et al. 1999) and the segregating populations generated will act as starting materials for developing improved lines with YMV resistance simultaneously paving the way for mapping the gene for YMV resistance with linked molecular marker. It is possible to develop molecular markers linked to MYMIV resistance to facilitate the genotyping of soybean germplasm for MYMIV reaction. Applying linked marker-assisted genotyping, plant breeders can carry out repeated genotyping throughout the growing season in absence of any disease incidence (Maiti et al. 2011).

A construct containing the sequences of Rep gene (566 bp) in antisense orientation has been used to produce MYMIV-resistant soybean plants, and the inheritance of transgene has been found to follow classical Mendelian pattern transgenic lines (Singh et al. 2013).

Vector Control

The management of the disease through prevention of population buildup of the vector can be possible. Spray of 0.1% metasystox, starting when the crop is about a month old or as soon as single diseased plant is seen in the field, can be useful in preventing severe incidence of the disease. However, control of the disease through control of vectors is often not very effective due to the fact that commonly recommended insecticides do not cause instant death of all individual vectors in the vector population and even a very few surviving population is capable of spreading the disease rapidly. Oil sprays can be more effective because they kill the insects within 15 min but they can be phytotoxic. Soil application of granular systemic insecticides at recommended doses can be a much better option for reducing vector population and delaying the appearance of the disease. Some fungal parasites of *B. tabaci* vector have been reported, which are potentially applicable for the development of biological control of the vector.

SOYBEAN CYST NEMATODE

Symptoms

The aboveground visible symptoms and definite signs of cyst nematode attack become detectable in a field only when the cyst content of the soil has gone very high. Foliar symptoms of SCN infection are not unique to SCN infection. In the first few crop seasons, after entry of the nematodes in a field, the disease goes undetected while the population of the cysts continues to rise. The symptoms at this stage could be confused with nutrient deficiency, particularly iron deficiency, stress from drought, herbicide injury, or another disease. The first signs of infection are groups of plants with yellowing

of leaves that have stunted growth. High population densities of the SCN can result in large portions of soybean fields with plants that are severely stunted and yellow. Small patches of poorly growing plants may appear in the field. The plants appear as if suffering from poor nutrition. Suspect fields usually have plants of different heights. Temporary wilting of plants occurs during hotter part of the day. Typical aboveground symptoms of heavy soil infestation are stunting and yellowing. Early senescence or maturation of the crop can be an indirect symptom of SCN.

When several crops of soybean are taken in the same field year after year, the patches of sick plants increase in dimension. The nematode feeds on the roots and root stunting, discoloration, and fewer nodules are belowground symptoms of SCN. The pathogen may also be difficult to detect on the roots, since stunted roots are also a common symptom of stress or other plant disease. Signs of root infection are the presence of adult females and white to brown cysts filled with eggs that are attached to root surfaces. Young females are small white and partly buried in the roots, with only part of them protruding on the surface, whereas older females are larger almost completely on the surface of the root and appear yellowish or brown depending on maturity. Once the cysts have matured, they turn brown and fall off the root.

Geographical Distribution and Losses

SCN is thought to be a native of Asia and has been a problem in China and northeastern Asian countries for centuries. The first documented report of damage by the SCN (*H. glycines* Ichinohe) was by S. Hori in Japan in 1915 (Davis and Tylka 2000). SCN was first reported in the United States in 1954 in North Carolina—an area known to import flower bulbs from Japan. It then spread with the expansion of soybean in the soybean belt (Illinois, Indiana, Iowa, Minnesota, Ohio, Missouri, Wisconsin) and adjacent states in the United States. Currently, this nematode causes more than U.S. $1 billion yield losses annually in the States alone, making it the most economically important pathogen on soybean (Liu et al. 2012). A 4-year study (2006–2009) done in the United States revealed that SCN (*H. glycines*) caused annual losses of $1.286 billion (128.6 million bushel). The SCN caused more yield losses than any other disease during 2006–2009 (Koenning and Wrather 2010). Yields may decrease slowly for a number of years as the population of SCN increases in the soil and infection of roots increases. SCN was detected in Colombia, South America, in the early 1980s and was soon thereafter found in Argentina and Brazil—two of the world's important soybean production countries. Yield losses can reach 100% in Brazil (Dias et al. 2009). The mean yield is reported to be 48% greater for the resistant cultivar compared with the susceptible cultivar in Iran (Heydari et al. 2012). SCN has also been reported from Egypt and Italy. In a survey of the top 10 soybean-producing countries in the world, SCN has been found to be the most damaging pathogen of soybean.

The penetration, feeding, and reproduction in soybean roots by the nematode result in direct yield losses and also allow other diseases to invade soybean roots. SCN can reduce soybean yield by more than 30% with no aboveground symptoms. When SCN infestation is severe, plants can become stunted and chlorotic and in some cases die resulting in up to 100% yield losses. In addition to causing yield loss directly, SCN also interacts with other pathogens (*F. virguliforme* and *Phialophora gregata*) making other diseases (SDS and BSR) worse during the same crop season.

Pathogen: *H. glycines* Ichinohe

Classification

Kingdom: Animalia
Phylum: Nematoda
Class: Chromadorea
Order: Tylenchida
Family: Heteroderidae

Subfamily: Heteroderidae
Genus: *Heterodera*
Species: *glycines* Ichinohe

The disease is caused by a microscopic roundworm, the plant-parasitic nematode, that changes shape as it goes through its life cycle, which forms cysts (overwintering structures) on soybean roots. Like all nematodes, the SCN (*H. glycines*) has six life stages—egg, four juvenile stages (J1–J4), and the adult stage. The duration of the SCN life cycle runs from 3 to 4 weeks, but this may be influenced by environmental conditions (mainly adequate temperature and moisture). The first-stage juvenile occurs in the egg; the worm hatches from an egg in the soil to produce the second-stage juvenile, or J2, nematode. The J2 is worm shaped, 375–520 μm long, and about 18 μm in diameter. It is the only life stage that can penetrate roots, and the third and fourth stages occur in the roots. The J2 enters the root moving through the plant cells to the vascular tissue where it feeds. The J2 induces cell division in the root to form specialized feeding sites. As the nematode feeds, in the root, juveniles become males or females and swell. SCN adults are sexually dimorphic, meaning that they are dissimilar in appearance. The females are swollen and sedentary, and the males are vermiform (worm shaped) and motile. The female eventually becomes flask shaped (0.4 mm in length × 0.12–0.17 mm in diam) and swells so much that its posterior end bursts out of the root and it becomes visible to the naked eye. In contrast, the adult male regains a wormlike shape (1.3 mm long × 30–40 μm in diam) and it leaves the root in order to find and fertilize the large females. Higher percentage of males is produced when the nematodes or host plants are under stress. Males do not feed, but they are required for sexual reproduction (copulation) with females that are exposed on the root surface. The male and juvenile stages must be extracted from soil or plant roots to be viewed under a microscope.

The fully developed yellowish-brown lemon-shaped female (0.6–0.8 mm in length × 0.3–0.5 mm in diam) after fertilization continues to feed as it lays 200–400 eggs in a yellow gelatinous matrix, forming an egg sac, which remains inside in its body, but some eggs may be laid in a gelatinous matrix extruded from the posterior (vulva) of the female. The female then dies. Eggs in the gelatinous matrix may hatch immediately, and the emerging second-stage juveniles may cause new infections. Subsequently as the gravid female dies, its cuticle becomes a brown, hardened structure (the cyst) that encases and protects hundreds of viable eggs. Cysts often fall from roots and remain free in the soil.

About 21–24 days is required for the completion of the life cycle of the nematode. Depending upon the environment, several generations of SCN can be completed in a typical soybean-growing season. A significant proportion of eggs that are retained within cysts are in a dormant state—they do not hatch until soybeans are planted for the next growing season. The overall body of the nematode is covered by a flexible, outer *cuticle*. The outside of the cuticle has a series of fine rings (annulations) that allow the cuticle to bend at any point along the nematode's body. The cuticle is composed mainly of the structural protein collagen, and the cuticle is molted four times to allow growth and maturation of the nematode. The *head* of the nematode can be recognized by the presence of a short, dark spear with basal knobs (the *stylet*) just inside the tip of the head. The stylet is hollow (like a hypodermic needle) and protrudes from the head when used by the nematode for feeding from plant cells and penetrating plant tissues. The very outer tip of the nematode head above the stylet (called the *lip* region) is slightly elevated, rounded, and darkened in J2 of SCN. In a relatively clear area just below the stylet, a round, muscular pumping organ called the metacorpus can be seen—the metacorpus pumps substances (i.e., food and secretions) up and down the esophagus of the nematode. Just below the metacorpus is another relatively translucent area that contains three esophageal glands that overlap the nematode's intestine on the ventral (stomach) side of its body. The intestine can be recognized as a fairly long, dark area extending from the esophageal glands to the tail of the nematode. The tail of SCN J2 tapers uniformly to a fine, rounded tip that is hyaline (Davis and Tylka 2000).

Epidemiology and Disease Cycle

Main source of survival of cyst nematode is the cyst. Eggs within the cyst can survive for 10 or more years. As with many plant-parasitic nematodes in soil, SCNs do not move far from the root zone. In most cases, the natural migration of SCN within a field is defined as *contagious*—small patches of infested areas that gradually enlarge to encompass significant areas of disease. The cysts are usually spread along with soil adhering to farm implements or anything that is contaminated with infested soil including seed-size clumps of dried soil within contaminated seed stocks. Surface drainage water, compost, shoes and feet of workers, movement of animals, and wind-borne dry soil are important means of spread. Even waterfowl and other birds feeding in infested fields may ingest cysts and carry them considerable distances. Diseased areas become much more pronounced in sections of soybean fields that are under environmental stress. It is possible that many soil factors may affect SCN reproduction and soybean yield loss, but only two soil factors are commonly associated with SCN damage and population densities—soil texture and soil pH. SCN is capable of infesting soils of all textures, but symptoms and yield loss generally are greater in sandy soils than medium- and fine-textured soils. SCN-infected roots are stunted and lack fine roots and, thus, can explore much less soil for water and nutrients than healthy roots. Also, coarse-textured soils do not hold water and some nutrients as well as medium- or fine-textured soils and SCN seem to cause greater damage to plants stressed by other factors, such as lack of water and/or minerals. The SCN population densities are more strongly related to high-pH soils (Rogovska et al. 2009, Pedersen et al. 2010). Among the microelement treatments, $FeCl_3 \cdot 6H_2O$ is the best one to inhibit J2 survival with the lowest value of LC50 (Zheng et al. 2010). A combination of soil compaction and real-time PCR enables rapid and sensitive quantification of SCN eggs in soil (Goto et al. 2009). SCN does not produce cysts containing eggs at a soil temperature of 33°C, although it does produce eggs at 25°C and 29°C. At soil temperature above 33°C for 200 h or longer, the egg reproduction ratio is significantly suppressed. After cultivation of resistant Peking, the egg number in the soil is significantly suppressed compared to that after Fuki (Uragami et al. 2005).

SCN has been reported to parasitize a broad range of host plants, encompassing nearly 150 legume and nonlegume genera representing 22 plant families. Several SCN host species are common winter annual weeds in U.S. soybean. The influence of winter annual weed management on SCN population densities has received little attention to date and warrants further investigation (Johnson et al. 2008). The SCN shows considerable degree of pathogenic variability all over the soybean-growing countries in the world (Dias et al. 2005, Rocha et al. 2008, Afzal et al. 2012, Asmus et al. 2012, Matsuo et al. 2012). This variability is large in Brazil, where 11 races (1, 2, 3, 4, 4+, 5, 6, 9, 10, 14, and 14+) have been found. Races 4+ and 14+ are found only in Brazil and differ from the classical 4 and 14 races, respectively, for their ability to parasite *Hartwig*, a North American soybean cultivar previously resistant to all races (Dias et al. 2009). Races are characterized by their ability to reproduce on certain soybean varieties. A system of designating races using the four differentials (Pickett, Peking, PI 88788, and PI 90763) has increased the number of potential races to 16 in the United States (Anonymous 2000).

When susceptible crop is planted, some hatching factor from the roots induces release of larvae. Hatching and emigration of larvae take place actively as a result of rise in temperature followed by host penetration and infection. The most rapid development and greatest female production occur between 20°C and 28°C. Male and female ratios do not differ in this range (Melton et al. 1986). However, the male-to-female ratio is the highest at 30°C–35°C (Rocha et al. 2008). Host penetration and infection occurs at a constant temperature of 20°C–22.2°C (Wang et al. 2009). These nematodes invade the root and partially reorganize root cell function to satisfy their nutritional demands for development and reproduction. After SCN hatch from eggs, the infective second-stage larvae penetrate primary roots or apical meristems of secondary roots. The larvae pierce their stylets into and feed off cells of the cortex, the endodermis, or the pericycle, causing the enlargement of these cells. The group of enlarged cells are called syncytia and serve as feeder cells for the nematode.

Syncytia often inhibit secondary growth of both phloem and xylem. Because a short portion of a root may be attacked by many larvae, the large number of syncytia that develop reduces the conductive elements and results in poor growth and yield of soybean plants, especially under stress of moisture (Wang et al. 2000, Alkharouf et al. 2006).

Disease Management

Once established in a field, SCN cannot be eradicated. However, there are various practices that can be implemented individually or in an IPM program to minimize SCN population densities at low to medium levels and maximize soybean yields in infested fields.

Host Plant Resistance

Effective management of this pathogen is contingent on the use of resistant cultivars. The genetic resistance is the most economical and accepted SCN control method by growers. Cultivars resistant to SCN can show greater yields in both high- and low-yielding environments and provide greater yield stability. These data support the selection of new cultivars that yield well at multiple locations and specifically cultivars with resistance to SCN for fields infested with SCN as a method to increase yield and yield stability (de Bruin and Pedersen 2008). However, host resistance must not be the only option because of the high genetic variability of the pathogen. This variability is large in most soybean-growing areas in the world (Dias et al. 2009).

Although more than 100 PIs (exotic varieties) have been identified with resistance to one or more SCN population designations, current resistant varieties trace to only a few PIs such as Pickett, Peking, PI 437654, PI88788, and PI 90763 from the soybean germplasm collection, which is also referred to as Hartwig resistance or the branded CystX® resistance (Anonymous 2008). The most widely used source of resistance is PI 88788. Because of the spread of multiple SCN races in Hokkaido, the Tokachi Agricultural Experiment Station (Japan) has bred soybeans for SCN resistance since 1953 by using two main resistance resources PI84751 (resistant to races 1 and 3) and Gedenshirazu (resistant to race 3) (Suzuki et al. 2012). It is confirmed that race 1 resistance in PI84751 is independently controlled by four genes, two of which are rhg1 and Rhg4. Suzuki et al. (2012) further classified the PI84751-type allele of Rhg1 as rhg1-s and the Gedenshirazu-type allele of Rhg1 as rhg1-g. I.

Having a variety with the correct source of resistance is the first step. Knowing the level of resistance is the second, and equally important, step. The level of resistance is given by the female index or resistance designation. In a general sense, the level of resistance is determined by how many resistance genes the variety has inherited from the original source of resistance. Both the source of resistance and the level of resistance are important for managing SCN in a field.

Resistance is described by no or limited reproduction of an SCN population on a given variety or genotype of soybean. This resistance is due to several (two, three, four, or more) genes being present and interacting in a soybean genotype. Soybean varieties labeled as resistant to SCN vary greatly in yield and in control of SCN. Both are determined by the genetics of the soybean variety and also the genetics of the SCN population in the field. The results of an HG-type test indicate how well a population will be controlled by the various sources of resistance used to develop soybean varieties. It is important to understand that the SCN designation describes the reaction of a population or group of individual nematodes with different genotypes to a source of resistance. Most field populations actually contain individual nematodes that would have different designations, but the population designation describes the average or majority reaction of the individual nematode genotypes in that population (Niblack et al. 2002). SCN-resistant varieties offer significant yield advantages (as much as 50% or more) over susceptible varieties when grown in heavily infested soil. However, variability of the pathogen enables some individuals to reproduce on resistant varieties thus making them less effective. To reduce the possibility of this happening, some researchers recommend that growers alternate the use of the soybean cultivars with different sources of SCN resistance and also that a

susceptible cultivar be grown once after all types of available resistance have been rotated. As far as possible, SCN-resistant varieties with other needed defensive traits, such as tolerance to iron deficiency chlorosis or resistance to SDS or *Phytophthora* root rot, should be preferred.

The HG-type test (*HG* represents *H. glycines*, the scientific name for SCN) is designed to give practical information about how well an SCN population in a field can reproduce on the various sources of SCN resistance. The *HG-type* system that has replaced the race system indicates which genetic sources of soybean resistance any given population of SCN can infect.

What population designation (race or HG type) represents the population of SCN individuals in the field is important to know. The most common population designation, for example, in Minnesota, is race 3 (one of 14 HG types). Knowing the population designation in a field is necessary in order to know what source of SCN resistance in the soybean would be most effective for that field (Niblack et al. 2002, Niblack 2005). Since 2003, the HG-type test has been adopted to replace the race test. This new test includes seven sources of resistance (germplasm lines) and the results are shown as a percentage, indicating how much the nematode population from a soil sample increased on each of the seven lines. This test indicates which sources of resistance would be good for the field being tested and which would be poor. Since the genetic sources of resistance are limited in commercially available soybean varieties, it is important to rotate these *sources of resistance* to delay the buildup of a virulent SCN population. Shift in virulence of SCN is associated with use of resistance from PI 88788. Rotation with alternative sources of resistance is recommended as a means to slow the adaptation to PI 88788 (Niblack et al. 2008). To delay SCN populations developing the ability to reproduce on SCN-resistant soybean varieties, producers should grow varieties with different sources of resistance in different years. If it is not possible to obtain the seed of an SCN-resistant variety with a source of SCN resistance different from what had been previously been used, rotate among different SCN-resistant varieties with the common source of SCN resistance, PI 88788 (Anonymous 2008).

The most common strategy applied by soybean genetic breeding programs in Brazil to introduce SCN resistance has been the selection of lines derived from populations resulting from crosses including adapted genotypes and North American cultivars with resistance derived from *Peking* (*Sharkey*, *Centennial*, *Padre*, *Forrest*, *Gordon*, among others) and/or the PIs 88788 (*Bedford*, *Linford*, *Fayette*, *Leflore*, etc.), 90763 (*Cordell*), and 437654 (*Hartwig*). The resistant cultivars are being developed along with the progress of the breeding programs and they, in turn, begin to replace with advantages of the North American resistant sources. Presently, there are about 50 soybean cultivars resistant to SCN in Brazil (Dias et al. 2009). Soybean germplasm lines S01-9364 (Reg. No. GP-350, PI 646156) and S01-9391 (Reg. No. 351, PI 646157) have value as parents in soybean improvement programs because of their broad resistance to SCN (*H. glycines*) populations (Liu et al. 2012).

Molecular Breeding for Resistance to SCN

Molecular mapping of QTL for resistance to SCN and MAS for breeding for SCN resistance have proven useful in order to assist in the development of SCN-resistant soybean cultivars at many major soybean breeding research institutes in the world (Arelli et al. 2010, Delheimer et al. 2010, Carter et al. 2011, Ferreira et al. 2011, Kim et al. 2011, Liu et al. 2011a,b, Mazarei et al. 2011, Vuong et al. 2011, Wu and Duan 2011, Arriagada et al. 2012, Yuan et al. 2012).

Recent advances in *H. glycines* genomics have helped identify putative nematode parasitism genes, which, in turn, will aid in the understanding of nematode pathogenicity and virulence and may provide new targets for engineering nematode resistance (Niblack et al. 2006). Real-time QPCR has been developed for screening for resistant cultivars, which can serve as a prelude to differentiation of resistance levels in soybean cultivars. With the QPCR assay, the time needed to differentiate highly resistant cultivars from the rest is reduced (Lopez-Nicora et al. 2012). This QPCR assay has the potential to replace the traditional female index-based screening and improve precision in determining infection levels.

Methods for MAS for SCN resistance have been identified (Young and Mudge 2002). Yields of the resistant cultivars are greater than those of the susceptible cultivars, except for the Peking source.

Compared with the susceptible cultivars, cultivars with *H. glycines* resistance from PI 88788 give a 13% increase in yield associated with a 15% increase in growth during R1–R5 growth stages. In cultivars with resistance from Hartwig, a 6% increase in yield is associated with a 4% increase in R1–R5 growth stages duration and increased seed-set efficiency. This work demonstrates that yield increases due to resistance to *H. glycines* can be attained by different physiological mechanisms associated with the different resistance sources and probably are controlled by different genes. This opens the possibility of pyramiding genes conferring resistance by different mechanisms (Rotundo et al. 2010). Recent advances in the study of the interaction between soybean and SCN at the genetic and genomic levels have been reviewed (Mitchum and Baum 2008). A total of 17 QTL mapping papers and 62 marker-QTL associations have been reported for resistance to SCN in soybean. SCN-resistant QTLs have been classified into three categories: suggestive, significant, and confirmed. Confirmed QTLs are credible and can be candidates for fine mapping and gene cloning. QTLs on linkage groups (LGs) G, A2, B1, E, and J are classified as confirmed. QTLs on LGs B2, C1, C2, D1a, D2, L, M, and N are classified into suggestive or significant. A relationship between soybean QTLs and SCN races has been reviewed (Guo et al. 2006).

Soybean PI 404198A is one of the newly identified sources that can provide a broad spectrum of resistance to SCN. QTL has been identified to be associated with resistance to SCN races 1, 2, and 5 in PI 404198A. LGs G and A2 are associated with resistance to race 1. Soybean PI 404198A may carry rhg1 on LG G, Rhg4 on LG A2, and a QTL on LG B1 (Guo et al. 2006). A SNP linked to the QTL of SCN resistance has been validated by comparing sequences amplified from *Hartwig*, a broad-based SCN-resistant line, and *Williams 82*, an SCN susceptible line (Gua et al. 2005, 2006).

Chemical Control

Chemical control with nematicides is not normally used because the economic and environmental costs are prohibitive. There are a few nematicides that are labeled for use against SCN, including the fumigant 1,3-dichloropropene (Telone) and the nonfumigants aldicarb (Temik or Bolster) and oxamyl (Vydate). When applied at planting, the effect of the nematicides may last long enough to provide an economic yield benefit (Schmitt et al. 2004). The performance of the nematicide will depend on soil conditions, temperatures, and rainfall. Yield and economic benefits generally are not guaranteed, but the chemicals are suggested to be applied at the soil depths of 5–15 cm, which can last for 1 month after soybean-seedling emergence for the effective management of the nematodes (Wang et al. 2009). Supplementing resistance with chemicals may improve soybean yield and/or nematode management, so a nematicide application, Aldicarb, {aldicarb[2-methyl-2 (methylthio) propionaldehyde *O*-(methylcarbamoyl) oxime]}, when included in the schedule, increases total plant biomass by 9% during R1–R5 soybean growth stages.

Cultural Control

SCN cannot reproduce if host plants are not present. Hence cultural practices, such as crop rotation, are useful as an effective tactic for SCN management. Because SCN is an obligate parasite (requires a living host), a crop rotation involving SCN nonhost plants like corn, alfalfa, small grains, sunflowers, flax, and canola can decrease the population of SCN (Jackson et al. 2005). For example, annual rotation of resistant soybean and corn results in the lowest SCN population density and produces the highest yield of both crops (Chen et al. 2001, 2007, Chen 2007). Similarly, the 2-year corn–soybean rotation generally results in increased soybean yield, decreased winter annual weed growth, and reduced SCN population density in comparison to when soybean is followed by soybean (Mock et al. 2012). In the North Central region of the United States, corn is almost exclusively used as a nonhost rotation crop with soybean (Miller et al. 2006). However, the data suggest that a single year of rotation of soybean with any other crops like sunflowers and flax before planting a susceptible soybean may not be sufficient in managing SCN (Miller et al. 2006). SCN-resistant soybean cultivars often are incorporated into a multiyear cycle of rotations with nonhost crops—this combination of practices is an excellent integrated management strategy

(Kulkarni et al. 2008, Dias et al. 2009). A certain percentage of SCN individuals can reproduce on resistant varieties. If sources of resistance are not rotated, these individuals can produce a SCN race shift. This will reduce the effectiveness of genetic resistance available in commercial soybean varieties.

Plants that have adequate moisture and nutrients are better able to withstand infection by SCN. In land infested with SCN, maintaining proper soil fertility and pH levels and minimizing other plant diseases, insect, and weed pests that weaken the plants are more critical to maximizing soybean yield than when land is noninfested.

The movement of soil can be best managed by following sanitation practices. If only certain fields on a farm are infested, planting and cultivating of infested land should be done only after noninfested fields have been worked. Soil on equipment should be thoroughly removed with high-pressure water or steam, if available, after working in infested fields. Also, seed grown on infested land should not be planted in noninfested fields unless the seed has been properly cleaned; SCN may be spread in the seed-size soil clumps mixed in with the seed (Davis and Tylka 2000, Schmitt et al. 2004, Donald et al. 2009).

Poultry litter at rates of 8 tons/ha when applied to SCN-infested soil results in the highest reduction in the number of SCN females and egg production (Lima et al. 2011). Anaerobically digested swine manure, which is actually the volatile fatty acid (VFA) manure, when applied to the soybean fields every 35 days, gives better results in reducing the SCN counts by 18%–34% (Xiao et al. 2007). Potassium fertilization at 150–600 mg/dm^3 (Pinheiro et al. 2009) and shallow tillage have been found to be advantageous to decrease the SCN population and to promote the suppressive effects of nonhost or trap crops, such as maize, crotalaria, and red clover (Tazawa et al. 2008).

Biological Control

Cysts and eggs of SCN are often found infected with one of several fungi such as *Fusarium*, *Verticillium*, *Neocosmospora*, *Dictyochaeta*, and more recently *Hirsutella minnesotensis* and *Hirsutella rhossiliensis* (Schmitt et al. 2004, Liu and Chen 2005). Biocontrol methods can play an important role in suppressing occurrence and damage of the nematodes (Chen et al. 2011). *H. minnesotensis* and *H. rhossiliensis* are endoparasites of nematodes, and their biological control potential against *H. glycines* is well known (Liu and Chen 2005). In general, percentage reduction of egg population density in the soil is negatively correlated with soil pH and positively correlated with sandiness. There appears to be no or weak correlation between egg reduction and organic matter. Soil pH and/or texture is important in influencing biocontrol effectiveness (Liu and Chen 2009). *Verticillium chlamydosporium* is another fungal BCA of SCN. Zn^{2+} stimulates the hatching of eggs of SCN. Cu^{2+}, Mn^{2+}, and Fe^{2+}, however, decrease hatching and Cu^{2+} could, therefore, be applied as a supplement to the biological control formulation (Xing et al. 2002). *H. minnesotensis* isolates vary in their efficacy in reducing the nematode population (Qian et al. 2011).

H. rhossiliensis controlled *H. glycines* more effectively in J2-infested soil than in egg-infested soil. Monitoring the population dynamics of a BCA in soil can be precisely studied with real-time PCR and bioassay (Zhang et al. 2008). Natural suppression of SCN exists and becomes increasingly attractive; however, ecological mechanisms leading to the suppressive state are rarely understood. Both bacteria and fungi are potentially involved in the soil suppressiveness to SCN: soil disturbance and biocide application may reduce natural soil suppressiveness that could be potentially associated with soil nematode community diversity and microbial enzyme activities (Bao et al. 2011).

Certain species of arbuscular mycorrhizal (AM) fungi could effectively inhibit the infection processes of SCN. It is proved that the tested AM fungi could significantly decrease SCN damage, reduce disease severity, the number of cysts on roots, the number of cysts and the second-stage juveniles (J2) in the rhizospheric soil, and the number of eggs per cyst. Among the AM fungi tested, *Glomus fasciculatum*, *Gigaspora margarita*, and *Glomus intraradices* are much more effective than *Glomus mosseae* and *Glomus versiforme* against the infection process of SCN (Li et al. 2002).

Effect of Plant Extracts

Mortality of SCN female induced by aqueous extract of the neem plant branches, leaves, and seeds is reported to be 99%, 97%, and 99.9%, respectively. The number of females on the root system when determined 30 days after the incorporation of 15 g of whole leaves/kg of soil or 10 g each of ground branches, whole seeds, and ground seeds/kg soil, the number of females recovered after incorporation of whole leaves, ground branches, whole seeds, and ground seeds has been found to be 1, 32, 9.1, and 0.8/root system, respectively, the differences being significantly different (5%). The number of females in the control roots could be 61, indicating the presence of toxic compounds in neem (Rodrigues et al. 2001). Overall, *Lolium multiflorum* is the most effective of all plant species tested for reducing populations of *H. glycines*, by increasing egg hatching of the nematode in the absence of a host, depleting lipid reserves of the juveniles, and inducing the lowest nematode parasitism of all nonhost residues studied (Riga et al. 2001).

OTHER DISEASES OF SOYBEAN

Brown Spot

Brown spot of soybean is caused by *Septoria glycines Hemmi* (teleomorph: *Mycosphaerella uspenskajae Mashkina & Tomilin*) and occurs in most soybean-growing regions in the world particularly in Argentina, Brazil, China, Pakistan, and the United States. Angular RB spots that vary in size from a pinpoint to 1/5 in. may appear on the lower leaves. Infected leaves turn yellow and fall prematurely. In severely infected fields, the lower half of the plant may lose all its leaves. The primary infection source of *S. glycines* is mainly from conidia within pycnidia surviving in plant residues infected in the previous year. The infection of *S. glycines* may be limited by the duration of water retention on the leaf, with a period of at least 24 h required. Warm, moist weather and poor drainage favor the spread of the disease. Management measures include the use of disease-free seed, crop rotation, deep burial of crop residue, and use of strobilurin foliar fungicides at R3 growth stage (Mirza and Ahmed 2002, Mantecon 2008, Carmona et al. 2010, Cruz et al. 2010).

Downy Mildew

The disease is caused by the fungus *Peronospora manshurica* (*Naumov*) *Syd*., which is of quarantine significance (Singh et al. 2003). It is the most widespread disease of soybean in the world. First, symptoms appear as indefinite yellowish-green areas on the upper leaf surface. Later, these areas become light- to dark-brown spots with yellow-green margins (Figure 9.9). In years favorable

FIGURE 9.9 Downy mildew of soybean. Note the initial symptoms on leaves. (Courtesy of Dr. Shrishail Navi, Iowa State University, Ames, IA.)

for the development of the pathogen, yields of susceptible cultivars may be considerably reduced. Disease symptoms may be systemic and local. Most typical symptoms occur on leaves, in the form of chlorotic spots, which necrose and coalesce with time. Conidiophores and conidia grow over the reverse side of the leaf. *P. manshurica* survive through oospores, which reside on seeds and plant residues. In the course of the growing season, *P. manshurica* proliferates by conidia (Vidic and Jasnic 2008a). Management measures include the growing of resistant cultivars. Soybean cultivar *AGSI29* is resistant to downy mildew. Marker OPH-021250 has been found to be present in 13 of 16 resistant soybean cultivars so investigated and absent in susceptible cultivars, thus confirming a potential for MAS for breeding for downy mildew resistance (Chowdhury et al. 2002). The use of healthy seed or seed treatment with fungicides based on metalaxyl, oxadyxil, and mancozeb could be useful in preventing the spread of inoculum through seed.

Purple Seed Stain

This disease is caused by the fungus—*Cercospora kikuchii* (Matsumoto & Tomoyasu) M. W. Gardner. It is reported to occur in almost all soybean-growing regions in the world. This disease often appears late in the season and can cause leaf blighting and staining of the seed. Yield losses are often minimal, but a reduction in seed quality can occur due to staining. In most cases, 7%–13% reduction in emergence can occur in the field. Leaves often have red to purple lesions, less than 1 cm in diameter, which become noticeable in August or early September. Infected seed has a distinctive purple discoloration (purple seed stain), varying from violet to pale purple to dark purple over part or all of the seed coat (Figure 9.10). This discoloration is often confined to the upper two layers of the seed coat. Size of the discoloration may vary from a small spot to the entire seed surface. The pathogen attacks other plant parts and overwinters in diseased leaves and stems as well as in infected seed. Premature defoliation may occur when leaves are severely infected. When infected seeds are planted, the fungus grows from seed coats and infects seedlings. This serves as a primary source of inoculum. Wet weather during the growing season favors the development of the disease. RH above 80% and temperature from 20°C to 24°C are more favorable for the germination of conidia and disease development (Kudo et al. 2011). The fungus overwinters in diseased crop residue as well as on infested seed.

There is a high degree of genetic variability and cercosporin production among isolates (Lura et al. 2011), and the population genetic structure of *C. kikuchii* is different between South America and Japan (Imazaki et al. 2006a). The disease management involves using a variety with greater tolerance. Three genotypes, AG5701 (Asgrow), TV59R85 (Terral), and PI80837, are among the more resistant cultivars to the disease (Jackson et al. 2008, Cai et al. 2009). Clean seed and a fungicide (azoxystrobin or carbendazim) seed treatment crop rotation and removal of residue to reduce infection have been potentially useful strategies in disease management (Imazaki et al. 2006b, Prasanth and Patil 2007).

Frogeye Leaf Spot

FLS, caused by *C. sojina* K. Hara, is a common disease of soybean in most soybean-growing countries of the world. Significant yield losses of soybean (10%–60%) have been attributed to FLS under hot and humid growing conditions (Mian et al. 2008). This disease usually appears late in the growing season and the economic impact is usually minimal. The fungus infects leaves, stems, and pods but is most conspicuous on the leaf. Symptoms occur in midseason and then become more severe after flowering. On the leaf, it causes an *eyespot* lesion composed of a gray or tan central area surrounded by a narrow RB margins. Lesions are 1–5 mm in diameter with a tan center and a dark-red/brown border. Older lesions coalesce, and leaves may appear ragged or with a slight slit in the center of the lesion. Badly infected leaves fall prematurely. The fungus is seed borne and also overwinters in residue and causes weak seedlings.

Eight genotypes such as ID, LMD, NLC, DI, PLLA, Cristalina, Davis, and Uberaba are the most resistant. The additive, dominant and epistatic genetic effects are important for the expression

Cercospora blight and purple seed stain (Cercospora kikuchii)

(a) Symptoms of cercospora leaf blight

(b) Purple stain on the seeds

FIGURE 9.10 Purple seed stain of soybean. Note the symptoms on leaf (a) and seed stain discoloration (b). (Courtesy of Dr. G.K. Gupta, ICAR-Directorate of Soybean Research, Indore, India.)

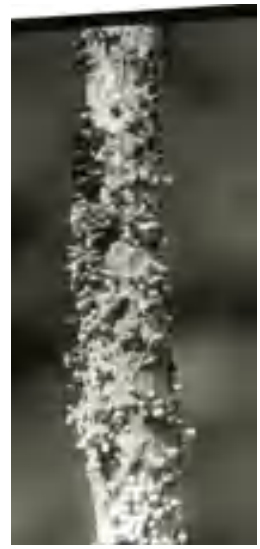
of resistance, although the additive genetic effect is reported to be the most important component. These cultivars can be recommended as parents in soybean breeding programs for enhanced resistance to *C. sojina* (Gravina et al. 2004). Advances in research on soybean resistance and inheritance and breeding of resistance against the fungal pathogen *C. sojine* [*C. sojina*] have been reviewed (Cao and Yang 2002). Results demonstrate that the resistance to *C. sojina* is controlled by a dominant gene or a gene block; additive genetic effect and dominance are involved; the effect of the environmental variation is minimum; and the interaction among the genes ranges from the partial to the complete dominance type, depending on the characteristic used in the evaluation of the resistance (Martins Filho et al. 2002). Advances in research on soybean resistance and inheritance and breeding of resistance against the fungal pathogen *C. sojine* [*C. sojina*] have been reviewed (Cao and Yang 2002). *C. sojina* is a dynamic pathogen with extensive virulence or race diversity. Twelve differentials and 11 races of the pathogen have been identified, which should provide the foundation for the identification and comparison of additional soybean resistance genes and new races of *C. sojina* (Mian et al. 2008). Management measures include planting disease-free seed and plowing under crop residue and crop rotation with nonhosts, such as corn or wheat.

Sclerotium Blight (Southern Blight)

Southern blight or southern stem blight is caused by the fungus *S. rolfsii* Sacc. This fungus survives in the soil on organic matter, is favored by hot weather stress, and is recognized by the appearance of white mold on stems at the soil surface causing rotting of stems and roots. Small tan to brown, *mustard seedlike* fruiting bodies (sclerotia) are produced within the white mold growth (Figure 9.11). The disease is most often seen in June, July, and August during very wet periods. Southern blight

Sclerotial blight (*Sclerotium rolfsii*)

(a)



(b)

FIGURE 9.11 Sclerotium blight of soybean. Note the presence of fungal growth and mustard seedlike sclerotia on affected plant. (a) White cottony mats of mycelium of pathogen in collar region of seedlings. (b) Reddish-brown sclerotia of pathogen on lower portion of stem of seedling. (Courtesy of Dr. G.K. Gupta, ICAR-Directorate of Soybean Research, Indore, India.)

is very common in fields with moderate to high levels of root-knot nematode (RKN). Occurrence of southern blight in a field is erratic and generally only individual plants are affected. However, in some instances, large numbers of plants may be killed. Plants may be affected at any stage of growth. The first symptom is sudden wilting and subsequent death. The sclerotia are the resting stage of the fungus and will persist in the soil for years. The fungus occurs widely in many soils and is capable of persisting on almost any type of organic matter. On the basis of oxalic acid (OA) production in culture filtrates and pathogenicity on different soybean varieties (cvs. Improved Pelican, Lee, Hardee, and Bragg), the isolates have been grouped into 12 races. Race I is more dominant than other races and highly virulent to all the soybean varieties. There appears to be a positive correlation between OA production and the virulence of the isolates of *S. rolfsii* (Ansari and Agnihotri 2000). It is difficult to manage the disease. Seed treatment with some fungicides such as carboxin and thiram may be effective to limited extent. But integrated approach involving rotation with other crops such as cotton or corn and soil amendments with organic matter can be effective in reducing the inoculum of the pathogen. Dried powders of kudzu (*Pueraria lobata*), velvet bean (*Mucuna deeringiana*), and pine bark (*Pinus taeda*) each at the rate of 25 g/kg stimulate increases in populations of antagonistic microorganism such as *Trichoderma koningii* and *Penicillium citreonigrum* and *Penicillium herquei* and are useful in reducing the incidence of the disease (Blum and Rodriguez-Kabana 2006b). *Bacillus thuringiensis* subsp. *israelensis* has been used to produce chitinase. The addition of chitinase (0.8 U/mg protein) causes increase in seed germination to 90%. *B. thuringiensis* chitinase may contribute to the biological control of *S. rolfsii* and other phytopathogenic fungi in soybean seeds in IPM programs (Reyes-Ramirez et al. 2004).

Diaporthe Pod and Stem Blight/Phomopsis Seed Mold

A complex of soybean diseases is caused by *Diaporthe/Phomopsis* species (D/P complex). D/P complex is grouped into two major taxa: *Diaporthe phaseolorum* var. *sojae* (*Lehman*) *Wehm.* (anamorph = *Phomopsis*) and *Phomopsis longicolla Thomas W. Hobbs.*, which are described as soybean pathogens. The first species includes three varieties: *D. phaseolorum* var. *sojae* (anamorph: *P. sojae*), the causal agent of pod and stem blight, and *D. phaseolorum* var. *caulivora* and *D. phaseolorum* var. *meridionalis*, agents of northern and southern stem canker, respectively. In addition to distinguishing interspecific and intraspecific variability, molecular markers allow the detection of differences among isolates within the same variety (Pioli et al. 2003). *D. phaseolorum* var. *caulivora* (northern stem canker) is the most economically important because it causes wilt and drying of plants during pod development and grain filling. Prematurely wilted plants yield 50%–62% less than healthy plants. *P. longicolla* is the most common and most damaging agent of soybean seed decay. The diseases caused by parasites from this D/P complex genus were first observed and described on soybean in the United States. Presently, they are widespread in most soybean production regions around the world (Li et al. 2004, Santos et al. 2011, Vidic et al. 2011).

Pod and stem blight is caused by the fungus *D. phaseolorum* var. *sojae* (sexual stage) also known as *P. longicolla* (asexual stage). Although plants are infected early in the season, symptoms do not become apparent until after midseason. The disease is identified by the numerous small, black fruiting bodies (pycnidia) appearing on stems and pods of infected plants. The pycnidia are arranged in linear rows on the stems, which is a useful diagnostic criterion to differentiate it from the brown spot and anthracnose diseases symptoms and signs. The pycnidia of the brown spot fungus and acervuli of anthracnose fungus do not occur in rows. RAPD and PCR-RFLP showed that significant variability exists within the population of *D. phaseolorum* var. *sojae*. Infected harvest residues and soybean seeds are the main sources of pathogen inoculum. Humidity and temperature (soil and air) are the main factors that affect the dynamics of fruiting body formation, spore release, establishment of infection, and the development of disease symptoms in soybean. The fungus infects seed and causes them to be shriveled, moldy, and smaller than normal. Seed may be infected but appear normal. Seed infection is the most serious phase of the disease. When infected seeds are planted, the

embryo is often killed before emergence or the seedlings are killed at an early stage. Delayed harvest results in an increased incidence of the disease, especially if rain or humid weather and warm conditions prevail. At this period of time, the pathogen is predominantly present in its asexual stage, *P. longicolla*. *P. longicolla* is the primary agent of seed decay and latent infections of seed, although the other members of this genus may cause identical symptoms (Mengistu et al. 2009b). It is characterized by fine cracks that usually develop near the hilum of the infected seed. A white or gray mold may be visible on the seed surface. The yield, grade, viability, and vigor of the seed can be reduced. Yield losses occur because severely infected seeds remain small and light and may be lost during harvest and cleaning operations. Isolation of *P. longicolla* from seed is negatively correlated with percentage of seed germination in irrigated environments but not in the nonirrigated environment (Mengistu et al. 2009b). The fungus overwinters in seed and crop debris. Spores of the fungus are splashed onto developing plants early in the season. Warm, wet, and humid weather during pod fill favors disease development. Varieties that mature late during the cool weather in the growing season or varieties that are short season for an area tend to mature earlier before environmental conditions become warmer and more favorable for seed mold should be preferred. Pod and stem blight can be controlled or reduced by integrating one or more of the control practices such as the use of planting pathogen-free seed, planting later, crop rotation, plowing under soybean debris, and a well-timed harvest.

Target Spot

Target spot is caused by the fungus, *Corynespora cassiicola* (Berk. & M. A. Curtis) C. T. Wei. It is found in most soybean-growing countries, particularly in Brazil and the United States. It is considered to be a disease of limited importance, although its incidence is increasing all over the tropical and subtropical regions. Under favorable climate conditions, it can cause serious damage to soybean. It has become an economically important disease in Brazil in the recent past (Teramoto et al. 2013) and assuming increasing importance in the southeastern United States (Koenning et al. 2006). Symptoms consist of the development of RB leaf lesions that are round to irregular varying from specks to mature spots, which are a centimeter or more in diameter. A dull green or yellowish-green halo commonly surrounds the lesions, which often become concentrically ringed at maturity, hence, the name target spot. Severely affected leaves fall prematurely. Microscopic examination of the lesions can reveal the presence of spores (conidia) typical of *C. cassiicola*. Conidia mostly three to five septate with a central hilum at the base and may range in size from 7 to 22 wide × 39 to 520 µm long. Dark-brown specks to elongated, spindle-shaped lesions form on the petioles and stems. Pod lesions are round and small but may enlarge and merge to cover the entire pod during wet or very humid periods. The fungus may sometimes grow through the pod wall and form small, blackish-brown lesions on the seeds. Large lesions form on the primary roots and growth of the secondary roots may be retarded. The *Corynespora* fungus overseasons in infected soybean debris and seeds and can survive in fallow soil for more than 2 years. The fungus can colonize a wide range of plant residues in soil as well as the cysts of the SCN. Leaf infections occur when free moisture is present on the leaves and the relative humidity is 80% or above. Heavy rainfall associated with hurricanes enhances the disease incidence during September 2004 in the southeastern United States (Koenning et al. 2006). Dry weather inhibits infection and colonization in both leaves and roots. Stems and roots first become infected in the seedling stage. Soil temperatures of 15°C–18°C are optimal for infection and disease development. The pathogen has an extremely wide host range and infects many plant species. There is an abundance of unrecognized genetic diversity within the species and provides evidence for host specialization on certain hosts such as papaya (Dixon et al. 2009). Management practices include the use of disease-resistant cultivars, sanitation involving destruction of crop residues, and avoiding soybean monoculture. Fungicides are rarely justified economically. Among the biological control agents, *T. harzianum* strain RMA-6

and *T. pseudokoningii* strain HMA-3 are reported to be the most effective and can be potentially useful in target spot disease management in soybean (Kaushal 2009).

Sclerotinia Stem Rot

Sclerotinia stem rot (*SSR*) or White mold disease caused by *Sclerotinia sclerotiorum* (Lib.) deBary is prevalent in all soybean-growing regions in the world, being most important in temperate regions and under cool conditions, often at intermediate altitude. Local epidemics outbreaks of the disease have been reported from Argentina, Brazil, Canada, Serbia, and the United States, when weather conditions are favorable for disease development (Vidic and Jasnic 2008b, Alvarez et al. 2012). It is most damaging in years with frequent and abundant rains in the summer. In some fields, more than 50% of plants are infected, causing significant yield reductions (Peltier et al. 2012). Symptoms occur on all aboveground plant parts in the form of thick, white, and soft mycelia and hence the disease is also referred to as *cottony soft rot*. The infected plant parts rot and the infected plants wilt and dry up. *S. sclerotiorum* is polyphagous. The fungus survives by sclerotia, which remain vital in the soil for several years. They germinate and give rise to the mycelium. Alternatively, under favorable weather conditions (humid and cool weather), bowl-shaped fruiting bodies (apothecia) form on the sclerotia. Infection follow colonization of injured or senescent tissue like flowers, cotyledons, or leaves, either by germinated ascospores from asci from the apothecia or directly from mycelium from sclerotia. *S. sclerotiorum* secretes OA and endo-polygalacturonase (endo-PG), which are important pathogenic factors in host plants (Favaron et al. 2004). Symptoms first appear as a watery-soaked lesion followed by cottony growth on the affected plant part with the formation of black irregular-shaped sclerotia. Epidemic development of the disease is favored by temperatures less than 21°C and secondary spread has been shown to occur at 18°C. Continuous moisture on leaves within the canopy or on infected flowers for a period of 48–72 h favors infection by ascospores. There is a potential for field to field dispersal of *S. sclerotiorum* and the majority of ascospores of *S. sclerotiorum* are deposited close to the source (apothecia), where a concentrated area or point source of *S. sclerotiorum* inoculum exists (Wegulo et al. 2000). Forecasting is based on soil moisture, canopy enclosure, senescing leaves, air and soil temperature, and the presence and number of apothecia.

Long-term crop rotation (corn–soybean rotations and compost amendment (Rousseau et al. 2007, Vidic and Jasnic 2008b)) with no soybean tillage (Gracia-Garza et al. 2002) are currently the major methods of controlling this disease. However, 1 year of moldboard plowing will bury sclerotia at least 10 cm in soil and delay the production of apothecia. How this affects SSR development depends on the other factors involved with disease development (Mueller et al. 2002). Fungicides such as thiophanate methyl are another option for the control of SSR but usually recommended in situations where susceptible cultivars must be grown or modification of cultural practices are not disease control options (Muller et al. 2004). The incidence of SSR can be reduced by planting partially resistant cultivars and by implementation of cultural practices that limit pathogen activity. *BCAs* such as *Coniothyrium minitans* CON/M/91-08 (product name: ContansReg. WG), *Streptomyces lydicus* WYEC 108 (ActinovateReg. AG), *T. harzianum* T-22 (PlantShieldReg. HC), *B. subtilis* QST 713 (SerenadeReg. MAX) (Zeng et al. 2012), *Sporidesmium sclerotivorum* [= *Teratosperma sclerotivora*] (Rio et al. 2002), *Clonostachys rosea* BAFC3874 (Rodriguez et al. 2011), and *B. amyloliquefaciens* strains ARP23 and MEP218 (Alvarez et al. 2012) all have been proved to be very effective in reducing the inoculum potential and SSR incidence in soybean.

Few genetic sources of resistance to the pathogen are available to breeders. Therefore, farmers have a continuing demand for new approaches to control the disease. The QTL associated with resistance to *S. sclerotiorum* in soybean genotypes PIs 391589A and 391589B have been identified (Arahana et al. 2001, Guo et al. 2008). SSR markers associated with resistance QTL mapped for SSR resistance may be useful for marker-assisted breeding programs in soybean (Vuong et al. 2008). Biotechnology opens a new avenue to manage this pathogen. Several strategies, including

detoxification, defense activation, and fungal inhibition, have potential to engineer *Sclerotinia resistance* (Lu 2003). Enzymes capable of degrading OA have been utilized to produce transgenic resistant plants. Transgenic soybean lines containing the decarboxylase gene (oxdc) isolated from a *Flammulina* sp. have been produced by the biolistic process. Molecular analysis reveals successful incorporation of the gene into the plant genome and shows that the OA decarboxylase (oxdc) gene has been transferred to the progeny plants (Cunha et al. 2010). An oxalate degrading enzyme, oxalate oxidase (OxO), in transgenic soybean has provided white mold resistance equivalent to the best commercial cultivars in a white mold–susceptible background (Donaldson et al. 2001, Cober et al. 2003).

Rhizoctonia Aerial/Foliar Blight

Rhizoctonia aerial/foliar blight of soybean is reported to occur in tropical and subtropical areas worldwide. It is becoming increasingly more important in Brazil (Ciampi et al. 2005, 2008), North Korea (Kim et al. 2005), and the southern United States particularly in Louisiana and North Carolina (Stetina et al. 2006) during prolonged periods of high humidity and high temperatures. This disease of soybean occurs with high disease severity of 50%–75% in tidal swamp land in south Kalimantan in Indonesia (Rahayu 2014). It has been estimated that the disease can cause about 70% losses of foliage and soybean pods. Extensive yield losses (40%–50%) have been reported in soybean when conditions favor disease development. However, Meyer et al. (2006) recorded that *Rhizoctonia* foliar blight of soybean causes higher yield reductions in the range of 60%–70%. Foliar symptoms often occur during late vegetative growth stages on the lower portion of the plant following canopy closure. Initially, leaf symptoms appear as water-soaked, grayish green lesions that turn tan to brown at maturity. The pathogen may infect leaves, pods, and stems in the lower canopy. RB lesions can form on infected petioles, stems, pods, and petiole scars. Long strands of weblike hyphae can spread along affected tissue and small, dark-brown sclerotia form on diseased tissue. Weblike hyphae of *R. solani* spreading along the stem of soybean becomes evident.

The Basidiomycete fungus *R. solani* Kuhn anastomosis group (AG)-1 IA (*Thanatephorus cucumeris* (Frank) Donk) is a major pathogen foliar blight of soybean all over soybean-growing regions in the world. But *R. solani* AG1-IB and AG2-3 are also reported to be the causes of foliar blight of soybean in Japan (Meyer et al. 2006). The pathogen overwinters as sclerotia in soil or plant debris from the preceding crop. During warm, wet weather, mycelium spreads extensively on the surface of plants, forming localized mats of *webbed* foliage. Spread from these localized areas can be rapid when conditions favor disease (high RH and 25°C–32°C). Because this pathogen also causes sheath blight of rice, soybean fields that follow rice with a history of sheath blight are likely to have high incidence of aerial blight. There is little host resistance to *R. solani* in soybean, but some cultivars are less susceptible than others. Planting the least susceptible and best adapted cultivar, rotating it with poor or nonhost crops such as corn or grain sorghum for 2 years, and avoiding narrow row widths and high plant populations are good management practices. When aerial blight is present in highly susceptible cultivars and environmental conditions are favorable for disease, preventive fungicide (strobulirin) applications are the most effective treatments in disease management. Several BCAs including *Trichoderma* species have been reported to be potentially useful in disease management. Nonpathogenic binucleate *Rhizoctonia* spp. (BNR) on the biocontrol of diseases caused by *R. solani* on many crops has been reported in the literature. BNR can induce resistance on soybean against the foliar blight caused by *R. solani* anastomosis group (AG) 1 IA (Basseto et al. 2008).

Powdery Mildew

Powdery mildew caused by the fungus *Microsphaera diffusa* Cooke & Peck (syns. *Erysiphe polygoni DC* and *E. glycines F. L. Tai*) is a minor but common disease of soybeans in many soybean-growing regions of the world particularly in Brazil (Knebel et al. 2006, Araujo et al. 2009) and the United

States (Grau 2006a) and under greenhouse conditions. In its early stages, powdery mildew may be recognized by the presence of small colonies of thin, light-gray or white fungus spreading rapidly on the upper surface of the leaf. Reddening of the underlying leaf tissue sometimes is evident. In time, the whitened areas of fungus enlarge but seldom coalesce to cover all the leaf surface. Many white, powdery patches form on both leaf surfaces and on the stems and pods about midseason. These areas may enlarge to cover much of the aboveground plant parts. Photosynthesis and transpiration are drastically reduced. On very susceptible cultivars, severely affected leaves may turn yellow, wither, and drop prematurely. Heavily infected pods usually contain shriveled, deformed, undeveloped, and flattened seeds. However, the soybean seeds do not become infected. Soybean planted late for a region will lose more yield to powdery mildew than early-planted soybeans. Yield losses ranging up to 14% have been attributed to the disease during certain years when cooler than normal temperatures prevail from flowering to maturity.

Infection occurs when microscopic asexual spores (conidia) land, germinate, and penetrate the epidermal cells. The conidia form several germ tubes, with the first attaching itself to the cells via an anchorage structure (appressorium). A thin filament (infection peg) forms under the appressorium and penetrates the host epidermis. This gives rise to the first feeding structure (haustorium), the only fungus structure found inside the host cells. The rest of the fungus body, or mycelium, grows superficially over the epidermal cells. Conidiophores (asexual fruiting structures) soon develop, giving rise to chains of conidia. Wind-borne conidia start new infections and repeat the disease cycle continuously until soybean plants tissues are available. Cool weather (18°C–24°C) favors disease development, while temperatures above 30°C arrest the growth and reproduction of the fungus. During rainy periods, conidia are washed away, temporarily delaying the secondary spread of the fungus. Speck-sized, black fruiting bodies (cleistothecia) sometimes are produced in mildew colonies late in the fall. Inside the cleistothecia, yellow ascospores (sexual spores) are produced in saclike structures called asci. It is believed that ascospores are released in the spring and serve as primary inoculum.

The only economical management method is to plant resistant soybean varieties. Certain varieties are susceptible in the seedling stage and express resistance about flowering time while others are resistant throughout their lifetime. A single dominant gene has been identified in soybean genotype PI 243540 that provides season-long resistance to powdery mildew, and the powdery mildew dominant resistance gene in PI 243540 has been mapped with PCR-based molecular markers. The map position of the gene is slightly different from previously reported map positions of the only known Rmd locus, which is tentatively called Rmd_PI243540, near the previously known Rmd locus on chromosome 16. The molecular markers flanking the gene will be useful for MAS of this gene (Kang and Mian 2010).

The yield increase due to fungicidal treatments such as thiophanate methyl is usually higher in soybean cultivars that are susceptible to powdery mildew (Hoffmann et al. 2004). Sewage sludge increments elicitation of phytoalexins in soybean and the severity of powdery mildew is reported to be reduced with an increase in the concentration of sludge in the soil and substrate (Araujo and Bettiol 2009).

Phyllosticta Leaf Spot

Phyllosticta leaf spot or leaf blight, caused by *Phyllosticta sojiecola* Massai (syn. *P. glycines*) and teleomorph *Pleosphaerulina sojicola* Miura, is a minor disease of soybean crop, rarely spreading beyond the first few trifoliate leaves. This fungal disease occurs throughout the soybean-growing regions in the United States (Yang 2002). The irregular marginal leaf scorch symptom exhibited on the lower leaves has been identified as *Phyllosticta* leaf spot. The infection starts at the leaf margin and progresses inward, forming an irregular, V-shaped area. Random leaf spots may also accompany the marginal necrosis. Numerous small, black specks (fungus fruiting bodies, or pycnidia) form in older lesions. The fungus may grow from the leaf blades into the petioles and then to

the stipules and stem tissues at the leaf scar. Superficial, light-gray, tan, or brownish lesions with a narrow, brown, or purplish border may form on the petioles, stems, and pods. With cool and moist conditions, pods and seeds can be infected, causing seed discoloration. The fungus produces numerous small spores, which can spread to healthy leaves and plants, thereby causing new infection. The fungus can survive on seeds and can be spread with infected seeds. If it is prevalent in seed fields, a seed health test may be conducted before saving the beans for seed. If disease is severe in a production field, consider the use of rotation and tillage to reduce infested residues for the next soybean crop.

Brown Stem Rot (*Phialophora gregata* f. sp. *sojae*)

BSR of soybean caused by *P. gregata* f. sp. *sojae* Kobayashi et al. occurs in many countries including Argentina, Brazil, Canada, Egypt, Japan, Mexico, the United States, and the former Yugoslavia (Gray and Grau 1999, Grau 2006b). Yield losses of 10%–30% are common for susceptible soybean varieties grown in management systems conducive for BSR development. There is no external evidence of the disease in the early reproductive stage and signs of early infection generally go unnoticed unless the stems are cut open and examined or only the mild strain of the pathogen is present. The onset of foliar symptoms typically occurs at growth stages R4 and R5 and foliar symptoms peak at R7. The pathogen causes stem and foliar symptoms that may not always occur together, depending upon pathotype, host genotype, and environmental conditions (Hughes et al. 2002, Malvick et al. 2003). Pathogen pathotype I (genotype A) causes browning of stems as well as foliar symptoms such as interveinal chlorosis, defoliation, and wilting. Symptomatic leaves have a shriveled appearance but remain attached to the stem. Pathotype II (genotype B) causes only browning of stems. Secondary symptoms of BSR are stunting, premature death, decrease in seed number, reduced pod set, and decrease in seed size.

Stem symptoms include brown discoloration of the pith and vascular tissue; foliar symptoms include interveinal necrosis and defoliation (Gray and Grau 1999). While all soybean genotypes may be susceptible to infection by the pathogen, soybean genotypes differ in expressing foliar symptoms. A lack of foliar symptoms is considered as resistance and has been a selection criterion in breeding programs for BSR resistance (Sebastian et al. 1986). Pathogen reproduces asexually by means of conidia inside host plants. Its sexual state has never been found. It is slow growing in culture and is thought to be a poor saprophytic competitor (Adee and Grau 1997). *P. gregata* f. sp. *sojae*, a soilborne vascular pathogen causing BSR of soybean, has been divided into two genotypes, designated as A (pathotype I) and B (pathotype II). These genotypes are differentiated by an insertion or deletion in the intergenic spacer (IGS) region of ribosomal DNA. The two genotypes differ in the type and severity of symptoms they cause and have displayed preferential host colonization. Pathotype I and pathotype II are based on variation in the IGS region of nuclear rDNA marker (Gray 1971, Hughes et al. 2009). The rDNA marker identifies genetically distinct populations. Pathotype I is the defoliating pathotype comprising population A, identifiable by the genotype A rDNA marker (Chen et al. 2000, Hughes et al. 2002), and preferentially infecting susceptible soybean cultivars (Chen et al. 2000, Malvick et al. 2003). Pathotype II is the nondefoliating pathotype comprising population B, identifiable by the genotype B rDNA marker (Chen et al. 2000, Hughes et al. 2002), and preferentially infecting certain resistant soybean cultivars (Chen et al. 2000, Malvick et al. 2003). Field isolation data showed that most isolates obtained from susceptible cultivars belong to population A, whereas most isolates obtained from resistant cultivars belong to population B (Chen et al. 2000, Hughes et al. 2002, Malvick et al. 2003, Malvick and Impullitti 2007). The two populations could be sympatric, residing not only in the same field but also in the same plants under field conditions. The first controlled experimental demonstration that a differential host preference of *P. gregata* f. sp. *sojae* exists toward different cultivars of the same host species has been given by Meng et al. (2005).

The *P. gregata* fungus produces no survival structures, but can overwinter as mycelium in decaying soybean residue previously colonized during the pathogen's parasitic phase. During overwintering,

conidia are produced; these conidia are the inoculum for new plants in the spring. Infection occurs through roots by growth stage V3 and progressively colonizes stems. Stem and foliar symptoms are most severe when air temperatures range between 15°C and 26°C during growth stages R4 to R6. Air temperatures in the 32°C range will suppress foliar symptom development. BSR is most severe when optimal soil moisture is present at R1 to R2 followed by dry soil conditions at R5 to R6. The severity of BSR is greater if soils are low in phosphorus and potassium and soil pH is below 6.5. *P. gregata* and *H. glycines*, the SCN, frequently occur together and there is evidence that the severity of BSR is greater in the presence of SCN.

The disease can be managed by the use of disease-resistant cultivars, and commercial soybean varieties have been improved dramatically for resistance to BSR. Most soybean cultivars with SCN resistance tracing to PI 88788 have various degrees of resistance to BSR. However, caution is advised for varieties with SCN resistance derived from Peking or Hartwig, the source of SCN resistance technology, as these two varieties are susceptible to BSR. Successful control of BSR has been obtained through crop rotation, especially if 2–3 years of nonhost crops are spaced between soybean. Soybean is the only known host grown; extended periods of cropping to nonhosts such as corn or small grains or forage legumes effectively lower the inoculum of *P. gregata*. The rate of inoculum decline is directly related to the rate of soybean residue decomposition. Early-maturing soybean cultivars escape the yield-reducing effect of BSR.

Bacterial Blight

Bacterial blight of soybean caused by *Pseudomonas syringae* pv. *glycinea* (Coerper) Youn et al. is the most common bacterial disease of soybeans and it occurs in all soybean-producing regions of the world. Although this disease is of limited importance, it is one of the first leaf spot diseases to appear on young plants (Jagtap et al. 2012b). Bacterial blight has been reported to cause significant yield reductions on susceptible cultivars under heavy disease pressure. In Europe, the disease has not caused great loss, but if the area of soybean production were to increase, losses might be expected on the scale seen in the United States, where in the period 1975–1977, it was by far the most damaging prokaryotic disease, causing an estimated annual average loss of $62 million (Kennedy and Alcorn 1980). In recent years, the disease has become economically important in Serbia in Europe (Ignjatov et al. 2007, 2008).

Bacterial blight is primarily a leaf disease, but symptoms can occur on stems, petioles, and pods. Leaf lesions are at first small, annular, water-soaked, tan-colored spots, which enlarge to 1–2 mm diameter and become dark brown to black with a dark center and a water-soaked margin and surrounded with a narrow yellowish halo. The halo is more noticeable on the upper leaf surface. Bacterial blight is easily confused with Septoria brown spot, a fungal disease that develops first on the lower leaves, whereas the bacterial blight leaf lesions develop first on upper young leaves. A simple test for bacterial blight is to hold infected leaves to the light; bacterial blight spots will be translucent. Lesions can coalesce to produce large irregular areas of dead tissues. The center of all lesions may drop resulting in shot-holed leaves, and the leaf may show ragged and torn appearance. Large black lesions may develop on stem. If the growing point of seedlings is affected, the plant usually dies.

The primary foci in crops derive from seed-borne infection that inhibit germination and on cotyledons cause marginal lesions that enlarge and become dark-brown necrotic. Often, the lesions are covered, particularly on their underside, with a film of a pale grayish bacterial slime that can dry to a thin silvery crust; less commonly, primary foci derive from overwintered infected crop debris. Secondary spread to infect young soybean leaves occurs by means of wind-driven rain and during cultivation or spraying when the foliage is wet at temperatures of 24°C–26°C. Hot, dry weather suppresses its development. There is evidence that the pathogen may be resident epiphyte in buds; this could provide a continued source of inocula. Infection occurs through natural openings on foliage and through wounds that occur commonly on sandy soils by abrasion with

sand particles. The bacteria may colonize leaf surfaces without producing symptoms until conditions become favorable. Rain splash and wind-driven aerosols can drive bacteria into natural leaf openings (stomata) causing rapid increases in disease. Psg PG4180 causing bacterial blight of soybean produces the phytotoxin coronatine (COR) in a temperature-dependent manner. COR consists of a polyketide, coronafacic acid (CFA), and an amino acid derivative, coronamic acid, and is produced optimally at 18°C, whereas no detectable synthesis occurs at 28°C. After spray inoculation, PG4180 causes typical bacterial blight symptoms on soybean plants when the bacteria are grown at 18°C prior to inoculation but not when derived from cultures grown at 28°C (Budde and Ullrich 2000).

Host plant resistance genes to bacterial blight in soybean germplasm and a number of physiologic races of *P. syringae* pv. *glycinea* (Psg) have been reported (Fett and Sequeira 1981, Zabala et al. 2006). The interaction of compatible and incompatible races of Psg with different soybean cultivars has been characterized (Cross et al. 1966, Ignjatov et al. 2007). Incompatible interactions lead to a cascade of plant responses (hypersensitive response [HR]) triggered by the action of a resistance gene R and the corresponding avirulent pathogen avr gene (Zabala et al. 2006). The Rpg4 gene may be involved in resistance to Psg in soybean; however, it has another useful function that somehow contributes to soybean productivity in modern agroecosystems, and thus, plant breeders have unintentionally increased its frequency in cultivated germplasm (Farhatullah Stayton et al. 2010). The Rpg4 locus is controlling bacterial blight resistance to *P. syringae* pv. *glycinea* race 4 (Farhatullah Groose et al. 2010). In the incompatible interaction, Psg PG4180 elicits the HR regardless of the bacterial preinoculation temperature (Budde and Ullrich 2000). The complex resistance responses in such incompatible plant–pathogen interactions have been characterized at the molecular level to a larger extent in the model plant *Arabidopsis thaliana* (Quirino and Bent 2003). Durable resistance is difficult to achieve because of many races of Psg.

There is no effective management for bacterial blight when an aggressive race, a susceptible cultivar, and weather favor disease development. However, resistance in susceptible soybean varieties can be induced by salicylic acid (SA), chitosan, beta-aminobutyric acid (BABA), and OA to bacterial blight disease. The concentration of 1000 μg BABA/mL induces the highest resistance among all the inducers. Soybean varieties and application methods all can vary the effect of induced resistance (Liu et al. 2008). Crop rotation to nonhosts such as corn, wheat, and other nonlegume species and tillage will help reduce inoculum. Comparatively narrow rotation should be sufficient to eliminate trash-borne infection from fields since the pathogen does not apparently survive in it for two seasons (Parashar and Leben 1972). Tillage where possible can also help reduce the survival of *P. syringae*–infested debris through burial and rapid breakdown of soybean debris. The epiphyte *P. syringae* pv. *syringae* 22d/93 (Pss22d), isolated from soybean leaves, had been characterized as a promising and species-specific biocontrol strain in vitro and in plant against Psg (Wensing et al. 2010). Seeds produced in heavily infected fields are likely to carry the pathogenic bacteria; hence, it is recommended that seeds should never be saved for sowing from the plants from affected fields. Copper fungicides are labeled for bacterial blight control on soybean, but application needs to be conducted early in the disease cycle to be effective.

Bacterial Pustule Disease

Bacterial pustule disease (BPD) is caused by *Xanthomonas axonopodis* pv. *glycines* (Nakano) Dye (syns. *Xanthomonas campestris* pv. *phaseoli* Smith, Dye) and *Xanthomonas phaseoli* (Smith) Dowson var. *sojensis* (Hedges) Starr and Burkholder. Although this bacterium is widely known as *X. campestris* pv. *glycines*, following DNA–DNA hybridization analysis (Vauterin et al. 1995), the bacterium has been renamed as *X. axonopodis* pv. *glycines* (Xag). It is an economically significant disease in most areas of the world in which the soybean crop is grown. It is widespread in many European countries causing seed losses of up to 28%. It has also become important in India (Khare et al. 2003), Korea (Van et al. 2007), Serbia (Balaz and Acimovic 2008), southern United States, and Thailand (Kaewnum et al. 2005). Yield losses of up to 40% have been reported in certain parts

of the world (Prathuangwong and Amnuaykit 1987). However, generally, it is far less economically important than bacterial blight caused by *P. syringae* pv. *glycinea*. The disease occurs typically on soybean foliage and symptoms include small, pale green spots with elevated pustules, which may develop into large necrotic lesions. Pustules are mainly formed by hypertrophy, but hyperplasia may also occur. The appearance of bacterial pustule varies from minute specks to large, irregular, mottled brown areas that arise when smaller lesion coalesces. The spots may enlarge and coalesce, leading to premature defoliation (Narvel et al. 2001). Symptoms on resistant soybean cultivars become visible in the form of small chlorotic spots, but not well-defined pustules or light green chlorosis. Those symptoms are sometimes confused with those of soybean rust. However, pustule lesions are characterized visually by small pustules surrounded by yellowing halos, whereas rust disease forms tan or brown lesion in which uredospores are formed and released through a central pore. The Xag is a motile, gram-negative rod (0.5–0.9 × 1.4–2.3 μm) with a single polar flagellum. Colonies on beef infusion agar are pale yellow, become deep yellow with age, and are small. It is seed borne and can also overwinter in infected host debris on the surface of soil (but less well in buried host debris) or in volunteer plants from infected seed. The pathogen can be transmitted from seed to the seedling. It enters through stomatal openings and proceeds intercellularly. Bulging of epidermal cells initially occurs. Infected cells become yellowish brown and later become deformed and disintegrated. The development of disease requires an optimum temperature of 27°C (+ or −2) with a minimum relative humidity of 83%. Pathogenic variations with regard to aggressiveness on soybean among isolates of Xag have been demonstrated (Ansari 2005, Kaewnum et al. 2005). The isolates differ in their ability to induce an HR on resistant varieties. Xag grows in the xylem vessels of the soybean plants and in the intercellular spaces of the leaves, causing pustule lesions. A major mechanism of this bacterium is the production of an extracellular polysaccharide or xanthan gum that is toxic to the plant tissues and cause them to be necrosis spots. This pathogen is not known to produce any extracellular toxins, degradative enzymes, or plant growth hormones.

The disease can be managed through the use of resistant cultivars. RT-PCR data suggest six candidate genes that might be involved in a necrotic response to Xag in resistant genotype PI 96188 (Van et al. 2007). A nonpathogenic mutant *the M715 mutant* (derived from the pathogenic wild-type strain YR32) shows promise as an effective BCA for BPD in soybeans (Rukayadi et al. 2000). Similarly, *B. subtilis* isolate 210 should be considered as a potential antagonistic agent for BPD of soybean (Salerno and Sagardoy 2003). *B. amyloliquefaciens* strain KPS46 selected as inducer of systemic resistance against Xag can be of potential use. Soybean seeds are treated with KPS46, SA, and harpin (synthetic resistance inducer) prior to sowing and challenged with Xag, the activity of PAL increases, and phenolics are found to accumulate in soybean leaf tissues giving protection to soybean plants against BPD (Buensanteai et al. 2007).

Soybean Mosaic Disease

Soybean mosaic disease (Figure 9.12) is caused by soybean mosaic virus (SMV) and it occurs worldwide causing mild to severe mosaic on susceptible cultivars. SMV infection adversely affects seed quality, oil content, and seed germination. Yield losses due to this disease generally range from 8% to 35%; however, losses as high as 94% have been reported. Infection in the early growth stages has the greatest risk of yield loss and reduced seed quality, compared to infection later in the season. Dual infection with other viruses, that is, mixed infection of SMV with Alfalfa mosaic virus, a common situation, increases the risk of yield loss and reduces seed quality (Malapi-Nelson et al. 2009). Tobacco streak virus and bean pod mottle virus (BPMV) have also been found in multiple infections (Fajolu et al. 2010). SMV and BPMV act synergistically. This means that mixed infections are more severe than single infections. Symptoms on plants with both viruses are very severe and terminal death may occur. Yield losses associated with combined infection can be as high as 66%–86%. The combined infection also increases the level of seed transmission of SMV. Of course, this combination effect is also related to the age of the plant when infection occurs.

FIGURE 9.12 Soybean mosaic symptoms on leaf. (Courtesy of Dr. Shrishail Navi, Iowa State University, Ames, IA.)

Common leaf symptoms of the disease are a mosaic of light and dark green areas that may later become raised or blistered, particularly along the main veins. The youngest and most rapidly growing leaves show the most symptoms, especially at cooler temperatures. Affected plants are stunted with rugose or crinkled leaves that become severely mottled and deformed. The trifoliate leaf blades become slightly narrowed and are puckered along the veins and curled downward. The mottling appears as light and dark green patches on individual leaves. Early-infected plants particularly are stunted with shortened petioles and internodes. Symptoms are more severe when temperatures have been cool (<24°C) and may be masked by warmer (>32°C) conditions. Affected plants produce fewer pods and seeds from infected plants can be mottled black or brown depending on hilum color. Not all infected plants produce mottled seed and seed mottling does not indicate that the virus is present in the seed. Seeds may show reduction in their size in comparison to seeds from healthy plants. Primary leaves of some cultivars may show necrotic local lesions, which merge into veinal necrosis followed by yellowing and leaf abscission.

The virus belongs to the genus *Potyvirus*, group IV (+)ss RNA, under the family Potyviridae. SMV is transmitted by infected seed, and soybean aphid species (*Aphis glycines*) and at least 32 aphid species, belonging to 15 different genera, transmit the SMV in a nonpersistent manner worldwide (Wang et al. 2006). In most varieties, seed transmission is less than 5%, but much greater than seed transmission for BPMV. Spread of the disease can also be done by soybean aphids, which can vector this virus. The timing and incidence of SMV infection depend largely upon the level of primary inoculum and aphid activity. SMV may be introduced into a virus-free region by planting infected seed. The pathogen is spread from plant to plant by aphids. The soybean aphid, *A. glycines*, the most common SMV vector, is the only aphid species that can establish colonies on soybeans. Once an aphid feeds on an infected soybean plant, it only takes a short time (seconds to a few minutes) for the insect to acquire the virus. As the virus-carrying aphids move and feed on healthy plants, the virus will be spread around. In the absence of soybeans, the virus can overwinter on a wide range of other hosts.

SMV is a flexuous rod consisting of positive-sense, single-stranded RNA. Numerous strains of the virus have been identified based upon reactions on a set of differential cultivars. In the United States, SMV has been classified into nine strains using differential reactions on eight soybean cultivars. The strains are currently known at G1 through G7, G7a, and C14. It is probable that additional strains exist, particularly in People's Republic of China and Japan (Zheng et al. 2008). SMV is sap and graft transmissible also. At least 32 aphid species, belonging to 15 different genera, transmit the

SMV in a nonpersistent manner. Virus isolates may show some vector specificity. Infected plants resulting from transmission through seed play an important role in SMV epidemiology. Such plants are primary inoculum sources for SMV. In most cultivars, seed transmission is less than 5%, but no transmission occurs in some cultivars while others can have levels as high as 75%. Infection of soybean plants with SMV has been reported to enhance *Phomopsis* spp. infection, which reduces seed quality. The use of SMV-resistant varieties prevent/reduce SMV and *Phomopsis* spp. seed infection (Koning et al. 2002).

Planting SMV-resistant soybean cultivars is the most economical practice to manage the disease. Several resistance genes have been identified and are effective against some, but not all, virus strains. Based on the differential reactions on a set of soybean cultivars, SMV has been classified into numerous strains. In the United States, nine strains, G1G7, G7a, and C14, are currently recognized. Additional strains have been identified in other countries such as Canada, China, Japan, and South Korea including strains that overcome all known resistance to the virus (Zheng et al. 2005, 2008). At least three independent loci (Rsv1, Rsv3, Rsv4) have been identified for SMV resistance. Multiple resistance alleles have been reported for the Rsv1 and Rsv3 loci (Liao et al. 2002, Zhen et al. 2008). The first dominant resistance gene identified in the soybean line PI 96983 has been designated as Rsv1. Single resistance genes in other cultivars, which confer differential reactions to strains G1 to G7, are found to be alleles at the Rsv1 locus and have been designated as Rsv1y, Rsv1m, Rsv1t, Rsv1k, Rsv1s, and Rsv1n. A new mutation in SMV resulting in overcoming Rsv4 resistance has been reported from Iran (Ahangaran et al. 2013). Some of the most promising soybean genotypes that are resistant to most strains of SMV in the Arkansas state in the United States are Ozark, USG 5002T and USG 5601T. Similarly, SMV-resistant soybean genotypes from India are JS71-05, KHSb2, LSb1, MACS58, MACS124, Punjab1, and VLS2 (Sharma et al. 2014). Soybean genotypes identified with high levels of resistance to SMV from Nigeria with disease incidence of 10% or less are TGx 1440-1E, TGx 1448-E, TGx 1479-1E, TGx 1446-3E, TGx 1371-1E, and TGx 1445-4E, TGx 1440-1E, and TGx.

At present, the use of SMV-free seed 1448-2E (Banwo and Adamu 2000) and avoiding late planting of soybean are the best control measures to preclude loss induced by SMV. Serological seed indexing techniques and/or grow-out tests can be used for virus detection in seed lots. Roguing, in addition to being generally impractical in the field, may not be very effective because of the tendency for symptoms in soybean to be masked above 30°C. Insecticides are not considered effective in reducing transmission of SMV by aphids. Aphids present at spraying are killed, but the field is quickly recolonized by winged aphids and virus transmission can resume. Aphids that contact insecticide residues on the leaf surface are killed but are still capable of virus transmission prior to death.

Bean Pod Mottle Disease (Bean Pod Mottle Virus)

BPMV (genus *Comovirus*, family *Comoviridae*) was first identified in soybean in 1951 in Arkansas (Walters 1958, Ross and Butler 1985) and has caused agronomic problems since this first finding in all production areas in the United States. Worldwide, it is also reported to occur in Brazil (Anjos et al. 1999), Canada (Michelutti et al. 2002), Ecuador (Zettler et al. 1991), Iran (Shahraeen et al. 2005), Nigeria (Ugwuoke 2002), and Peru (Fribourg and Perez 1994). Maximum losses occur when plants are infected at the seedling stage. Yield losses from BPMV alone may reach 2%–20% depending on planting date and geographical area. Yield loss assessments on southern soybean germplasm have revealed reductions ranging from 3% to 52% (Ziems et al. 2007). BPMV produces further financial loss for soybean producers because it causes increased seed coat mottling, which is an irregular pattern or streaking of the hilum associated with BPMV infection. The effects on yield and seed coat mottling are increased when there is a dual infection of BPMV and *SMV*, which causes yield losses >80%. Symptoms on infected soybeans may vary depending on the variety. Foliage symptoms range from mild chlorotic mottling, leaf rugosity in the upper canopy to puckering and severe mosaic in lower leaves, terminal necrosis, and death. A common symptom of BPMV

infection is uneven crop maturity or *green stem* in which stems and leaves remain green, even though pods have matured. Young leaves in the upper canopy often have a green-to-yellow mottling that may fade and then redevelop later in the growing season. The green stems are difficult to cut during harvesting. However, it has been shown recently that green stem is independent of BPMV infection when random plants were tested for BPMV at growth stage R6 (Hobbs et al. 2006). In severe cases, malformed leaves and pods may be produced. Infected leaves show reduced turgidity resulting in curling. A reduction in pod set often occurs in infected plants that have undergone moisture stress during dry periods. Infected seed coats, similar to SMV infection, are mottled with brown or black streaks extending from the hilum.

BPMV has a bipartite positive-strand RNA genome consisting of RNA1 (approximately 6.0 kb) and RNA2 (approximately 3.6 kb) that are separately encapsidated in isometric particles 28 nm in diameter (Lomonossoff and Ghabrial 2001). BPMV RNA-1 codes for five mature proteins required for replication, whereas RNA-2 codes for a putative cell-to-cell MP and the two coat proteins (L-CP and S-CP). Separation of segments can be achieved by density gradient centrifugation into three components: top (T), middle (M), and bottom (B). The middle component contains a single RNA1 molecule, whereas the bottom component has RNA2 and the top particle lacks nucleic acid. BPMV is heat stable with a temperature inactivation point of 70°C. Its dilution end point in fresh plant extract is 10,000 and its longevity in vitro is 62 days at 18°C. Unlike SMV, BPMV does not spread very efficiently in seed. The virus is primarily transmitted by the bean leaf beetle (*Cerotoma trifurcata*). The virus has a wide host range among legumes and will be transferred to bean leaf beetles that feed on infected legume plants. The virus can be spread by mechanical injury, especially under wet conditions. The virus has been found in overwintered bean leaf beetle adults that may survive in grass, leaf litter, or even rocks and colonize soybeans as seedlings emerge. Because most flight events of beetles are limited to about 30 m, it is likely that BPMV spread is restricted within and between fields.

The use of soybean cultivars resistant or tolerant to BPMV infection would be the most practical approach. However, currently, no soybean lines have been identified with resistance to BPMV. Soybean cultivars with feeding deterrents against bean leaf beetle may not be sufficient to reduce BPMV incidence in the field. Transgenic soybean lines expressing the BPMV coat protein are resistant to BPMV infection. But this resistance has not been incorporated into commercial soybean cultivars (Reddy et al. 2001). Current management recommendations for reducing BPMV infection include the application of insecticides to manage bean leaf beetle populations to reduce the potential for virus movement. The recommended management of BPMV has been solely based on vector population dynamics, and not on BPMV disease. Delayed soybean planting date has been suggested to manage BPMV. Delayed planting is supposed to help soybean escape the migration period of beetle vectors of the virus.

Root-Knot Disease (*Meloidogyne* species)

Several genera of nematodes parasitize soybean worldwide, and the highest economic impact is attributed to SCNs—*H. glycines* (as described earlier in pages 397–405), root-knot nematodes (*Meloidogyne* species), lesion nematode (*Pratylenchus* species), and reniform nematode (*Rotylenchulus* species).

Root knot nematodes (RKNs) are biotrophic parasites of the genus *Meloidogyne*. The four most common species are *Meloidogyne incognita* (*Kofoid and white*) *Chitwood*, *Meloidogyne hapla Chitwood*, *Meloidogyne javanica* (*Treub*) *Chitwood*, and *Meloidogyne arenaria Chitwood*, but only *M. incognita* and *M. hapla* have been found to be important on soybeans worldwide. *M. hapla*, the northern RKN, is generally considered less damaging on soybean than the southern RKN, the *M. incognita* in the United States (Westphal and Xing 2006). Susceptible soybean plants can be infected at any stage of development. Infected plants are stunted and chlorotic. The interaction effect of the nematode (*M. javanica*) with other soilborne pathogens such as *R. solani* and *M. phaseolina* causes significant decrease in soybean plant height and dried plant weight, especially when

nematodes are inoculated 1 week before both fungi and rate of severely damaged plants can reach to about 47.62% and 64.62%, respectively (Stephan et al. 2006). The aboveground symptoms of root-knot disease can be easily confused with other soil-related plant growth–suppressing factors. To confirm RKN infection, it is necessary to excavate root systems and examine them for root galling. Nematode-induced galls consist of globular, irregular deformations within the root system. These swellings are easily distinguished from nodules that are a normal part of soybean root systems. These normal nodules result from infection by beneficial, symbiotic bacteria that fix atmospheric nitrogen for the plant. Beneficial nodules are nearly spherical structures about 1/4 in. in diameter that are attached to the outside of roots. RKN galls, on the other hand, range from 1/8 to 1 in. in diameter and are swellings of the root itself. RKNs are obligate parasites, but they can survive as eggs in the soil for several years. These eggs contain the nematodes in their infective stage: second-stage juveniles. When soil conditions are favorable (when soil temperatures are more than 50°F) and a susceptible host plant is grown, juveniles hatch from the eggs and move through soil in search of host plant roots. When a juvenile finds a suitable root location, generally near the growing tip, it penetrates the root and becomes sedentary. After several molts, a juvenile develops into a mature female, which in turn produces an egg mass containing several hundred new nematode eggs in a gelatinous matrix deposited on the outside of the root. At this point, juveniles either immediately hatch from their eggs or remain dormant within the egg until infection conditions are favorable. The gelatinous matrix is thought to protect the eggs from soil organisms that might otherwise consume the eggs and suppress the nematode's initial inoculum level.

Concerning RKN management, soybean faces the same economic losses and difficulties as other crops. Despite the use of management strategies such as crop rotation with nonhosts and sanitation practices aimed at reducing initial inoculum, sustainable and long-lasting pest management strategies are in high demand. One of the strategies is to deploy novel sources of RKN resistance in soybean breeding programs, for example, using the soybean line PI 595099 (Accession NPGS/GRIN: G93-9223), which is resistant against specific strains and races of nematode species, including *M. javanica*, *M. incognita*, *M. arenaria*, and also the SCN. Another alternative is to introduce genetic modifications in soybean plants to obtain RKN resistance interactions (Bird et al. 2009, Beneventi et al. 2013). Increasing amount of auxin-induced reactive oxygen species (ROS) accumulation in cells in the nematode-inoculated soybean genotype (PI 595099) has an immediate effect on halting pathogenesis. The host coordinate and modulate defenses mostly by the interplay between auxin (Aux), gibberellin (GA), and jasmonate (JA) (Beneventi et al. 2013).

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