Diseases of Sunflower

(Carl Bradley, Sam Markell and Tom Gulya)

Sunflower *(Helianthus annuus)* is unique in that it is one of the few crop plants that are native to North America. The genus *Helianthus* comprises more than 60 annual and perennial species, with one to several species found in every state. Since wild sunflower is a native plant, a native population of diseases and insects that can attack cultivated sunflower also is present in most areas of the U.S. Sunflower is also unusual in that it is both a field crop, with two distinct subtypes (oil and confection), and is grown as a garden flower and for the cut-flower industry. At least 30 diseases, caused by various fungi, bacteria and viruses, have been identified on wild or cultivated sunflower, but fortunately, only a few are of economic significance as far as causing yield losses. When considering sunflowers as an ornamental plant, even small spots on the foliage are enough to reduce marketability, and thus proper disease identification is necessary to decide upon appropriate disease management. See Appendix 1 for a listing of all known sunflower diseases that occur in the world.

The most important diseases in the northern Great Plains are Sclerotinia wilt, Sclerotinia head rot and downy mildew. Rust, especially on confection sunflowers, and Phomopsis stem canker are important in some years, but are of less overall concern. Phoma black stem is almost universally prevalent, but is not thought to cause yield losses. Leaf diseases caused by *Alternaria*, *Septoria* and powdery mildew, head rots caused by *Botrytis* and *Erwinia*, and Verticillium leaf mottle are diseases that either are observed infrequently in the northern Great Plains or have not occurred with sufficient intensity to be considered serious.

The most important sunflower disease in both the central Great Plains (Kansas, Nebraska, Colorado, Texas) and California is Rhizopus head rot. Sclerotinia head rot and wilt, downy mildew and Phoma black stem are of sporadic importance. In the central Great Plains, rust can be severe on sunflower grown under center pivot irrigation, and Phomopsis stem canker has been of concern in years of plentiful rainfall. Under drought conditions, charcoal rot is also of concern in the central Great Plains. In California, rust, followed by various stalk rots, are the most prevalent diseases after Rhizopus head rot. For complete details on the incidence and severity of sunflower diseases, visit the National Sunflower Association Web site (www.sunflowernsa.com), where the proceedings of the annual "Sunflower Research Workshop" are posted.

The production of ornamental sunflowers in the U.S. is scattered across at least 41 states, and includes both field and greenhouse production. Greenhouse-grown sunflower is prone to *Pythium* and *Phytophthora* root rot and *Botrytis* blight, as well as the range of sunflower-specific pathogens. Field-grown ornamental sunflower, especially in the Midwest, is prone to the same pathogens that attack oilseed sunflower. Sclerotinia wilt, rust, powdery mildew, Rhizopus head rot, southern blight and root knot nematodes are cited as the major diseases. Since cosmetic appearance is so important on ornamental crops, even diseases considered as minor on an oilseed sunflower crop are of consequence to ornamental sunflower. Thus, obscure diseases, such as leaf smut, cocklebur rust and petal blight, have been cited as causing losses to ornamental sunflower in the U.S. and abroad.

Effective control measures for most sunflower diseases are:

- Planting resistant hybrids
- A minimum rotation of four years between successive sunflower crops
- Seed treatment for control of downy mildew and damping-off
- Tillage to bury crop residue that may harbor pathogens
- Foliar fungicides for rust and other foliar diseases

Many sunflower diseases are controlled by single dominant genes for resistance (e.g., downy mildew, Verticillium leaf mottle). Most sunflower hybrids in the U.S. have resistance to Verticillium leaf mottle, several races of downy mildew and several races of rust. Unfortunately, the rust and downy mildew pathogens continue to evolve and new races are found periodically. This requires commercial seed companies to add new genes to their hybrids so their hybrids can remain resistant to the ever-changing pathogens. Some other disease organisms, such as *Sclerotinia*, require multiple genes for resistance, which makes development of resistant hybrids much more difficult. Great strides have been made in making sunflower hybrids more tolerant of both Sclerotinia wilt (root rot) and Sclerotinia head rot. No current hybrids can be considered immune to either type of *Sclerotinia* infection, but then again, breeders have so far been unable to develop immune varieties for other Sclerotinia-susceptible crops. Disease reaction varies widely among hybrids, and growers are encouraged to consult seed companies and Extension or public research personnel for recommended disease-resistant varieties. Producers also should remember that disease response is highly influenced by environment. Disease evaluations made in only one location may not accurately predict a hybrid's performance in all areas.

Crop rotation helps reduce populations of many important sunflower pathogens in the soil. Most sunflower diseases are caused by pathogens specific to sunflower. *Sclerotinia*, however, attacks many crops, and susceptible crops (such as mustard, canola, crambe, soybean and dry edible bean) should be interspersed in rotation with corn and cereals, which are nonhosts of *Sclerotinia*. Rotation time away from sunflower is influenced by the occurrence and severity of diseases noted in the current year. Regular monitoring of fields and maintaining accurate records are also important in determining rotation practices. Rotation will have a minimal effect on foliar diseases since the pathogen spores may be carried by wind from distant fields. Currently, only two fungicides are employed for foliar disease control, with several others in the registration process. Consult state Extension publications for more details.

The arrangement of the sunflower diseases in the following section is based on time of appearance during the growing season and the plant part affected. Early season diseases (e.g., downy mildew and apical chlorosis) are covered first, followed by foliar diseases (including virus diseases), stalk and root infecting diseases (including nematodes) and finally, head rots and other diseases of mature plants.

For additional information and photos of sunflower diseases, visit the National Sunflower Association Web site (www.sunflowernsa.com) and the Web site of the USDA Sunflower Research Unit in Fargo, N.D. (www.ars.usda.gov/main/site_main.htm?modecode=54420520), as well as the heavily referenced chapter on sunflower diseases in "Sunflower Technology and Production" edited by A.A. Schneiter and published by the Agronomy Society of America as Monograph No. 35. For current information on the disease resistance of commercial hybrids, please consult NDSU publication A652, "Hybrid Sunflower Performance Testing," available in hard copy or on the Web at www.ext.nodak.edu/extpubs/plantsci/rowcrops/a652.pdf.

I. Early Season Diseases

Downy Mildew

Description: Downy mildew has been observed on cultivated and wild sunflower throughout the U.S. and was quite common before the advent of resistant hybrids and the use of fungicides as seed treatments. It is most serious in areas with flat topography or heavy, clay soils that foster waterlogged conditions conducive for disease development.

Typical systemic symptoms in seedlings include dwarfing and yellowing (chlorosis) of the leaves (Figure 70) and the appearance of white, cottony masses (fungal mycelium and spores) on the lower and sometimes upper leaf surface during periods of high humidity or dew (Figure 71). Most infected seedlings are killed, but those that survive will produce stunted plants with erect, horizontal heads with little, if any, seed (Figure 72). When seedlings are infected several weeks after emergence, or a fungicide seed treatment inhibits rather than prevents infection, the plants usually start showing symptoms at the four-, six- or eight-leaf stage. This situation is referred to as "delayed systemic infection." These plants are characterized by some degree of stunting, with typical downy mildew leaf symptoms starting at some level in the plant (with lower leaves appearing normal). If susceptible plants are exposed to the mildew pathogen after the seedling stage, they also may develop a thickened, clublike root and become stunted, but may not show foliar symptoms. All infected plants serve to perpetuate the pathogen in the soil and are more prone to drought stress and lodging.

Sunflower plants also display localized foliar lesions due to airborne downy mildew spores. The infected spots are generally small, angular lesions (delimited by veinlets) with white sporulation on the underside of the lesion. These local lesions may coalesce into larger lesions, but they rarely result in a systemic infection, and thus have minimal impact upon yield.

Dwarfing and distortion of leaves also are symptoms typical of herbicide drift damage, especially from 2,4-D and related phenoxy compounds, and may be confused with downy mildew symptoms (Figure 73). Herbicide damage, however, never will exhibit the white appearance (fungal mycelium and spores) on the underside of the leaves nor the chlorosis typical of downy mildew.

Figure 70. Dwarfing and discoloration of sunflower resulting from infection by downy mildew. (D.E. Zimmer)

Figure 71. Lower surface leaves on downy mildew infected plants frequently exhibit a white cottony growth of fungus. (D.E. Zimmer)

Figure 72. Plants infected with downy mildew seldom produce heads; when they do, the heads do not nod but remain erect and produce little or no seed. (T. Gulya)

Disease Cycle: Downy mildew is caused by the obligate fungus *Plasmopara halstedii,* which is soil-borne, wind-borne and seed-borne. Sunflower plants are susceptible to systemic infection before the seedling roots exceed 2 inches in length. This short period may range up to a maximum of two to three weeks, depending on soil temperature and moisture. Cool, water-saturated soil during this period greatly favors infection. The fungus may persist in the soil for five to 10 years as long-lived oospores. While this downy mildew fungus does not infect any crops other than sunflower and Jerusalem artichoke, weeds in the Compositae family, such as marsh elder, are susceptible, and thus may serve as reservoirs for the fungus.

Sunflower planted on land with no previous sunflower history occasionally has shown downy mildew infection. There are three principle ways downy mildew may occur in fields with no previous history of sunflower. Windblown and soil-borne spores account for the majority of such infections. Spores of the fungus occurring on volunteer sunflower or wild annual sunflower, even a few miles distant, may be blown to newly planted fields and result in substantial infection under favorable conditions of cool, water-logged soils. Spores also may adhere to soil particles and move to neighboring fields during dust storms. Water running through an infested field also may carry mildew spores into a previously disease-free field.

Modern seed production practices, coupled with stringent inspection of certified seed, virtually eliminate the possibility of introducing downy mildew into a "clean" field via infected seed. Seed from infected plants is generally either nonviable or so light in weight that it is separated during seed processing. A slight possibility remains that viable seed may be produced on infected plants. These seeds may produce healthy plants, systemically infected seedlings or plants with latent infection in which the fungus is localized in the roots and does not produce symptoms on the leaves. These latent infections, however, will help perpetuate the disease in the field.

Figure 73. Damage to sunflower from 2,4-D or growth regulator type herbicides may be mistaken for downy mildew symptom. (D.E. Zimmer)

Damage: Severely infected plants may die before or shortly after emergence or in the seedling stage. The few plants reaching maturity seldom produce viable seed. Heads on these plants typically face straight up, rendering them extremely vulnerable to bird feeding.

Yield losses from downy mildew can be substantial, depending on the percentage of infected plants and their distribution within the field. If infected plants are scattered randomly throughout a field, yield losses probably will not be observed unless infection exceeds 15 percent. Sunflower have excellent compensating ability when healthy plants are adjacent to infected plants. When the disease is in a localized area, such as a low spot in a field, and all plants are infected, the resultant yield loss is much greater.

Management: The continued discovery of new downy mildew races and the occurrence of mildew strains resistant to Apron XL (mefenoxam) and Allegiance (metalaxyl) seed treatment have altered management strategies. When these seed treatments were effective, seed companies had no need to develop resistant varieties. Now most seed companies are developing hybrids that use multirace immunity, which should be effective despite the development of new races. Not all hybrids in a company's lineup will have downy mildew resistance, however. At least two dozen races of the fungus have been identified in the U.S., but annually, usually two to three races make up the majority of races. Fortunately, lines released by the USDA are available that confer resistance to all known races.

With the appearance of downy mildew strains that are insensitive to metalaxyl and mefenoxam fungicide seed treatments (Apron XL, Allegiance), efforts are under way to find replacement seed treatments. The fungicide azoxystrobin (Dynasty) recently has been labeled for use on sunflower as a seed treatment

for downy mildew suppression. As other effective chemicals are registered for use as sunflower seed treatments, the most effective management strategy would be to use two fungicides at the same time. A two-fungicide treatment will improve disease control and probably be less costly, in addition to delaying the development of fungicide resistance in downy mildew. Seed-applied fungicides will protect against root infection by all races, and thus will augment protection offered by resistant hybrids.

Seed applications, however, will not protect against foliar infection. Since the fungicides are water-soluble, they can be washed off shallow-planted seed with excessive rainfall, resulting in poor disease control. Selection of downy mildew-resistant hybrids is the most effective way of controlling downy mildew. Seed companies are actively incorporating genes that control all known races, and these hybrids will be immune, at least until a new race emerges. Additional management practices that minimize downy mildew problems include extended crop rotations, eradication of volunteer sunflower, avoiding poorly drained fields or those with excessive low areas and delaying planting until warm soil temperatures foster rapid seedling growth.

Apical Chlorosis

Description: Apical chlorosis is the one of two bacterial diseases of sunflower that is noticed with any regularity in the U.S. The causal organism is *Pseudomonas syringae* pv. *tagetis*. Apical chlorosis is striking and seldom goes unnoticed. The major symptom of the disease is the extreme bleaching or chlorosis of the upper leaves (Figure 74). Apical chlorosis may be distinguished from iron chlorosis or nitrogen deficiency by the complete lack of green pigment and the uniformity of the chlorosis. With mineral deficiencies, the veins characteristically remain green. In addition, the white leaves affected by apical chlorosis never will "regreen," while those due to mineral deficiencies will.

Disease Cycle: Apical chlorosis occurs only during the vegetative growth stage when leaves are actively expanding. It is most severe in young seedlings during cold weather and in water-logged soils.

Damage: Plants affected by apical chlorosis usually will produce new green leaves in several weeks with little discernible effect other than striking white leaves in the middle of the plant. Thus, yield reductions due to apical chlorosis are minimal. However, if long periods of cold spring temperatures coincide with water-logged soils, seedlings affected by apical chlorosis may die. Yield losses still will be minimal as healthy neighboring plants should compensate for the dead seedlings.

Management: No hybrids are completely immune to apical chlorosis. The only control recommendation at present is to follow a four-year rotation to avoid increasing the population of the bacteria in the soil. Roguing (identifying and removing) infected plants in seed production fields will eliminate the disease and minimize the possibility of infected seed. This same bacterium will produce apical chlorosis on other Compositae weeds, such as thistles, ragweed, other *Helianthus* species (including cultivated Jerusalem artichoke), marigold and zinnia. Thus, controlling volunteer sunflowers and *Compositae* weeds, as potential disease reservoirs, will help minimize soil populations of this bacterium.

Figure 74. Apical chlorosis is characterized by an extreme bleaching or chlorosis of the upper leaves. (William K. Pfeifer)

II. Foliar Diseases

Rust

On most crops, rust refers to a single fungal species. On sunflower, the main fungus causing rust is *Puccinia helianthi*, which is worldwide in distribution and causes economic losses. In North America, four other *Puccinia* species are found on wild and cultivated sunflower: *P. canaliculata* (nutsedge rust), *P. enceliae, P. massalis* and *P. xanthii* (cocklebur rust), plus one rust from another genus, *Coleosporium helianthi*. The following discussion will deal mainly with *P. helianthi*, with minimal descriptions of the other rust fungi.

Description: Rust occurs in all sunflower production areas of the U.S. and Canada and also is widespread on wild sunflower. Most oilseed hybrids have had good resistance to the prevailing rust races, but changes in the rust population in the last decade have resulted in greater rust severity and occasionally in substantial losses in seed yield or seed quality. Confection hybrids are generally more susceptible to rust (and other diseases) than oilseed hybrids. At least 25 different rust races have been identified in the U.S., which makes breeding for rust resistance a challenge. Rust, incited by the fungus *Puccinia helianthi*, is characterized by cinnamon-colored spots or uredial pustules, which primarily occur on the leaves (Figure 75) but also on the stems, petioles, bracts and the back of the head under severe infestations. The initial appearance of rust is determined by adequate rainfall and warm temperatures, so the disease usually occurs in late summer in the northern Great Plains. The uredial pustules turn black with the advent of cool temperatures as the brown urediospores are replaced by black overwintering teliospores.

Disease Cycle: This rust completes its entire life cycle on sunflower and does not require an alternate host, as do some cereal rusts. *Puccinia helianthi* overwinters on plant debris as teliospores (thick walled, resting spores). These spores germinate in the spring to produce basidiospores that infect volunteer seedlings or wild sunflowers. This initial infection results in the formation of pycnia (generally on the underside of leaves), which, in turn, leads to aecial pustules, generally on the upper surfaces of leaves or cotyledons. The aecia are small (1/8 inch), orange cup-shaped pustules that may occur singly or in small clusters (Figure 76). The aeciospores are spread by wind to other sunflower plants, where they initiate the cinnamon-brown uredial pustules. The uredial stage is the repeating portion of the rust life cycle. Rust multiplies rapidly under favorable conditions of warm temperatures and either rain or dew. Thus, even in dry years, if night temperatures are low enough to promote dew formation on leaves, this minimal amount of leaf wetness will be sufficient to initiate rust infection. Excessive rates of nitrogen fertilization and abnormally high seeding rates result in excessive foliage, which increases humidity within the canopy and favors rust development.

Damage: Rust not only reduces yield, but also reduces oil, seed size, test weight and kernel-to-hull ratios. Late-planted fields of susceptible hybrids are generally more severely damaged by rust than earlier-planted fields. Irrigated fields also are apt to have more severe infection as the constantly wet leaves provide an ideal environment for the rust fungus to multiply.

Figure 75. Rust occurs most commonly on leaves and after flowering. The cinnamon-red pustules produce summer spores; the black pustules occur late fall and produce overwintering spores. (D.E. Zimmer)

Figure 76. Aecial cups of *Puccinia helianthi*. (T. Gulya)

Management: The most effective way to avoid loss from rust is by planting rust-resistant hybrids. With sunflowers grown under center pivot irrigation, night irrigation fosters more rust infection since the spores germinate best in the dark. Headline (pyraclostrobin) and Quadris (azoxystrobin) are registered for control of rust on sunflower, and additional fungicides may become registered. Refer to the most current edition of the "North Dakota Field Crop Fungicide Guide" (PP-622) available from the NDSU Extension Service to view fungicide products registered on sunflower. The injury threshold (i.e., disease severity above which fungicide spraying is warranted to minimize yield loss) developed for using tebuconazole is when the rust severity on the upper four leaves is **3 percent** or greater. High rust severity on lower leaves has less impact upon seed yields, as the upper leaves are the ones supplying most of the photosynthate for the developing seeds.

Sunflower rust, like cereal rusts, occurs as many different "physiological races," which constantly change. Thus, hybrids selected for rust resistance eventually become susceptible as rust races change. Seed companies continually are testing their hybrids in different locations to determine rust resistance against the various races from region to region. Rust evaluations made under conditions of natural infection or with artificial inoculation of specific races also are done by university research centers, and this information frequently is found in the NDSU publication "Hybrid Sunflower Performance Testing" (A652). A few other management practices, besides rust-resistant hybrids, that can minimize rust are available. Destruction of volunteer plants and wild annual sunflower occurring in the vicinity of commercial fields as early in the spring as possible will reduce sources of inoculum. High rates of nitrogen fertilizer and high plant populations both foster dense canopy development that in turn create ideal conditions for rust infection, and thus should be avoided if rust is of concern.

Other Rusts: As mentioned previously, four other *Puccinia* species and one *Coleosporium* species of rust can infect sunflower in parts of the U.S. None of them have yet to be reported to be of economic significance on cultivated sunflower, but they might be considered potential nuisance foliar pathogens on ornamental sunflowers. As sunflower production expands into new areas, especially in the south-central and southwestern U.S., the possibility of other *Puccinia* species attacking cultivated sunflower increases.

Puccinia xanthii, commonly known as cocklebur rust, is easy to distinguish from *P. helianthi* based on pustule size. This rust is microcyclic, with only telia and basidiospores, and does not exhibit the five spore stages of a full-cycle (macrocyclic) rust such as *P. helianthi.* On sunflower, the telial pustules are few in number, range from 1/4 to 3/8 inch in diameter, are distinctly puckered (convex) and bear a layer of dark brown spores only on the underside of leaves. As the teliospores germinate in place, the spore layer changes from brown to gray. As little as two to three hours of dew at temperatures of 68 to 77 F is sufficient for *P. xanthii* infection. Sunflower is minimally susceptible to this rust. The main host for this rust is cocklebur (*Xanthium* species), with some authors also listing ragweed *(Ambrosia)*. This rust has been seen only once on sunflower, and only in North Dakota.

Puccinia enceliae and *P. massalis* are two rusts that are recorded on wild *Helianthus* species in the southwestern U.S. and have been shown to infect cultivated sunflower in greenhouse tests. No reports of them being identified on or causing yield losses to cultivated sunflower are known. *Puccinia massalis* is reported only on Texas blueweed *(Helianthus ciliaris)* in the Rio Grande Valley of Texas and New Mexico. Uredial pustules are indistinguishable from those of *P. helianthi.* Positive identification of *P. massalis* requires microscopic examination of teliospores for the placement of germ pores. *Puccinia enceliae* occurs on wild *Helianthus* and dessert shrubs in the genera *Viguiera* (goldeneye and resin bush), *Encelia* (brittlebush) and *Tithonia* (Mexican sunflower) in the desert regions of western U.S. and northern Mexico. Uredial and telial pustules of *P. enceliae* on sunflower are similar in appearance to those of *P. helianthi.* Positive identification of *P. enceliae* requires microscopic examination of teliospores. Neither of these two *Puccinia* species has been observed on sunflower in the northern Great Plains.

Puccinia canaliculata is a full-cycle, heteroecious rust that has its aecial stage on sunflower and cocklebur *(Xanthium strumarium),* and its uredial and telial stage

on sedges of the genus *Cyperus*. This rust had been considered as a possible mycoherbicide for control of the noxious weed yellow nutsedge. On sunflower, the aecial pustules look very similar to the telial pustules of *P. xanthii* in shape, with the main difference that the *P. canaliculata* aecia on the underside of the leaf are orange, as compared with the dark brown of the telia of *P. xanthii.* This rust has been observed in Kansas in a sunflower field infested with nutsedge. Since the rust requires both hosts to complete its life cycle, elimination of the nutsedge effectively will limit this rust infection of sunflower.

C*oleosporium* species are commonly referred to as pine needle rusts and are heteroecious rusts (requiring two different hosts to complete their life cycle). *Coleosporium helianthi* has its uredial and telial stages on sunflower, and the remainder of its life cycle on two- and three-needle pines, such as Jack pine, Virginia pine and loblolly pine. The rust is of minimal economic importance in pine plantations and never has been observed on cultivated sunflower. On Jerusalem artichoke (and other perennial *Helianthus* species), *C. helianthi* infections can be quite severe and potentially cause yield losses. Preliminary greenhouse tests have shown that cultivated sunflower is susceptible, with little differences between commercial hybrids. The most effective way to prevent *C. helianthi* infection on sunflower is to avoid planting near shelter belts or areas with two-needle pines.

Albugo or "White" Rust

Description: White rust is one of the rarest sunflower diseases in North America, but is considered a potentially serious disease in countries such as South Africa and Argentina. Despite the word "rust" in the common name, this disease is caused by a pathogen more closely related to downy mildew. White rust has been recorded on sunflower in North America only in western Kansas, eastern Colorado and adjacent Nebraska during the late 1990s and seldom is seen in this area. In North Dakota, it has been recorded on ragweed *(Ambrosia* spp.*),* various sage and sagebrush *(Artemisia* spp.*)* and goatsbeard *(Tragopogon)*. White rust has not been recorded on either wild sunflower or cultivated sunflower in the northern Great Plains or in Canada on sunflower.

Foliar lesions, consisting of raised, chlorotic pustules up to 3/8 inch in diameter, are the most commonly seen symptom on sunflower in the U.S. (Figure 77). A dull, white layer of spores forms in these pustules on the underside of the leaf, which could be mistaken for local lesions caused by downy mildew. If the Albugo pustules are numerous enough, they will merge as they enlarge, and entire areas of the leaf will turn necrotic as secondary fungi colonize the pustules.

One striking feature often seen with *Albugo* is that a single horizontal "layer" of leaves in the crop canopy usually is affected, with a sharp demarcation of healthy leaves above and below the affected leaves. This delimited layer of infected leaves suggests that environmental conditions and *Albugo* spores were

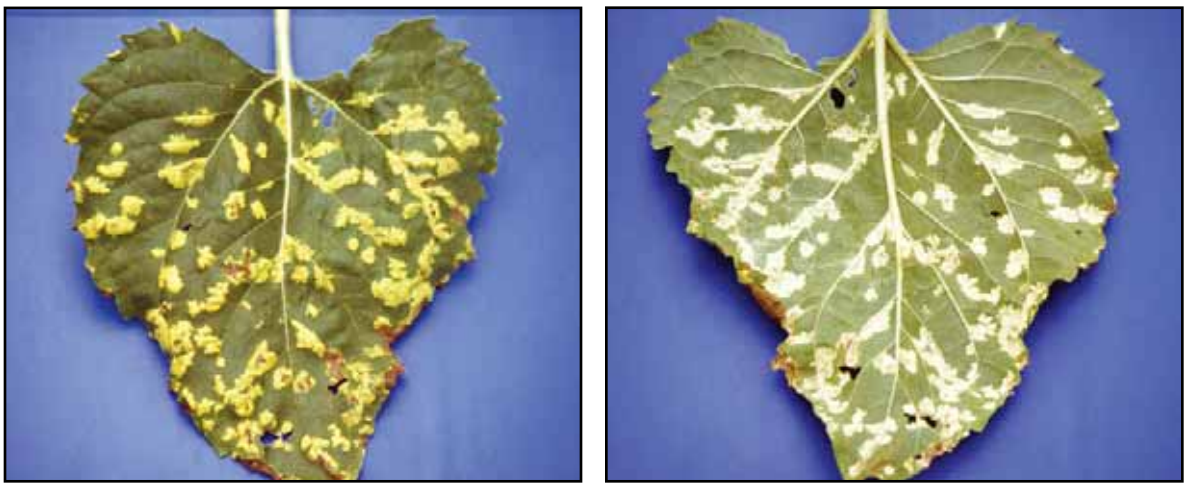

Figure 77. Albugo lesions on upper leaf surface (left) and lower leaf surface (right). (T. Gulya)

present for a very short time, thus only one layer of leaves shows symptoms. *Albugo* also can cause lesions on stems, petioles, bracts and the back of heads. The lesions on stems (Figure 78), petioles and the back of the heads are much different from those on leaves, and appear to be dark, bruiselike lesions. This appearance is due to the presence of black oopsores just beneath the epidermis. No white sporangia ever are seen in the stem or petiole lesions. Stem lesions often are colonized by other fungi and may lead to stalk rot and lodging by the secondary fungi. Petiole lesions may girdle the petiole and cause the affected leaf to wilt, thus causing considerable damage and yield loss.

Disease Cycle: The causal agent of white rust is the obligate fungus *Albugo tragopogonis*, which recently has been reclassified as *Pustula tragopogonis.* In addition to sunflower, this fungus infects cocklebur *(Xanthium),* groundsel *(Senecio),* marsh elder *(Iva),* ragweed *(Ambrosia)* and several other weedy Composites. The fungus appears to have host-specific races, however, so white rust occurring on one host genus may not infect other genera or cause only minimal infection. *Albugo* overwinters as oospores in infected plant debris. Rain splashes the oospores onto nearby seedlings, where they germinate to form motile zoospores that enter through stomates. The pustules that develop contain masses of dry, white asexual sporangia. The pustules rupture to release sporangia that are windblown to other plants to continue leaf infection. Sporangia also can initiate infection on stems, petioles and bracts. Optimal conditions for *Albugo* infection are cool nights (50 to 60 F) with rain or dew, but lesion development is favored by warm days (70 to 80 F).

Damage: Foliar infection by *Albugo* seldom causes yield loss, although the symptoms are quite noticeable. Lesions on stems and petioles, which fortunately are seen seldom in the U.S., are much more serious. Petiole lesions lead to defoliation, with significant yield losses, and stem lesions may become colonized by other fungi and result in lodging. *Albugo* infections on the head may result in seed infection, which would be of concern in seed production fields.

Management: Field trials have shown that sunflower plants can tolerate a high proportion of foliage infection by *Albugo* before yield losses are observed. Stem and petiole lesions caused by *Albugo* are more serious, as stem lesions can result in lodging and petiole lesions will result in defoliation. In countries where *Albugo* is serious, commercial seed companies have made the effort to develop resistant hybrids. In the U.S., no information is available regarding the reaction of hybrids to white rust. Evaluations of USDA breeding material have shown that resistance probably is controlled by several genes, each governing infection of leaves, stem, petioles and heads. Since *Albugo* is an Oomycete, like downy mildew, the same fungicides that control downy mildew have been effective against white rust. Thus, seed treatment with metalaxyl or mefenoxam will offer protection against both systemic infection and foliar infection for a limited time. Rotation is of limited use with *Albugo,* as the spores can be windblown from adjacent fields.

Figure 78. Albugo lesions on stems. (T. Gulya)

Alternaria Leaf and Stem Spot

Description: Alternaria leaf spot is a ubiquitous disease on senescing leaves and generally of little concern, but under warm, humid conditions it can be a serious defoliating disease. The Midwest has two main species of *Alternaria: A. helianthi* and *A. zinniae*, of which *A. helianthi* is the more prevalent and more serious. In addition, several other *Alternaria* species have been reported on sunflower, including *A. alternata, A. helianthicola, A. helianthinficiens* and *A. protenta. Alternaria helianthi* and *A. zinniae* both produce dark brown spots on leaves. These spots are irregular in size and shape with a very dark border and a gray center (Figure 79). The spots on young plants may have a yellow halo. Leaf lesions may coalesce, causing leaves to wither. Stem lesions begin as dark flecks that enlarge to form long, narrow lesions (Figure 80). These stem lesions often coalesce to form large blackened areas, resulting in stem breakage. Stem lesions are distributed randomly on the stem and are not associated with the point of attachment of the leaf petiole. Brown, sunken lesions also may form on the back of the head, especially following any mechanical damage such as that caused by hail or birds. The leaf and stem lesions caused by the various *Alternaria* species are similar and thus not diagnostic. Therefore, microscopic examination is required to distinguish which *Alternaria* species is present.

Disease Cycle: All *Alternaria* fungi overwinter on diseased stalks. They can be seed-borne at low levels, although seed is a relatively unimportant source of the inoculum under most conditions. Seedling blights caused by *Alternaria* may develop when sunflower plants emerge in rainy weather on *Alternaria*-infested soil. However, plants at the flowering to maturing stage are more susceptible than plants in the vegetative or budding stage. Safflower and cocklebur also can be alternate hosts of *A. helianthi.*

Disease development in *A. helianthi* is favored by 77 to 82 F temperatures and at least 12 hours of wet foliage. Extended wet periods of three to four days can cause serious losses as the spots enlarge.

Damage: The primary damage that all *Alternaria* species cause is the leaf blights that lead to defoliation which increases the potential for yield loss. In the northern Great Plains, the climate is usually not conducive for *Alternaria* epidemics, and *Alternaria* generally affects only the lower, senescing leaves. However, in warmer climates with plentiful rainfall, the potential for defoliation by *Alternaria* species and subsequent yield loss is much greater. In addition to the direct yield loss caused by foliar *Alternaria* infection, this fungus also has been noted to cause blemishes on the achenes. While this damage may be only superficial, if the achenes are on confection sunflowers destined for human consumption, the impact of "achene blemish" can be significant. This achene

Figure 79. Leaf lesions caused by *Alternaria hellanthi.* (B.D. Nelson)

Figure 80. Stem lesions caused by *Alternaria hellanthi.* (T. Gulya)

blemish, currently noted only on sunflower grown in Israel, looks very similar to "kernel black spot," which is caused by feeding by the tarnished plant bug *(Lygus)*, with no involvement from *Alternaria.*

Management: *Alternaria* leaf blights are considered a major disease in subtropical sunflower growing areas, where yield losses may range from 15 percent to 90 percent, but are much less serious in temperate areas of the U.S. However, severe epidemics have been observed on sunflower in the eastern and southeastern portions of the U.S. Management practices to minimize *Alternaria* problems include crop rotation and burying of infested crop refuse to hasten decomposition. Consult university Extension publications, such as NDSU's PP-622, "Field Crop Fungicide Guide," for current recommendations of foliar fungicides registered for use on sunflower for *Alternaria* control. Seed treatments with metalaxyl or mefenoxam offer no control of *Alternaria* seedling blights.

Septoria Leaf Spot

Description: Septoria leaf spot develops first on the lower leaves and spreads to the upper leaves. The spots begin as water-soaked areas (greasy green in appearance). The spots become angular, with tan centers and brown margins (Figure 81). A narrow yellow halo often surrounds young spots. Mature leaf spots may contain tiny black specks, the fungal fruiting bodies (pycnidia), which are visible with a 5X to 10X hand lens. The presence of pycnidia is the best means of distinguishing leaf spots caused by *Septoria* from those caused by *Alternaria.*

Disease Cycle: Septoria leaf spot can be caused by two *Septoria* species, *S. helianthi.* and *S. helianthina*, of which the former is the major species in the U.S. *Septoria* can be seed-borne and also can survive on infected sunflower crop refuse. *Septoria* leaf spot may appear anytime during the growing season and is favored by moderately high temperatures and abundant rainfall. As such, the disease potentially is more severe in southern growing areas, compared with the northern Great Plains.

Damage: In the temperate climate of the Midwest, Septoria leaf spot usually causes little damage. Severe *Septoria* infection may cause some defoliation, but if this affects only the lower leaves on mature plants, the impact upon yield will be minimal.

Management: Crop rotation, incorporation of sunflower residue and clean seed are the best means of managing *Septoria* leaf spots. Although resistance to *Septoria* has been identified in breeding material, the infrequent occurrence of Septoria has not warranted the development of resistant hybrids.

Figure 81.Septoria leaf spot. Note small black pycnidia in lesions. (T. Gulya)

Powdery Mildew

Description: Powdery mildew, caused by the fungus *Erysiphe cichoracearum*, can be found in most fields after full bloom. The symptoms are distinctive and easy to recognize: a dull white to gray coating of the leaves, starting as individual circular spots and eventually merging to cover the entire leaf (Figure 82). This coating is the scant mycelial growth of the fungus on the leaf surface. Severely infected areas senesce prematurely and dry up. Normally the lower leaves are more heavily infected than the upper leaves. In other countries, two other powdery mildew fungi have been documented on sunflower: *Sphaerothecia fuliginea* and *Leveillula taurica.* These both exist in the U.S., but to date have not been documented on sunflower.

Disease Cycle: Powdery mildew seldom is seen until late in the growing season, as senescing leaves are most susceptible to infection. While leaves are the most common plant part affected, powdery mildew also will form on bracts and the backs of heads. The powdery coating seen is a combination of scant mycelia and spores of the asexual stage, which is referred to as *Oidium.* As the season progresses, the fungus forms small (pinhead-sized) black cleistothecia, the sexual fruiting bodies.

Damage: Powdery mildew generally occurs late enough in the season that control measures are not needed. Sunflower cultivars differ widely in reaction to powdery mildew. On ornamental sunflower, especially those grown in the greenhouse, powdery mildew is common. Although the main impact of the disease is cosmetic, this alone can cause economic losses.

Management: Powdery mildew is seldom a problem on cultivated sunflower in the Midwest, but may be of concern in more humid areas and on the southern Plains states. On ornamental sunflowers, powdery mildew can be minimized by adequate air movement, allowing the leaves to dry, and by the use of registered fungicide sprays specific for powdery mildew.

Diseases Caused by Viruses and Phytoplasmas

Description: Several viruses have been reported on sunflower from other countries and the warmer regions of the U.S. (Florida), but no reports of viruses occurring on sunflower in the northern Great Plains have been confirmed. Wild sunflower is a host of *Tobacco ring spot virus* in the Rio Grande Valley, and *Cucumber mosaic virus* has been reported on sunflower in Maryland. Viruses reported on sunflower outside the U.S. include *Tobacco streak virus, Tomato big bud virus, Sunflower rugose mosaic virus,* and *Tomato spotted wilt virus.* Confirmation/identification of a virus as the causal agent is based on observation of the viral particles using an electron microscope, detailed chemical analysis of the viral components or serological identification.

The only well-documented virus found on sunflower in the U.S. is *Sunflower mosaic virus (SuMV),* currently found only in southern Texas on both cultivated sunflower and wild *Helianthus* species. Symptoms of SuMV are a mottled pattern of light green and normal green areas on the leaf, referred to as mosaic (Figure 83). Affected plants may die in the seedling stage or live to maturity, with all leaves affected.

Aster yellows is a disease at first believed to be caused by a virus but which has since been identified as a phytoplasma. Phytoplasmas are living cells, in contrast to viruses, and are intermediate in size between viruses and bacteria. Symptoms on sunflower include yellowing of leaves and/or the head, which often occurs in sectors. A characteristic symptom is a wedge-shaped portion of the head that remains green and bears small leaves rather than floral parts (Figure 84), a condition termed “phyllody.”

Figure 82. Powdery mildew with fungus producing white, powdery spores on leaf surface. (Reu V. Hanson)

Figure 83. Sunflower mosaic virus. (T. Gulya)

Figure 84. Aster yellows. Note wedged shaped portion of head which remains green. (Donald Henne)

Disease Cycle: SuMV is spread primarily by aphids but is also seed-borne to a small extent. Seedlings less than a month old are the most susceptible, and mosaic symptoms appear within a week after aphid transmission. Affected leaves will retain the mosaic pattern for the life of the plant, but no stunting due to the virus is discernible.

The aster yellows phytoplasma is transmitted only by the aster leaf hopper *(Macrosteles quadrilineatus)* and occurs on a wide variety of plants. Symptoms generally appear at flowering, and affected heads will show the symptoms for the remainder of the summer.

Damage: SuMV, which currently is found only in Texas, can substantially reduce yield in individual plants. No fields have been observed with high incidence. This disease is also of quarantine significance, and many countries will not accept seed from fields with any level of SuMV. Aster yellows, which occurs throughout the Midwest, is sporadic in occurrence and is generally more of a novelty than of economic consequence.

Management: As both SuMV and aster yellows are spread by insects, the easiest means of minimizing both diseases is to control their insect vectors. Varietal differences to aster yellows have been noted. In contrast, no resistance to SuMV is available in commercial hybrids, although resistance has been found in wild *Helianthus* species from Texas.

Other Miscellaneous Foliar Diseases

The diseases mentioned above are the most likely leaf diseases to be encountered in the main sunflower-producing areas of the Midwest. As sunflower production expands into other areas, a possibility exists of other fungi causing leaf spots. Some of the fungi recorded to cause leaf spots on wild sunflower in other areas of the U.S. include *Ascochyta compositarum, Cercospora helianthi, C. pachypus, Colletotrichum helianthi, Entyloma compositarum* (leaf smut), *Epicoccum neglectum, Itersonilla perplexans, Myrothecium roridum, Phialophora asteris* (Phialophora yellows), *Phyllosticta wisconsinensis* and *Sordaria fimicola.* Check out the "Sunflower Diseases" chapter by Gulya et al. in "Sunflower Technology and Production," published by the Agronomy Society of America, for more details on these minor foliar pathogens.

III. Stalk- and Root-infecting Diseases

Sclerotinia Wilt

Description: Sclerotina wilt usually is observed first as plants start to flower. The first symptoms include a sudden wilting (Figure 85) with no other leaf symptoms, and a characteristic stalk lesion at the soil line. The length of time from the first sign of wilt to plant death may be as little as four to seven days. The stalk lesions that form at the soil line are tan to light-brown and eventually may girdle the stem (Figure 86). Under very wet soil conditions stalks and roots may be covered with white mycelia and hard black structures called sclerotia (Figure 87). Sclerotia are irregular-shaped structures which range in size and shape from spherical and 1/8 inch in diameter to cylindrical or Y-shaped and up to 1 inch in length. Sometime a series of dark "growth" rings produced by the daily extension of the fungus can be observed.

Disease Cycle: *Sclerotinia sclerotiorum* overwinters as sclerotia in the soil or in plant debris. When sunflower roots grow near sclerotia, the sclerotia are stimulated to germinate, and the resulting mycelium infects the lateral roots. The fungus grows along the root system to the tap root and up into the stem, and the plant wilts and dies. Contact between roots of adjacent plants within rows allows the fungus to spread from plant to plant. The fungus does not move between conventionally spaced rows. Sunflower is the only crop that *S. sclerotiorum* consistently infects through the roots. Other susceptible crops are infected mainly by spores on above-ground parts of the plant.

Sclerotia are formed in the decayed stem pith and on the roots as the plant dies. These sclerotia then are returned to the soil during tillage operations and serve as sources of inoculum for the next susceptible crop. Sclerotia can survive in the soil for five or more years, with a portion of them dying each year if they fail to infect a host. The higher the inoculum density (i.e., the number of sclerotia in the soil), the longer the period a

Figure 85. Sudden wilting is a characteristic symptom of Sclerotinia wilt. (B.D. Nelson)

Figure 86. Basal canker formed from *Sclerotinia* wilt infection. (B.D. Nelson)

Figure 87. Dense white mold may form on the surface of the basal canker. Hard black bodies called sclerotia also form on the outside and the inside of stems. (D.E. Zimmer)

field will remain infested. Soil moisture and temperature during the growing season are not critical factors affecting wilt incidence. Plant population, in the range from 15,000 to 30,000 plants per acre on 30- or 36-inch rows, is not a factor affecting disease incidence, although solid seeding should be avoided. Lodging of wilted plants, however, increases at high plant populations due to smaller stem diameter.

Damage: Historically, wilt is the most prevalent of the Sclerotinia diseases, with the disease found in one out of two fields in the Dakotas, and about 3 percent of the crop affected. Wilt occurs whenever sunflower is planted on *Sclerotinia*-infested soil and can cause severe yield loss. Infected plants die rapidly, and if this occurs before the seed is fully mature, the result is a loss of seed yield accompanied by lower test weight and lower oil content. Since plant death occurs late in the season, healthy adjacent plants have little opportunity to compensate for the loss of the *Sclerotinia*-infected plant. On the average, infected plants yield less than 50 percent of healthy plants. Equally important, however, is that Sclerotinia wilt leads to increased numbers of sclerotia in the soil, thus contaminating the field for all susceptible crops that would be in the rotation.

Management: Sclerotinia wilt is a difficult disease to manage for several reasons. First, the fungus has a very broad host range and basically is able to infect all broadleaf crops to some extent. Rotation to minimize *Sclerotinia* is most effective with cereal grains and corn. Second, since the sclerotia can persist in the soil for long periods of time, long rotations away from broadleaf crops are necessary to minimize sclerotial populations in the soil. This may not be feasible because repeated cropping of cereal grains will lead to a buildup of cereal diseases, especially Fusarium head blight. Sunflower breeders have strived to increase the level of resistance to Sclerotinia wilt and tremendous improvements have been made, but no immune hybrids are on the market yet. Based on several years of tests with artificial inoculations, commercial hybrids with extremely good levels of resistance are available. Information on hybrid ratings for Sclerotinia stalk rot resistance is found in NDSU publication A-652, "Hybrid Sunflower Performance Testing."

Lastly, chemical control is not an option since the sclerotia are low in number and scattered throughout the soil. The most effective chemicals would be soil fumigants used for nematocide control, and even if the chemicals were registered on sunflower, the cost would be prohibitive. Two management strategies hold promise to minimize, but not eliminate *Sclerotinia.* One is the use of biocontrol. Various fungi, termed mycoparasites because they feed upon other fungi, have been shown to attack *Sclerotinia.* One such fungus is *Coniothryium minitans*, which is found in the registered product called "Contans." If this product is applied to the soil (and preferably to the crop before disking), the mycoparasite *Coniothryium* will colonize the sclerotia and kill the sclerotia in several months rather than years. This will allow shortened rotations to be used, but replanting a *Sclerotinia*-infested field to a susceptible crop (e.g., dry bean, canola, sunflower) the following year still is inadvisable.

Another management option is tillage, and this is at the center of two schools of thought. One opinion is that deep tillage (inversion of the soil profile via moldboard plowing) will put the sclerotia deep into the soil in an anaerobic environment where they are more prone to bacterial degradation and are out of the plant root zone. If deep tillage is used, producers should practice reduced tillage in the following years to prevent bringing the buried sclerotia back to the surface. The second school of thought is to let the sclerotia remain on the soil surface, where they are subject both to weathering and attack by other fungi. No conclusive evidence is available to show that either no-till or deep tillage produces significantly less Sclerotinia wilt with sunflower, although research on soybeans strongly favors no-till to reduce sclerotia numbers.

Sclerotinia Middle Stalk Rot and Head Rot

Description: Middle stalk rot is the disease least often caused by *Sclerotinia*, and is first observed in the middle to upper portion of the stalk at or before flowering. Midstalk rot begins with infection of the leaf, and the fungus progresses internally through the petiole until it reaches the stem (Figure 88). Symptoms of *Sclerotinia* leaf infection are not unique enough to identify the fungus, but once the stem lesion forms, the symptoms are identical with the lesion formed by root infection. The characteristic pith decay and formation of sclerotia both within the stem and sometimes on the exterior are highly diagnostic. The stalk usually lodges at the lesion site and the leaves above the canker die. With time, the fungus completely disintegrates the stalk, and the affected area will have a shredded appearance, as only the vascular elements of the stem remain.

The first symptoms of head rot usually are the appearance of water-soaked spots or bleached areas on the back of the heads. The fungus can decay the entire head, with the seed layer falling away completely, leaving only a bleached, shredded skeleton interspersed with large sclerotia (Figure 89). These bleached, skeletonized heads, which resemble straw brooms, are very obvious in the field, even from a distance. During harvest, infected heads often shatter and any remaining seeds are lost. The large sclerotia in the heads may be 0.5 inch (12 mm) or greater in diameter and many are harvested along with the seed (Figure 90).

Disease Cycle: If soil is very wet for seven to 14 days, sclerotia in the upper several inches of soil can germinate to form small mushrooms called apothecia. These apothecia produce ascospores for a week or more. The ascospores can originate within the sunflower field or can be blown in from adjacent fields. Thus, sunflower fields with no history of Sclerotinia can became affected by head and middle stalk rot. Apothecia are not usually observed until after the crop canopy has completely covered the rows. Apothecia are more likely to form in crops with dense canopies, such as small grains, and the resultant spores can be blown a distance to nearby sunflower fields (Figure 91). Ascospores require both free water (dew or rain) and a food base such as dead or senescing plant tissue to germinate and infect. The fungus cannot penetrate unbroken

Figure 88. Middle stalk rot occurs via ascospore infection. (B.D. Nelson)

Figure 89. Head rot showing skeleton head filled with sclerotia. (B.D. Nelson)

Figure 90. Sunflower seed contaminated with sclerotia.

Figure 91. Apothecia in field of soybean plants. (B.D. Nelson)

tissue. Midstalk infection may result from either leaf infection or infection at the leaf axil. Head infection actually starts as ascospores colonize the dead florets and pollen on the face of the head. Thus, when lesions are seen on the back of the head, several weeks have elapsed since infection took place.

Damage: Middle stalk rot is the least often seen phase of Sclerotinia diseases. Head rot incidence fluctuates dramatically, dependent entirely upon weather conditions. In dry years, head rot is entirely absent, while in years and locations where rainfall is frequent during and after flowering, head rot may be present in nearly all fields to some degree. Currently the incidence of head rot and wilt in the Dakotas is about equal, with approximately 3 percent of the crop affected by each disease. Yield loss from head rot on an individual plant may range from minimal to total loss since the affected head may disintegrate and drop all of the seed on the ground prior to harvest. Intact but diseased heads will have light and fewer seeds, with lower oil concentration, and also will shatter during harvest. The sclerotia that form in the diseased stalks and heads are returned to the soil at harvest, thus contaminating the field for subsequent broadleaf crops.

Management: The same comments made about Sclerotinia wilt also apply to head rot management, with some exceptions. Since ascospores that cause head rot can be blown into a field, rotation will have less consistent impact upon head rot, even though it may reduce the levels of sclerotia in the field. Anything that can minimize the crop canopy will help modify the environment necessary for ascospore infection. Thus, lower plant populations will facilitate more air movement and hasten leaf drying. Moderate levels of nitrogen fertilization also will minimize excessive foliage, but this needs to be counterbalanced with adequate fertilization to optimize yields.

One of the most important tools for managing all Sclerotinia diseases is monitoring the incidence of Sclerotinia diseases on any preceding crop. If *Sclerotinia* is observed on a crop, the grower then knows that planting any susceptible crop in that field the next year is imprudent. The number of years necessary to rotate away from susceptible broadleaf crops (while the population of sclerotia declines) depends upon the initial *Sclerotinia* incidence. As a general rule of thumb, most researchers suggest that four years away from broadleaf crops should reduce the sclerotial population to below a threshold level. Since sunflower is the only crop prone to root infection, and root infection can happen even in dry years, sunflower obviously would be the worst crop choice to plant in a known infested field, with dry bean and canola following closely behind. As stated earlier, the best crops to break the *Sclerotinia* cycle are monocots (small grains, corn, sorghum).

Sunflower hybrids do exhibit different degrees of susceptibility to head rot, but no totally resistant hybrids are available. An extensive testing program is under way to evaluate commercial hybrids for head rot resistance. Several university research centers have established mist-irrigated plots, which when coupled with artificial inoculations with ascospores, have produced high levels of infection to accurately assess hybrid disease response. After the most resistant hybrids have been tested in multiple locations, this information will be published in the NDSU publication "Hybrid Sunflower Performance Testing" (A652), also available online at www.ag.ndsu.edu/pubs/plantsci/rowcrops/a652.pdf. No chemical is registered for control of head rot in the U.S. Even if a chemical were registered, it would have to be applied as a preventative because when symptoms become visible, the infection already took place two to three weeks earlier and the fungus has become well-established in the head.

For more information on sclerotinia, consult NDSU Extension Service publication "Sclerotinia Disease of Sunflower" (PP-840), also viewable on the Internet at www.ag.ndsu.edu/pubs/plantsci/rowcrops/pp840w.htm.

Stem Rots Caused by *Sclerotinia Minor* and *Sclerotium Rolfsii*

Description: Stem rots caused by *Sclerotinia minor* or *Sclerotium rolfsii* are very similar in appearance and will be covered together. *Sclerotinia minor* on sunflower is seen in California and the southern Great Plains, but has not been reported in the northern Great Plains. Stem rot caused by *Sclerotium rolfsii,* also called Southern blight, primarily is observed on sunflower in warm climates such as California, Florida and irrigated fields in the southern Great Plains. *Sclerotium rolfsii* has not been observed on sunflower in the northern Great Plains, but has been noted in nursery stock in Minnesota, suggesting it could become established in the northern latitudes. Both fungi have a very broad host range encompassing many broadleaf crops.

The symptoms of both *Sclerotinia minor* and *Sclerotium rolfsii* are outwardly very similar to the root infection and wilt caused by *Sclerotinia sclerotiorum.* Affected plants have a water-soaked lesion on the stem at the soil line that turns light brown. Under humid conditions, white mycelium also may be found on the lesion. In some instances, the lesion also may have a series of dark rings due to the diurnal growth pattern of the fungus. Plants infected with either fungus wilt and die suddenly, without any distinctive foliar symptoms, within a week of the onset of wilting. The major field sign to distinguish S. *sclerotiorum* from *S. minor* is the size and shape of the sclerotia. In *S. minor,* the sclerotia are always round and generally less than 1/12 inch (2mm) in diameter (Figure 92), and thus much smaller than sclerotia of *S. sclerotiorum.* Sclerotia of *Sclerotium rolfsii* are also round and the same size (< 1/12 inch) as those of *S. minor,* but *Sclerotium rolfsii* sclerotia are tan to light brown. Sclerotia of these two fungi can be found within pith tissue and on the surface of the tap root.

Disease Cycle: Both fungi overwinter in infected plant debris or can persist for several years as free sclerotia in the soil. The sclerotia germinate in response to root exudates to form mycelia that infect roots of adjacent sunflower plants. As the fungus moves up the root system and reaches the tap root and the stem, it produces toxins and oxalic acid that cause the plant to wilt. Both fungi are capable of infecting adjacent sunflower plants by root-to-root spread. Neither fungus produces any spores to initiate leaf or head infection, in contrast to *S. sclerotiorum.*

Damage: Both *Sclerotinia minor* and *Sclerotium rolfsii* are as potentially damaging as *S. sclerotiorum,* with the caveat that the first two species only cause a root rot and subsequent wilt. Plants infected near anthesis will suffer substantial seed losses, and if the affected plants lodge, further yield losses occur. Additionally, the sclerotia produced by these two fungi will contaminate the field and make subsequent crops of other host plants prone to infection.

Management: Crop rotation, elimination of infected residue and weed control will help reduce disease caused by either fungus. Since both fungi are highly aerobic, deep plowing (>12 inches), especially if complete inversion of the soil profile is possible, will remove most of the sclerotia from the root zone and place the sclerotia where they are most vulnerable to attack by soil bacteria. Differences in hybrid susceptibility have not been investigated in the U.S. because neither disease is present in the major sunflower production areas.

Figure 92. Sclerotia of *Sclerotinia sclerotiorum* (left) and *S. minor* (right) from sunflower stalks. Ruler at top in centimeters. (T. Gulya)

Charcoal Rot

Description: Charcoal rot is caused by *Macrophomina phaseolina*, a fungus that attacks about 400 plant species, including sunflower, dry bean, soybean, corn and sorghum. Charcoal rot is found throughout the Great Plains, but the disease is most common and severe in southern areas such as Texas, Kansas and Nebraska. Charcoal rot has been found on sunflower in western North Dakota and north-central South Dakota recently, and also on corn and soybean in both states. Charcoal rot generally appears after flowering but seedling blights have been reported. Symptoms on stalks appear as silver-gray lesions near the soil line (Figure 93), which eventually decay the stem and tap root, leaving a shredded appearance. Stems are hollow and rotted, and lodge easily. Plants show poor seed fill and undersized heads. Seed yield, test weight and oil concentration are reduced. Numerous tiny black fungus bodies, called microsclerotia, form on the outside of the stalk and in the pith. To the unaided eye, the microsclerotia look like pepper grains; with a 5X to 10X lens they are clearly distinguishable as black, spherical sclerotia. Another unique characteristic of charcoal rot is the compressing of pith tissue into horizontal layers, like a stack of separated coins (Figure 94). This is a diagnostic characteristic of the disease.

Disease Cycle: The primary source of inoculum is sclerotia in the soil, but *Macrophomina* also can be seed-borne. Upon stimulation by nearby root exudates, the sclerotia germinate to form mycelium that colonizes the roots. *Macrophomina* may colonize roots early in the season, but disease symptoms do not manifest themselves until anthesis. Once the root system is colonized, the fungus enters the stem and colonizes the vascular system, resulting in wilt and partial degradation of the pith. Disease development is favored by soil temperatures above 85 F. Moisture stress during the post-flowering period greatly favors disease development.

Damage: Post-flowering stresses due to high plant population or drought coupled with heavy applications of nitrogen fertilizer, hail or insect damage promote disease development and accentuate the impact of charcoal rot. Yield losses can be significant if disease incidence is high, as infected plants die before seed set is complete.

Management: Crop rotation, balanced fertilizer programs and practices to reduce moisture stress all help minimize the impact of charcoal rot. Certain hybrids offer some resistance, possibly through drought tolerance. Since the fungus also attacks corn, sorghum and soybeans, not growing sunflower and these crops in successive years on the same ground would be advisable if charcoal rot has been observed.

Figure 93. Silver grey discoloration of lower stem caused by charcoal rot compared with healthy green stalk (left). (T. Gulya)

Figure 94. Charcoal rot affected stalk split apart to reveal characteristic compression of pith into layers. (T. Gulya)

Texas Root Rot

Description: Texas root rot, or cotton root rot, is a soil-borne fungal disease found only in Texas, New Mexico, Arizona, southeastern California and northern Mexico. The causal agent, *Phymatotrichopsis omnivora* (synonym: *Phymatotrichum omnivorum*) has a very broad host range of more than 1,800 species of broadleaf herbaceous crops and weeds. On sunflower, as with most crops, the initial symptom is wilting followed quickly by death of affected plants. The disease in Texas develops in late spring and usually occurs in circular spots in the field, which enlarge after rain or irrigation. No diagnostic symptoms appear on leaves or stems, but white mycelial strands on roots, visible with a 5X to 10X hand lens, are characteristic of this fungus. Following rain or irrigation, the fungus may produce white mycelial mats up to 12 inches in diameter and up to ¾ inch thick on the soil surface.

Disease Cycle: The Texas root rot fungus survives for many years in the soil as sclerotia. These germinate to produce mycelial strands that can grow some distance in the soil until root contact is made. This ability also allows the fungus to spread from plant to plant in a row via overlapping root systems. The disease is most severe in moist, warm (80 F or higher at a 1-foot depth) soils that are high pH and high clay content. Sclerotia, at first white and turning tan to dark brown, are round and generally greater than 1/12 inch in diameter. Sclerotia may form in the soil away from plant roots. On roots, the sclerotia may form at irregular intervals, giving the appearance of a string of beads.

Damage: Like other root-infecting, stalk-rotting fungi, *Phymatotrichum* can cause considerable yield losses if plants are infected near bloom. Even if seed yield is not reduced, the fungal infection will lower oil content and result in lower test weight. In field trials in western Texas, disease incidences up to 60 percent were observed, which cut yields by at least half.

Management: The fungus persists in the soil for many years as sclerotia, which are the propagules that infect subsequent crops. Resistance has not been observed in sunflower. The best disease management is rotation with nonhosts, such as corn, sorghum, small grains or grass. Planting after cotton or alfalfa, especially where Texas root rot was observed, would be the worst-case scenario.

Phoma Black Stem

Description: Phoma black stem, caused by the soil-borne fungus *Phoma macdonaldii*, is characterized by large, jet black lesions on the stem, sometimes reaching 2 inches in length. In addition, the fungus produces lesions on the leaves, on the back of the head and at the base of the stalk. The typical stem lesions originate with leaf infections that progress down the petiole to the stalk. Under favorable conditions, the leaf wilts, the petiole turns uniformly black and the stem lesions expand to form a large, shiny, black patch with definite borders (Figure 95). Small circular fruiting bodies of the fungus are produced on the surface of the stem, but these are inconspicuous to the naked eye and require a hand lens to observe.

Disease Cycle: *Phoma* infection occurs throughout the growing season, although it usually is not noticed until the stem lesions become obvious later in the summer. The fungus overwinters in infected debris and conidia are spread by splashing rain. Insects such as *Apion* and *Cylindrocopturus* stem weevils also can carry *Phoma* spores both internally and externally. Adult weevils feeding on the leaves cause leaf lesions, while contaminated larvae spread the fungus as they tunnel throughout the stem. Disease transmission through infected seed is of minor importance.

Figure 95. Large black lesions associated with the point of attachment of the leaf to the stem is a characteristic symptom of Phoma black stem.
(D.E. Zimmer)

Damage: *Phoma* black stem is the most widespread stalk disease noted on sunflower in the northern Great Plains, but yield losses attributable solely to *Phoma* generally are considered minimal. Infected plants may produce smaller heads with reduced seed yield and oil. *Phoma* stem lesions are generally superficial and do not result in pith damage or lodging. However, if stem weevil larva tunneling spreads *Phoma* spores in the pith, extensive pith degeneration can occur.

Management: No control measures are totally effective. A four-year rotation to other crops will minimize the concentration of *Phoma* within the soil. Control of stem weevils can help reduce transmission of the fungus. However, such control is not recommended solely for management of *Phoma.* No hybrids have been identified as being immune to the disease, but some hybrids are more tolerant than others.

Phomopsis Stem Canker

Description: *Phomopsis* stem canker, caused by *Phomopsis helianthi* (sexual stage = *Diaporthe helianthi*) is a serious disease that first was observed in Europe in the late 1970s and in the U.S. in 1984. The distinguishing feature of the disease is the large tan to light brown lesion or canker that typically surrounds the leaf petiole (Figure 96). Compared with *Phoma* black stem, the *Phomopsis* lesion is much larger, reaching 6 inches in some cases, is brown rather than black and typically has a sunken border. *Phomopsis* also causes more extensive pith degradation than *Phoma*, so the stalk may be crushed with moderate thumb pressure. Phomopsis-infected plants also are more prone to lodging than Phoma-infected plants.

Figure 96. Phomopsis is characterized by the large light brown lesion or canker which typically surrounds the leaf petiole. (T. Gulya)

Disease cycle: The fungus overwinters predominantly as perithecia of *Diaporthe* in infected plant debris. The ascospores released from the perithecia are rain splashed or windblown onto leaves. The infection starts on the margins of lower leaves. A brown necrotic area develops and may be bordered by a chlorotic margin. The infection spreads down through the veins to the petiole and finally to the stem. These symptoms look similar to those of *Verticillium* leaf mottle, but with *Verticillium*, the necrotic areas are **between** the veins. Stem lesions usually do not appear until flowering. Girdling stem lesions result in wilting and make the plant more prone to lodging. The disease may be difficult to identify when both *Phoma* and *Phomopsis* are present, in which case the stem lesion may be intermediate in color between the black lesion associated with *Phoma* and the brown typically associated with *Phomopsis.* In these cases, microscopic identification of the fungus is necessary.

Damage: Phomopsis stem canker has been found in both the central and northern areas of the Great Plains. Since the fungus is specific to sunflower, it likely would not be found in areas without a history of sunflower production. The disease is most severe under conditions of prolonged high temperatures and high rainfall. Yield losses result from smaller heads and lighter seed, and from lodging due to weakened stems, which can be quite extensive.

Management: Since the fungus overwinters in infected sunflower debris on the soil surface, thorough disking in the fall to bury plant residue and crop rotation can reduce disease incidence and severity. Leaving crop residue on the soil surface would foster the best development of *Phomopsis.* Most U.S. sunflower companies are trying to incorporate some levels of Phomopsis resistance into their hybrids, using parental lines developed in Europe, where the disease is particularly severe. No U.S. commercial hybrids are immune to Phomopsis stem canker, nor are any fungicides registered in the U.S. for control of *Phomopsis.* Please consult NDSU publication A-652 for information on ratings of commercial hybrids to *Phomopsis.*

Verticillium Leaf Mottle

Description: Verticillium wilt, or more accurately, leaf mottle, is caused by the soil-borne fungus *Verticillium dahliae.* The fungus has a wide host range and causes wilt of several other cultivated plants and weeds. Potato is the other important crop host of *Verticillium* in the northern Great Plains. Verticillium leaf mottle typically causes necrosis between the main leaf veins with yellow margins. The contrast between the necrotic tissue surrounded by chlorosis and the healthy green leaf tissue is striking and quite diagnostic. Symptoms begin on the lower leaves and progress slowly upward (Figure 97) and may encompass all leaves. Affected leaves rapidly become completely dry, but do not wilt to the same degree as with *Sclerotinia* wilt. Thus, the term leaf mottle may be more appropriate than *Verticillium* wilt. Symptoms usually are not observed until flowering, but under severe conditions, they may occur as early as the six-leaf stage. The vascular system of infected plants may be discolored brown, visible as a brown ring in a cross-section of the stem. The pith of severely diseased plants is blackened with a layer of tiny black fruiting bodies (microsclerotia). These microsclerotia are much smaller than microsclerotia of charcoal rot and are not visible with a hand lens. Under a microscope, Verticillium microsclerotia are irregular to club-shaped (< 0.1 mm long), while charcoal rot microsclerotia are more uniformly spherical and larger (0.1 to 1.0 mm in diameter). Another fungus, *Phialophora asteris,* causes quite similar symptoms on sunflower. This fungus does not form microsclerotia, which is one way to distinguish it from *Verticillium.*

Life Cycle: *Verticillium* overwinters as mycelium or microsclerotia in infected plant debris. The microsclerotia germinate in response to root contact and colonize the root system. As the fungus reaches the tap root and lower stem, toxins produced by the fungus are translocated to the leaves to produce the chlorotic and necrotic areas between the veins. *Verticillium* remains within the stem tissue and cannot be isolated from symptomatic leaves. The fungus is isolated most easily from stems and petioles of infected plants. No involvement of conidia occurs in disease development, although the fungus does produce conidia in culture.

Damage: Sunflowers infected with *Verticillium* usually die before seeds are completely mature, and thus yield losses result from smaller head size, lighter test weigh and reduced oil concentration. The stems of *Verticillium*-infected plants are weakened as the pith shrinks, and are more prone to lodging.

Management: Resistance to *Verticillium dahliae* is controlled by a single dominant gene (V-1), and most U.S. oilseed hybrids contain this resistance. However, a new strain of *Verticillium* that is able to overcome the V-1 gene recently was identified both in the U.S. and Canada. Thus, hybrids that previously were considered resistant have shown symptoms of Verticillium wilt due to infection by this new strain. Confection hybrids as a group are more susceptible to *Verticillium* than are oilseed hybrids. Verticillium leaf mottle is a serious disease on lighter soils with a history of sunflower cropping, and is seen less frequently on heavy, clay soils. This disease will cause some yield loss each time a susceptible crop is planted, as the fungus can persists for five to 10 years as microsclerotia.

Figure 97. Plants infected with Verticillium wilt show interveinal necrosis with yellow margins. (T. Gulya)

Bacterial Stalk Rot

Description: Bacterial stalk rot occurs sporadically in the Great Plains and generally is not considered a major sunflower disease. The pathogen is *Erwinia carotovora,* a bacterium that causes soft rot on potato and other vegetables. Typical disease symptoms are stem discoloration (dark brown to black), often centered on a petiole axil, and a wet, slimy, soft rot of internal stem tissue. A pungent odor, reminiscent of rotting potatoes, is also characteristic. Due to pressure from the gas produced by the bacteria, the stem may be split open. Symptoms may extend down the stem into the roots. The bacterium also can infect the head, causing a wet, slimy rot of the receptacle. Infected stems are prone to lodging, and infected heads quickly fall apart. After the plant dies, the affected stalk or head dries up and may leave little indication of prior mushy rot.

Disease Cycle: The main source of the bacteria is infested plant residue on the soil surface. The bacteria enter the plant through wounds caused by insects, hail and windblown sand; they cannot penetrate unbroken epidermis like fungal pathogens. Extended wet and warm periods favor disease development. Most diseased plants are observed later in the season. Young plants are more resistant to stalk decay than plants nearing senescence. Varietal differences in susceptibility to bacterial stalk rot are reported.

Damage: Bacterial stalk rot is seen infrequently. Plants affected by this disease will be killed and produce little or no seed, but disease incidence within a field is generally low.

Management: While differences in resistance have been observed, little information is available on the resistance of current hybrids. Control of stem feeding insects will minimize the potential of insect transmission.

Nematode Diseases

Description: Nematodes are microscopic, nonsegmented roundworms that can cause serious damage to the roots of many crops. While many different types of nematodes have been found in sunflower fields, their economic impact on the plant is undocumented or highly variable.

Genera of nematodes that have been reported on sunflower in the northern Great Plains include *Heliocotylenchus, Tylenchrohynchus, Paratylenchus, Hoplolaimus* and *Xiphinema;* the first two genera are the most widely distributed in North Dakota, while *Paratylenchus* is the dominant nematode in South Dakota. All these nematodes are ectoparasites, meaning that they feed either on the root surface or burrow partially into the roots. Root-knot nematodes *(Meloidogyne* spp.), damaging pests of many crops, have been reported on sunflower in Florida and in warm areas of other countries but have not been recorded on sunflower in the upper Great Plains states. Sunflower is not a host for the soybean cyst nematode *(Heterodera glycines),* making sunflower suitable for rotation with soybean where the cyst nematode is a problem.

Disease Cycle: Nematodes overwinter in the soil as eggs and colonize plant root systems throughout the growing season. Symptoms caused by nematodes are not distinctive and mimic those due to drought and nutrient deficiencies. In severe infestations, the foliage wilts and turns yellow, and stunting may occur. The pattern of affected plants in the field may have little or no relationship with topography. Examination of roots is necessary to prove nematode damage. Identification of nematodes to genus requires their extraction from soil and roots, followed by microscopic examination.

Damage: High populations of nematodes have caused yield reductions in greenhouse studies.

Management: Applications of nematocides in field trials have produced variable yield responses. No nematocides are registered for use on sunflower, and the potential cost return makes their use questionable. Tolerance to nematode damage appears related to the extensive root system of sunflower.

IV. Head Rots and Diseases of Mature Plants

Head Rots, Other Than *Sclerotinia*

Description: Several head rots (other than *Sclerotinia*) occur on sunflower in the U.S., and these are caused by several fungi, including *Rhizopus, Botrytis* and the bacterium *Erwinia*, covered previously.

Rhizopus head rot, caused by *Rhizopus arrhizus* and *R. stolonifera*, was considered a sporadic disease in the Great Plains, but recent surveys have shown it to be the most widespread disease in the central Great Plains (Figure 98). Initial symptoms are brown, watery spots on the receptacle. *Rhizopus* species rot the soft tissues of the head, turning it brown and mushy. A threadlike, whitish fungal mycelium develops on and within the receptacle. Tiny black pinhead-sized fruiting structures (sporangia) form within the head tissue, giving the appearance of pepper grains.

Botrytis head rot is caused by the widespread fungus *Botrytis cinerea*, and is distinguished from *Rhizopus* by the gray "fuzz" on the heads (caused by mycelium and spores). Heads affected by *Botrytis* eventually will disintegrate and may contain small sclerotia, similar in size to those caused by *Sclerotinia*.

Bacterial head rot caused by *Erwinia carotovora* is rare in the Great Plains. This disease is characterized by a slimy, wet, brownish rot of the head with no fungus growth or spores in the tissues. Often such heads have a putrid odor.

Disease Cycle: *Rhizopus* enters the head through wounds caused by hail, birds and insects and has been associated with head moth and midge damage. The susceptibility of heads increases from the bud stage up to the full bloom and ripening stages. Disease development is most rapid in warm, humid weather. Once the head is fully colonized and all tissue killed, the head dries up and becomes hard and "mummified."

Botrytis infects sunflower heads during cool, wet weather and requires organic debris, such as flower parts or senescing tissue, to initiate growth. Late attacks start from the senescent petals and head bracts and may be serious during a wet fall and late harvesting. Head rot symptoms start as brown spots on the back of the head, which is identical to the initial symptoms of all head-rotting fungi. These spots become covered with gray powdery *Botrytis conidia*, giving the head a "fuzzy" appearance. These spores generally form on the surface tissues and not inside the tissues, as with *Rhizopus*. In wet weather, the infection spreads throughout the tissues, and the head becomes a rotten, spongy mass.

Bacterial head rot, as with bacterial stalk rot, is caused when windblown or rain-splashed bacteria fall on wounds in the head caused by hail, insects or birds. No fruiting structures are associated with bacterial diseases, but the putrid odor associated with bacterial rotting is distinctive enough for identification.

Damage: Disease incidence for all three described head rots is generally low throughout the northern Great Plains, while Rhizopus head rot is common in the High Plains. Individual heads affected by any of the head rots will have lighter test weight seed, lower oil content and reduced seed yield. In severe cases, the affected heads may be entirely lost. Seeds from heads infected by any of these fungi or bacteria will have higher free fatty acid content, resulting in a bitter taste. Thus, head rots of confection sunflower may cause losses due to lowered quality factors, even in cases where actual seed yield losses are minimal.

Management: Insect control may help minimize *Rhizopus* head rot, but will not offer as much disease reduction as fungicide sprays. In the U.S., no fungicides are registered for control of any head rot, except Sclerotinia. No practices are recommended to control bacterial head rot, other than to minimize head-feeding insects that might transmit the bacterium.

Figure 98. Rhizopus head rot is characterized by a dark brown, peppery appearance of tissues in the receptacle. (T. Gulya)