DISEASES

EARLY SEASON DISEASES

Downy Mildew

Downy mildew is capable of killing or stunting plants, reducing stands and causing severe yield losses during wet years.

Host Crops
Sunflower

Biology

Downy mildew is caused by a soil-borne, wind-borne and seed-borne fungi *Plasmopara halstedii*, which can survive for up to 10 years in soil. Under cool, water-saturated soil conditions, the spores germinate upon contact with sunflower roots, enter, and spread throughout the entire plant. This is systemic infection. Systemic infection occurs only when the roots are less than 2 inches long when they contact the fungi. Sunflower plants surviving the initial infection will produce white spores on the underside of chlorotic areas on leaves. The white spores are air-borne, and upon landing on sunflower leaves may cause secondary infections. Secondary infections are most common when sunflower leaves remain wet for prolonged periods of time. Plants are susceptible to secondary infections for a much longer period of time versus systemic infections via root infection. As infected plants rot and are tilled into the soil, the fungus forms the resting stage which will germinate during favorable conditions in subsequent years.

Figure 38. Seedling Sunflower with downy mildew.

Figure 39. Downy Mildew causes stunting and heads to face straight upwards.
Symptoms
Symptoms can vary considerably depending on amount of inoculum, the age and host organ attacked and environmental factors. Symptoms can be broadly classified as those caused by systemic or localized infections. Root infection of seedlings or young plants results in systemic infection. Severely infected plants may die before or shortly after emergence or in the seedling stage. Typical systemic symptoms (Figure 38) in seedlings include dwarfing, yellowing of the leaves and the appearance of white, cottony masses on the lower and sometimes upper leaf surface during periods of high humidity. When seedlings are infected several weeks after emergence, or a fungicide seed treatment inhibits rather than prevents infection, the plants start showing symptoms at the four-, six-, or eight-leaf stage; this is termed ‘delayed systemic infection’. These plants may or may not develop typical downy mildew leaf symptoms, but are typically stunted with thickened, club like roots. The heads of the few plants reaching maturity face straight upwards and seldom produce viable seed (Figure 39). Airborne downy mildew spores can cause localized small, angular foliar lesions with the white fungal growth on the underside of the leaf. These infections generally have minimal impact on yield.

Yield losses may be substantial. If infected plants are dispersed randomly throughout the field, yield losses probably will not be observed unless infection exceeds 15 percent. Neighboring plants can compensate for severely infected plants by growing larger heads. When the disease is in a localized area, such as a low spot in a field and all plants are infected, yield loss can be substantial.

Scouting Techniques
Infected plants can most often be found alone or in standing water.

CONTROL TIPS
Resistant varieties are available; however, due to the development of new races, resistance may not be a sufficient management tool in all fields. Fungicide seed treatments can be an effective management tool for downy mildew, but as with genetic resistance, the pathogen has developed resistance to metalaxyl and mefanoxam, two of the most commonly used fungicides. Other seed treatment options are available and more will be labeled in the future. Refer to the current issue of the Guide to Crop Protection for updated information on products and rates for application. Other management procedures are to control weed hosts which include wild and volunteer sunflowers to help reduce inoculum.
FOLIAR DISEASES

Rust

Early infection of susceptible varieties can decrease head size, seed size, oil content and yield. Confection hybrids are more susceptible than oil-seed types.

Hosts

Wild sunflower and cultivated sunflower

Biology

Sunflower rust is caused by *Puccinia helianthi*. Infection can occur at any time during the growing season, depending on the inoculum source and environmental conditions. Conditions favorable for infection are free water on the leaves, either from rainfall or dew, and warm temperatures. A minimum of two hours of wet leaves is sufficient for rust infection; six to eight hours of leaf wetness results in the maximum amount of infection. Sunflower rust spores overwinter on the debris from infected sunflower leaves and stems of previous crops. In the spring spores germinate to infect volunteer seedlings, wild sunflower or new young plants in nearby fields. Under favorable conditions, spore production and infection can occur continuously within a year. The ‘repeating stage’ is the most damaging, as multiple waves of spores are spread by wind to other fields as conditions allow. As the crop ripens, the spores form the overwintering stage which can then re-infect following sunflower crops.

Symptoms

The first signs of rust usually appear when sunflowers are at or past bloom as environmental conditions within the crop canopy are more favorable for infection. The aecial stage (Figure 41) of rust appears late spring to summer as clusters of orange cups. The most common stage of rust (uredinia) (Figure 42) is often observed within two weeks of the aecia. These pustules are small (0.1 to 1 mm), cinnamon-brown, can be rubbed of easily and occur on both the upper and undersides of leaves. As the disease progresses, uredinia may be found on the upper leaves, stem and bracts of the sunflower plant. In response to temperature, the uredinia convert to telia (black spots on the upper surface of leaves) at the end of the season which do not rub off and are the overwintering structures.

Economic Threshold

Rust severity on the upper four leaves is 3 percent or greater.
CONTROL TIPS

The most effective way to avoid loss from rust is by planting rust-resistant hybrids. Most oilseed and confectionary hybrids have good to excellent resistance to most races of rust. Rotation to other crops is also a useful tool to minimize infections, as sunflower rust only infects sunflowers. If possible, avoid planting next to a field that had sunflowers last year; manage wild sunflowers or volunteer sunflowers as they are hosts for the disease. Early seeding and short season hybrids can also help minimize disease severity. Any cultural practice which fosters a dense canopy, such as high plant stands and high nitrogen fertilization, which traps dew, increases chances of a severe rust infection and should be avoided if risks are high. Foliar fungicides are registered for rust. Consult the current edition of Guide to Crop Protection for products.

Figure 41. Aecia cups on the underside of sunflower leaf.

Figure 42. Rust uredinia develop on the under-and upper-leaf surfaces.
Alternaria leaf spot is a ubiquitous disease on senescing leaves and generally of little concern. Under warm and humid conditions, it can however become a serious defoliating and yield reducing disease.

Host Crops
Sunflower, safflower and cocklebur

Biology
Alternaria fungi overwinter on diseased stalks and may be seed-borne at low levels. Seedling blights caused by Alternaria may develop when sunflower plants emerge in rainy weather in Alternaria-infested soil. Alternaria spores are spread by wind and splashing water. The fungi require free water for 4 hours for infection to occur. Leaf symptoms are most frequently observed after flowering as the dense canopy is conducive to infection. Once established, the disease can develop quickly under favorable conditions. In western Canada, climate is not typically conducive for Alternaria epidemics and generally only the lower senescing leaves are affected.

Symptoms
Dark brown irregular spots with dark brown to purple borders and a gray center develop on leaves (Figure 43). The spots on young plants may have a yellow halo. Leaf lesions may coalesce, causing leaves to wither. Stem lesions begin as dark flecks then enlarge to form long, narrow lesions. The stem lesions often join to form large blackened areas which may result in stem breakage. The lesions are located randomly along the stem and not associated with a petiole.

CONTROL TIPS
Crop rotation and burying infested crop residue to hasten decomposition helps minimize Alternaria infection.

Figure 43. Alternaria leaf lesions close up.
**Septoria Leaf Spot**

Septoria is widely distributed on sunflowers but usually causes little damage. In severe instances it can cause defoliation of the lower leaves.

**Host Crops**

Cultivated and Wild Sunflower

**Biology**

Septoria is caused by the fungi *Septoria helianthi*. The fungi survive on infected crop residue and can also be seed-borne. The disease can appear any time during the growing season with initiation favored by moderately high temperatures and abundant rainfall.

**Symptoms**

Septoria develops first on the lower leaves and spreads upwards through the canopy. The lesions (up to 15 mm in diameter) begin as water-soaked areas which are greasy green in appearance. The spots become angular to diamond shaped, with tan centers and brown margins. Young spots are often surrounded by a narrow halo that gradually merges with the surrounding green tissue (Figure 44). Mature spots contain small black specks or fruiting bodies. This is the best way to distinguish Alternaria from Septoria. The lesions may coalesce in later stages and the leaf may wither and die.

**CONTROL TIPS**

Crop rotation, incorporation of sunflower residue and use of clean seed are the best management practices to manage Septoria.

**Figure 44. Septoria lesions contain small black fruit bodies.**

**STALK AND ROOT- INFECTING DISEASES**

**Sclerotinia Wilt and Basal Rot**

Infected plants die rapidly, and if the plant dies prior to seed maturity it results in yield loss, lower test weight, and lower oil content.

**Host Crops**

Sclerotinia has a very wide host range of over 360 species, which includes sunflowers, canola, mustard, dry beans, field peas, lentils and potatoes.
Biology

Sclerotinia overwinters as *Sclerotinia sclerotiorum* in the soil or on plant debris. Sclerotia are irregularly shaped structures which range in size and shape from spherical and 1/8 inch in diameter to Y-shaped and up to 1 inch in length. The sclerotia bodies can survive in the soil for 5 or more years. As sunflower roots grow near sclerotinia in the soil, the sclerotia are stimulated to germinate producing mycelium which infects the lateral roots. Sclerotia form in the decayed stem pith and on the roots as the plant dies. The sclerotia are returned to the soil during tillage operations and serve as sources of inoculum for the next susceptible crop.

Soil moisture and temperature during the growing season are not critical factors affecting the rate of incidence of sclerotinia wilt.

Symptoms

Wilt (Figure 45) can appear at any time between emergence and maturity, but is more prevalent around flowering. Sudden wilt of the plant occurs when infected roots cannot uptake adequate water to meet the demands of the plant. Examination of the stem-root area will reveal a tan-brown, water soaked lesion at the soil surface (Figure 46). The stalks and roots may become covered with white mycelia and hard sclerotia bodies develop under very wet soil conditions.

CONTROL TIPS

Management of sclerotinia is difficult because of the wide host range, but rotation to cereals and corn is the most effective to minimize sclerotinia in the soil. Sunflower hybrids with some levels of resistance are available. The use of a mycoparasites which feed upon other fungi has shown to attack Sclerotinia. One commercially available is *Coniothyrium minitans* (Contans). This mycoparasite can kill sclerotia in several months rather than years.

Figure 45. Sclerotinia wilt.
Figure 46. Sclerotinia Basal rot lesion.
**Sclerotinia Mid-Stalk Rot**

Mid-stalk rot is the disease least often caused by Sclerotinia. Lodging can cause complete yield loss on a per plant basis.

**Biology**

Under wet soil conditions, the sclerotia in the soil can germinate to form small mushrooms called apothecia that produce air-borne spores. The spores can originate in the sunflower field or can be blown in from adjacent fields. Spores can move by rain splash, various insect carriers and up to 1 km by air. The spores require free water and senescing plant tissue to germinate and infect plants. Mid-stalk infection may result from leaf infection or infection at the leaf axil.

**Symptoms**

Infection can occur anytime from seedling to maturity depending on the presence of infecting spores and favorable environmental conditions. Mid-stalk rot begins with infection of the leaf, and the fungus progresses internally through the petiole until it reaches the stem. The leaf lesions are not unique enough to identify the fungus, but the stem lesions are identical to that formed by root infection; tan colored and water-soaked (Figure 47). The sclerotia can develop within the stem or on the exterior of the stem. Leaves above the lesion wilt, and the stalk eventually disintegrates becoming shredded as only vascular components of the stem remain (Figure 48).

**CONTROL TIPS**

The comments made about Sclerotinia wilt also apply to mid-stalk rot management. Cultural practices to avoid high plant densities by reducing populations and high nitrogen fertilization help lower the incidence of infection. Shallow or zero-tillage practices may aid in faster deterioration as sclerotia are left on the ground surface and subject to increased environmental deterioration. Resistance genes have been the most effective approach, and breeding for higher tolerance continues.

Figure 47. Sclerotinia mid-stalk rot lesion

Figure 48. Sclerotia bodies develop within the stem tissue or on the surface of the stem.
Phoma Black Stem

Phoma is the most widespread stalk disease in the northern Great Plains, but yield losses are considered minimal.

Host Crops
Cultivated sunflower

Biology
The fungus (*Phoma macdonaldii*) overwinters in infected debris. Phoma infection occurs throughout the growing season; however, it is not usually noticed until the stem lesions become obvious later in the summer. Leaves can be infected by wind carried spores, rain splashed spores or spores transmitted by insects. Stem weevils are suspected in transmitting the fungi internally and externally. Adult weevils feeding on the leaves cause leaf lesions whereas contaminated larvae spread the fungus as they tunnel throughout the stem. Leaf lesions are not distinctive and can be confused for Alternaria. The leaf infections progress down the petiole to the stalk. Under favorable conditions, the leaf wilts, the petiole turns uniformly black, and the stem lesions expand.

Symptoms
Large, jet black lesions develop on the stem (Figure 49), sometimes reaching about 4 to 5 cm in length. The lesions are uniformly black and shiny with definite borders. Small circular fruiting bodies of the fungus are produced on the surface of the stem but these require a hand lens to see. The fungus may also produce lesions on the back of the head, on the leaves, and at the base of the stalk. Stem lesions do not result in pith damage or lodging and are generally regarded as superficial lesions. If stem weevil larva tunneling spreads Phoma spores within the pith, extensive pith degeneration can occur.

CONTROL TIPS
Cultural practices to minimize Phoma includes a four year rotation to minimize the inoculum load in the soil, delayed planting and avoiding high plant populations and high nitrogen fertilization. Control of stem weevils can help reduce transmission of the fungus but insecticide application is rarely economically justified. Some hybrids are more tolerant than others, but none are immune to the disease.

Figure 49. Phoma black stem lesion
Phomopsis Stem Canker

In recent years Phomopsis has become a very prevalent disease. Yield losses result from smaller heads, lighter seed and lodging due to weakened stems.

Hosts
Sunflower

Biology
The fungi (*Phomopsis helianthii*) overwinter on infected plant debris and spores are rain splashed or windblown onto leaves. The infection initiates at leaf margins of lower leaves, developing into a brown necrotic area bordered by a chlorotic margin. The infection spreads down through the veins to the petiole and finally to the stem. The symptoms are similar to those of Verticillium leaf mottle; however, with Verticillium veins remain green. Stem lesions usually do not appear until flowering. The disease is most severe under conditions of prolonged high temperatures and high rainfall.

Symptoms
First symptoms initiate on lower to middle leaves after flowering as necrotic spots with a chlorotic border on leaf margins. The stem lesions begin as a small brown sunken spot but enlarges rapidly becoming a large tan to light brown lesion or canker centered on a leaf petiole. Lesions are much larger than Phoma black stem, sometimes reaching 6 inches and brown rather than black. Black fruiting bodies (pycnia) form on infected tissue. Phomopsis causes extensive pith degradation and the stalk can usually be crushed under moderate thumb pressure. Phomopsis infected plants are more prone to lodging than Phoma infected plants.

CONTROL TIPS
Thorough incorporation of the sunflower stalks into the soil to bury residue and crop rotation can reduce disease incidence and severity. Crop residue left on the soil surface fosters development of Phomopsis. Hybrids resistant to Phomopsis, developed using parental lines from Europe where the disease is particularly severe, are commercially available.

Figure 50. Phomopsis Stem Canker
Verticillium Leaf Mottle

Verticillium can significantly reduce sunflower yield especially on lighter soils. Quality may also be affected through decreased oil content and seed size.

Host Crops

Various plant species can be infected including sunflowers, potatoes, alfalfa and sweet clover

Biology

The fungus (*Verticillium dahliae*) is seed- and soil-borne. It survives as very small, black, resting bodies (microsclerotia) in diseased sunflower debris and broadleaved weeds, persisting for 5 to 10 years. The microsclerotia germinate in response to root exudates. The root tips of sunflower plants are invaded and, eventually, all parts of the plant become infected. The fungus produces toxins which are translocated throughout the plant causing the chlorotic and necrotic areas between the veins. When the plant dies, the fungus produces black fungal bodies that return to the soil with the plant debris.

Symptoms

Symptoms usually are not observed until flowering, but may appear at the six-leaf stage under severe conditions. Initial symptoms begin as tiny chlorotic flecks that increase in size with time developing into extensive inter-veinal yellowing (Figure 51). Symptoms begin on the lower leaves and progress slowly up the plant and may eventually infect all leaves. Affected leaves quickly become dry. The vascular system may be discolored brown, apparent as a ring around the pith in cross section. In severely infected plants, the pith is blackened with a layer of tiny black fruiting bodies.

CONTROL TIPS

Most oil-seed hybrids contain resistance to Verticillium whereas confection hybrids are generally more susceptible. However, a new strain has recently been identified in Canada and the U.S. which is able to overcome the source of resistance in these varieties. The disease is more severe on lighter land with a history of sunflower cropping than on heavy, clay soil. Rotation is important on lighter soil with a history of sunflower cropping to reduce inoculum load in the soil.

Figure 51. Verticillium Leaf Mottle
HEAD ROT AND DISEASES OF MATURE PLANTS

Sclerotinia Head Rot

Head-rot is considered the most important disease affecting sunflower production, causing yield and quality loss.

Hosts

Sunflower, canola, mustard, dry beans, field peas, lentils, potatoes

Biology

Sclerotinia head-rot (Figure 52) is quite variable, usually occurring late in the season and influenced primarily by the amount of rainfall from flowering through to harvesting. As with sclerotinia mid stalk rot, head-rot is caused by air-borne spores produced by apothecia (small mushrooms) either within the field or blown from a neighboring field. The spores require free water and a food base such as dead or senescing plant tissue to germinate and infect. Ascospores colonize the dead florets and pollen on the face of the head. After infection it takes several weeks until the appearance of brown lesions on the back of the head.

Symptoms

The first symptoms of head rot usually are the appearance of water-soaked spots or bleached areas on the fleshy back of the head. The fungus can decay the entire head, with the seed layer falling away completely, leaving only a bleached, shredded vascular system interspersed with large sclerotia. The bleached, skeletonized heads resemble straw brooms and are very obvious in the field even from a distance.

Yield loss from head rot on an individual plant can range from minimal to total loss if the head disintegrates and drops all the seed to the ground prior to harvest. Intact but diseased heads will have light and fewer seeds, with lower oil content and will shatter during harvest.

CONTROL TIPS

Sunflower hybrids exhibit variable range of susceptibility to head rot, but no hybrids are commercially available with adequate resistance. Fungicides may help reduce the incidence of head rot, with applications made preventively since several weeks lapse from infection to symptom development. Consult the current edition of the Guide to Crop Protection for product information.
**Rhizopus Head Rot**

Rhizopus is a very widespread disease in the central Great Plains. Affected heads will have a lighter test weight, lower oil content and reduced seed yields.

**Host Crops**

Many hosts for Rhizopus including sunflower, beets and tobacco

**Biology**

Susceptibility of the head increases from the bud stage up to the full bloom and ripening stages. Spores are disseminated by wind, rain and insects. Rhizopus enters the head through wounds caused by hail, birds, insects and has been associated with sunflower midge damage. Rapid disease development occurs in warm, humid weather. Once the head is fully colonized and all tissue killed, the head dries up and becomes ‘mummified’.

**Symptoms**

Initial symptoms are similar to other head rot diseases; brown, sunken, water soaked lesions on the back of the head. As the lesions enlarge, the interior of the head becomes mushy (Figure 53). The interior of the head becomes filled by mycelium interspersed with black fruiting bodies resembling pepper grains. Once the head dries up it is characterized by a dark brown, peppery appearance of tissues in the receptacle and becomes very hard.

**CONTROL TIPS**

Controlling head insects is the most efficient method to minimize incidences of Rhizopus head rot. Genetic resistance has been identified, but severity of the disease has not warranted intensive research. Rotation does not help reduce the incidence of Rhizopus due to the number of Rhizopus species. No fungicides are registered for control of Rhizopus head rot.

*Figure 53. Rhizopus Head Rot*