DUPONT PIONEER FIELD FACTS

Sclerotinia Diseases in Sunflower

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Causal Organism, Importance and Geographical Distribution

Sclerotinia diseases are caused by the soil-borne fungal pathogen *Sclerotinia sclerotiorum* which is often called the "white mold" fungus. This pathogen is a serious threat to sunflower production all over the world. It is probably the most common and widespread pathogen affecting sunflower globally. The fungus can infect the sunflower plant (root, stem and head) during all stages of plant development resulting in several types of symptoms. Thus, *S. sclerotiorum* causes two disease phases (Sclerotinia Stem Rot on the one hand, and Mid-stalk Rot and Head Rot on the other hand), depending on the mode of germination of the overwintering sclerotia. Myceliogenic germination produces mycelia that infect the roots and the basal part of the stem along the soil surface causing root rot, basal stem rot, and wilt of the sunflower plant. Carpogenic germination of the sclerotia produces apothecia and ascospores, which become airborne and infect sunflower heads and stems, causing head rot, mid-stem infection, and breakage. Depending on the environmental and climatic conditions, one disease can be promoted more than the other. Stem rot finds good conditions of infection under cool to moderately warm and wet weather conditions, while head and mid-stalk rot is favored in warmer and wet environments. *S. sclerotiorum* is a cosmopolitan pathogen that can infect more than 360 plant species belonging to 25 genera with major hosts in the Cruciferae, Leguminosae and Solanaceae families. The vast majority of susceptible species are broadleaf plants (Dycotyledons). Among the agronomic species of importance are soybeans, dry beans, lentils, peas, potatoes, canola and sugar beets, as well as numerous vegetable crops such as cabbage and carrots. Many broadleaf weeds are also susceptible and support reproduction of the fungus. The impact of Sclerotinia diseases varies from year to year and region to region based on soil moisture levels and environmental conditions. Depending on the year, root rot or head rot can be more commonly found and the associated yield penalties can vary depending on the weather conditions. It is assumed that head rot is most damageable with yield losses of 10-20% reported in most years. However, epidemics with up of 80% of plants affected have been observed.

Symptoms

Sclerotinia Stem rot

A water-soaked lesion develops at the base of the plant. The lesion becomes a grayish green to brown canker that often girdles the stem. As decay progresses, the stalk becomes bleached and has a shredded appearance. The decayed portion may extend a foot or more up the stem. Generally speaking, wilting is first observed in fields immediately prior to flowering. The pith is decays at the base of the stem and, black resting bodies called sclerotia (about 1/8 to 1/4 inch in diameter) can be observed. The sclerotia are produced by the pathogen and provide a positive identification of the disease. Infected plants lodge easily. During wet weather, white mycelium (mold) often develops at the base of the stem, hence the name "white mold". Figure 1 shows the typical symptomatology of Sclerotinia stem rot at different stages.

Figure 1. Symptoms of Root Rot Sclerotinia showing the initial infection in the base of the stalk with the presence of mycelium, first formation of sclerotia, wilting of plants and the appearance inside of the stalk full of sclerotia

Seed Metering

Sclerotinia Middle Stalk Rot

Middle stalk rot is usually observed around flowering time in the middle to mid-upper portion of the stalk. It begins as a brownish to grey water-soaked lesion, most commonly found at

or near the leaf node. A canker develops around the stalk, and the decayed tissue often has a wet, pulpy consistency. Usually the stalk falls over at the point of decay and the tissues above the canker die. Often a dense white mycelium and some sclerotia will be produced both inside and outside the stalk, especially during wet weather. Eventually the affected tissues become bleached and have a shredded appearance. Sometimes a leaf will be infected with *Sclerotinia* and the fungus will cause decay of the petiole and eventually reach the stem to cause middle stalk rot (Figure 2).

Figure 2. Symptoms of Midstalk Rot Sclerotinia showing initial infection in the stem from the petiole, breakdown of the stem and the presence of sclerotia when the plant is dead and dry

Sclerotinia Head Rot

The first symptoms of head rot are usually either the appearance of a white fungus mycelium growing over the flower parts or the development of water-soaked spots on the receptacle (the fleshy part of the head). The fungus grows profusely within the receptacle, causing its decay, and produces white mycelium and many large black sclerotia. The receptacle is usually bleached and is easily distinguished from a healthy head. The entire receptacle can rot and the seed layer falls away, leaving only a bleached, shredded skeleton which consists of vascular bundles interspersed with large sclerotia. These bleached skeleton heads are very obvious in the field, often from a distance. When harvested, infected heads often just shatter and all remaining seed is lost. During wet weather the fungus will grow over the seeds and form large net-like sclerotia which can cover the front of the head. The seeds usually do not decay but many are empty. The large sclerotia in the heads are harvested along with the seed. Sclerotia mixed in with seed confirm that a field had head rot. Figure 3 illustrates the characteristic Sclerotinia head rot symptoms.

Disease Life-Cycle and Epidemiology

S. sclerotiorum survives in the soil during the wintertime as sclerotia. They are hard, black to brown, irregular-shaped fungal bodies that are produced during the fungal colonization of plant tissue and remain viable in the soil for many years (Figure 1, 2 and 3 show sclerotia). The sclerotia are involved in the infection through two mechanisms:

- Sclerotia can germinate directly, producing the typical white mycelium that is characteristic for the disease Sclerotinia Stem Rot or Sclerotinia Wilt.
- Sclerotia can produce apothecia, which in turn can produce air borne spores which initiate the diseases Sclerotinia Head Rot and Middle Stalk Rot.

Figure 3. Symptoms of Head Rot Sclerotinia starting from the apothecia formation, initial infection in the back of the head, the infection is progressing, it can cover all the head observing the classical white mycelium of the fungus, it forms the sclerotia and the head can be completely destroyed

Figure 4 presents the life cycle of *S. sclerotiorum* showing the different diseases caused by the pathogen. More detailed information about the disease cycle can be obtained from this website: http://www.sclerotia.org/lifecycle/

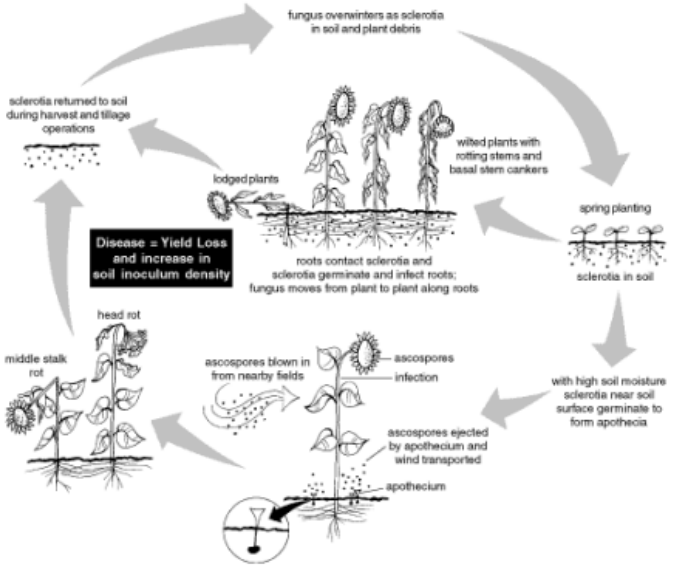

Figure 4. Disease life cycle of Sclerotinia wilt, middle stalk and head rot of sunflower

Sclerotinia Wilt

Upon contact with sunflower roots in early summer, the sclerotia germinate underground by producing the typical white mycelium. It directly penetrates the root, and then grows up the lateral root into the main tap root. The mycelium also grows down the other lateral roots, eventually causing decay of the entire root system. Contact between the roots of adjacent plants allows the fungus to spread from plant to plant. The root to root spread is especially important in sunflowers due to the shallow root system. The fungus generally does not move between rows. The first signs of Sclerotinia wilt appear when plants are starting to flower. However, the majority of the wilted plants are observed after flowering and commonly found in clumps (because of the root to root transmission of the fungus). The

time between the first sign of plant wilt and plant death may be as little as 4-7 days. After a short time of mycelium growth, hard black structures called sclerotia are produced. The most important factor impacting the incidence of Sclerotinia Wilt is the concentration of sclerotia in the soil. Research carried out in the USA has described that an inoculum density of 1.0 sclerotia per liter of tillage layer soil can result in about 65% wilted plants.

Sclerotinia Middle Stalk Rot

In addition to infecting roots through direct germination (by means of mycelium), sclerotia produce airborne spores through a mushroom-like structure called apothecia resulting in Middle Stalk Rot symptoms. At the end of the spring or beginning of summer, after canopy closure, sclerotia produce apothecia if the soil is very wet for 7-14 days. Apothecia development is favored by dense canopies and management practices that maintain high soil moisture such as irrigation. The apothecia then continuously produce ascospores for up to a week if the soil moisture remains high. The spores are forcibly ejected into the air in large numbers, and carried by the wind onto other sunflower plants. After the ascospores land on leaves, petioles and stems of sunflowers, they can remain viable for up to a week depending on temperature, relative humidity and shading from UV light. Temperatures above 25ºC and humidity above 35% decrease survival time. Ascospores require a film of water and a food base to germinate and grow before infecting the tissue. They may also penetrate through wounds caused by insects or hail. On the stem, the ascospores come in contact with food and water in the stem-leaf axil and infection occurs. Substances like sucrose produced in glands on leaves, petioles and stem may provide the needed nutrients for leaf infection. The dependence on free moisture for leaf and stem infection is the major reason that middle stalk rot occurs only sporadically. Serious outbreaks do occur in wet growing seasons or under irrigation. This mode of infection can also produce another symptomatology in the terminal bud. It has been described frequently in France. Since infection can lead to destruction of the terminal bud, and thus of the capitulum, there can be important yield loss.

Sclerotinia Head Rot

The infection of the sunflower head follows the same mechanism as the middle stalk rot. Ascospores are the infection agent and the same environmental conditions (water film and nutrients) promote infection. Therefore, it normally occurs only sporadically unless the growing season is very wet. In the case of head rot, ascospores use the flower parts as a nutrient base to begin growth. They penetrate the receptacle and cause decay of the entire head. The pathogen can infect the seed and exists as a mycelium in the seed coat, but infected seed is considered of minor importance for the disease.

Control and Management

S. sclerotiorum is one of the most difficult pathogens to control. Extensive research has been devoted to continuously improve partial resistance but complete resistance to the pathogen does not currently exist. An integrated control program that includes the following is recommended: (1) use of hybrids with a good tolerance profile, (2) cultural practices that avoid high planting densities and a 3 to 4 year crop rotation with non-host crops, (3) timely application of fungicides, and (4) implementation of agronomic practices that can produce a deterioration of sclerotia on the soil surface. Circumstances and economic feasibility determine which combination of methods can achieve the desired control.

There is some evidence that minimum or reduced tillage keeps sclerotia on or near the soil surface which promotes their microbial degradation, whereas deep burial of sclerotia promotes their survival. Tillage operations can also redistribute the sclerotia throughout the soil which can actually increase disease incidence by creating a more uniform distribution of the fungus within a field. Crop rotation for 3-4 years with non-host crops reduces the number of sclerotia in the soil and minimizes the impact of root and stem base infections. Crop rotation may not have a major impact on head rot and mid-stem infections because airborne ascospores can blow in from nearby fields. High crop density and high nitrogen fertilization contribute to the formation of a dense canopy, which raises the humidity level and creates an environment favorable for carpogenic germination of sclerotia. Wide plant spacing allows air movement, thus drying the soil surface and reducing the chances for apothecial formation and ascospore viability.

Sources of partial resistance to Sclerotinia head rot and stalk rot have been identified and are continually incorporated into commercial sunflower hybrids. Hybrids with a low frequency of stalk rot are not necessarily the same as those with low frequency of head rot. Different genetic factors are involved in the root and stem base infection versus the head rot infection mechanisms. For more information about the hybrids with the Sclerotinia tolerance package most adapted to your area, please refer to your local DuPont Pioneer contact.

Foliar fungicide applications have limited efficacy against sclerotinia stem rot. However, when applied preventatively, foliar fungicides can be very effective against Sclerotinia head and middle stalk rot. Specifically, picoxystrobin shows very good efficacy against sclerotinia head rot**.**

Bibliography

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