University of Idaho Extension

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White mold of potatoes

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Introduction

White mold of potatoes, also called Sclerotinia stem rot, is prevalent in Idaho and the Pacific Northwest. The disease is caused by the fungus *Sclerotinia sclerotiorum* (Lib.) de Bary (Discomycetes, Helotiales).

White mold is favored by moist conditions and commonly occurs in fields with overhead irrigation, especially under a center pivot. Disease development is enhanced by agricultural practices that promote extensive canopy growth, keep humidity and free moisture in the crop canopy for extended periods of time, and reduce wind movement.

Symptoms

White mold symptoms begin as water-soaked lesions 14 to 20 days following row closure, depending on the cultivar and cultural practices. Lesions usually first appear in the intersections between the stem and branches or on branches and stems in contact with the soil. These become quickly covered with a white, cottony growth that can spread rapidly to nearby stems and leaves when moisture is present for several hours (figure 1).

As lesions expand, they girdle stems, causing foliage to wilt (figure 2). White mold is also often accompanied by bacterial stem rot, especially under wet conditions (figure 2). When conditions become dry, lesions dry out and turn beige, tan, or bleached white and become papery-looking. As infected tissue decays, hard, irregularly shaped resting structures called sclerotia form on the inside and outside of decaying tissue (figure 3). The sclerotia are usually 1/4 to 1/2 inch in diameter, start out white to cream-colored, and gradually turn black with age (figure 4). Stems are frequently hollowed out by the fungus, leaving a papery shell covering numerous sclerotia. The sclerotia are released from the papery shells and eventually fall to the ground as infected stems dry out and the host plant dies. The sclerotia can survive in the soil for several years.

Figure 1. White mold lesions (L) usually first appear on branches and stems in contact with the soil. These areas become covered with a white cottony growth (arrows) and wilt.

Figure 2. White mold lesions spread rapidly and can girdle stems, causing foliage to wilt. White mold is also often accompanied by bacterial stem rot, especially under wet conditions.

No stem rot symptoms are observed on below-ground tissues (roots, tubers, or stolons) even when stems are infected with white mold.

Figure 3. As infected tissue decays, hard resting structures called sclerotia (arrowheads) form inside and outside the decaying lesions (L). An apothecium (A) can be seen next to the infected stem.

Figure 4. In late spring, sclerotia germinate to produce small, pink to beige, mushroom-like discs called apothecia.

Disease cycle

White mold is caused by the soilborne fungus *Sclerotinia sclerotiorum*. This pathogen causes disease in more than 400 plant species. Alternate host species in Idaho include dry beans, peas, alfalfa, and some common weeds found in potato fields such as redroot pigweed, common lambsquarters, and nightshade.

Sclerotinia sclerotiorum overwinters from one growing season to the next as sclerotia and can survive in the soil for several years. Sclerotia require a conditioning period of cool temperatures before they can germinate. These chilling requirements are usually met during Idaho winters.

In late spring, sclerotia within 1 to 2 inches of the soil surface germinate, forming small, pink to beige, flat to cup-shaped, mushroom-like disks called apothecia (figures 3 and 4). Each apothecium then produces as many as 8 million ascospores (sexual spores), which are the primary source of inoculum in potato. Under favorable conditions of cool and wet weather, the ascospores are forcibly ejected into the air and can disperse throughout an entire field or to adjacent fields by wind and air currents. In Idaho and the Pacific Northwest, the peak period of ascospore release has been found to coincide with full bloom of potatoes.

Apothecia are frequently found in fields planted with winter wheat, even though this crop is considered a weak host for the fungus. However, apothecia are usually only found in wheat fields if a susceptible host such as potatoes or beans were grown in the field the previous year. Wind currents sweeping across these wheat fields to neighboring potato fields may carry ascospores and thus act as an alternate source of inoculum.

Ascospores are similar in size to pollen and are easily dispersed by air currents and trapped by potato blossoms. In high-humidity and low-air-movement environments, ascospores germinate to form hyphae, which infect flowers, leaves, and stems. As infected flowers, leaves, and stems senesce and drop off the plant, they fall to the ground or are trapped in the canopy. The fungus then grows from the senescing tissues onto healthy stems and leaves in the lower parts of the canopy, causing further infection.

Under warm and dry soil conditions, which are not favorable for the production of apothecia and ascospores, sclerotia may germinate and produce mycelia that grow and infect adjacent plant tissues within a radius of about 1/8 inch, occasionally infecting even potato plant stems that are not touching the soil surface. As healthy tissue is infected, water-soaked

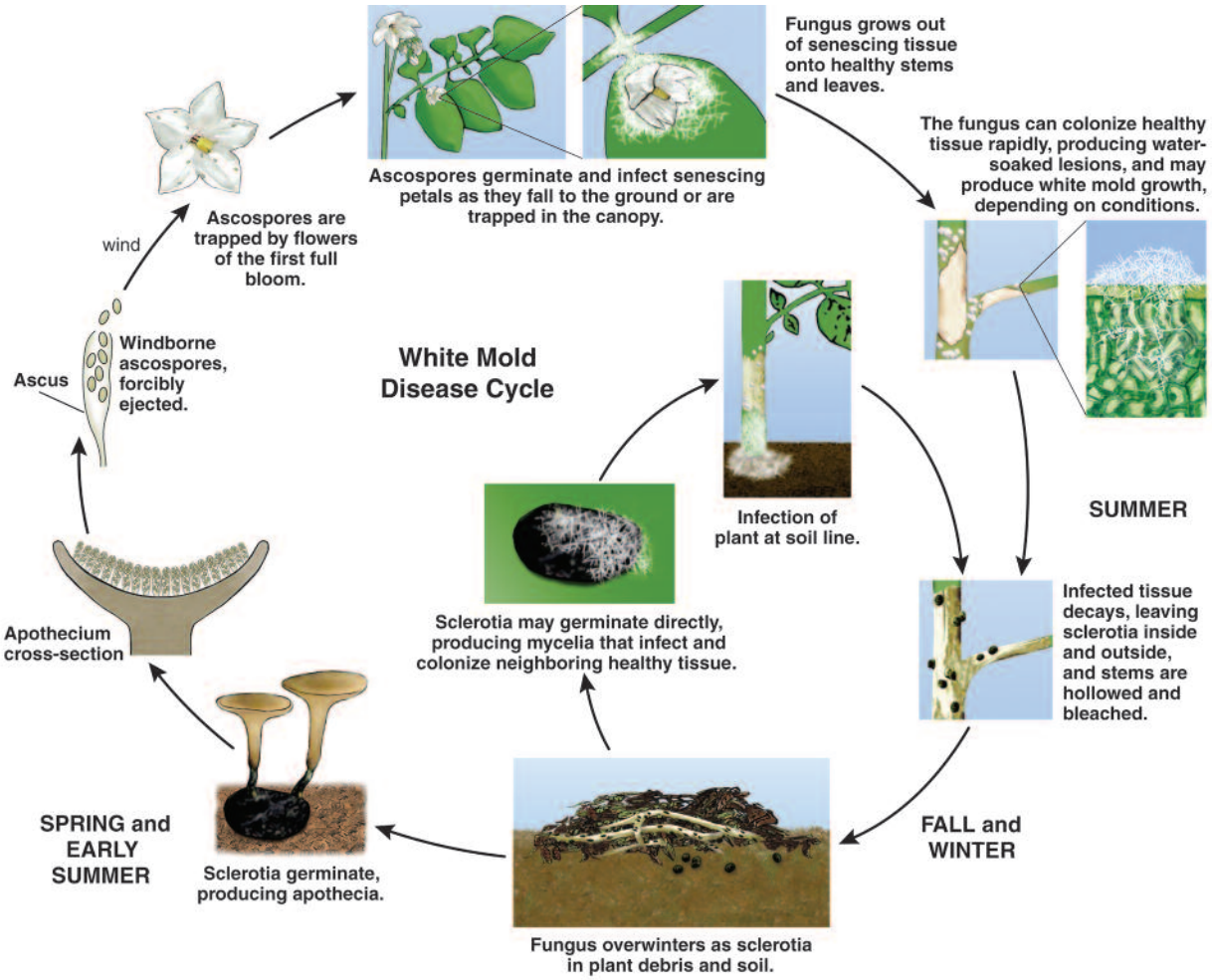

Figure 5. The disease cycle of the white mold pathogen, *Sclerotinia sclerotiorum.*

lesions with white, cottony growths appear, and sclerotia form in the decaying infected tissue. The disease cycle (figure 5) is repeated when a susceptible host grows in close proximity to the sclerotia. Although mycelia from sclerotia can infect potato foliage and stems directly, ascospores cannot, even under ideal conditions.

Monitoring and control

Effective management of white mold requires implementation of an integrated disease management approach. The disease has been controlled primarily through the use of cultural practices and foliar fungicides; however, biological control methods are available.

Cultural control

Cultural practices minimizing white mold in potato include eradication of weed hosts and crop rotation with nonsusceptible hosts such as corn or weak hosts such as small grains. *Sclerotinia* sclerotia can survive at significant inoculum levels for several years as they may be buried fairly deeply within the soil. During rotations to nonhost crops, sclerotia near the soil surface will germinate but, lacking a suitable host, the fungus will be unable to continue its life cycle. As such, the longer a field is out of potatoes the lower the white mold inoculum levels become. If a field has a history of white mold, avoid rotating to susceptible hosts such as beans or alfalfa as these rotations will increase the numbers of sclerotia in the soil.

Fertility management that prevents excessive canopy development will also suppress white mold. Cultivars that naturally produce thick, dense canopies that retain moisture are at higher risk of white mold than those that produce sparser canopies.

Proper irrigation management is critical in the control of white mold because the disease is favored by high humidity and free moisture in the crop canopy. Irrigation strategies that reduce humidity and free moisture in the canopy and allow the soil surface to dry will help decrease white mold. Irrigating in cool, cloudy weather should be avoided, and irrigation should be timed to allow plants to dry before nightfall where possible.

Biological control

Use of the biological control agent *Conithirium minitans*, a parasite of *S. sclerotiorum* sclerotia, to reduce the numbers of sclerotia in the soil has yielded conflicting results. In Wisconsin, foliar application of *C. minitans* to bean has significantly reduced white mold incidence in beans. In studies conducted at the University of Idaho, white mold incidence on potato plants sprayed with *C. minitans* was also significantly lower than on the nontreated controls. However, no reduction in apothecial numbers was observed in treated potato fields in the Columbia Basin of Washington state. Numbers of apothecia emerging in fields neighboring and at a distance from potato trial fields seem to be higher in the Columbia Basin than in Idaho, which may explain the differing Washington and Idaho results.

If applied prior to the planting of rotational crops susceptible to *S. sclerotiorum*, *C. minitans* is likely to reduce the in-field inoculum, especially if applications are repeated over a number of seasons. Use of this biological control agent is not compatible with fumigation because fumigation kills *C. minitans* as well as *S. sclerotiorum*. However, studies at the University of Idaho showed that it is compatible in a rotation where conventional fungicides are used to control white mold.

Deep tillage and hilling of fields will redistribute sclerotia across soil profiles, thus bringing to the top sclerotia that have not been exposed to the biological control agent. Therefore, it is essential to repeat the application of *C. minitans* over a number of years to reduce the sclerotia bank accumulated in the field. Even if a field is not intended for potato production, it may be of benefit to reduce the sclerotia bank because ascospores can migrate to neighboring and more distant fields and result in high disease incidence.

Chemical control

The most widely planted commercial potato cultivars are equally susceptible to white mold. In the absence of resistant cultivars, chemical control with fungicides remains the most effective management tactic. Omega (fluazinam) and Endura (boscalid) are effective fungicides that are registered for use against white mold in Idaho. Recently registered fungicides include Luna Tranquility (fluopyram + pyrimethanil), Aproach (picoxystrobin), and Vertisan (penthiopyrad). Application of any of these fungicides at initial full bloom is effective in reducing the number of infected stems. However, application of the same fungicides at or prior to row closure was found to be less effective.

For more information, please visit:
http://www.idahopotatodiseases.org

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