Extension Bulletin E-2989 • New • May 2007

MICHIGAN Potato Diseases

White Mold

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White Mold

Sclerotinia sclerotiorum (Lib.) de Bary
(Discomycetes, Helotiales)

Introduction

White mold, also called Sclerotinia stem rot, is prevalent in the Pacific Northwest, but in Michigan it is of secondary importance except in wet seasons or under excessive irrigation. The disease is enhanced by very moist conditions and is especially common in fields with overhead irrigation. Agricultural practices that promote extensive canopy growth and keep relative humidity and free moisture in the crop canopy for extended periods of time and reduce wind movement enhance disease development.

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 (Fig. 1). These become quickly covered with a white cottony growth that can spread rapidly to nearby stems and leaves if moisture is present for several hours. As lesions expand they can girdle stems, causing foliage to wilt (Fig. 2). White mold is also often accompanied by bacterial stem rot, especially under wet conditions (Fig. 2). When conditions become dry, lesions dry out and turn beige, tan or bleached white and papery-looking. As infected tissue decays, hard, irregularly shaped resting structures called sclerotia form on the inside and outside of decaying tissue (Fig. 3). Sclerotia are usually 1/4 to 1/2 inch in diameter, start out white to cream-colored and gradually turn black with age (Fig. 4). Stems are frequently hollowed out by the fungus, leaving a papery shell covering numerous sclerotia. Sclerotia eventually fall to the ground as infected stems dry out and the host plant dies. No stem rot symptoms are observed on below ground tissues – i.e., roots, stolons or tubers.

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

Disease cycle

White mold is caused by the soil-borne fungus *Sclerotinia sclerotiorum*. The pathogen causes disease in more than 400 plant species. Host plants in Michigan include dry beans, soybeans, alfalfa, peppers and tomatoes, as well as some common weeds associated with potato production, such as

Figure 2. White mold lesions spread rapidly and can girdle stems, causing foliage to wilt.

Figure 3. As infected tissue decays, hard resting structures called sclerotia (S) form inside (a) and outside (b) the decaying tissue.

lambsquarters, pigweed and nightshade. *Sclerotinia sclerotiorum* overwinters from one growing season to the next as sclerotia that can also survive in the soil for several years. Sclerotia require a conditioning period of cool temperatures before they can germinate, but these chilling requirements are normally met during Michigan winters.

In late spring, sclerotia within 1 to 2 inches of the soil surface germinate when the crop canopy shades the ground and soil moisture remains high for several days. After germination, small, pink to beige, flat to cup-shaped mushroomlike disks called apothecia are formed (Fig. 5). The fungus does not produce conidia (asexual spores) but ascospores (sexual spores) in the apothecia (Fig. 5). Ascospores are the primary source of inoculum in potato. Millions of ascospores (up to 8 million) are formed in each apothecium, and under favorable conditions of temperature and humidity (cool, wet weather), they are forcibly ejected into the air (Fig. 5). Ascospores are dispersed by wind and air currents and can be spread throughout an entire field or to adjacent fields. Apothecia frequently occur in winter wheat fields that are cropped after potatoes, beans or another susceptible host, and ascospores may be carried by wind currents from these fields to neighboring potato fields. In the Pacific Northwest, the peak period of ascospore release has been found to coincide with initial full bloom of potatoes. Ascospores are similar in size to pollen and, when being dispersed by air currents, can be trapped by potato blossoms. In high humidity and low air movement environments, ascospores germinate and infect these flowers and other contaminated plant parts as they senesce and drop to the ground or are trapped in the canopy. The fungus then grows out of the senescing tissues onto healthy stems and leaves in the lower parts of the canopy.

When warm and dry soil conditions are not favorable for the production of apothecia and ascospores, sclerotia may germinate directly, producing mycelia that grow and infect adjacent (within about 1/8 inch) plant tissues, occasionally affecting the crown area of potato plants early in the growing season. Though mycelium from sclerotia can infect the crown area of potato stems, ascospores are unable to infect potato foliage or stems directly, even under ideal conditions. As the fungus colonizes healthy tissue, it produces water-soaked lesions with white cottony growth, and sclerotia form in the decaying infected tissue as described above. The disease cycle is repeated when a susceptible host grows in close proximity to the sclerotia.

Monitoring and control

Effective management of white mold requires implementation of an integrated disease management approach. The disease can be controlled primarily through the use of cultural practices and foliar fungicides.

Cultural control

Cultural practices such as eradication of weed hosts and crop rotation with non-susceptible hosts such as corn or weak hosts such as small grains will help minimize Sclerotinia rot in subsequent potato plantings. However, *Sclerotinia* sclerotia can survive for several years in the soil, so it may be necessary to grow non-susceptible hosts in long rotations to reduce inoculum levels significantly. During these rotations sclerotia will germinate, but the fungus will not have a suitable host to infect and will not be able

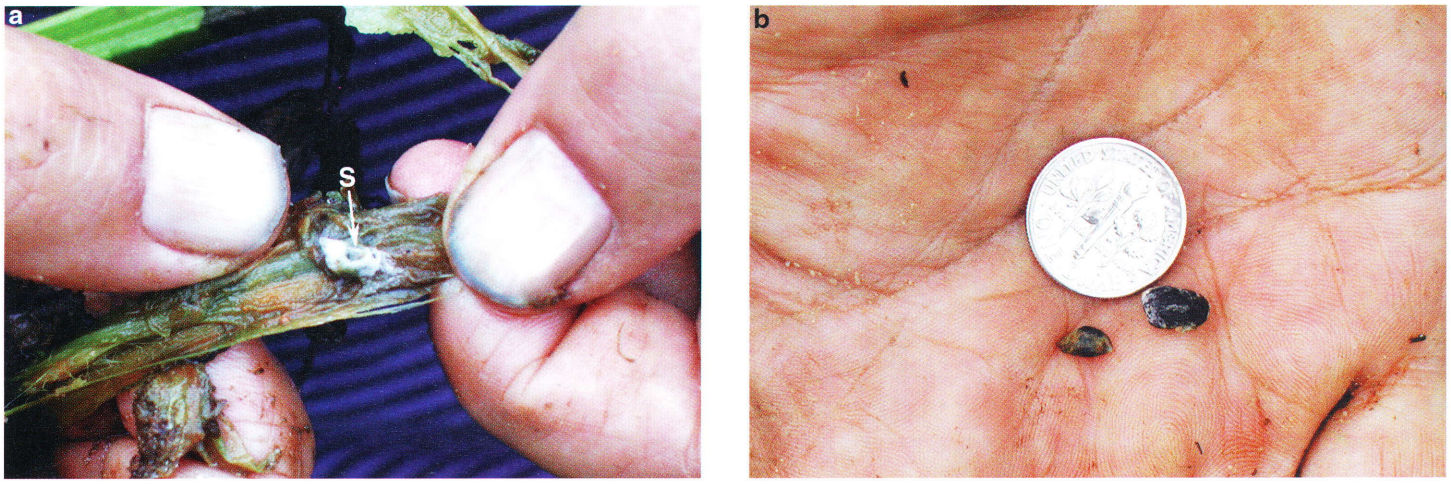

Figure 4. Sclerotia (S) are usually ¼ to ½ inch in diameter, start out white to cream-colored (a) and gradually turn black with age (b).

to continue its life cycle. If a field has a history of white mold, avoid rotating into susceptible hosts such as dry beans, soybeans, alfalfa or canola.

Good fertility management to prevent excessive canopy development will also suppress white mold. Cultivars that naturally produce thick, dense canopies are at higher risk of white mold than those that produce sparser canopies.

As this disease is favored by high humidity and free water in the crop canopy, proper irrigation management

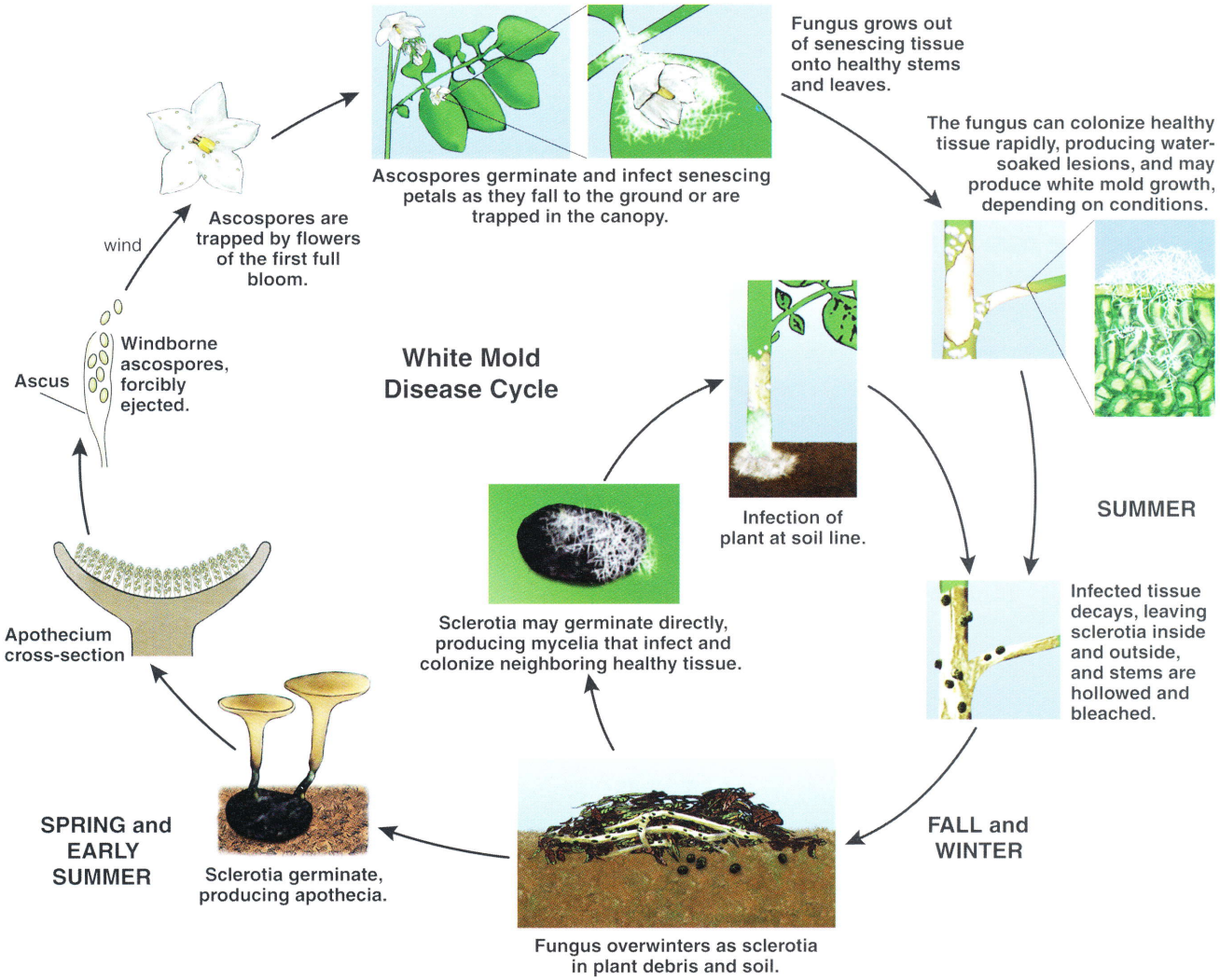

Figure 5. The disease cycle of the white mold pathogen, *Sclerotinia sclerotiorum*.

is a critical factor in dealing with potential white mold problems. 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 time to dry before nightfall. Cultural practices that help to prevent late blight disease development will also help in white mold management.

Biological control

Use of the biological control agent *Conithirium minitans*, a parasite of *S. sclerotiorum* sclerotia, to reduce the sclerotia bank in the soil has yielded conflicting results between the regions where the experiments were conducted. In Wisconsin, applications of *C. minitans* in bean fields have repeatedly reduced white mold incidence significantly on bean plants. Conversely, no reduction in apothecial numbers was observed in potato fields in the Columbia Basin of Washington state, so disease incidence was not affected. Furthermore, the migration of ascospores generated from apothecia emerging in neighboring and more distant fields to potato fields seems to be more substantial in the Pacific Northwest than in Wisconsin. Nevertheless, 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 the applications are repeated over a number of seasons. Use of this biological control agent is not compatible with fumigation. 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 cultivated commercial cultivars of potato are equally susceptible to Sclerotinia stem rot. In the absence of resistant cultivars, chemical control with fungicides remains the most effective management tactic. Effective fungicide products include Iprodione (a.i., iprodione), Botran (dichloran), Omega (fluazinam), Topsin (thiophanate-methyl) and Endura (boscalid). Field, greenhouse and in vitro experiments have shown no significant differences in the effectiveness of these compounds. Application of these fungicides at initial full bloom is effective in reducing the number of infected stems. However, application of the same fungicides made at or prior to row closure following label recommendations was found to offer erratic protection at best.

Photos, text editing, design and page layout by P.S. Wharton; illustrations by Marlene Cameron.
For more information, please visit: **http://www.potatodiseases.org**.

This publication is part of a new series of bulletins on potato diseases in Michigan. Funding for this publication was provided by Project GREEEN, MSU Extension, the Michigan Agricultural Experiment Station and the Michigan Potato Industry Commission.

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