

Potato White Mold

SLIDE 1:

This is Steve Johnson, University of Maine Cooperative Extension, bringing you this information on Potato White Mold. It is possible to receive a Maine Board of Pesticides Control recertification credit for this presentation. As this presentation is approximately a half hour, another presentation would also have to be viewed to get a credit. Additionally, a test must be passed with a minimum of 80 percent correct answers on each presentation. While there is no charge for viewing this information, there is a charge for taking each test, whether the tests are passed or not.

SLIDE 2:

Potato white mold is caused by *Sclerotinia sclerotiorum* (Lib.) de Bary. This fungus has a very wide host range.

SLIDE 3:

The fungus produces white and fluffy mycelium and also produces hard, black, irregularly-shaped sclerotia. The sclerotia are varied in size and range from very small to over one-half inch in diameter. The sclerotia are the resting stage and the pathogen overseasons in this form. Apothecia emerge from sclerotia and are about one quarter inch in diameter. Apothecia are formed at the soil surface and range from funnel-shaped to flat. Their colors include pale orange, pink, light tan, and white. From the apothecia, wind-dispersed ascospores are released and initiate primary infections.

SLIDE 4:

The white mold pathogen affects at least 148 genera of plants and includes over 400 species of plants. It is common on beans, peas, sunflowers, canola, and soybeans. To a lesser extent, white mold does affect potatoes.

SLIDE 5:

Some crops are more susceptible to white mold than others; dry beans are more susceptible than soybeans. Broadleaf crops are susceptible to white mold during the bloom stages as the flower petals are the site of primary infection.

SLIDE 6:

White mold has been reported to reduce yield by as much as 50 percent in some crops. In Maine, losses in potatoes never approach this level.

SLIDE 7:

Potato stem lesions are almost always associated with a previous food source for the pathogen. Flower petals are the most common site of primary infection for the white mold pathogen. The spore tends to need a food source to develop on before a potato stem is infected. Frequently, stem lesions are associated with parts of infected blossoms that may have fallen onto the stem. A white, cottony mycelium of the fungus may be observed on water-soaked lesions on the stems. Decaying stems, leaves, or leaflets that are infected by the pathogen can be associated with stem lesions.

SLIDE 8:

The lesions are commonly found in canopy where conditions are cool and moist. New lesions can occur when stem contact occurs with an existing lesion. As the disease progresses, the mycelium may not be apparent on the stems.

White mold symptoms in potatoes can be in the form of wilting of the affected stem. Oxalic acid is secreted by the pathogen, and causes opening of stomata leading to water loss and wilting. Wilting symptoms are difficult to accurately assess in the early stages of this disease.

SLIDE 9:

As white mold progresses, the lesions on the stem often will turn a bleached white. Often there is soft rot associated with the diseased stems. This may occur where the stem comes into contact with the soil.

A diagnostic characteristic of white mold is the presence of a small, black fungal structure called a sclerotium. This is the resting body of the pathogen and serves to allow the pathogen to persist from year to year. Sclerotia can be found inside affected stems. Tuber infection is rare.

SLIDE 10:

Potato stem lesions can also be associated with decaying leaves that fall onto the stems deep in the canopy as seen in this photograph. White mold lesions are starting to develop in several places along the center stem in this photograph.

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on the soil surface will germinate; 2 tenths of an inch below the soil surface, half will germinate; 4 tenths of an inch below the soil surface, a quarter will germinate; 7 tenths of an inch below the soil surface, 10% will germinate; and 9 tenths of an inch below the soil surface, only 5% of the sclerotia will actually germinate into apothecia.

Depth in soil (inches)	Emergence (%)
0.0	60
0.2	50
0.4	25
0.7	10
0.9	5

SLIDE 13:

Sclerotia germinate by producing apothecia, which are the cup-shaped structures holding the reproductive spores for the fungus, *Sclerotinia sclerotiorum*. They are produced when conditions of low soil temperature and high soil moisture occurs for 2 to 3 weeks. The warmer the temperature, the faster this occurs. You can see from the chart below:

Temperature (°F)	Days to Apothecia
50	58
59	42
68	25

SLIDE 14:

Most apothecia are produced faster under high light conditions than under low light or dark conditions. An increase in ground cover or shading increases the number of apothecia. Apothecia will produce spores for about a week. Increased apothecia numbers lead to increased ascospores which leads to increased white mold.

SLIDE 15:

Up to 100 apothecia can be produced per sclerotia and each apothecia can produce up to 2×10^8 per spores.

That's 200,000,000

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or

200 million spores

Photo by Dave Lambert, University of Maine

SLIDE 16:

The mean temperature in Presque Isle for July 2008 was 68.7°F; July 2009, it was 64.6°F; and in July 2010, it was 69.9°F. This gives a 3-year average of 67.7°F, which relates to a low of 57°F and a high of 78°F.

Apothecia germination is reported to occur from 320 to 1,814 GGD units (no base, °F) under high light. This is from 4 to 28 days at a mean temperature (GDD) of 67.7°F.

With low light, like under a canopy, apothecia germination is reported to occur from 1,400 to 3,128 GDD units (no base, F). This is from 22 to 48 days at a mean temperature (GDD) of 67.7°F.

What this means, that germination can occur from 4 to 28 days under high light or from 22 to 48 days under low light in a potato canopy situation.

SLIDE 17: This is a graph of the Mean Temperature (F) for July 2009 in Presque Isle. This is information relating to the apothecia germination from the previous slide.

SLIDE 18:

Once sclerotia produce apothecia, spores, called ascospores, are produced in asci, which are located in the apothecia.

Ascospores do not infect green potato tissue. They can infect blossoms and cause disease with a 25 percent relative humidity which is rather dry.

Ascospores don't dry out easily and can infect flowers after 6 days of drying or 12 days on a leaf or a petal. Temperatures above 71°F for 48 hours will kill the ascospores.

SLIDE 19:

As with any disease, there has to be a host, pathogen, and environment triangle--the host being a susceptible potato host, in this case; the pathogen being the *Sclerotinia sclerotiorum*; and the environment being the cool moist condition. The spores (ascospores) are released into the air. Free moisture

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and high relative humidity are required for ascospore germination and tissue colonization.

Because of this, symptoms are typically seen after row closure as relative humidity in the crop canopy will increase.

Flower petals, again as I have said before, are the most common site of primary infection for the white mold pathogen. The spore tends to need a food source to develop before a potato stem is infected.

SLIDE 20:

Prolonged flowering periods by variety characteristics or lowered temperatures will increase the window for infection by the white mold pathogen. This is more relevant for crops like canola than potatoes.

The temperature range for blossom infection is 59 – 86°F. Outside of this range, there is little blossom infection.

White mold development in the canopy can occur under wide temperature range from 41 to 79°F.

SLIDE 21:

Once infection has been established, the pathogen produces characteristic white cottony mycelium. The mycelium is capable of spreading within the canopy and inciting more disease.

Sclerotia are formed as a survival mechanism, so these are not often found until the food source is exhausted in the local area of infection or on the plant as a whole.

The process of harvesting the potatoes serves to shatter the potato stems, releasing the sclerotia from inside the infected potato stems. Sclerotia left on the soil surface appear to survive as well as those buried in the soil.

SLIDE 22:

White mold has not been well controlled with crop rotation. Usually four or more years of a nonhost must be grown to reduce inoculum levels.

Currently, the most effective method of controlling white mold on potatoes is by timely application of fungicides. Fungicides should be applied before

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the fungus attacks the plant and before visible symptoms are noticed. Applications at early bloom are more effective than those applied at late bloom.

Potato varieties differ in resistance to the pathogen. Superior is one of the most susceptible potato varieties in our area.

SLIDE 23:

Wheat, barley, and oats are nonhosts for the white mold pathogen. However, sclerotia in grain fields may produce apothecia and ascospores which can then travel to susceptible host crops. Ascospore dispersal can be as far as 3 miles but 90 percent of the dispersal is within 500 feet of the apothecium.

Studies from Washington State showed ascospores come from outside the potato planting rather than within the potato planting. Also, no deposition gradient was detected. Their conclusions were that inoculum from a source outside of the potato field is responsible for the white mold.

SLIDE 24:

Dense canopies with high humidity and over 1 inch of rain within a 1-2 week period before the onset of flowering with temperatures in the 60's and 70's are conditions that favor white mold.

SLIDE 25:

In some crops:

Increased rain accumulation in the first half of June leads to increased white mold later in the season.

Decreased temperature in July leads to increased white mold later in the season.

Increased number of rainy days in June has led to increased white mold later in the season, but there isn't a lot of consistency.

SLIDE 26:

Heavy rain early then dew later may encourage infections later on in the season.

The key elements for white mold field risk is:

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Field history of white mold. Has the field had white mold before and the rain accumulation two weeks before flowering?

SLIDE 27:

White Mold (*Sclerotinia sclerotiorum*) can be a greater problem with increased plant populations. Shown is data from soybeans planting densities:

Plant Population per acre	White Mold Percent Incidence
125,000	37
175,000	45
225,000	50
275,000	53

SLIDE 28:

As a general rule, the earlier the maturing the potato variety, the more susceptible the variety is to white mold.

When possible, avoid putting susceptible varieties into fields that have a history of white mold.

SLIDE 29:

Cultural Management of White Mold:
Rotation for nonhosts:

Usually four or more years of a nonhost must be grown to reduce inoculum levels.

SLIDE 30:

Chemical Management of White Mold:

White mold can be controlled with chemicals. Fungicides need to be applied at flowering. Some materials benefit from a second application 7 to 14 days later.

Starting applications after flowering has begun has resulted in reduced efficacy.

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Chemical Management of White Mold:

Shown here are some current recommendations for White mold:

Endura: 5.5 to 10 oz per acre

Omega: 5.5 to 8 oz per acre

If one application of fungicide is to be made:

Omega will out perform Endura.

If two applications are to be made:

Endura will out perform Omega.

It is simply a cost question.

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Chemical Management of White Mold:

With Endura:

If less than the full rate of 5.5 oz per acre are used, the control will drop off.

Omega will also control late blight.

Serenade has been tried. The controlled results have not looked promising.

There may be some new products on the horizon, but they are not registered as of today.

SLIDE 33:

The fungus *Coniothyrium minitans* is a hyperparasite of *Sclerotinia sclerotiorum*, the causal agent of white mold of potatoes. The hyper parasite produces 1, 3 glucanase which lyses *Sclerotinia sclerotiorum* sclerotia. In this slide, containers with *Sclerotinia sclerotiorum* sclerotia are shown. The photograph of the vial on the left has only sclerotia. They are the black structures on the bottom of the vial. The photograph of the vials on the right have *C. minitans* added to solution with the sclerotia. The vials have been shaken to show the varied states of sclerotia breakdown that this

hyperparasite of *Sclerotinia sclerotiorum* sclerotia can do. Control can be as erratic as the vials in the photograph on the right.

SLIDE 34:

A control product named Contans has the hyperparasite *C. minitans* as an ingredient. The use rate of Contans is at 1 to 4 pounds per acre. There are some things to keep in mind if you are choosing to use this product.

SLIDE 35:

The soil must be between 50°F and 80°F during the application.

If the soil is disturbed, the application must be repeated.

Water can serve to incorporate the material in the top soil layer, but the surface sclerotia are far more colonized than buried sclerotia. This is more than likely an early fall application rather than a spring application.

SLIDE 36:

The less plant residue on the soil surface, the better the control has been with this hyperparasite.

Again, best results have been reported when there is eight weeks between application and planting, likely not making a spring application, with the temperature requirements.

The material can be used to reduce soil contamination by killing overwintering sclerotia (mainly those in the top 1 inch of the soil).

SLIDE 37:

The pathogen hyperparasite can infect 90% of the sclerotia in the top layer and, therefore, reduce the apothecia by 90%.

Some studies have shown a 50% reduction in white mold with the use of this hyperparasite.

The control can be erratic.

SLIDE 38:

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Temperatures below 45°F have resulted in slow rates of sclerotia infection by *C. minitans*.

The optimal conditions for growth and development of *C. minitans* are 68°F and relative humidity above 98%.

SLIDE 39:

C. minitans survived in the soil for two years and spread to adjacent control plots and infected sclerotia within those plots.

In some studies, despite the fact that the inoculum potential of *S. sclerotiorum* was reduced by *C. minitans* treatment, no disease control was obtained where disease levels were low or where disease levels were high.

SLIDE 40:

The fall inoculations of *C. minitans* have resulted in negligible numbers of sclerotia remaining viable after one month.

Coniothyrium minitans – This was not effective in Washington State where more ascospores originated from outside the potato planting than from within the potato planting.

SLIDE 41:

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