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report on
PLANT
DISEASE

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WHITE MOLD OF TOMATO

White mold (also known as Sclerotinia blight), caused by the fungus *Sclerotinia sclerotiorum,* is a disease of tomato whenever cool conditions prevail during crop development. The pathogen causes disease in more than 360 plant species, including many vegetable crops.

SYMPTOMS

White mold is usually first noted on tomato about the time of flowering. Infection usually begins in leaf axils or in stem joints where flower petals are fallen. Water-soaked areas develop at these sites. The stems are subsequently invaded, which become light gray, and plants dye (Figure 1). During cool, moist weather, cottony mycelium is often evident on diseased stems. Sclerotia are produced on mycelia mat. They also readily form inside stems, assuming a shape elongated in the direction of the stem cavity (Figure 2 and Figure 3A). The fungus may also enter plants at the soil line, especially if senescent tissue is present. Mycelial growth accounts for some plant-to-plant spread, especially in cases of lush growth. Fruit may also be infected. Typically, infected fruit is gray and rapidly breaks down in watery rot. A ring of sclerotia often develops around the calyx.

Figure 1. White mold of tomato, caused by *Sclerotinia sclerotiorum.*

DISEASE CYCLES

The pathogen overwinters as sclerotia in soil. Sclerotia on the surface or in the top 2-3 cm of soil germinate and produce sexual reproductive bodies (apothecia). Each sclerotiorum produces one to several apothecia (Figure 3B). Apothecia are cup-shaped and white, yellow, or brown (Figure 3D). Cylindric-clavate asci are produced on apothecia (Figure 3E). Temperatures of 52-59°F (11-15°C) are favorable for the formation of apothecia. Ascospores are ejected from asci and are carried by wind currents onto the plants. Once sufficient fungal growth has taken place on the dead or senescent tissue,

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healthy stems are invaded. Long periods (16-72 hr) of continuous wetness are required for both ascospore infection of senescent tissue and subsequent infection of living plant part. Any condition that contributes to poor air circulation and the retention of moisture is likely to aggravate the disease. The disease intensity is higher in low-laying areas.

Figure 2. White mold (*Sclerotinia sclerotiorum*). Note white mycelium on stem (left) and sclerotia inside stem (right).

DISEASE MANAGEMENT

There is no host resistance to white mold of tomato. Every effort must be taken to avoid overly dense plant canopies, as poor air circulation aggravates white mold. Rotation with crops other than tomato, pepper, potato, and snap bean may help reduce levels of initial inoculum. Control of tomato white mold is based primarily on the application of effective fungicides prior to colonization of tissues. Application of a broad-spectrum fumigant is effective in destroying sclerotia in soil. However, with full-bed plastic mulch commonly used in the culture of fresh-marked tomatoes, there is a considerable amount of unfumigated soil between beds. Providing large space between the plants results in lower incidence of whit mold. For the up-to-date recommendation on fungicide use for control of tomato white mold, refer to the current edition of publication number C1373, "Midwest Vegetable Production Guide for Commercial Growers" (www.btny.purdue.edu/pubs/id/id-56/). This publication is available from ITCS, University of Illinois, 1917 S. Wright St., Champaign, IL 61820; or call 1-800-345-6087.

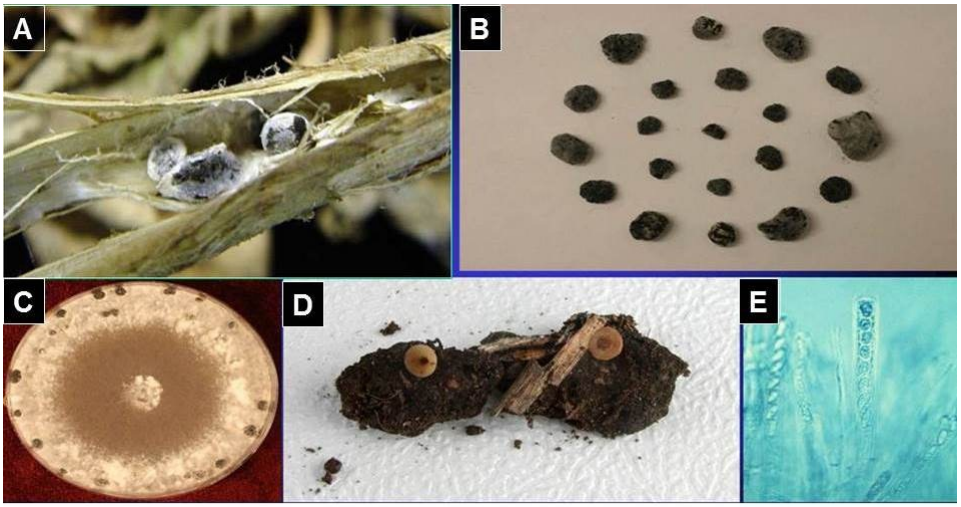

Figure 3. Tomato infection with *Sclerotinia sclerotiorum*: A, infected tomato stem; B, sclerotia of the pathogen; C, colony and sclerotia of *S. sclerotiorum* on potato dextrose agar; D, apothecia (courtesy C. A. Bradley); E, asci and ascospores (courtesy H. F. Schwartz).