Crop Knowledge Master **Fungi**

Sclerotium rolfsii

southern blight, southern wilt... (Plant Disease Pathogen)

Hosts Distribution Symptoms Biology Epidemiology Management Reference

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HOSTS

S. rolfsii has an extensive host range; at least 500 species in 100 families are susceptible. The most common hosts are the legumes, crucifers, and cucurbits.

Known hosts in Hawaii include: carnation (*Dianthus caryophyllus* L.), corn or maize (*Zea mays* L.), eggplant (*Solanum melongena* L.), florist's chrysanthemum (*Chrysanthemum morifolium* Ram.), ground cherry or poha (*Physalis peruviana* L.), okra (*Hibiscus esculentus* L.), beans (*Phaseolus* sp.), *Spathiphyllum* sp., sugar cane (*Saccharum officinarum* L.), sweet pepper (*Capsicum frutescens* L.), sweet potato (*Ipomoea batatas* (L.) Poir), sweet william (*Dianthus barbatus* L.), taro (*Colocasia esculenta* (L.) Schott), tomato (*Lycopersicon esculentum* Mill.), tuberose (*Polianthes tuberosum*), watermelon (*Citrullus vulgaris* Schrad.), and winter squash (*Cucurbita maxima* Decne.).

Other reported hosts (worldwide) include: alfalfa, amaryllis, artichoke, banana, bean, beet, Brussels sprouts, cabbage, canteloupe, carrot, cauliflower, celery, chrysanthemum, coffee, cotton, cucumber, delphinium, endive, escarole, garlic, ginger, gourd, iris, lettuce, mango, muskmelon, mustard, narcissus, onion, parsley, southern pea, peanuts, pineapple, potato, pumpkin, radish, rhubarb, soybean, squash, tobacco, tulip, turf (i.e., golf greens, bermudagrass and crabgrass), turnip, and yam.

The fungus persists in many weed hosts as well.

DISTRIBUTION

S. rolfsii commonly occurs in the tropics, subtropics, and other warm temperate regions, especially the southern United States, Central and South America, the West Indies, southern European countries bordering the Mediterranean, Africa, India, Japan, the Philippines, and Hawaii. The pathogen rarely occurs where average winter temperatures fall below 0 C.

SYMPTOMS

S. rolfsii primarily attacks host stems, although it may infect any part of a plant under favorable environmental conditions including roots, fruits, petioles, leaves, and flowers. The first signs of infection, though usually undetectable, are dark-brown lesions on the stem at or just beneath the soil level; the first visible symptoms are progressive yellowing and wilting of the leaves. Following this, the fungus produces abundant white, fluffy mycelium on infected tissues and the soil. *Sclerotia* of relative uniform size are produced on the mycelium: roundish and white when immature then becoming dark brown to black. Mature sclerotia resemble mustard seed. The fungus occasionally produces basidiospores (the sexual stage of reproduction) at the margins of lesions and under humid conditions, though this form is not common.

Seedlings are very susceptible and die quickly once they become infected. Older plants that have formed woody tissue are gradually girdled by lesions and eventually die. Invaded tissues are pale brown and soft, but not watery.

BIOLOGY

Synonyms of *Sclerotia rolfsii* include: *Athelia rolfsii* (Curzi) Tu and Kimbrough (Sexual stage) and *S. delphinii* (Synonyms for the sexual stage: *Corticium rolfsii, Pellicularia rolfsii*).

S. rolfsii grows, survives, and attacks plants at or near the soil line. Before the pathogen penetrates host tissue it produces a considerable mass of mycelium on the plant surface, a process which can take 2 to 10 days. Penetration of host tissue occurs when the pathogen produces an enzyme which deteriorates the hosts' outer cell layer. This results in tissue decay, further production of mycelium and the formation of *sclerotia.* The latter two rely upon favorable environmental conditions.

Sclerotia undergo either hyphal or eruptive germination. Hyphal germination is characterized by the growth of individual strands of hyphae from the sclerotial surface while eruptive germination is characterized by plugs or aggregates of mycelium bursting through the sclerotial surface. The quantity of mycelial growth and the energy needed for infection is dictated by the type of *sclerotial* germination that takes place. A food base of nonliving organic matter must be present for hyphally germinating sclerotia to infect host tissue because mycelial growth is sparse. However, mycelium from eruptively germinating *sclerotia* can infect host tissue without requiring an exogenous food base.

S. rolfsii is able to survive (and thrive) within a wide range of environmental conditions. Growth is possible within a broad pH range, though best on acidic soils. The optimum pH range for mycelial growth is 3.0 to 5.0, and *sclerotial* germination occurs between 2.0 and 5.0. Germination is inhibited at a pH above 7.0. Maxiumum mycelial growth occurs between 25 and 35 C with little or none at 10 or 40 C. *Sclerotial* formation is also greatest at or near the optimum temperature for mycelial growth. Mycelium is killed at 0 C, but *sclerotia* can survive at temperatures as low as -10 C. High moisture is required for optimal growth of the fungus. *Sclerotia* fail to germinate when the relative humidity is much below saturation. However, there are some studies which assert that sclerotia germinate best at relative humidities of 25-35 %. One review summed it up by stating that soil moisture studies are difficult to interpret. Mycelial growth and sclerotial germination occur rapidly in continuous light, though they will occur in darkness if other conditions are favorable.

Occasionally *S. rolfsii* has a sexual fruiting stage which develops on the margins of lesions and in locations that are shaded from the sun. Two or four thin-walled colorless spores are borne on short spines at the ends of slightly enlarged short threads. To what extent this stage aids in the reproduction and spread of the organism under field conditions is unknown. The spores are so light that if produced in large quantities they could be carried long distances in the air. This stage is not frequently seen in the

field and is not believed to be of primary importance in disease transmission.

EPIDEMIOLOGY

S. rolfsii can overwinter as mycelium in infected tissues or plant debris. It usually persists as *sclerotia. Sclerotia* are disseminated by cultural practices (infested soil and contaminated tools), infested transplant seedlings, water (especially through irrigation), wind, and possibly on seeds. In addition, a small percentage of sclerotia may survive passage through sheep and cattle, and thus, could be spread through fertilizers.

MANAGEMENT

NON-CHEMICAL CONTROL

Control of *Sclerotium* diseases is difficult and depends on a combination of cultural, biological and/or chemical methods. Good cultural practices include roguing, eliminating weed hosts, and avoiding crop injury during cultivation. A dense canopy increases disease incidence, thus increasing plant spacings can help keep infection down. A delayed planting date may also help reduce disease incidence if planting is timed so that the dense canopy forms after temperatures fall so that infection is not as likely. Also, keeping plant bases free of dead leaves (and weeds) will deny the pathogen a food source, helping to keep disease incidence down.

Crop Rotation:

Because *S. rolfsii* has such a broad host range, crop rotation has less of a chance of being successful as there are few resistant crops. There are some grasses and grains that are not as susceptible to the fungus that help in reducing soil inoculum levels. Onion is susceptible to *S. rolfsii,* however, some cultivars have been shown to reduce the viability of *sclerotia* when cultivars are planted in winter when temperatures are too low for disease development. A significant increase in yield and reduction in disease incidence was reported for summer peanut crops when appropriate onion cultivars were planted the previous winter. It has been postulated that onion exudates cause the pathogen to become susceptible to antagonistic microflora in the soil.

Plowing:

Deep plowing (at least 20 cm) with a moldboard extention inverts soil so that organic matter, sclerotia, and plant debris are buried at least 10 cm beneath the surface. This helps to eliminate inoculum when plowing occurs just prior to planting. Buried soil must not be re-surfaced during the growing season.

Amendments:

Compost, oat, or straw added to the soil has been shown to limit disease incidence. The addition of an amendment may increase populations of antagonistic soil microorganisms (see biocontrol section). This method may be reasonable for small-scale farms and greenhouses, but is probably not practical for large farms unless it is combined with crop rotation.

Soil Solarization:

Soil solarization or solar heating is a relatively recent method for controlling *S. rolfsii* inoculum. *Sclerotia* grown in vitro are still viable after 12 hours at 45 C, but are killed in 4-6 hours at 50 C and in 3

hours at 55 C. Covering soil with transparent polyethylene sheets during the hot season increases soil temperatures and kills *sclerotia* when the temperature under the sheets get hot enough for an appropriate length of time. Most field trials have achieved *sclerotia* degradation at 1 cm, but eradication at greater depths usually did not occur. In addition, this method requires immediate planting, which excludes crops that are planted in spring because temperatures are not high enough to affect *sclerotia.* Soil solarization combined with the addition of *Trichoderma harzianum* (a mycoparasite, see biocontrol section) has been shown to decrease disease incidence more than either treatment alone. However, the practicality of soil solarization is questionable. First, the length of time of solarization may be limited; a trial in Arizona reported that the tarps disentigrated after 6 weeks. Second, it is not known what affect solarization has on the existing soil microflora and what affect any microflora change would have on the crop. Third, it is not known what affect solarization has on non-target and/or temperature-tolerant pathogens.

Black Plastic Mulch:

Mulching with black plastic has been shown to reduce disease incidence and perhaps provide greater crop yields. Black plastic mulch (BPM) prevents or reduces the "bridge" of dead tissue between the soil and plant and may increase temperatures, conserve soil moisture, and help control weeds for a higher crop yield. BPM alone and BPM with floating row covers both provide better control than no treatment. Disease incidence can still be high, but significantly lower than no treatment. BPM alone and BPM with floating row covers combined with a chemical treatment (PCNB) provides even better control.

Biological Control:

A number of antangonistic fungi have been shown to provide control against *S. rolfsii* in controlled experiments, though field trial results vary. Some of the commonly used organisms are: *Trichoderma harzianum, T. viride, Bacillus subtilis, Penicillium* spp., and *Gliocladium virens.*

Trichoderma spp. are known mycoparasites of a number of plant pathogens. *T. harzianum* colonizes *S. rolfsii* hyphae, disrupts mycelial growth, and kills the organism. Field studies where effective control was obtained, involved application rates in the range of 140 to 600 to 1500 kg/ha. However, the populations of the pathogen and the antagonist were not monitored over time. *T. viride* has been shown to provide good control, especially when used in combination with an herbicide or pesticide. When combined with EPTC (an herbicide) in autoclaved soil, *S. rolfsii* activity was diminished, eventhough EPTC alone stimulated growth of the pathogen. In natural soil, the effectiveness of *T. viride* was reduced in the presence of EPTC, indicating the involvement of other soil microorganisms. *T. viride* in combination with PCNB has been shown to provide good disease control and better yield in artificially inoculated field plots (tomato) than in non-inoculated, untreated field plots. *T. viride* without PCNB provided statistically similar disease control but a lower yield. PCNB alone was less effective than PCNB with *T. viride* or *T. viride* alone.

Gliocladium virens have been shown to rapidly degrade *S. rolfsii* strain SR-1 in soil. *G. virens* will colonize *S. rolfsii* strain SR-3 but *sclerotia* can germinate under good conditions. The different reactions between *S. rolfsii* strains may be due to the size of the *sclerotia* (SR-3 sclerotia are up to 15-20 times larger than SR-1 sclerotia) and the amount of melanin in the sclerotial rind (SR-3 sclerotia has more melanin). In vitro studies show that varying concentrations of *G. virens* provide a corresponding variation in the germination of *S. rolfsii* (SR-1) sclerotia, but all concentrations result in a low percentage of *S. rolfsii* infection. In most cases there were always sclerotia that germinated but the pathogen did not infect plant tissues. *G. virens* had little effect on the germinability and infectivity of *S. rolfsii* (SR-3). Data such as these suggest considerable specificity in biocontrol due to differences in susceptibility of strains of the same pathogen to a single biocontrol strain, in addition to specificity due to various strains of a biocontrol agent.

CHEMICAL CONTROL

Control measures include chemical disinfection of vegetative propagation material, adjustment of soil pH by liming, adjustment of fertilizer regime, and use of herbicides for weed control. Formalin, chlorobromopropene and methyl bromide are among the most promising fumigants for treatment of seed beds or fields for valuable crops.

Pre-plant chemicals and application techniques: fumigants such as metamsodium (Vapam), Vorlex, methyl bromide, and chloropicrin, when applied to soil, reduce southern blight incidence.

Tomato:

Six fungicides tested on tomato (Aatopam-N, Aldrex T, Calixin M, PCNB, captan and captafol) at 200 mg a.i.l-1, applied as pre- and post- inoculation soil drenches. Only pre-inoculation soil drenches were effective, and only PCNB effectively reduced disease severity when applied 10 days before inoculation.

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