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Sclerotium rolfsii; Causative organism of southern blight, stem rot, white mold and sclerotia rot disease

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ABSTRACT

Sclerotium rolfsii is a soil borne pathogen that causes stem rot disease on plants. It primarily attacks host stems including roots, fruits, petioles and leaves under favourable conditions. It commonly occurs in the tropics, subtropics and other warm temperate regions of the world. Common hosts are legumes, crucifers and cucurbits. On a global perspective, estimated losses of 10 – 20 million dollars associated with S. rolfsii have been recorded with yield depletion ranging from 1 – 60% in fields. Sclerotia serve as primary inoculum for the pathogen and are spread to uninfected areas by wind, water, animals and soil. Control measures include excluding the pathogen from the area, plant removal, soil removal, soil treatment, heat, solarization, chemical soil treatment, cultural practices, resistance and transgenic plant resistance, plant treatment, crop rotation, amongst others. Despite considerable research on this pathogen, its control continues to be a problem.

Keywords: *Sclerotium rolfsii,* stem rot, white mold, stem blight.

INTRODUCTION

Sclerotium rolfsii is a destructive soil borne plant pathogen which causes Southern blight disease on a wide variety of plants. In 1928, the United States Department of Agriculture reported that *S. rolfsii* and root knot nematode caused more damage in southern USA than any other Pathogen [1] (Hagan,1999). In recent years, *Sclerotium rolfsii* has been especially damaging on tomatoes in Benue State, Nigeria, Peanut, tomato in the southeast USA, and sugar beet in California. Despite considerable research on this pathogen, its control continues to be a problem.

Sclerotia of *S. rolfsii* on tomato fruits in Chile Island, Makurdi, Benue State, Nigeria

***S. rolfsii* causing stem rot of tomato plants in Benue state, Nigeria**

History

The species was first described in 1911 by Italian mycologist Pier Andrea Saccardo, based on specimens sent to him by Peter Henry Rolfs who considered the unnamed fungus to be the cause of tomato blight in Florida in 1892. The specimens sent to Saccardo were sterile, consisting of hyphae and sclerotia. He placed the species in the old form genus *Sclerotium*, naming it *Sclerotium rolfsii*. It is, however, not a species of *Sclerotium* in the strict sense. In 1932, Mario Curzi discovered that the teleomorph (spore-bearing state) was a corticioid fungus and accordingly placed the species in the form genus *Corticium*. With a move to a more natural classification of fungi, *Corticium rolfsii* was transferred to *Athelia* in 1978.

Tomato fruit destroyed by *S. rolfsii* in Chile Island, Makurdi, Benue State, Nigeria

Classification

Sclerotium rolfsii is the anamorphic stage of the pathogen which is under group Incertae sedis, as the teleomorph stage i.e. the sexual stage is rarely observed. The teleomorph, *Athelia rolfsii* (Curzi) C.C. Tu & Kimbr. is a basidiomycete classified in the following order:

Kingdom	:	Fungi
Phylum	:	Basidiomycota
Subphylum	:	Agaricomycotina
Class	:	Agaricomycetes
Order	:	Atheliales
Family	:	Atheliaceae
Genus	:	*Athelia*

Binomial name

Athelia rolfsii

(Curzi) C.C. Tu & Kimbr.

Synonyms
Corticum rolfsii Curzi
Pellicularia rolfsii (Curzi) E. West
Botryobasidium rolfsii (Curzi) Venkatar
Sclerotium rolfsii anamorph sacc.
Hypochnus centrifuges (Weinm.) Lev.
Rhizoctonia centrifuga Lev.

[18] (Bowen *et al.,* 2010) Carried out phylogenetic analysis of Agaricomycetes based on a six-locus nuclear data set and proposed a new order; Amylocorticiales. Based on sequence identity, they placed *Athelia rolfsii* in order Amylocorticiales. Taxonomic problems related to classification of *Sclerotium rolfsii* needs to be resolved.

Economic Significance
In 1892, Peter Henry Rolfs first published a description of a new disease on tomato where some fields in Florida showed a greater than 70% loss. The fungus was named *Sclerotium rolfsii* by Saccardo in 1911[2] (Grecher, 1995). Descriptions of *S .rolfsii* disease in Connecticut, Louisiana, North Carolina, Japan, Ceylon, and India were published in the early 1900s. In 1928, the United States Department of Agriculture (USDA) reported that *S. rolfsii* and root-knot nematode caused more damage in southern states than any other pathogen. In the first half of the 20th century, peanut production sustained losses of 10-20 million dollars annually due to this pathogen. Losses of 25-50% were not uncommon in 1938-1947. By 1944, the pathogen was known to occur in 24 states. By 1966, there were almost 2000 publications on the pathogen in various locations around the world, but mostly from tropical and subtropical areas [1] (Hagan, 1999).

From a global perspective, peanut crops sustain higher losses than any other agricultural crop. In 1959, the United States Department of Agriculture estimated losses from $10 million to $20 million associated with *S. rolfsii* in the southern peanut-growing region, with yield depletions ranging from 1-60% in fields in the NC coastal plains region. During the middle of the 20th century, *S. rolfsii* was controlled to some degree by fumigation or soil applied fungicides. These chemicals are often too expensive and too toxic for many situations, and future uses of fumigants are being restricted due to environmental concerns. Today, control of this fungus disease is still the subject of many research projects involving chemicals, biological agents, soil amendments, cultural modifications, disease physiology, nutrition studies, and cultivar/variety resistance [3] (Singh and Dwivedi,1991). Despite these efforts, *S. rolfsii*, like many other soil borne fungal disease agents, continues to be a difficult pathogen to control. The wide host range, prolific growth, and ability to produce persistent sclerotia contribute to the large economic losses associated with the pathogen.

Sclerotia of *S. rolfsii* on tomato fruit in Chile Island, Makurdi, Benue State, Nigeria

***S. rolfsii* on tomato fruit and soil in Chile Island Makurdi, Benue State, Nigeria**

***S. rolfsii* on Tomato fruit in Chile Island, Makurdi, Benue State, Nigeria**

Geographical 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 winter temperatures fall below 0^{o}C [4] (Punja, 1985).

Host and Host range

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 [5] (Punja, 2005).

Although no worldwide compilation of host genera has been published, over 270 host genera have been reported in the United States alone. 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.), 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*), water melon (*Citrullus vulgaris* Schrad.), and winter squash (*Cucurbita maxima* Decne.).

Other reported hosts (worldwide) include: alfalfa, amaryllis, artichoke, banana, bean, beet, Brussels sprouts, cabbage, cantaloupe, 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 [6] (Aycock, 1996).

The fungus persists in many weed hosts as well.

Signs and Symptoms

S. rolfsii primarily attacks host stems, although it may infect any part of a plant under favourable environmental conditions including roots, fruits, petioles, leaves, and flowers. 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 (sexual stage of reproduction) at the margins of lesions and under humid conditions, though this form is not common [7] (Taylor and Rodriguez, 1999).

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.
The first symptom usually noticed by the homeowner or grower is wilt. Wilted plants often decline and die rapidly as a result of an extensive lower stem rot.

Sclerotia and mycelia of *S. rolfsii* on tomato fruit and stem in Chile Island, Makurdi

Lower Stem Rot

On tomato, disease begins with a small, water-soaked lesion on the lower stem at or near the soil surface. The lesion spreads rapidly to girdle the stem. Lower stems (crowns) often become rotted, but rotting may not extend completely throughout the crown tissues. Hosta plants may or may not completely die as a result of infection. On mature pepper and tomato, the stem cortex several centimetres (inches) above and below the soil surface will decay, but the stem central cylinder does not decay. As the lower stem decay develops, plants usually remain erect and foliage wilts. On many host plants, wilted leaves gradually become brown and remain hanging on the plant [8] (Pattmark *et al.*, 1996).

Stem rot caused by *S. rolfsii* on tomato plants in Chile Island, Makurdi, Benue State

As lower stems decay, a white mat of mycelium develops at the lesion site. This white mat will often spread out onto the nearby soil surface. Shortly after the mycelia mat develops, small (0.5-1 mm), white, round, fuzzy mycelia bodies begin to appear. These mustard-seed-sized structures, called sclerotia, soon become smooth and light tan,

brown or black in colour. Sclerotia serve as overwintering bodies and may be seen in the mycelium, on diseased tissues above or below ground, on soil surfaces, or in soil crevices [9] (Tsahouridou and Thanassoulopoulos, 2002).

S. rolfsii **on stem and root of tomato plants in Chile Island, Makurdi, Benue State, Nigeria**

Root Decay

On some plants, such as tomato, pepper, and sweet potato, root infection may follow crown infection [10] (Bowen *et al.*, 1992). On apples, roots are the primary infection site and crown rot develops subsequently. Usually the characteristic white mycelia mat and sclerotia develop near and on infected crown tissues or in and around roots close to the soil surface. The leaves eventually die, and branch dieback develops.

S. rolfsii **causing foot and root rot of tomato stem in Chile Island, Makurdi, Benue State**

Fruit

Tomato fruit and other fruit at or near the soil surface may become infected with *S. rolfsii*. Soft, water-soaked, sunken, slightly yellowish lesions develop. These lesions quickly spread throughout most or all of the fruit, which will eventually collapse. Coarse white mycelium develops with sclerotia. Although symptoms vary with the host affected, infection is usually restricted to plant parts in contact with the soil. *S. rolfsii* attacks stems, roots, leaves, and fruit.

Fruit and other fleshy organs near the soil surface may become infected with *S. rolfsii*. Soft, water-soaked, sunken, slightly yellowish lesions develop. These lesions quickly spread throughout most or all of the fruit, which will eventually become soft and collapse within 3 to 4 days of infection. The skin of the fruit often crack open and fine white mycelium and developing sclerotia spreads over the surface and quickly fills lesion cavities.

Tomato stem and fruit infected with *S. rolfsii* in Chile Island, Makurdi, Benue State

Tomato fruits affected by *S. rolfsii* in Chile Island, Makurdi, Benue state, Nigeria

***S. rolfsii* causing decay of tomato fruits in Chile Island, Makurdi, Benue State**

Tomato fruit infected with *S. rolfsii* in Chile Island, Makurdi, Benue State, Nigeria

Biology

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 [11] (Fery and Dukes, 2002). This results in tissue decay, further production of mycelium and the formation of *sclerotia.* The latter two rely upon favourable environmental conditions.

Sclerotia undergo either hyphal or eruptive germination. Hyphal germination is characterized by the growth of individual strands of hyphae from the sclerotia surface while eruptive germination is characterized by plugs or aggregates of mycelium bursting through the sclerotia surface. The quantity of mycelia growth and the energy needed for infection is dictated by the type of *sclerotia* germination that takes place. A food base of nonliving organic matter must be present for hyphally germinating sclerotia to infect host tissue because mycelia growth is sparse. However, mycelium from eruptive germinating *sclerotia* can infect host tissue without requiring an exogenous food base [12] (Mehan *et al*., 1994).

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 mycelia growth is 3.0 to 5.0, and sclerotia germination occurs between 2.0 and 5.0. Germination is inhibited at a pH above 7.0. Maximum mycelia growth occurs between 25 and 35^{o}C with little or none at 10 or 40^{o}C. Sclerotia formation is also greatest at or near the optimum temperature for mycelia growth. Mycelium is killed at 0^{o}C, but *sclerotia* can survive at temperatures as low as -10^{o}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 humidity of 25-35 %. One review summed it up by stating that soil moisture studies are difficult to interpret. Mycelia growth and sclerotia germination occur rapidly in continuous light, though they will occur in darkness if other conditions are favourable [13] (Edmund *et al.*, 2003).

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 colourless 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.

S. *rolfsii* on stem and fruit in Chile Island (Makurdi), Benue State, Nigeria

Tomato stem and fruit infected with *S. rolfsii* in Chile Island (Makurdi), Benue State

Epidemiology

Sclerotia serve as the principle overwintering structures and primary inoculum for disease. Persisting near the soil surface, sclerotia may exist free in the soil or in association with plant debris. Those buried deep in the soil may survive for a year or less, whereas those at the surface remain viable and may germinate in response to alcohols and other volatiles released from decomposing plant material. Thus, deep ploughing serves as a cultural control tactic by burying sclerotia deep in the soil. High temperatures and moist conditions are associated with germination of sclerotia. High soil moisture, dense planting, and frequent irrigation promote infection.

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.

Temperature and moisture are very important factors in the spread and development of this pathogen. Hyphal growth occurs over a temperature range of 8-40ºC / 46-104º F, but optimal growth and sclerotia production occurs between 27-35ºC / 81-95ºF. In addition to temperature effects, hyphal growth and sclerotia germination require a water-saturated soil. High humidity also favours fungal development. At 27ºC / 81ºF on Potato Dextrose Agar, the hyphal growth rate of *S. rolfsii* has been observed to be 0.8-0.9 mm per hour. Sclerotia form after 5-7 days. Host penetration and infection will proceed optimally at 27-30ºC / 81-86ºF, provided that moisture and high humidity are present [14] (Tu and Kimbrough, 1978).

Current season spread of *S. rolfsii* within a planting occurs by mycelia growth from infected plants, plant debris, or sclerotia. Long distance spread occurs as a result of movement of infected plant material or infested soil. Studies have shown that sclerotia may pass through the digestive tract of cattle or sheep and still be viable. Mycelium does not usually survive below freezing temperatures, but sclerotia are known to survive in locations where below freezing temperatures have occurred.

Dissemination

Sclerotia spread to uninfected areas by wind, water, animals, and soil. Mycelium is carried to new places by transplants and infected seeds.

***S. rolfsii* on tomato fruit and soil in Chile Island, Makurdi, Benue State, Nigeria**

Management/Control

Excluding the Pathogen from an Area

Only pathogen-free plants, cuttings or seeds should be purchased from a reputable dealer. In a greenhouse, all pots and planting equipment must be clean and pathogen-free, and planting media also must be pathogen-free. If field planting is involved, areas that do not have a history of *S. rolfsii* should be selected. Even though southern blight is not common in areas with cold winters, the disease may be a problem during the growing season if infected transplants from greenhouses or warmer regions are planted [15] (Takahashi, 1927).

Plant Removal

As with many plant diseases, removal of infected plants is an important aspect of disease control. *Sclerotium rolfsii* usually causes infection at the lower stem (crown) section of the plant, and once infection takes place, removal of the whole plant is necessary. Prompt removal of infected plants will help prevent the addition of abundant fungal inoculum to the soil. If infected plants are allowed to remain in an area, these plants will serve as a continuous source of inoculum. Plant removal is a practical measure in landscapes, gardens, nurseries, and greenhouses, but not in field situations [16] (Townsend and Willets, 1954).

Soil Removal

Sclerotia are reported to survive in the soil for 3 - 4 years. In gardens or landscapes, some localized soil removal and replacement may help to control *S. rolfsii*. The grower should take care not to increase the distribution of the fungal inoculum in an area when transporting soil. In nurseries and greenhouses, used potting media should not be saved for new plants. Plant debris and used media should be thoroughly cleaned out of production areas after each crop is removed. Recycled containers should be thoroughly cleaned so as to remove all old pot media.

Soil Treatment

Treating the soil with heat (including solarization), fungicides or fumigants, cultural manipulations, organic amendments, fertilizers, or biological treatments may help to control southern blight.

Heat

In some large nurseries or greenhouses, it may be possible to treat beds or bulk soil with aerated steam. All areas must be brought to a temperature of 160-180^{o}F for 30 minutes. Treated soil should be stored away from contaminated areas. Even after steam treatment, some sclerotia may survive and losses may occur.

Solarization

Solarization for southern blight control has been beneficial in select situations in the southern United States. Successful solarization requires that the soil is prepared properly for planting. Adequate soil moisture must be present. Clear plastic sheeting, 0.025 - 0.4 mm thick, must be applied to the area for 4-8 weeks, depending upon the time of year. For example, plastic applied during the month of June in Alabama would require a shorter exposure time of 4 weeks due to the high temperatures. Treated areas should receive direct and full sunlight. Soil solarization will significantly reduce viable sclerotia. It will also help control other soil borne diseases, plant parasitic nematodes, and some weeds. To be effective in control of southern blight, solarization must be repeated every year. Soil solarization is expensive, and it is not practical for use in large field situations [17] (Weber, 1931).

Chemical soil treatments

Soil fungicides or fumigants have been successfully used to control *S. rolfsii*. The soil fungicide pentachloronitrobenzene (PCNB) has been used on peanuts and some other crops since the 1940s. In a recent peanut trial, azoxystrobin applied as pre plant and post plant furrow treatments significantly controlled southern blight. However, applying fungicides to soil may require large quantities of chemical which is not practical in many situations. Also, fungicide effectiveness is not always consistent from year to year. With some ornamentals in commercial production situations, the fungicides azoxystrobin, flutolanil, flutolanil + thiophanate-methyl, and/or tebuconazole are labelled for pre- and post plant drenches. Fumigants, such as metam sodium or dazomet (granular), are toxic to sclerotia and mycelium in the soil. But, even after treatment with fumigation, some sclerotia survive, and treatments must be repeated annually. These fumigant chemicals are restricted-use products and may only be applied by certified applicators. The toxicity and cost of fumigants limit their usefulness in many situations. Methylbromide was used for many years to help control Southern blight but it is no longer available, due to its detrimental effects on the ozone layer of the atmosphere. In addition to the more conventional soil treatments of fungicides or fumigants, a soil insecticide (chlorpyrifos) is also labelled for use as a soil treatment to control *S. rolfsii* in certain peanut cropping situations.

Cultural practices

Cultural modifications for management of *S. rolfsii* in the landscape include deep ploughing, lime additions, aerification, and thatch removal. In some situations, deep ploughing will provide disease control. At depths below 20-30 cm [8-12 in.], sclerotia do not survive longer than 45 days. Also, deep ploughing removes sclerotia from contact with root tissues. Although deep ploughing is effective the first time, it may not work in subsequent years because sclerotia are returned to the upper soil layer. Keeping the soil pH at 6.5 by the addition of lime will help to prevent rapid fungal growth. Aerification of the soil and removal of thatch or other plant debris will also aid in suppressing *S. rolfsii* growth [18] (Bowen *et al*., 2010). In greenhouses, areas should be kept open with good plant spacing to help keep relative humidity low. Also, keeping the temperature below 25ºC / 77ºF and maintaining well-aerified plant media will discourage fungal growth. Nurseries must be designed to carry drainage water away from container areas.

Soil amendments, fertilization, biological agents and plant-produced chemicals.

Soil can be treated with organic amendments, fertilizers, or biological agents to help control *S rolfsii*. The addition of organic amendments such as compost, oat or corn straw, or cotton gin trash to soil sometimes reduces southern blight incidence and development. This effect may be due to the increase of toxic ammonia and/or the increase of certain soil microorganisms in the soil. Furfuraldeyde, an organic (sugar derivative) amendment, has been shown to change the soil microflora, and this change has been related to a decrease of *S. rolfsii* in the soil in lab and

greenhouse studies. Also, neem oil and pine bark extracts or pine bark powders have resulted in reduced growth of *S. rolfsii*. To date, these amendments have not been widely used for disease control, but future refinements may allow their use in the field.

Fertilizer studies have shown that treatments with ammonium, calcium nitrate or calcium sulphate may help control southern blight. Increased nitrogen may inhibit sclerotia germination, alter host susceptibility, or alter the soil microorganisms.

Biological control of *S. rolfsii* has been achieved to some degree with bacteria (*Bacillus subtilis*), actinomycetes, a mycorrhizal fungus, or certain *Trichoderma* species. There have been several studies to attempt to explain the mechanism for *Trichoderma* inhibition of *S. rolfsii*. Many studies have shown disease control by biological agents in laboratory and greenhouse tests, but disease control is less effective in the field. When control is seen in field studies, the required quantity of the biological product may be very high and not practical in most agronomic situations. In addition to the above, certain compounds in some aster roots and mustards are being studied for their inhibitory activity against this pathogen. At the present time, soil amendments, fertilization products, biological agents and plant-produced chemicals have not been widely adopted for southern blight control.

Crop Rotation

When soil borne plant pathogens are involved, crop rotation is a common and preferred method of disease control. This practice is not used much with southern blight because of the unusually wide host range of *S. rolfsii*. Corn is reported to be a non-host of this pathogen. When infested fields are rotated to corn, inoculum levels in the soil slowly decrease, and lower disease incidence will be seen in subsequent years. Some field research has shown that peanut rotations with cotton, 'Pensacola' bahia grass (*Paspalum notatum*), or switch grass (*Panicum virgatum*) resulted in a decreased incidence of white mold (stem rot) in peanut fields.

Plant Treatments

Plant treatments with fungicides for control of southern blight are generally protective in nature. Most fungicides are labelled for use on select ornamentals, vegetables, or some field crops. On peanuts, good disease control has been achieved by foliar applications of azoxystrobin products, flutolanil, or tebuconazole. PCNB is a fungicide that was used for many years to control *S. rolfsii* on some ornamentals, peanuts, some vegetables, and as a seed treatment. The product is available as a granular or liquid formulation. In 1998, PCNB no longer provided control of *S. rolfsii* in some Texas peanut fields. Fungal isolates tolerant to PCNB had developed. Southern blight control in peanut usually involves protective treatments with fungicides. However, good disease control has also been achieved with the insecticide chlorpyrifos, which is hydrolyzed into a fungicidal product, or with the nematicide ethoprop (mode of action unknown). For many years the only fungicide labelled to control southern blight on aucuba and several other ornamentals was PCNB. We now have the fungicides azoxystrobin, flutolanil, flutolanil + thiophanate-methyl, and tebuconazole in addition to PCNB available for use on select ornamentals for control of southern blight. Many of these products are labelled for pre- and post plant drench or broadcast application.

Resistance and Transgenic Plant Resistance

The use of resistant varieties or cultivars is always a much preferred method of disease management or control. Unfortunately, many common host plant species of *S. rolfsii* do not contain cultivars or varieties that exhibit high levels of resistance to this fungus. Nevertheless, research in this area continues. Some recent cultivar studies were done on Hosta, peanut, and cowpea. In 2003, 18 Hosta cultivars in a greenhouse study were evaluated for their resistance to *S. rolfsii* infection and disease development. Variable symptom severity resulted, but complete resistance to *S. rolfsii* was not reported. Recent inoculation studies with peanuts and cowpeas showed there were differences in disease susceptibility between some cultivars. And, preliminary results from a recent laboratory study with transgenic carrots indicated a reduced susceptibility to *S. rolfsii*.

Management of Southern blight is difficult when inoculum levels are high and conditions are conducive to the pathogen. Avoiding the disease by selecting fields that are free of *S. rolfsii* is the most successful method of control. Crop rotations of two years or more to a non-host crop like corn or small grains will help to prevent build-up of inoculum and disease problems.

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