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Unraveling the biology, effects and management methods of *Sclerotium rolfsii* infection in plants for sustainable agriculture

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ABSTRACT: *Sclerotium rolfsii* is an economically important soil-borne fungal pathogen that affects various crops worldwide, causing diseases such as blight, collar rot, crown rot, damping-off, *Sclerotium* wilt, and stem rot. It primarily attacks stems but can also infect roots, fruits, petioles, leaves, and flowers under favorable conditions. Initial symptoms include yellowing and wilting of leaves, followed by the appearance of white, fluffy mycelium on infected tissues and soil. The fungus forms round, white sclerotia that mature into dark brown or black structures resembling mustard seeds. These sclerotia enable *S. rolfsii* to survive unfavorable conditions for up to seven years in soil. The fungus has an extensive host range of over 500 species across 100 plant families. *S. rolfsii* thrives at or near the soil line. Before penetrating host tissue, it produces a considerable mass of mycelium on the plant surface over 2 to 10 days. The pathogen secretes enzymes that degrade the host's outer cell layer, leading to tissue decay, further mycelium production, and sclerotia formation. Managing *S. rolfsii* is challenging due to its broad host range and persistent sclerotia. Biological control methods have been explored, focusing on bacterial and fungal antagonists. *Pseudomonas* and *Bacillus* species have been studied for their ability to inhibit hyphal growth and reduce sclerotia germination. Additionally, antagonistic fungi, including *Gliocladium virens*, *G. roseum*, *Glomus fascicatum*, *Penicillium pinophilum*, *Gigaspora margarita*, and various arbuscular mycorrhizal fungi, have shown promise in suppressing *S. rolfsii*.

Keywords: *Athelia rolfsii*; Blight; Grey rot fungus; Mycelium; Sclerotia; *Sclerotium rolfsii*.

1. INTRODUCTION

Sclerotium rolfsii (Curzi) C.C. Tu & Kimbr. is an economically important soil borne pathogenic fungi capable of infecting several host crops in different regions of the world [1]. It is associated with variety of plants diseases such as blight, collar rot, crown rot, damping off of seedlings, *Sclerotium* wilt and stem rot. On onions and garlic, the pathogen is known to cause bulb rot resulting in complete yield loss in tropical climates and up to 65% in temperate climate [2,3]. Yield loss of 80% on pods has been reported on groundnut infected with *S. rolfsii* [4]. Similarly, over 70% of crop loss has been recorded on tomato and chili [5]. According to Wydra [6], infection by *S. rolfsii* is severe on crops from the Solanaceae family, legumes, cucurbits and other vegetables.

The wide host range, prolific growth, and ability to produce persistent sclerotia contribute to the large economic losses associated with the pathogen. Plants parts that are directly in contact with infected soil become infected. Affected plants develop round-elliptical basal lesions, brown-tan colored with a whitish centre and produce sclerotic. Diseased plants wilt and die after producing chaff seeds [7]. Management of *S. rolfsii* has been very challenging due to its extensive host range and ability to form resting stage (sclerotia) in soil to overcome unfavourable conditions [7].

2. ORIGIN OF *SCLEROTIUM ROLFSII*

Sclerotium rolfsii was named by Saccardo in 1911 in recognition of Rolf's pioneer work in Florida referred to in Tu and Kimbrough (1978). Initially, *Sclerotium* was a genus assigned to the artificial class of fungi imperfecti that included many diverse species. These species were grouped in one genus because of their shared phenotypic characteristics. They generally form small, tan to dark-brown/black, spherical sclerotia, that function as survival structures. As more became known about these fungi and their sexual states, some were reclassified into other genera, either in the Basidiomycota or the Ascomycota, and sometimes renamed according to their teleomorph. In 1931, Cruzi first described the perfect stage of *S. rolfsii* and *Corticium rolfsii* was the first teleomorph name, referred by Tu and Kimbrough, who in 1978, proposed to classify the pathogen in the genus *Athelia* and the binominal name, *Athelia rolfsii* (Cruzi) Tu and Kimbrough, has been used since. Some *Sclerotium* species still have no known sexual state, but by exploring molecular identification methods each species can now be assigned to the right genus. For example, based on sequence analysis of the rDNA large subunit (LSU) and internal transcribed spacer (ITS) regions, some *Sclerotium* species were renamed and moved from Ascomycota to Basidiomycota, whereas others, including *Sclerotium denigrans* and *S. perniciosum*, were moved from the Basidiomycota to the Ascomycota [8].

3. IDENTIFICATION OF *SCLEROTIUM ROLFSII* ON INFECTED PLANT AND GROWTH MEDIA

Colonies of *S. rolfsii* can be readily distinguished on infected plant material or artificial media by gross morphological characteristics. Rapidly growing, silky-white hyphae tend to aggregate into rhizomorphic cords [9]. In culture, agar media are rapidly (2-3 days) covered with mycelium, including aerial hyphae. The optimum temperature for hyphal growth and sclerotial formation is 27-30ºC [10]. Xu et al. [11] showed that the difference in temperature in the southern and northern parts of the United States of America affects survival of sclerotia. As a result, the severity of *S. rolfsii* in the southern part was higher than that in the northern part. Sclerotia (0.3-3.0 mm diameter) begin to develop after 4-7 days of growth when hyphae cluster together as a compact mass. After an initial white appearance, the sclerotia quickly become dark brown [10] (Figure 1).

4. 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 [13].



Figure 1. (a, b) Pure cultures of *Sclerotium rolfsii,* (c) Sclerotia collected on old cultures of *S. rolfsii* isolate [12].

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 [13].

5. EPIDEMIOLOGY, SURVIVAL AND SPREADING OF *SCLEROTIUM ROLFSII*

Sclerotia are the primary structures that enable *S. rolfsii* to survive periods of unfavourable conditions. Sclerotia are formed from masses of hyphal strands. Mature sclerotia include an outer layer (rind), an underlying cortical layer, and a central medullary region comprised of loosely interwoven hyphae. Sclerotia of *S. rolfsii* survived from 2 months to 7 years in field soil depending on experimental conditions. Climatic factors such as temperature and moisture may interact in influencing survival of *S. rolfsii* under field conditions. 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 [7].

6. *SCLEROTIUM ROLFSII* 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 [14]. 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; okra (*Hibiscus esculentus* L.), beans (*Phaseolus* sp.), sugar cane (*Saccharum officinarum* L.), sweet pepper (*Capsicum frutescens* L.), 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.), 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 squash pumpkin (*Cucurbita maxima* Decne.) [14].

Other reported hosts (worldwide) include: bean, beet, Brussels sprouts, soybean, squash, tobacco, tulip, cabbage, cantaloupe, carrot, cauliflower, celery, chrysanthemum, escarole, garlic, ginger, gourd, iris, coffee, cotton, cucumber, delphinium, endive, lettuce, mango, muskmelon, mustard, turnip, narcissus, onion, parsley, southern pea, alfalfa, amaryllis, artichoke, banana, peanuts, yam, potato, pumpkin, radish, rhubarb, turf (i.e., golf greens, bermudagrass and crabgrass), and pineapple [15]. Uwaidem et al. [12], reported that vine rot and wilt disease of *Telfairia occidentalis* that is prevailing in the study area (Abak, Nigeria) is caused by *Athelia rolfsii* (*S. rolfsii*) (Figures 2 and 3).

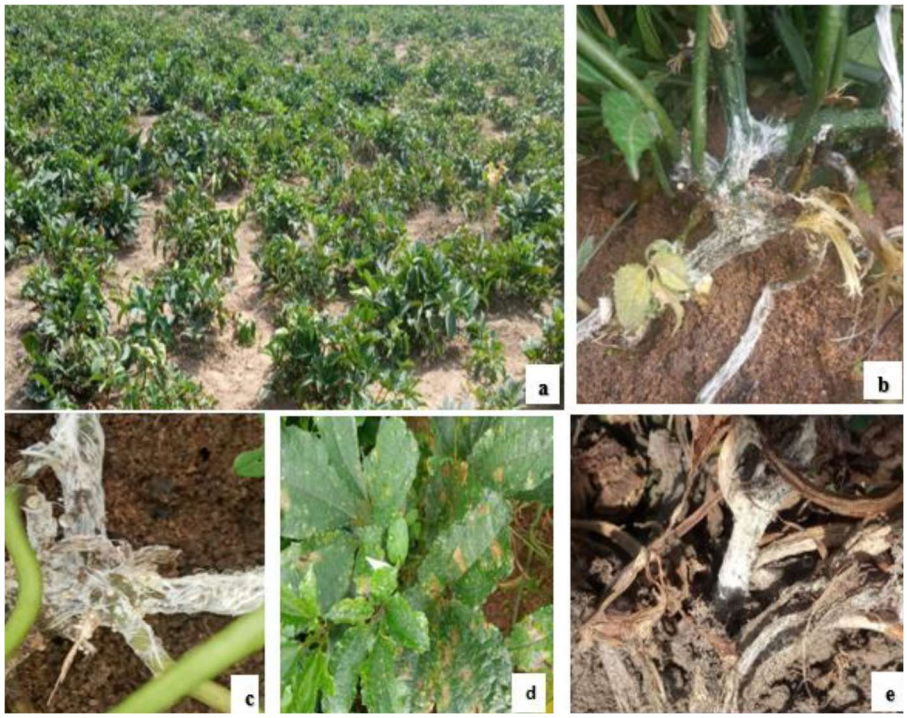


Figure 2. (a) Overview of *S. rolfsii* infected plot (b, c, d) symptoms of vine rot and patches on leaves caused *S. rolfsii* infection on *T. occidentalis* (e) wilted [12].



Figure 3. Symptoms of vine rot and patches on leaves caused *S. rolfsii* infection on *Arachis hypogea*.

7. BIOLOGY AND INFECTION MECHANISM OF *SCLEROTIUM 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 [16]. This results in tissue decay, further production of mycelium and the formation of sclerotia. The latter two rely upon favourable environmental conditions. During infection, *S. rolfsii* secrete oxalic acid and tissue degrading enzymes such as cellulose. Oxalic acid is corrosive to tissues of hundreds of genera of plants. Oxalic acid can combine with calcium in plant tissues, removing it from association with the pectic compounds in plant cell walls, lowering cell wall pH, and thereby favouring activity of the cell wall-degrading enzymes end opolygalacturonase and cellulose [17].

Massive mycelial growth on plant tissues produces large quantities of oxalic acid that facilitate penetration of hyphae into tissue [15]. Oxalic acid and tissue-degrading enzymes work together to break down cell walls, resulting in tissue maceration. The pathogens then absorb nutrients from the macerated tissue. Maceration interrupts transport of water and nutrients in plant tissues, thereby causing wilting, yellowing, and necrosis.

8. ECONOMIC IMPORTANCE OF *SCLEROTIUM ROLFSII*

S. rolfsii, a soilborne fungal pathogen, causes southern blight and is economically significant in agriculture, particularly in tropical and subtropical regions. The fungus has a broad host range and infects over 500 plant species, including economically valuable crops such as peanuts, vegetables, and Jerusalem artichokes. According to United State Department of Agriculture, *S. rolfsii* and root knot nematode caused more damage in different regions of the world 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 [18].

8.1. Economic Impact on Crop Production

S. rolfsii is a major pathogen in peanut (*Arachis hypogaea*) production, causing up to 80% yield losses in some cases. For instance, between 1988 and 1994 in the United States, the pathogen was responsible for significant economic damage in peanut farming, prompting research into effective control strategies [19]. The pathogen threatens vegetable crops, especially in areas where the use of soil fumigants like methyl bromide has been reduced. In Florida, its impact on fresh-market vegetables has highlighted the importance of integrated disease management practices [20]. Infection by *S. rolfsii* in Jerusalem artichoke (*Helianthus tuberosus*) can lead to yield losses of up to 60%. This crop is especially vulnerable to stem and tuber rot caused by the pathogen, necessitating the implementation of targeted disease management strategies [21].

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 [22]. 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 [22].

9. MANAGEMENT/CONTROL METHODS

S. rolfsii's broad host range and persistence in soil make management challenging. Traditional fungicides are often economically unviable for small-scale farmers, and the pathogen's ability to survive in soil for extended periods complicates eradication efforts. Integrated approaches, such as using resistant crop varieties, cultural practices, and biological control agents, have shown promise [23].

Recent studies have explored endophytic fungi as biocontrol agents. For instance, certain endophytes have demonstrated the ability to inhibit *S. rolfsii* growth, providing a potential environmentally sustainable alternative to chemical fungicides [24].

9.1. 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 inch.], 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 [25].

Aerification of the soil and removal of thatch or other plant debris will also aid in suppressing *S. rolfsii* growth [25]. 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.

9.2. 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 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. Crop rotation can reduce *S. rolfsii* inoculum, but its effectiveness is limited by the pathogen's wide host range and survival in soil for extended periods [26].

9.3. Resistant Cultivars

Because it is not easy to breed cultivars highly resistant to *S. rolfsii*, disease-tolerant cultivars may be used as a component of an integrated control effort. In the mid-90s, two cultivars, i.e. Toalson and Southern Runner, were developed which are less susceptible/partially resistant to *S. rolfsii* and more recently, additional partially stem rot resistant groundnut cultivars have been released including C-99R [27], Florida MDR 98 [28], Georgia-03L [29], Georgia-07W [30], and Florida-07 [31]. The use of these cultivars is still limited, possibly due to the relatively low level of resistance.

9.4. 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. 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°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.

9.4.1. Chemical Control

Strobilurin fungicides like pyraclostrobin have demonstrated high efficacy against *S. rolfsii*. Establishing baseline sensitivity of pathogen isolates to these fungicides helps ensure effective application and delays resistance development [32]. Other fungicides that are used to control stem rot disease include difenoconazole, carbendazim, flusilazole and chlorothalonil [33]. When difenoconazole was tested in combination with *Trichoderma harzianum*, a biocontrol agent of *S. rolfsii,* no reduction of the effect of *T. hazianum* was observed [33]. Although fungicides can protect groundnut plants from infection by *S. rolfsii*, chemical control should be gradually minimized because of its potential harmful effects to the environment. Therefore, integration of several different control measures is proposed to provide sustainable management of *S. rolfsii* and other diseases of groundnut. In this context, biological control can be an alternative or supplement to current management practices for *S. rolfsii* [34]. Natural compounds, such as physcion, have shown antifungal activity by disrupting the cell membrane of *S. rolfsii*, making them potential candidates for sustainable management [35].

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. Covering moist soil with polyethylene sheets to trap solar energy has proven effective in reducing *S. rolfsii* populations and disease incidence [36].

9.4.2. Biological Control

Application of beneficial microorganisms to soil, seeds or planting materials has been proposed as a sustainable and supplementary approach to control plant diseases. The most widely studied microorganisms with antagonistic activity against plant pathogens and with beneficial effects on plant growth, belongs to the bacterial genera *Bacillus, Pseudomonas, Rhizobium*, or the fungal genus *Trichoderma* [37]. Beneficial microbes such as *Trichoderma* spp. and *Pseudomonas fluorescens* suppress *S. rolfsii* through mechanisms like mycoparasitism and competition. These agents have been successfully used to manage southern blight in crops such as peanuts [38].

Fungal biocontrol agents may directly or indirectly kill sclerotia or mycelium of *S. rolfsii*. Lectins produced by *S. rolfsii* were proposed to serve as recognition factors for fungal biocontrol agents [39]. Among the fungal biocontrol agents, *Trichoderma* species are the most widely studied. In a direct interaction, hyphae of *Trichoderma* penetrate the rind and the cortex of sclerotia and lyse the medullar tissue. Degraded sclerotia

become dark, soft and disintegrate under slight pressure [39] and it was shown that chitinase and β-1,3-glucanase play a role in the interaction between *Trichoderma harzianum* and *S. rolfsii* [39]. Next to *Trichoderma*, several other fungal genera have been tested for their ability to control diseases caused by *S. rolfsii* on bean, carrot, chilli, ginger, wheat, lentil, sesame, soybean, sugar beet, sunflower, tomato, or groundnut. These antagonistic fungi include *Gliocladium virens, G. roseum, Glomus fascicatum, Penicillium pinophilum, Gigaspora margarita* and also *S. rolfsii*.

For biocontrol of *S. rolfsii*, several bacterial genera and species have been studied. Most of them belong to the genera *Pseudomonas* and *Bacillus*. *Pseudomonas* strains can restrict in vitro hyphal growth or reduce germination of sclerotia of *S. rolfsii* [34, 40]. Although pseudomonads are well-known for the production of a diverse array of antimicrobial compounds, including 2,4-diacetylphloroglucinol, pyrrolnitrin, pyoluteorin, rhizoxins, phenazines and lipopeptides the role of these or other bioactive compounds in biocontrol of *S. rolfsii* has not been studied in detail [40]. Also, for most of the tested *Pseudomonas* strains, there is a lack of knowledge on the genes involved in their activity against *S. rolfsii*. Next to *Pseudomonas*, several *Bacillus* species and strains have been studied for their efficacy to control stem rot disease of groundnut. Pretreatment of groundnut seeds with *Bacillus subtilis* protected groundnut seeds against *S. rolfsii* and significantly increased the number of pods [40]. Also, for the *Bacillus* strains and other bacterial genera tested to date, little or no knowledge is available on the fundamental mechanisms involved in their activity against *S. rolfsii*. Moreover, most of these studies were conducted under controlled conditions and only few studies were performed under field conditions. Recent studies show that certain endophytic fungi can effectively inhibit the growth of *S. rolfsii*, providing an environmentally friendly alternative to chemical fungicides [38].

9.4.3. Integrated Disease Management (IDM)

Integrated strategies combining resistant crop varieties, biological control agents, judicious fungicide use, proper irrigation, and sanitation practices have proven most effective against *S. rolfsii* [26]. These approaches ensure reduced reliance on chemical inputs while maintaining crop productivity.

10. CONCLUSION

S. rolfsii is a soil borne necrotrophic fungal pathogens capable of infecting several host crops with significant yield loss. In tropical and temperate climates, infection reaches threshold level with varying symptoms across different host crops. The presence of whitish cottony mycelial mat and white-brown sclerotia on infected plant parts are general symptoms associated with infection by *S. rolfsii*. Management of *S. rolfsii* has been challenging due to its extensive host range and its ability to form resting stage (sclerotia) in soil to overcome unfavourable conditions. The biology, mechanism of infection and management methods of *S. rolfsii* has been extensively studied, however control methods capable of mitigating infection by *S. rolfsii* is being investigated. The use of biological components, its interaction and mode of action has been promising. Biological agents are cheap, safe, and reliable and are capable of replacing the use of chemical control methods. However, their mode of application may require farmers to be trained by experts in the field of biological control.

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