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REVIEW

Sclerotinia sclerotiorum (Lib.) de Bary: A Cosmopolitan Plant Pathogen

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Sclerotinia sclerotiorum (Lib.) de Bary is a destructive necrotrophic fungal pathogen widely recognized for causing economically important diseases in numerous agricultural crops. The pathogen is considered a cosmopolitan fungus because of its global distribution and ability to survive and infect hosts under diverse climatic conditions. It produces characteristic survival structures known as sclerotia, which enable the fungus to persist in soil and plant debris for several years, thereby serving as a primary source of inoculum in subsequent cropping seasons. *S. sclerotiorum* has an exceptionally broad host range, infecting more than 400 plant species including economically important crops such as sunflower, soybean, rapeseed–mustard, canola, beans and several vegetables. The pathogen is responsible for a range of diseases such as white mold, stem rot, head rot and Sclerotinia rot, which often lead to significant yield and quality losses worldwide. Effective management of the disease requires an integrated approach, including cultural practices, resistant cultivars, biological control agents such as *Coniothyrium minitans* and judicious use of fungicides. Understanding the biology, epidemiology and management strategies of *S. sclerotiorum* is therefore essential for developing sustainable disease management practices in diverse cropping systems.

Keywords : Cosmopolitan fungus, host range, necrotrophy, sclerotia, *Sclerotinia*

INTRODUCTION

Plant pathogenic fungi are among the most important biological agents responsible for losses in global agriculture and food production (Agrios, 2005). These fungi infect numerous agricultural and horticultural crops, causing considerable declines in both yield and produce quality (Doehleman *et al*. 2017). Depending on their mode of nutrition and host interaction, fungal pathogens may follow biotrophic, necrotrophic or hemibiotrophic lifestyles, which help them invade plant tissues and evade host defense systems. Necrotrophic fungi are regarded as especially damaging because they destroy host cells and obtain nutrients from dead plant tissues, often leading to severe disease outbreaks and significant economic losses when environmental conditions are favorable (Dean *et al*. 2012; De Cal *et al*.2022).

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Among necrotrophic fungal pathogens, *Sclerotinia sclerotiorum* (Lib.) de Bary is regarded as one of the most destructive and economically important plant pathogens worldwide because of its extensive host range and ability to cause severe yield losses in numerous crops (Boldizsár *et al*. 2026). The pathogen causes diseases commonly known as white mold or Sclerotinia rot in more than 400 plant species, including soybean, sunflower, canola, common bean and several vegetable crops (Xu *et al*. 2018). Under cool and humid environmental conditions, epidemics of the disease can result in substantial reductions in crop productivity and major economic losses in temperate agricultural regions (O'Sullivan *et al*. 2021; Cohen, 2023). One of the distinguishing characteristics of *S. sclerotiorum* is its ability to produce hardened survival bodies called sclerotia, which allow the pathogen to persist in soil and infected plant residues for extended periods and serve as a major source of primary inoculum during subsequent cropping seasons

(Han *et al*. 2023; Hossain *et al*. 2023). The pathogen's prolonged survival potential, wide host range and highly aggressive necrotrophic infection behavior make its management particularly challenging in intensive agricultural production systems (Xu *et al*. 2018; 2019).

S. sclerotiorum is recognized as a cosmopolitan fungal pathogen because of its worldwide occurrence and remarkable capacity to infect a wide range of host plants (Hossain *et al*. 2023). Its extensive host range greatly contributes to its persistence, spread and epidemiological success in different cropping systems (Xu *et al*. 2018). The pathogen is responsible for destructive diseases such as white mold, stem rot, head rot and soft rot in crops including soybean, sunflower, rapeseed-mustard, canola, peanut, chickpea, lentil, common bean, tomato, potato, cabbage, lettuce and several other vegetables (Hossain *et al*.2023). Infection by *S. sclerotiorum* frequently results in extensive tissue maceration, lodging, premature plant death and significant deterioration in both yield and market quality (Xu *et al*. 2018). In susceptible crops such as soybean, canola and sunflower, disease outbreaks under cool and humid environmental conditions can cause yield losses ranging from 10–20% under moderate disease pressure to more than 50% during severe epidemics (Derbyshire and Denton-Giles, 2016; O'Sullivan *et al*., 2021). The broad host adaptability, prolonged survival through sclerotia formation and highly aggressive necrotrophic infection strategy collectively make *S. sclerotiorum* one of the most economically important fungal pathogens in modern agriculture (Hossain *et al*. 2023; Xia *et al*.2019). Due to wide host range, long-term survival through sclerotia and favorable environmental requirements for disease development, *S. sclerotiorum* continues to pose a major challenge for crop protection. Therefore, understanding its biology, host interactions and epidemiology is essential for developing effective and sustainable management strategies in modern agricultural systems.

Taxonomy and morphological characteristics of S. sclerotiorum (Lib.) de Bary

Taxonomic classification

S. sclerotiorum (Lib.) de Bary is a well-known soil-borne fungal pathogen belonging to the family Sclerotiniaceae. The pathogen is classified within the phylum Ascomycota, a large group of fungi characterized by the production of sexual spores (ascospores) inside specialized sac-like structures known as asci. The taxonomic classification of *S. sclerotiorum* is presented below :

Kingdom	:	Fungi
Phylum	:	Ascomycota
Class	:	Leotiomycetes
Order	:	Helotiales
Family	:	Sclerotiniaceae
Genus	:	*Sclerotinia*
Species	:	*sclerotiorum*

Members of the genus *Sclerotinia* are characterized by the formation of hardened survival structures called sclerotia, which enable the fungus to persist in soil or plant debris for several years. Among the species in this genus, *S. sclerotiorum* is the most widely distributed and economically important due to its extensive host range and ability to infect numerous agricultural crops (Purdy, 1979; Bolton *et al*., 2006).

Morphological features

The morphology of *S. sclerotiorum* includes vegetative structures such as mycelium, survival structures known as sclerotia and sexual reproductive structures including apothecia and ascospores. These structures play crucial roles in the pathogen's survival, reproduction and disease development.

Mycelium

The vegetative body of *S. sclerotiorum* consists of a network of hyaline, septate and branched hyphae that collectively form the mycelium. Under suitable environmental conditions, the fungus develops abundant white, cottony mycelial growth on infected plant tissues and artificial culture media. The hyphae are typically thin-walled and capable of rapidly colonizing the host tissues. During infection, the pathogen secretes various enzymes and organic acids that facilitate the

degradation of plant cell walls, leading to tissue maceration and disease symptoms (Bolton *et al*. 2006; Derbyshire and Denton-Giles, 2016).

Sclerotia

One of the most distinctive morphological characteristics of *S. sclerotiorum* is the formation of sclerotia, which are compact, melanized survival structures produced within infected plant tissues or on their surfaces. Sclerotia are typically irregular, elongated to rounded in shape and black on the exterior with a white internal medulla. Their size may vary from a few millimeters to more than one centimeter depending on the host and environmental conditions. These structures enable the pathogen to survive during unfavorable environmental conditions such as drought, cold temperatures or absence of host plants. Sclerotia can persist in soil for several years and serve as the primary source of inoculum in subsequent cropping seasons (Purdy, 1979; Willetts and Wong, 1980).

Apothecia and ascospores

Under favorable environmental conditions, particularly adequate soil moisture and moderate temperatures, sclerotia germinate carpogenically to produce apothecia, which are small, cup-shaped fruiting bodies. Apothecia are usually light brown to tan in color and are borne on slender stalks that emerge from germinating sclerotia present in the soil. Within the apothecia, numerous asci are formed, each containing eight ascospores. The ascospores are hyaline, unicellular and ellipsoidal in shape. These spores are forcibly discharged into the air and can be dispersed by wind over considerable distances. Once deposited on susceptible host tissues, particularly senescent or wounded plant parts, ascospores germinate and initiate infection. This airborne dispersal of ascospores plays a crucial role in the epidemiology and widespread distribution of Sclerotinia diseases in agricultural systems (Bolton *et al*., 2006; Derbyshire and Denton-Giles, 2016).

Host range and geographical distribution

S. sclerotiorum is recognized as one of the most polyphagous plant pathogenic fungi, possessing an exceptionally broad host range among cultivated and wild plant species. The pathogen has been reported to infect more than 400 plant species belonging to over 75 plant families, which contributes significantly to its persistence and widespread occurrence in diverse agricultural ecosystems (Boland and Hall, 1994; Purdy, 1979). This extensive host range allows the fungus to survive and multiply on a variety of crops as well as on numerous weeds, which often act as alternative hosts and reservoirs of inoculum.

In rapeseed-mustard and canola, the pathogen causes Sclerotinia stem rot, which is considered one of the most destructive diseases affecting oilseed crops in many parts of the world. Similarly, in sunflower, it causes stem rot, head rot and basal stalk rot, leading to significant yield and quality losses. Soybean is another major host where the pathogen causes white mold, a disease capable of causing severe epidemics under cool and humid environmental conditions (Bolton *et al*., 2006). Leguminous crops such as common bean, chickpea and lentil are also vulnerable to infection by *S. sclerotiorum*, resulting in stem rot and white mold symptoms. In addition, the pathogen infects several vegetable crops, including potato, tomato, cabbage, lettuce, carrot and pepper. A wide range of ornamental plants, such as chrysanthemum, marigold and sunflower ornamentals, can also be affected by the fungus. The ability of *S. sclerotiorum* to infect both field and horticultural crops highlights its importance as a major constraint in agricultural production systems (Bolton *et al*. 2006; Derbyshire and Denton-Giles, 2016).

S. sclerotiorum is considered as a cosmopolitan pathogen due to its presence in almost all major agricultural regions of the world. The pathogen has been reported in North and South America, Europe, Asia, Africa and Australia, indicating its broad adaptability to different environmental conditions. Though the disease occurs in diverse climates but it is particularly prevalent in temperate and subtropical regions, where moderate temperatures and high humidity favor disease development and pathogen survival (Purdy, 1979; Bolton *et al*. 2006). The global distribution of the pathogen is closely associated with the cultivation of susceptible crops such as soybean, sunflower, canola and various vegetable crops. In many

countries, favorable environmental conditions during the growing season promote the germination of sclerotia and the production of airborne ascospores, which facilitate rapid disease spread within crop fields. As a result, epidemics of Sclerotinia diseases have been frequently reported in regions with cool, moist weather conditions, which enhance ascospore release and infection (Derbyshire and Denton-Giles, 2016). Because of its broad host range, long-term survival through sclerotia and wide environmental adaptability, S. sclerotiorum continues to be a major pathogen affecting numerous agricultural crops across different climatic zones worldwide.

Disease symptoms

Diseases caused by *S. sclerotiorum* are commonly known as white mold, Sclerotinia rot or stem rot, depending on the host plant and the plant part affected. Although the severity and appearance of symptoms may vary among crops, the disease generally exhibits a number of characteristic signs, including water-soaked lesions, white cottony mycelial growth, stem rot and plant wilting and th**e** formation of black sclerotia on infected tissues. These symptoms have been reported in several economically important crops such as soybean, sunflower, rapeseed-mustard and various vegetable crops (Bolton *et al*. 2006; Kabbage *et al*. 2015).

Water-soaked lesions

The initial stage of infection caused by *S. sclerotiorum* is typically characterized by the appearance of small water-soaked lesions on leaves, stems, flowers or pods. These lesions often develop on senescent or injured plant tissues where airborne ascospores germinate and penetrate host surfaces. Under conditions of high humidity and moderate temperatures, the lesions expand rapidly and become soft, pale brown and irregular in shape. As infection progresses, the pathogen secretes several cell wall-degrading enzymes such as polygalacturonases, cellulases, hemi-cellulose and proteases along with oxalic acid, which facilitate the breakdown of host tissues, leading to maceration and collapse of infected plant parts (Kabbage *et al*. 2015; Bolton *et al*. 2006).

Cottony white mycelial growth

A characteristic symptom of *S. sclerotiorum* infection is the development of abundant white, cottony mycelial growth on infected tissues. The fluffy mycelium may cover stems, leaves, flowers and pods, particularly under moist environmental conditions that favor fungal growth. This dense mycelial growth spreads rapidly across plant surfaces and penetrates internal tissues, resulting in extensive rotting of affected plant parts. The conspicuous white fungal growth associated with infected tissues has led to the common name “white mold” for diseases caused by this pathogen in many crops (Peltier et al. 2012; Derbyshire and Denton-Giles, 2016).

Stem rot and wilting

As the pathogen continues to colonize host tissues, it frequently causes **stem rot**, which may girdle the stem and interfere with the normal transport of water and nutrients within the plant. Infected stems often become soft, bleached and water-soaked. As the disease progresses, affected plants may exhibit wilting, lodging and eventual death, particularly when infection occurs during flowering or reproductive stages. Such symptoms are commonly observed in crops such as soybean, sunflower and *Brassica* species, where severe infections can lead to significant yield losses under favorable environmental conditions (Bolton *et al*. 2006).

Formation of black sclerotia

Another distinctive diagnostic feature of S. sclerotiorum infection is the formation of black sclerotia, which are hardened survival structures produced either inside hollow stems or on the surface of infected tissues. These sclerotia are generally irregular or elongated in shape, black externally and white internally. Their size may vary depending on the host plant and environmental conditions. Sclerotia play a critical role in the survival of the pathogen by enabling it to persist in soil and plant debris for extended periods, thereby serving as an important source of inoculum for future disease outbreaks (Willetts and Wong, 1980; Bolton *et al*., 2006). Overall, the typical disease progression caused by *S.*

sclerotiorum involves the development of water-soaked lesions followed by the growth of cottony mycelium, stem rot, plant wilting and the formation of sclerotia, which facilitate long-term survival and spread of the pathogen in agricultural ecosystems.

Disease cycle

The disease cycle of *S. sclerotiorum* is closely associated with its ability to survive in soil through specialized structures and to produce airborne spores that facilitate infection of susceptible host plants. The pathogen exhibits a complex life cycle involving survival, germination, dispersal and infection phases. Environmental conditions such as temperature, humidity and canopy moisture play an important role in determining the development and spread of the disease in agricultural systems (Bolton *et al*. 2006; Derbyshire and Denton-Giles, 2016).The life cycle has been depicted in Fig.1.

Survival through sclerotia

The primary survival structure of *S. sclerotiorum* is the sclerotium, a compact and melanized structure formed within infected plant tissues or on their surfaces. After the infected plant tissues decay, these sclerotia fall to the soil and can remain viable for several years. The hardened outer rind of the sclerotia protects the fungal cells from unfavorable environmental conditions such as drought, extreme temperatures and microbial antagonism. Because of their long-term survival capability, sclerotia serve as the principal source of inoculum for subsequent disease outbreaks in susceptible crops (Willetts and Wong, 1980; Bolton *et al*. 2006;).

Myceliogenic and carpogenic germination

Sclerotia of *S. sclerotiorum* can germinate through two distinct mechanisms, myceliogenic germination and carpogenic germination. In myceliogenic germination, the sclerotia produce

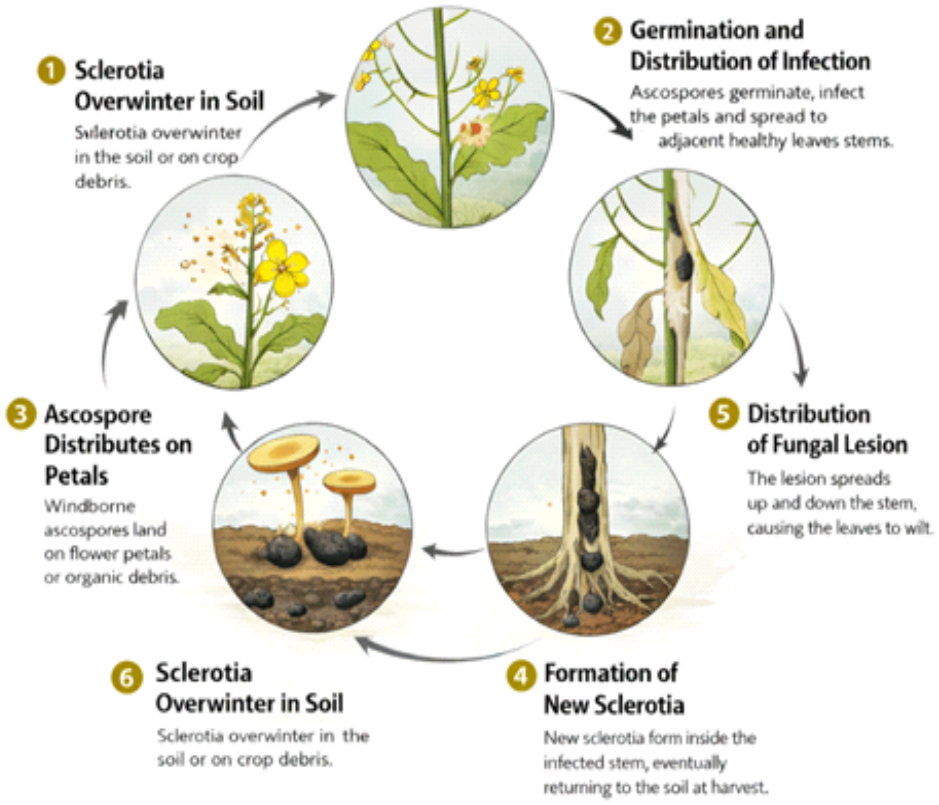

Fig 1 : Disease cycle of *S. sclerotiorum* on different host crops

vegetative hyphae that directly infect plant tissues in contact with the soil. This type of germination is more common in certain host-pathogen interactions where infection occurs at the soil surface or near the base of plants. In contrast, carpogenic germination results in the production of specialized fruiting bodies known as apothecia. This form of germination generally occurs when sclerotia are located near the soil surface and environmental conditions are favorable, particularly adequate soil moisture and moderate temperatures. Carpogenic germination is considered a major source of primary inoculum in many crop systems affected by Sclerotinia diseases (Purdy, 1979; Bolton *et al*. 2006; Clarkson *et al*. 2014).

Role of edaphic and environmental factors on myceloid and carpogenic germination of sclerotia

Environmental and edaphic factors play a significant role in regulating both myceliogenic and carpogenic germination of *S. sclerotiorum* sclerotia, thereby strongly influencing disease initiation and epidemic development. Sclerotia can germinate myceliogenically through direct hyphal emergence from the sclerotial rind or carpogenically through the production of apothecia that release airborne ascospores (Hossain *et al*. 2023). Soil temperature, moisture, aeration, pH, organic matter content and crop canopy microclimate are among the major factors affecting these germination processes. Carpogenic germination is generally favored under cool and moist soil conditions, where temperatures ranging from 10-20°C along with prolonged soil moisture stimulate apothecial formation and ascospore production (Zamani-Noor *et al*. 2024; O'Sullivan *et al*. 2021). High soil moisture, extended wetness periods and dense crop canopies further increase humidity near the soil surface, thereby enhancing carpogenic germination and subsequent disease outbreaks. In contrast, myceliogenic germination is commonly stimulated by fluctuating soil moisture, nutrient availability, physical damage to sclerotia and environmental stress conditions that induce direct hyphal growth from sclerotia (Hossain *et al*. 2023). Soil texture and microbial activity also influence sclerotial viability and germination, as suppressive soils enriched with antagonistic microorganisms can reduce survival and infectivity of the pathogen (Hossain *et al*., 2023). Recent epidemiological investigations have demonstrated that interactions among soil temperature, moisture availability and crop canopy architecture largely determine the predominance of either myceliogenic or carpogenic germination under field conditions, thereby affecting the epidemiology and management of Sclerotinia diseases (O'Sullivan *et al*. 2021; Zamani-Noor *et al*. 2024).

Production of apothecia and airborne ascospores

During carpogenic germination, sclerotia produce small, cup-shaped fruiting bodies called apothecia, which arise on slender stalks emerging from the soil surface. Each apothecium contains numerous asci and each ascus typically produces eight ascospores (Agrios, 2005; Bolton *et al*. 2006**)**. These ascospores are forcibly discharged into the air and dispersed by wind over considerable distances. Once deposited on susceptible host tissues, especially senescent flowers or leaves, the ascospores germinate and initiate infection. The presence of nutrient-rich senescent tissues often facilitates successful colonization of the host plant. Airborne ascospore dispersal is therefore considered a critical step in the epidemiology of Sclerotinia diseases in many crops such as soybean, sunflower and rapeseed-mustard (Bolton *et al*. 2006; Peltier *et al*.2012; Clarkson *et al*. 2014).

Environmental factors influencing disease development

Environmental conditions play a crucial role in the development and severity of diseases caused by *S. sclerotiorum*. Moderate temperatures, typically ranging from 15-25°C, combined with high relative humidity and prolonged leaf wetness, provide ideal conditions for apothecia formation, ascospore release and infection. Dense crop canopies that retain moisture further promote disease development by creating a humid microclimate favorable for fungal growth. Soil moisture also strongly influences the germination of sclerotia and the production of apothecia.

Continuous rainfall or irrigation during the flowering stage of susceptible crops often leads to higher disease incidence due to increased ascospore production and infection opportunities. Consequently, environmental conditions that favor prolonged canopy wetness and moderate temperatures are closely associated with the occurrence of epidemics in many agricultural regions (Bolton *et al*., 2006; Clarkson *et al*. 2014; Derbyshire and Denton-Giles, 2016). Overall, the ability of *S. sclerotiorum* to survive in soil as sclerotia, produce airborne ascospores through carpogenic germination and infect a wide range of host plants under favorable environmental conditions contributes significantly to its success as a widespread and destructive plant pathogen.

Epidemiological advances of S. sclerotiorum

Recent epidemiological studies on *S. sclerotiorum* have significantly improved understanding of disease development, survival mechanisms, host interaction and environmental factors associated with epidemic outbreaks. The pathogen primarily survives through the formation of sclerotia, which can persist in soil and infected crop debris for several years and germinate either myceliogenically or carpogenically depending on environmental conditions (Hossain *et al*., 2023). Epidemiological investigations have shown that temperature, prolonged leaf wetness, high canopy humidity and dense crop canopies play crucial roles in ascospore production, dispersal and successful infection establishment (Clarkson *et al*. 2014; O'Sullivan *et al*. 2021). Cool temperatures ranging between 15-25°C along with extended periods of moisture are considered highly favorable for disease initiation and rapid epidemic development (O'Sullivan *et al*. 2021). Recent studies have further highlighted the role of changing climatic conditions and intensive cropping systems in the increasing prevalence and geographic spread of *S. sclerotiorum* in many agricultural regions worldwide (Derbyshire and Denton-Giles, 2016; Hossain *et al*. 2023). In addition, molecular, epidemiological and genomic studies have enhanced understanding of pathogen diversity, virulence evolution and host adaptation, thereby contributing to the development of improved disease forecasting and integrated management strategies (Xu *et al*. 2018; Xia *et al*. 2019).

Pathogenicity and infection mechanism

S. sclerotiorum is a destructive necrotrophic fungal pathogen that infects host plants by killing plant tissues and subsequently colonizing the dead cells. The infection process involves a combination of biochemical and molecular mechanisms that enable the pathogen to overcome host defenses and degrade plant tissues(Fig.2). Among the most important factors contributing to the pathogenicity of *S. sclerotiorum* are the production of **oxalic acid**, secretion of cell wall-degrading enzymes and its characteristic necrotrophic lifestyle (Bolton *et al*. 2006; Kabbage *et al*. 2015).

Role of oxalic acid

Oxalic acid is considered one of the most important virulence factors produced by *S. sclerotiorum*. During infection, the pathogen secretes oxalic acid into host tissues, where it plays multiple roles in disease development. One of its primary functions is the acidification of host tissues, which creates favorable conditions for fungal growth and enhances the activity of enzymes involved in plant cell wall degradation. In addition, oxalic acid chelates calcium ions from plant cell walls, weakening the structural integrity of the cell wall and facilitating tissue maceration. Oxalic acid also interferes with plant defense responses by suppressing the oxidative burst that normally occurs during pathogen attack. This suppression of host defense mechanisms promotes successful colonization of plant tissues by the pathogen. Experimental studies using mutants deficient in oxalic acid production have demonstrated a significant reduction in pathogenicity, confirming the central role of this compound in disease development (Cessna *et al*., 2000; Kim *et al*. 2008).

Cell wall degrading enzymes

The pathogenicity of *S. sclerotiorum* is further enhanced by the production of cell wall-degrading enzymes (CWDEs) that facilitate penetration and colonization of host tissues. These enzymes break down structural components of plant cell walls, including cellulose, hemicellulose and pectin. Important enzymes produced by the

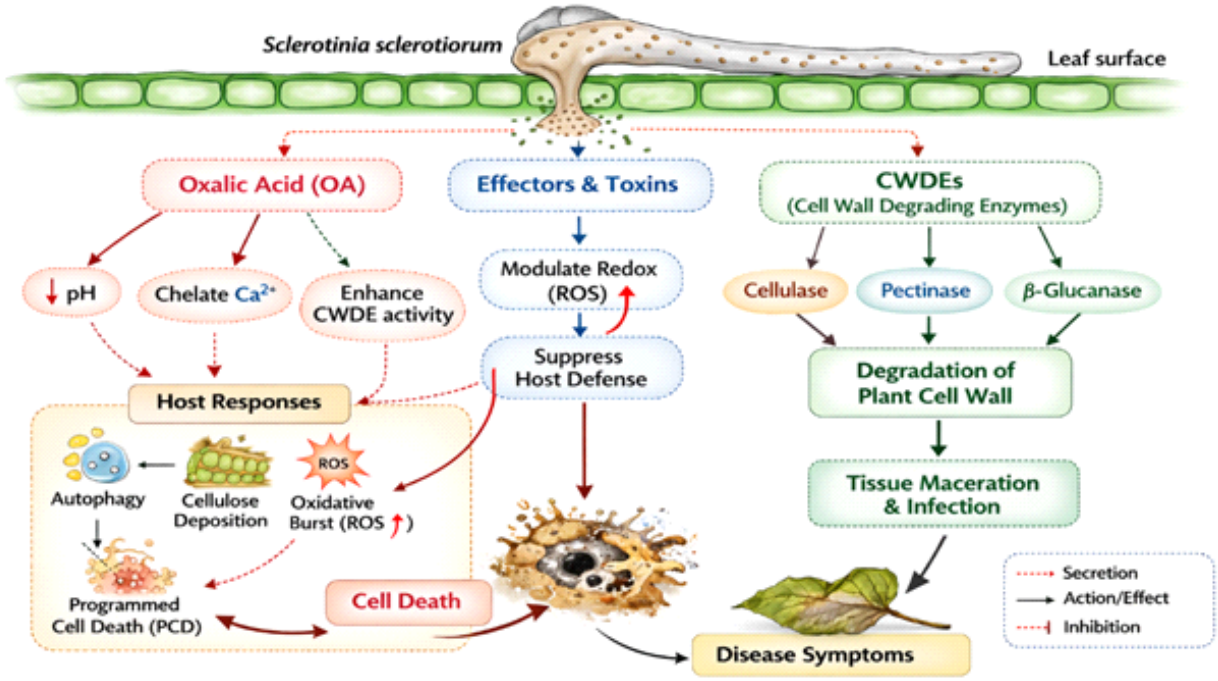

Fig 2 : Mechanism of pathogenicity in *S. sclerotiorum*

pathogen include pectinases, polygalacturonases, cellulases and hemicellulas**es**, which collectively degrade plant cell walls and cause the soft rot symptoms characteristic of Sclerotinia diseases. The activity of these enzymes is often enhanced under acidic conditions created by oxalic acid secretion, which further accelerates tissue degradation and disease progression. The coordinated production of CWDEs enables the pathogen to rapidly invade and colonize host tissues, resulting in extensive plant damage (Liang *et al*. 2015; Bolton *et al*.2006).

Necrotrophic nature of pathogen

S. sclerotiorum is classified as a necrotrophic pathogen, meaning that it kills host cells and derives nutrients from the dead tissues. This infection strategy differs from that of biotrophic pathogens, which depend on living host cells for survival. The pathogen often initiates infection on senescent or weakened plant tissues, such as aging flowers or injured plant parts, where it establishes initial colonization. Recent research suggests that *S. sclerotiorum* may briefly exhibit a biotrophic-like phase during early infection before switching to a necrotrophic phase. During this transition, the pathogen manipulates host defense mechanisms and eventually triggers extensive cell death through the combined action of oxalic acid, enzymes and other virulence factors. This ability to induce host cell death and efficiently utilize the resulting nutrients contributes to the pathogen's wide host range and high virulence (Kabbage *et al*. 2015; Liang and Rollins, 2018; Bolton *et al*. 2006). Thus, the pathogenic success of *S. sclerotiorum* results from the combined effects of oxalic acid production, enzymatic degradation of plant cell walls and its necrotrophic infection strategy, which together enable the pathogen to invade, colonize and destroy host plant tissues.

Molecular pathogenicity of S. sclerotiorum

The molecular pathogenicity of *S. sclerotiorum* involves a complex interplay of virulence factors, secreted effectors, cell wall-degrading enzymes and signaling pathways that collectively facilitate host colonization and disease development. Recent studies have demonstrated that the pathogen initially establishes a transient biotrophic-like interaction with host tissues before shifting to an aggressive necrotrophic phase characterized by extensive host cell death (Xu *et al*. 2018; Zhu *et al*. 2024). Oxalic acid is

considered one of the major pathogenicity determinants of *S. sclerotiorum* because it acidifies host tissues, suppresses oxidative burst responses, alters host redox balance and enhances the activity of cell wall-degrading enzymes during infection (Xu *et al*. 2018; Daniel *et al*. 2024). The pathogen also secretes several hydrolytic enzymes, including polygala cturonases, cellulases, pectinases and cutinases, which degrade plant cell walls and promote tissue maceration and fungal invasion (Derbyshire *et al*. 2022). In addition, small secreted effector proteins such as SsNEP2 and other pathogenicity-associated proteins have been reported to interfere with host immune responses and induce programmed cell death, thereby enhancing fungal virulence (Yang *et al*. 2022; Derbyshire *et al*. 2022). Advances in genomics, transcriptomics and secretome analysis have further revealed that coordinated regulation of pathogenicity genes, reactive oxygen species metabolism and environmental pH modulation play critical roles in the successful infection strategy of *S. sclerotiorum* (Derbyshire *et al*. 2022; Zhu *et al*. 2024). These molecular insights have considerably improved understanding of host-pathogen interactions and may contribute to the development of durable disease management strategies.

Management strategies

Effective management of diseases caused by *S. sclerotiorum* is challenging because of the pathogen's wide host range and its ability to survive in soil through long-lived sclerotia. Therefore, successful control generally requires the integration of multiple strategies, including cultural practices, biological control agents, chemical fungicides and integrated disease management approaches. The combination of these strategies helps reduce the survival of sclerotia, limit pathogen spread and minimize disease incidence in susceptible crops (Derbyshire and Denton-Giles, 2016; Peltier *et al*. 2012). Some of the recommended management practices have been outlined in Table 1.

Cultural practices

Cultural practices play an important role in reducing the inoculum potential of *S. sclerotiorum* and limiting disease development in the field.

- **Crop rotation** with non-host crops such as cereals can significantly reduce the population of sclerotia in soil. Continuous cultivation of susceptible crops often favors the buildup of the pathogen, whereas rotation with non-host species interrupts the disease cycle and lowers inoculum levels (Bolton *et al*. 2006).

- **Deep ploughing** is another useful cultural practice that helps bury sclerotia deeper into the soil profile. Sclerotia located at greater soil depths are less likely to germinate and produce apothecia, thereby reducing the amount of airborne inoculum available for infection (Subbarao *et al*. 1996).

- **Field sanitation** is also essential for minimizing disease spread. Removal and destruction of infected plant debris help reduce the number of sclerotia returning to the soil. Proper crop residue management and improved field hygiene can therefore play a significant role in lowering disease pressure in subsequent cropping seasons (Derbyshire and Denton-Giles, 2016).

Biological control

Biological control has gained increasing attention as an environmentally friendly approach for managing *S. sclerotiorum*. Several antagonistic microorganisms have shown promising results in suppressing the pathogen. One of the most well-known biological control agents is *Coniothyrium minitans*, a mycoparasitic fungus that specifically attacks and degrades sclerotia of *S. sclerotiorum*. This fungus colonizes the sclerotia in soil and reduces their viability, thereby decreasing the primary inoculum responsible for disease outbreaks (Whipps and Gerlagh, 1992). Species of *Trichoderma* are also widely studied as biological control agents against soil-borne pathogens. These fungi suppress *S. sclerotiorum* through several mechanisms, including competition for nutrients and space, production of antifungal metabolites and secretion of cell wall–degrading enzymes that inhibit pathogen growth (Harman *et al*. 2004; Verma *et al*. 2007). The use of such antagonistic microorganisms provides an eco-friendly alternative for disease management.

Table 1: Recommended management practices for *S. sclerotiorum* on different host crops

Host crop	Disease	Recommended management practices	References
Soybean *(Glycine max)*	Sclerotinia stem rot / white mold	Resistant cultivars, wider row spacing, crop rotation, fungicides (boscalid , fluazinam), and biocontrol agents such as *C. minitans* and *Trichoderma* spp.	Peltier *et al*. (2012); Li *et al*. (2025); Khambhati and Chen (2025)
Rapeseed-mustard (*Brassica* spp.)	Stem rot/white mold	Crop rotation, deep ploughing, sanitation, fungicide sprays and bioagents like *Bacillus subtilis* and *Trichoderma* spp.	Wang *et al*. (2024)
Sunflower (*Helianthus annuus*)	Head and stem rot	Resistant hybrids, field sanitation, crop rotation, proper irrigation management and fungicide application during flowering.	Rothmann *et al* . (2019); Talukder *et al*. (2022)
Common bean (*Phaseolus vulgaris*)	White mold	Screening of resistant genotypes along with fungicidal management has been found effective.	Pascual *et al* . (2010); Teixeira *et al*. (2019)
Pea (*Pisum sativum*)	White mold	Use of resistant genotypes, crop rotation, sanitation and fungicide application has been reported effective for management of white mold in pea.	Islam *et al*. (2021); Ashtari Mahini *et al*. (2020)
Lettuce (*Lactuca sativa*)	Lettuce drop	Application of fu ngicides such as boscalid, fluazinam, fluopyram + trifloxystrobin and iprodione effectively reduced lettuce drop.	Matheron and Porchas (2019)
Tomato (*Solanum lycopersicum*)	Stem rot	Integrated use of fungicides, compost amendments, sanitation and chemical inducers has been found effective.	Gomaa *et al*. (2016); Mazumdar (2021)
Cucumber (*Cucumis sativus*)	Watery soft rot/white mold	Co-application of *B. amyloliquefaciens* and salicylic acid significantly improved cucumber resistance against *S. sclerotiorum* and promoted plant growth.	Hathurusinghe *et al*. (2025)

Chemical control

Chemical control through fungicides remains an important component of disease management in crops susceptible to *Sclerotinia* diseases. Several fungicides belonging to different chemical groups have been reported to be effective in reducing disease incidence. Commonly used fungicides include boscalid, fluazinam, iprodione, procymidone and thiophanate-methyl, which inhibit fungal growth and prevent disease development when applied at appropriate growth stages (Mueller *et al*. 2002; Bolton *et al*. 2006; Peltier *et al*. 2012). Fungicide applications are generally most effective when applied during the flowering stage, which coincides with the period of highest susceptibility to infection by airborne ascospores. However, reliance solely on chemical control is not recommended because repeated use of fungicides may lead to the development of resistant pathogen populations and may also have environmental implications (Mueller *et al*. 2002; Derbyshire and Denton-Giles, 2016).

Integrated disease management approach

Because of the persistent nature of *S. sclerotiorum* in soil and its ability to infect a wide range of hosts, integrated disease management (IDM) is considered the most effective approach for controlling Sclerotinia diseases. IDM involves the combined use of cultural, biological and chemical methods to reduce disease incidence and minimize yield losses. For example, crop rotation and sanitation practices can reduce the buildup of sclerotia in soil, while biological control agents such as *Coniothyrium minitans* help destroy existing sclerotia (Whipps and Gerlagh, 1992; Gerlagh*et al*., 1999; Bolton *et al*. 2006). Timely fungicide applications during susceptible growth stages can further suppress disease development.

The integration of these strategies helps maintain disease levels below economic thresholds while promoting sustainable crop production (Bolton *et al*. 2006; Peltier *et al*. 2012; Derbyshire and Denton-Giles, 2016).

Future research needs

Despite considerable progress in understanding the biology and management of *Sclerotinia sclerotiorum* (Lib.) de Bary, the pathogen continues to cause substantial yield losses in many economically important crops worldwide. Its broad host range, prolonged survival through sclerotia and adaptability to diverse environmental conditions make disease management particularly challenging. Therefore, future research should emphasize the development of sustainable and integrated strategies that combine host resistance, biological control and advanced molecular approaches. One important area of research is the development of **resistant cultivars**. Breeding for resistance to *S. sclerotiorum* has proven difficult because strong qualitative resistance is rarely found in most crop species and resistance is generally quantitative and influenced by multiple genes. Nevertheless, identification of new resistance sources from wild relatives and germplasm collections may help improve resistance breeding programs. The application of modern molecular tools such as quantitative trait locus (QTL) mapping, marker-assisted selection and genomic approaches may accelerate the development of cultivars with enhanced tolerance to Sclerotinia diseases (Bolton *et al*. 2006; Derbyshire and Denton-Giles, 2016).

Another promising research direction involves the improvement of biological control strategies. Antagonistic microorganisms such as *Coniothyrium minitans* and *Trichoderma* species have demonstrated the ability to suppress *S. sclerotiorum* by parasitizing sclerotia, competing for nutrients or producing antifungal metabolites. However, the field performance of biological control agents can be inconsistent due to environmental variability and soil conditions. Future studies should focus on developing more stable formulations, improving delivery systems and identifying additional microbial antagonists with higher efficacy under field conditions. Understanding the ecological interactions among the pathogen, biocontrol agents and the host plant will further enhance the practical application of biological control methods (Whipps and Gerlagh, 1992; Harman *et al*. 2004). Advances in molecular research and epidemiological modeling also offer new opportunities for improving disease management. Molecular studies aimed at identifying genes involved in pathogen virulence, host resistance and plant defense mechanisms can provide valuable insights into the infection process and may lead to the development of novel control strategies. In addition, epidemiological models that integrate environmental conditions, crop growth stages and pathogen biology can help predict disease outbreaks and guide timely implementation of control measures. The integration of molecular tools, ecological studies and predictive models will therefore be essential for improving the understanding and management of Sclerotinia diseases in the future (Bolton *et al*. 2006; Clarkson *et al*. 2014; Derbyshire and Denton-Giles, 2016).

CONCLUSION

Sclerotinia sclerotiorum (Lib.) de Bary is a destructive and cosmopolitan fungal pathogen that causes significant quality and quantity losses in a wide range of agricultural and horticultural crops worldwide. Its broad host range, long-term survival through sclerotia and ability to produce airborne ascospores contribute to its persistence and rapid spread under favorable environmental conditions. The pathogen infects plants through complex pathogenic mechanisms involving oxalic acid production, secretion of cell wall-degrading enzymes and a necrotrophic mode of infection that leads to extensive tissue damage. Environmental factors such as moderate temperatures, high humidity and prolonged canopy wetness further enhance disease development and epidemic outbreaks. Effective management of *S. sclerotiorum* therefore requires a comprehensive understanding of its biology, epidemiology and host interactions. Cultural practices such as crop rotation, deep ploughing and field sanitation can help reduce inoculum levels, while biological control agents like *Coniothyrium minitans* and *Trichoderma* species provide environmentally sustainable options for suppressing the pathogen. In addition, timely application of fungicides during critical crop growth stages can reduce disease severity. However, due to the persistence and adaptability

of the pathogen, reliance on a single management strategy is often inadequate. Consequently, the integration of cultural, biological and chemical approaches through integrated disease management practices offers the most effective and sustainable strategy for minimizing the impact of Sclerotinia diseases. Continued research on resistant cultivars, improved biological control systems and advanced molecular and epidemiological studies will further strengthen future efforts to manage this important plant pathogen.

DECLARATION

Conflict of Interest. Authors declare no conflict of interest.

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