NARDI FUNDULEA, ROMANIA ROMANIAN AGRICULTURAL RESEARCH, NO. 43, 2026
www.incda-fundulea.ro; doi.org/10.59665/rar4382 First Online: July, 2026. DII 2067-5720 RAR 2026-181

Sclerotial Persistence in Soil-Borne Fungal Pathogens: Molecular Resilience, Climate-Driven Adaptation, and Microbiome-Integrated Biocontrol

Wael F. Shehata[1], Safaa A. M. Yousef[2], Othman Al-Dossary[1], Bader Alsubaie[1], Ahmed H. Ismail[3], Gamal A. El-Shaboury[4], Sally Negm[5], Mohamed A. Abou-Zeid[6*]

[1]Department of Agricultural Biotechnology, College of Agriculture and Food Sciences, King Faisal University, Al-Ahsa 31982, Saudi Arabia

[2]Mycology Research and Plant Disease Survey Department, Plant Pathology Research Institute, ARC, Giza 12619, Egypt

[3]Department of Agribusiness and Consumer Sciences, College of Agriculture and Food Sciences, King Faisal University, P.O. Box 400, Al-Ahsa 31982, Al-Hofuf, Saudi Arabia

[4]Department of Biology, College of Science, King Khalid University, Abha 61421, Saudi Arabia

[5]Health Specialities, Basic Sciences and Their Applications Unit, Applied College, Mahayil Asir, King Khalid University, Abha, 62529, Saudi Arabia

[6]Wheat Diseases Research Department, Plant Pathology Research Insitute ARC, Giza 12619, Egypt

[*]Corresponding author. E-mail: m.abouzeid@arc.sci.eg

ABSTRACT

Sclerotia are specialized survival structures that allow soil-borne fungal pathogens to persist between seasons, tolerate environmental stress, and sustain recurrent disease pressure in cropping systems. This review synthesizes molecular, structural, ecological, and management aspects of sclerotial resilience, with emphasis on *Sclerotinia sclerotiorum* and other emerging sclerotia-forming or sclerotium-like pathogens. It highlights regulatory networks governing sclerotial development and persistence, including the Velvet complex, calcium-mediated morphogenesis, and DHN-melanin biosynthesis. The melanized rind is considered a key survival determinant because it reduces environmental stress, microbial degradation, and the penetration of fungicides. The review also examines how temperature, soil moisture, drought, and climate variability influence sclerotial viability, pathogen epidemiology, and the reliability of disease management. It further evaluates the declining durability of fungicide-based control in the context of protected survival structures, reduced sensitivity, and the development of resistance. As an alternative, the review proposes an integrated biological framework involving metabolically primed *Trichoderma* spp., abiotic elicitors, arbuscular mycorrhizal fungi, and host-defense activation. These strategies target persistent inoculum and the plant–rhizosphere interface by promoting enzymatic degradation of sclerotial tissues, microbial antagonism, and induced resistance. Finally, formulation stability, climate-adaptive biocontrol strains, and microbiome-informed delivery systems are identified as priorities for field-resilient disease management.

Keywords: *Sclerotial* persistence, melanized rind, *Trichoderma* spp., biological control, climate-resilient disease management.

INTRODUCTION

Sclerotia-producing soil-borne fungi remain major threats to crop protection because their compact survival structures persist in soil and sustain recurrent disease pressure. Among them, *Sclerotinia sclerotiorum* is particularly destructive, infecting more than 700 plant species across oilseed, vegetable, and horticultural systems (Boland and Hall, 1994; Bandara et al., 2020). Persistence as long-lived sclerotia drives repeated seasonal outbreaks and constrains chemical control, because sclerotia tolerate fungicides, ultraviolet radiation, desiccation, and microbial antagonism, while repeated fungicide use has selected resistant populations, including carbendazim-resistant isolates (Gupta et al., 2014; Bandara et al., 2020; Shang et al., 2024). Similar concerns apply to other sclerotia-forming pathogens, including *Neopestalotiopsis*, *Macrophomina phaseolina*, and *Sordaria sclerogenia*, which cause diverse diseases and persist under heat, drought, or low temperature (Gupta et al., 2020; Rebollar-Alviter et al., 2020; Yousef et al., 2025b). Management must therefore account for contrasting ecological niches, including cool, humid environments favoring *S. sclerotiorum* and hot, dry conditions

Received 4 May 2026; accepted 25 July 2026.

preferred by *M. phaseolina* (Gupta et al., 2014; Rebollar-Alviter et al., 2020). This review synthesizes sclerotial ecology, resilience, and control, emphasizing *Trichoderma*-based synergistic strategies and environmental stress effects on sustainable, immunity-oriented crop protection.

REVIEW METHODOLOGY

Search Strategy and Information Sources

A structured search of PubMed, Web of Science, Scopus, and Google Scholar covered peer-reviewed studies and authoritative reviews published between 2014 and 2026. Priority was given to sclerotial biology and molecular biocontrol, especially transcriptional regulation, the Velvet complex, and environmentally induced melanization linked to sclerotial persistence.

Keywords and Search Terms

Boolean search strings covered molecular, ecological, and applied aspects of the topic. Pathogen-related terms included "sclerotia-forming fungi", *Sclerotinia sclerotiorum*, *Sclerotium rolfsii*, *Macrophomina phaseolina*, and *Sordaria sclerogenia*. Additional terms targeted persistence mechanisms ("melanized rind structure", "DHN-melanin pathway", "SCD1", "THR1", "transcription factor SsSMR1", "Velvet complex"), management strategies ("metabolic priming of Trichoderma", "tripartite plant-microbe interactions", "induced systemic resistance", "DAMP-triggered immunity", "abiotic elicitors"), and environmental drivers ("climate change effects on sclerotial survival", "heat-induced melanization", "sustainable soil sanitation").

Inclusion Criteria

Studies were included when they advanced mechanistic understanding or practical management of sclerotia-forming pathogens. Priority was assigned to molecular regulation of sclerotial formation, persistence, and degradation, as well as to integrated strategies involving *Trichoderma* spp., arbuscular mycorrhizal fungi, and abiotic elicitors. Comparative evidence across pathogens was retained, with in vitro, greenhouse, and field validation prioritized. Approximately 33 studies were synthesized to examine the link between sclerotial resilience and biologically based management.

BIOLOGY AND ECOLOGICAL RESILIENCE OF SCLEROTIA-FORMING FUNGI

Molecular Mechanisms of Morphogenesis and Antagonistic Intervention

Sclerotial development is tightly regulated and supports survival and disease establishment in many soil-borne fungi. The Velvet complex (VeA, VelB, and LaeA) links development with secondary metabolism; its disruption markedly impairs or abolishes sclerotial formation (Calvo and Cary, 2015; Huang et al., 2025). Calcium signaling also shapes early morphogenesis by controlling hyphal differentiation and, consequently, sclerotial initiation, abundance, and maturation (Pan et al., 2024).

Against these structures, *Trichoderma* spp. act beyond direct mycoparasitism. Their antagonism reflects metabolic plasticity, as abiotic elicitors such as salicylic acid and selected organic salts can stimulate the production of antifungal metabolites, including volatile organic compounds and phenolic derivatives absent under basal conditions (Yousef et al., 2018). This response is relevant to sclerotial pathogens, whose structural defenses often restrict conventional antagonism. Scanning electron microscopy has shown severe sclerotial damage after exposure to antagonistic *Trichoderma* strains, including disruption of the melanized rind and collapse of internal cellular organization, thereby weakening persistence and increasing susceptibility to biological degradation (Yousef et al., 2025a).

Structural Resilience: The Melanized Shield

Mature sclerotia comprise three layers: an outer pigmented rind, a cortical layer, and an inner medulla (Ordóñez-Valencia et al., 2015). This architecture supports soil persistence, particularly through the rind, where DHN-melanin accumulates under

regulation by genes such as SCD1 and THR1 (Liang et al., 2018). The melanized outer layer functions as a durable barrier, enhancing survival under adverse conditions.

Functionally, the rind reduces ultraviolet injury, restricts fungicide penetration, and limits enzymatic degradation by non-adapted or unprimed soil microorganisms (Salazar-García et al., 2022). These features make it a central determinant of sclerotial resilience and a major obstacle to effective control.

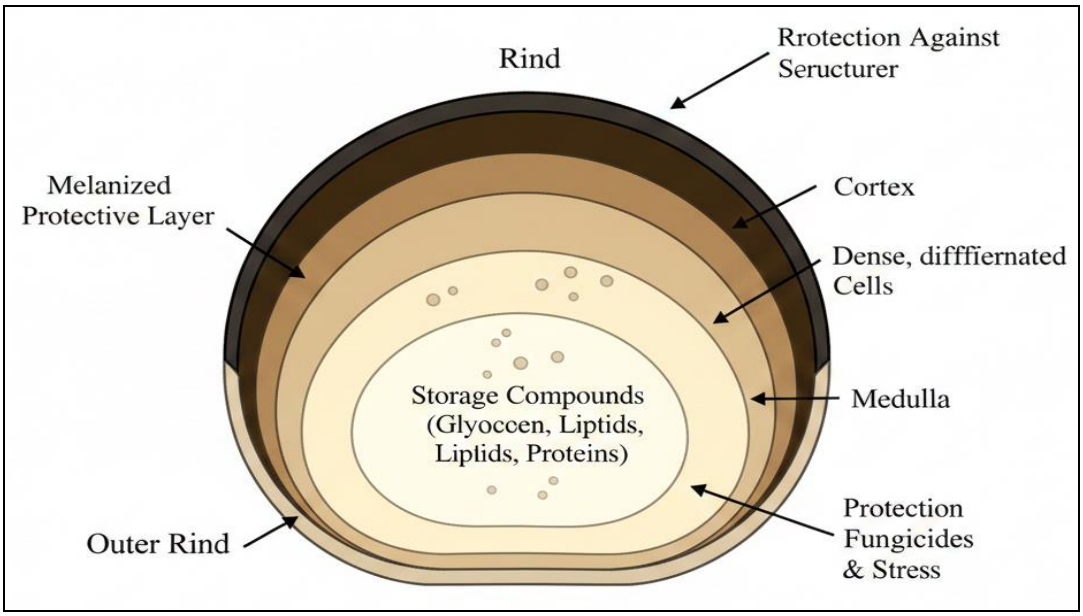

Figure 1. Diagrammatic cross-section of a *Sclerotinia sclerotiorum* sclerotium showing its three-layered structure: melanized rind, cortex, and medulla. The outer rind provides protection against fungicides and environmental stress, while the medulla contains storage compounds such as glycogen, lipids, and proteins (adapted from Shang et al., 2024).

Environmental Adaptation and Climate Change Dynamics

The resilience of *Sclerotinia sclerotiorum* depends on maintaining sclerotial viability under environmental change. Temperature and soil moisture regulate development and disease expression: cool, humid conditions generally favor carpogenic germination and infection, whereas long-term persistence depends on sclerotia remaining intact in fluctuating soils (Butler et al., 2009; Cohen, 2022; Hossain et al., 2023). Rind melanization, therefore, acts as an adaptive trait that increases tolerance to oxidative stress, microbial attack, and environmental exposure, sustaining the soil inoculum reservoir across variable climates (Figure 2).

Accordingly, *S. sclerotiorum* epidemiology is better interpreted through climate variability than static conditions. Temperature and moisture shifts can alter local suitability for survival, germination, and spread, affecting spatial and seasonal disease expression (Cohen, 2023). Because sclerotia persist in soil, the pathogen can exploit transient favorable conditions. Rhizosphere interactions with *Trichoderma* spp. may support resilience by improving nutrient uptake, modulating root-associated microbial activity, and priming induced systemic resistance, particularly under abiotic stress (Hussein et al., 2024; Jung et al., 2024). These findings support climate-informed management integrating pathogen persistence, host responsiveness, and biocontrol performance.

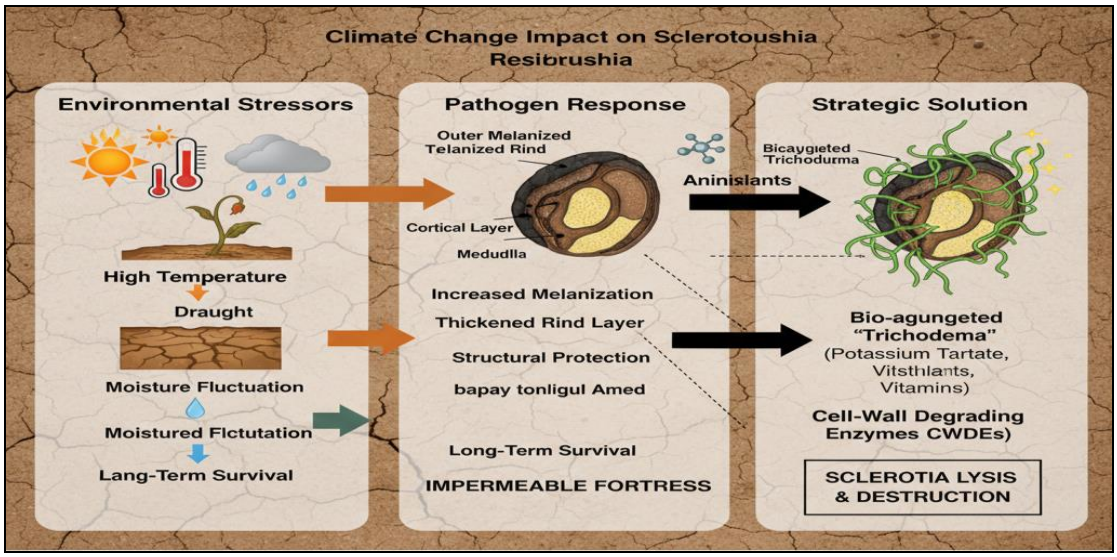

Figure 2. Effects of environmental stressors on the structural resilience and persistence of fungal sclerotia. The diagram shows how abiotic stressors, particularly high temperatures, drought, and fluctuations in soil moisture, induce physiological adaptations in the pathogen. These conditions promote rind melanization and cortical thickening, thereby enhancing long-term survival in soil and increasing resistance to microbial degradation.

Synergistic Potential of Abiotic Inducers and Biocontrol Agents

Because sclerotia persist in soil for extended periods, their suppression usually requires integrated rather than single-component management. Abiotic inducers such as salicylic acid may complement biological control by enhancing host defense readiness and antagonist performance. As a regulator of systemic acquired resistance, salicylic acid can restrict disease by reinforcing defense expression against invading pathogens.

In these systems, *Trichoderma* acts beyond direct antagonism. Besides mycoparasitism and secretion of cell wall-degrading enzymes, it can activate host resistance through hormone-responsive signaling. Current evidence indicates that this protection is largely ISR-related, with possible SAR-like components depending on host, strain, and environment. Combined with abiotic defense activators, *Trichoderma* may prime host immunity while weakening sclerotial structures through enzymatic and metabolic attack. Sclerotial degradation may also release damage-associated molecular patterns, thereby strengthening local and systemic responses. Thus, combining defense-activating compounds with effective biological antagonists may reduce inoculum and improve host responsiveness before pathogen establishment.

PATHOGENICITY AND GLOBAL ECONOMIC IMPACT

Versatile Infection Strategies and Host Colonization

Sclerotia-forming pathogens infect hosts through coordinated biochemical and structural mechanisms. In *Sclerotinia sclerotiorum*, oxalic acid acidifies host tissues, alters redox balance, and suppresses early defenses, including oxidative burst and callose deposition, favoring necrotrophic growth. The pathogen also secretes cell wall-degrading enzymes, including glycoside hydrolases, that promote penetration, tissue maceration, and lesion expansion.

The shift from dormant sclerotia to active germination converts persistent inoculum into primary infection, making this stage a key biological intervention target. Antagonists such as *Trichoderma* can inhibit germination through lytic enzymes, mycoparasitism, and inhibitory metabolites, compromising propagule integrity before colonization. The economic importance of these pathogens reflects durable survival structures and aggressive infection strategies, underscoring the need for integrated

molecular, ecological, and biological management approaches (Figure 3).

Although *Sclerotinia sclerotiorum* remains the most consequential sclerotia-forming pathogen, other fungi with persistent survival structures are becoming important in high-value crops. Recent ultrastructural investigations of *Sordaria sclerogenia* have described distinctive perithecial morphogenesis, rosette-like ascospores, virulence-associated metabolites, and mechanisms of root invasion, broadening current understanding of structurally differentiated soil-associated fungal pathogens (Yousef et al., 2026). These pathogens cause fruit rot, shoot dieback, root disease, and post-harvest decay in specialty systems, extending sclerotial or sclerotium-like survival biology beyond the classical *Sclerotinia* pathosystem. Their effects include reduced yield and quality, shorter shelf life, and higher management costs under recurrent disease pressure. Repeated outbreaks increase fungicide dependence and selection for reduced sensitivity; in *S. sclerotiorum*, documented carbendazim resistance underscores the need to diversify management beyond single-site chemistries.

Molecular Orchestration of Pathogenesis

In sclerotia-forming fungi, pathogenicity is closely linked to developmental regulation. The Velvet system, particularly the VeA-VelB-LaeA complex, coordinates differentiation, secondary metabolism, and infection-related traits. In *S. sclerotiorum*, disruption of this complex severely impairs sclerotial development and alters genes involved in cell wall degradation and infection, indicating integration between sclerotiogenesis and virulence. Calcium signaling also influences early morphogenetic transitions linked to sclerotial formation. Although these pathways are mechanistically important, their direct use as management targets remains prospective and requires pathosystem-specific validation. Their convergence on development and virulence makes them plausible targets for future RNA-based, microbial-interference, or signaling-disruption strategies.

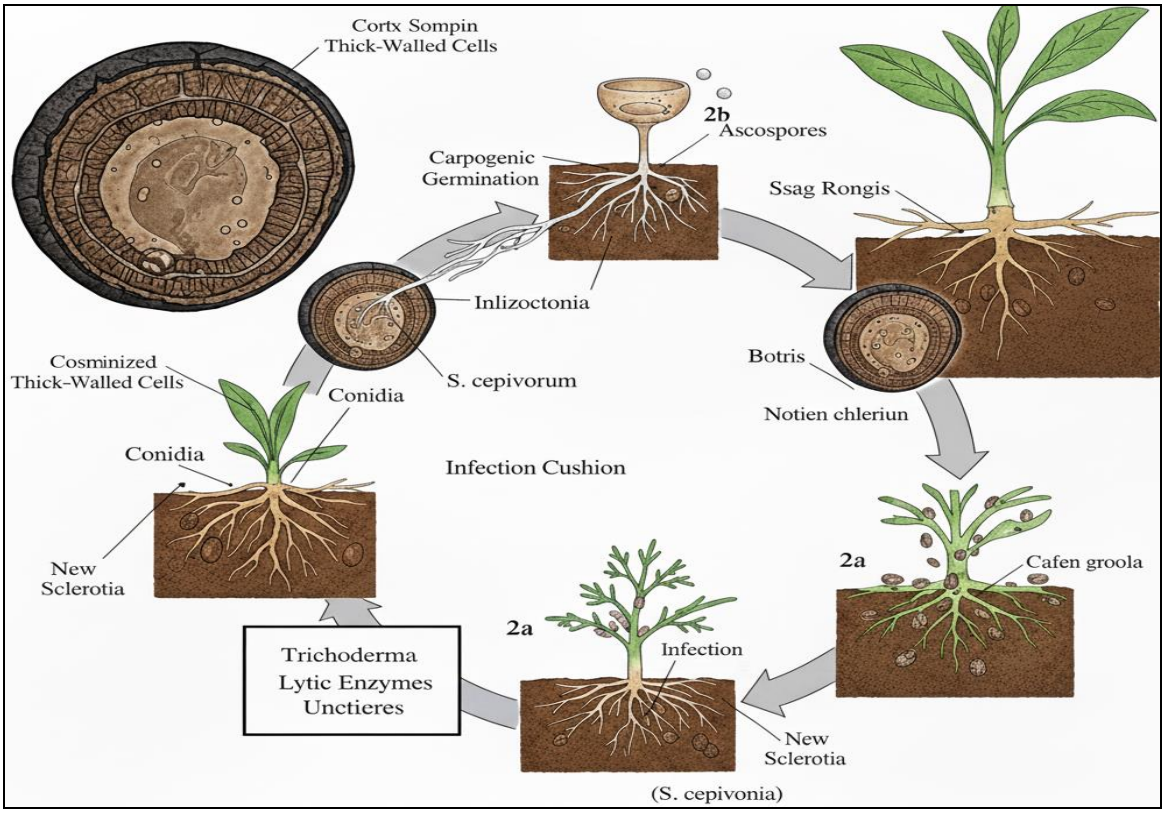

Figure 3. Schematic representation of the pathogenic life cycle of sclerotia-forming fungi and antagonistic intervention by *Trichoderma* spp. The diagram depicts sclerotial germination, host colonization, and secondary inoculum production, together with the inhibitory action of *Trichoderma* lytic enzymes on pathogen structural integrity. 4.2. Emerging Pathogens and Economic Consequences.

LIMITATIONS OF CONVENTIONAL CHEMICAL CONTROL AND THE RISE OF RESISTANCE

The Resistance Crisis: Structural and Genetic Barriers

Control of sclerotia-forming pathogens has long relied on synthetic fungicides, particularly benzimidazoles and dicarboximides, but efficacy is increasingly constrained by structural protection and adaptive resistance. The melanized rind shields internal tissues and likely reduces fungicide activity against dormant survival structures. Although efficacy varies with compound, dose, timing, and target stage, intact sclerotia remain a major cause of incomplete suppression.

Repeated use of single-site fungicides imposes strong selection pressure on pathogen populations. In *S. sclerotiorum*, carbendazim resistance is documented, and reduced sensitivity to other fungicide classes has been reported in some populations. Because resistance varies among active ingredients, compound-specific interpretation is preferable to broad generalization. These patterns make resistance management essential in fungicide-based programs targeting sclerotia-forming pathogens.

Ecological Consequences and Microbiome Disruption

Heavy reliance on fungicides also has ecological costs. Chemical exposure can disrupt non-target soil microbial communities, affecting nutrient cycling, natural disease suppression, and beneficial root-associated interactions. Because *Trichoderma* spp. and arbuscular mycorrhizal fungi support disease suppression, nutrient acquisition, and stress tolerance, their disruption may weaken rhizosphere buffering capacity and indirectly favor pathogen persistence.

These limitations support a shift from fungicide-centered control to integrated strategies combining biological antagonists with host-defense activators or physiological inducers. Such approaches target active infection and survival structures driving recurrence.

INTEGRATED MANAGEMENT: MICROBIOME ENGINEERING AND SYNERGISTIC STRATEGIES

Trichoderma and Biostimulant Synergy

Integrated management increasingly emphasizes rhizosphere optimization rather than simply adding biocontrol agents. *Trichoderma harzianum*, applied alone or in combination with *Bacillus subtilis*, improved plant vigor and reduced disease pressure under field conditions, supporting its potential as a component of integrated disease management systems (Petcu et al., 2023). Recent greenhouse evidence further demonstrates that integrating *Trichoderma asperelloides* with chitosan and biosynthesized silver nanoparticles can strongly suppress onion white rot caused by the persistent sclerotia-forming pathogen *Sclerotium cepivorum***,** although field-scale validation and assessment of non-target effects remain necessary (El-Debaiky et al., 2026). Abiotic supplements or biostimulants may metabolically prime *Trichoderma* spp., enhancing physiological readiness in a strain- and formulation-dependent manner. Abiotic supplements or biostimulants may metabolically prime *Trichoderma* spp., enhancing physiological readiness in a strain- and formulation-dependent manner. This is plausible because *Trichoderma* is a multifunctional plant-beneficial fungus that combines growth promotion, resistance induction, antibiosis, and mycoparasitism, all of which are shaped by environmental and nutritional context.

Such priming likely enhances, rather than redefines, the existing antagonistic machinery of *Trichoderma*. Under suitable conditions, it may increase the production of hydrolytic enzymes, antimicrobial metabolites, and volatile compounds, thereby improving biomass production, sporulation, or antifungal performance. This is relevant against sclerotial pathogens, whose melanized survival structures resist single-mechanism suppression. Microscopic observations show *Trichoderma*-mediated directional growth, attachment, hyphal coiling, and appressorium-like structures

enabling localized enzymatic disruption of resistant propagules.

The rationale for combining *Trichoderma* with arbuscular mycorrhizal fungi lies in their complementary functions. AMF enhance nutrient acquisition and influence host transcriptional responses, whereas *Trichoderma* provides direct antagonism and defense priming. With selected elicitors, these organisms form a multilayered system that links inoculum suppression, rhizosphere reinforcement, and induced resistance to durable control of persistent soil-borne pathogens.

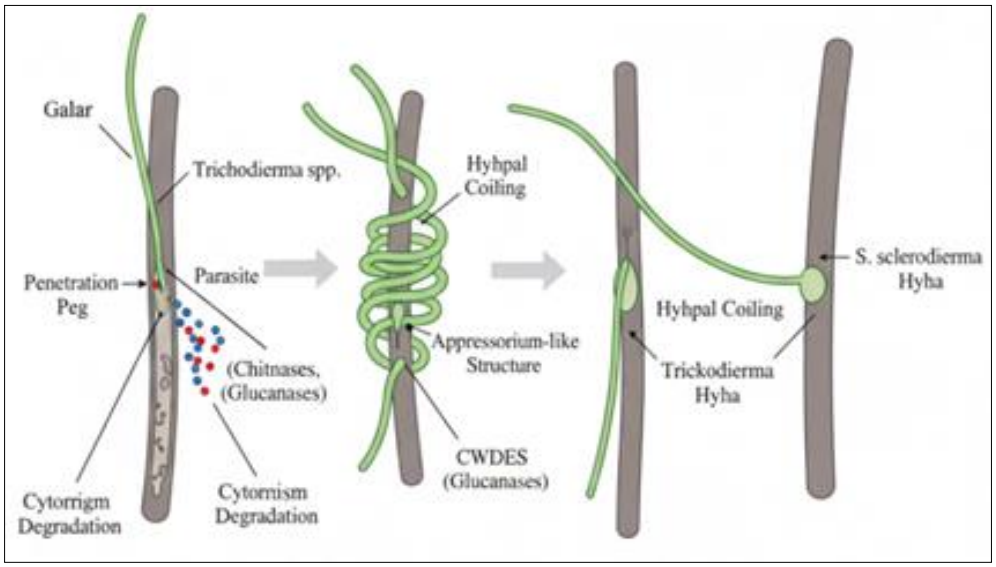

Figure 4. Schematic representation of the synergistic mycoparasitic action of biostimulant-augmented *Trichoderma* spp. against *S. sclerotiorum*. (A) Biostimulant-induced priming enhances the metabolic readiness of *Trichoderma* mycelia. (B) The recognition phase involves close physical interaction and intensive hyphal coiling around the pathogen. (C) Appressoria-like structures form at points of contact, enabling mechanical and enzymatic penetration. (D) The coordinated action of cell wall-degrading enzymes (CWDEs) drives progressive degradation of the melanized sclerotial rind and cortical tissues, leading to sclerotial viability loss. (Based on mechanisms established in Yousef et al., 2016, 2018, 2025).

Enzymatic Deconstruction of the Sclerotial Fortress

Effective biological control of sclerotia-forming pathogens depends largely on disrupting the sclerotial rind. In *Trichoderma*-mediated mycoparasitism, this occurs mainly through cell wall-degrading enzymes, especially chitinases and β-1,3-glucanases, which support host recognition, wall hydrolysis, and nutrient acquisition. As these barriers degrade, resistant propagules lose structural integrity, reducing survival and germination potential.

Microscopic observations often show erosion, pitting, cracking, and collapse of sclerotial tissues after enzymatic attack. However, such damage should be described as substantially reducing viability and germination rather than achieving complete soil decontamination, which depends on inoculum density, soil conditions, antagonist persistence, and in situ colonization. Enzymatic degradation remains a central mechanism by which *Trichoderma* destabilizes the melanized barrier underlying long-term inoculum persistence.

Induction of Systemic Resistance (ISR)

Beyond direct antagonism, *Trichoderma* can strengthen plant defense by inducing or priming systemic resistance. After root colonization, treated plants often exhibit greater activation of defense-related enzymes and signaling pathways, enabling faster responses to pathogen challenge. Across pathosystems, *Trichoderma*-based treatments have been associated with increased peroxidase and polyphenol oxidase activity and broader activation of pathways linked to phenylpropanoid metabolism and

pathogenesis-related responses (Figure 5). Cross-pathosystem evidence also indicates that chitosan can activate salicylic acid-associated defenses, increase phenolic accumulation, and enhance the activities of chitinase, β-1,3-glucanase, peroxidase, and polyphenol oxidase, supporting its potential role as a complementary resistance elicitor (Mahmoud et al., 2025).

This response is valuable because *Trichoderma* suppresses pathogens through mycoparasitism and enzymatic degradation while physiologically priming the host against subsequent invasion. Specific marker genes such as PAL1 and CHI II should be retained only when directly supported by cited studies; otherwise, it is more rigorous to state that *Trichoderma* activates defense-associated enzymatic and transcriptional networks consistent with induced systemic resistance. Under variable environments, combined inoculum suppression and immune priming remain important components of sustainable management for persistent soil-borne pathogens.

Figure 5, proposed model for ISR triggered by bio-augmented *Trichoderma* spp.

The figure depicts a multistage rhizosphere-driven defense response in which bioaugmented Trichoderma enhances the production of elicitor-active compounds during root colonization, generates root-to-shoot systemic signals, and promotes distal defense activation, including increased peroxidase, polyphenol oxidase, phenolic compounds, and resistance-associated genes. This model represents "systemic vaccination" as a mechanistic interpretation of current evidence rather than a fully resolved pathway.

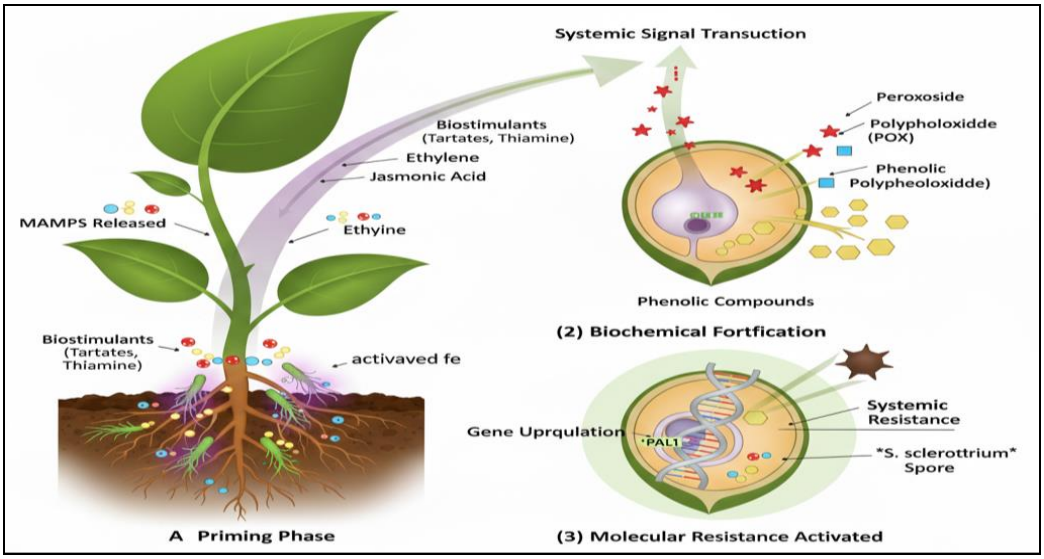

Figure 5. Proposed model of induced systemic resistance (ISR) triggered by bio-augmented *Trichoderma* spp. The numbered stages depict a systemic "vaccination" effect: (1) priming and recognition, in which rhizosphere interactions between *Trichoderma* and biostimulants activate molecular elicitors; (2) systemic signaling, involving long-distance movement of defense signals from roots to distal leaves; and (3) defense execution, marked by rapid accumulation of biochemical markers (POX, PPO, and phenolics) and upregulation of resistance genes (*PAL1* and *CHI II*), culminating in systemic protection.

Comparative Evaluation of Management Strategies

This section summarizes the shift from chemical control to bio-augmented strategies. As shown in Table 1, integrating *Trichoderma* with biostimulants can reduce sclerotial viability and improve plant fitness more effectively than traditional approaches.

DISCUSSION AND SYNTHESIS: MECHANISTIC INSIGHTS AND STRATEGIC IMPLICATIONS

Persistence in sclerotia-forming pathogens, particularly *Sclerotinia sclerotiorum*, reflects an integrated survival strategy rather than structural defense alone. The melanized rind

protects the medulla from physical, chemical, and biological stress, enabling long-term soil persistence. Environmental instability, including temperature extremes, fluctuations in moisture, and oxidative stress, may enhance melanization and cortical reinforcement, extending longevity and complicating eradication (Butler et al., 2009; Sousa Melo et al., 2019; Cohen, 2022). This explains the limited success of fungicide-based control: benzimidazoles and dicarboximides are constrained by poor access to protected tissues and by resistance or reduced sensitivity in pathogen populations (Gupta et al., 2014; Salazar-García et al., 2022). Continued chemical dependence may also disrupt the soil microbiome by suppressing antagonists such as *Trichoderma* spp. and beneficial symbionts, including arbuscular mycorrhizal fungi (AMF), weakening rhizosphere buffering and favoring pathogen re-establishment (Talmo and Ranjan, 2025).

Table 1. Comparative analysis of control strategies against sclerotia survival structures

Strategy	Mechanism of Action	Key Advantages	Limitations	Key References
Chemical Control	Inhibition of mycelial mitosis and respiration.	Rapid initial suppression of vegetative growth.	Soil toxicity; minimal penetration of melanized rind; emergence of MDR strains.	Butler et al. (2009); Gupta et al. (2014)
Traditional Biocontrol	Mycoparasitism and competition for space/nutrients.	Environmentally sustainable; eco-friendly.	High variability under field conditions; slow action against mature sclerotia.	Silva et al. (2022); Ziedan (2022)
Enhanced Biostimulation (Trichoderma + Inducers)	Metabolic priming; synergistic activation of CWDEs (GH family); host immune priming (ISR/SAR).	Maximum degradation of melanized shield; effective under fluctuating climates.	Requires specialized formulation and precise timing.	Yousef et al. (2018, 2025a); El-Sharkawy et al. (2025); Lee et al. (2026)
Climate-Based Modeling	Predictive analytics based on precipitation and thermal thresholds.	Early-warning system for precision application.	Requires high-resolution climate data; site-specific validation.	Cohen (2022); Mapfumo et al. (2024)

Table 2. Evidence map for *Trichoderma*-based priming against sclerotial pathogens: from in vitro assays to greenhouse and field validation

Evidence axis (what is being claimed/tested)	In vitro evidence (lab/plate/microscopy)	Greenhouse/growth-chamber evidence (plant-pathogen outcomes)	Field evidence (multi-site realism)	References
1. Direct antagonism & mycoparasitism of sclerotia	Hyphal attachment/coiling; SEM evidence of rind erosion; inhibition in dual-culture assays	Reduced disease severity after soil/root treatment; improved plant vigor under challenge inoculation	Often limited or heterogeneous; requires multi-location replication	Yousef et al. (2016, 2018, 2025c); Silva et al. (2022)
2. Cell-wall-degrading enzymes (CWDEs) targeting sclerotial tissues	Increased chitinase/beta-1,3-glucanase/protease activity; structural pitting/cracking of rind	Lower sclerotial viability/germination potential translated into reduced infection	Rarely quantified directly in the field; usually inferred from disease suppression	Khan et al. (2020); Yousef et al. (2025b)
3. Induced systemic resistance (ISR)/ host priming	Upregulation of defense markers (phenolics, PPO/POX); defense-gene induction (e.g., PAL, CHI)	Reduced lesion expansion/necrosis; improved tolerance under pathogen pressure	Field-scale durability depends on cultivar x environment x formulation	El-Sharkawy et al. (2025); Zeilinger et al. (2016)
4. "Stacked priming": Trichoderma + elicitors/biostimulants	Enhanced antagonism and/or metabolite/VOC profiles; stronger suppression in dual-culture assays	Improved growth + stronger defense readiness; synergy depends on dose/timing	Promising but under-validated; needs robust agronomic trials	Yousef et al. (2025a); El-Sharkawy et al. (2025)
5. Environment-dependent performance & decision support (timing/forecasting)	Not primarily a lab axis; relies on datasets and mechanistic understanding	Used to interpret stress-condition outcomes and consistency	Strongest when built on repeated seasonal datasets and management contexts	Mapfumo et al. (2024)

Note: Evidence strength typically decreases from controlled assays (in vitro) to open-field complexity; "field evidence" should be reserved for replicated trials under agronomic conditions.

Bio-enhanced *Trichoderma* spp. offers an alternative by combining direct antagonism with host- and rhizosphere-mediated effects. Abiotic inducers, including B-vitamins, antioxidants, and organic acids, appear to enhance growth, sporulation, and secretion of cell wall-degrading enzymes and antifungal metabolites (Yousef et al., 2016, 2018, 2025a). Primed *Trichoderma* also forms intensive hyphal coiling and appressoria-like structures that disrupt the melanized rind, reducing viability of the pathogen's main survival structure (Yousef et al., 2018). Its value increases with AMF and the host plant, coupling suppression with stronger immune competence, including upregulation of PAL1, CHI II, and HQT, and increased peroxidase and polyphenol oxidase activity (El-Sharkawy et al., 2025). Salicylic acid may further increase pathogen susceptibility by destabilizing fungal growth (Yousif, 2019).

Key uncertainties remain, especially the field stability of metabolically enhanced *Trichoderma*, consistency across abiotic environments, and interactions with native microbiota. Future work should prioritize delivery systems, including encapsulation and nano-enabled carriers, and should incorporate metagenomic and metabolomic analyses. Overall, evidence supports biologically integrated, climate-aware management that weakens survival structures, restricts establishment, and strengthens host defense (Table 2).

Strategic Frontiers for Future Research

Translating *Trichoderma*-based biocontrol into field-ready, climate-resilient management requires progress in formulation, strain performance, and rhizosphere ecology. Precision delivery is critical because inconsistent post-application survival and activity limit field efficacy. Nano-enabled or biodegradable carriers may protect *Trichoderma* propagules and metabolic inducers from desiccation, ultraviolet exposure, and microbial competition, while improving placement near sclerotial inoculum during germination and early infection. Formulation, therefore, determines whether laboratory success translates into field performance.

A second priority is climate-adaptive strains. Because temperature extremes, irregular rainfall, and water limitation affect pathogen and antagonist, future work should target *Trichoderma* strains with stronger thermotolerance, osmotic resilience, rhizosphere competence, and sustained hydrolytic-enzyme and antifungal-metabolite production under stress. Evidence from thermally stressed root-rot pathosystems indicates that endophytic microbial antagonists and metabolically active biocontrol fungi may retain disease-suppressive functions under elevated-temperature conditions, supporting the selection of climate-adapted strains for future formulations (Farag et al., 2026; Fouad et al., 2026). A third frontier is the integration of rhizosphere microbiomes. Metagenomic, metatranscriptomic, and metabolomic approaches can clarify interactions between introduced *Trichoderma* and indigenous microbiota and their effects on soil suppressiveness and persistence. Together, these priorities support precision microbiome engineering and scalable, biologically integrated crop protection.

CONCLUSIONS

Reliance on synthetic fungicides is becoming increasingly unsustainable in pathosystems dominated by persistent sclerotia-forming fungi. The structural protection of sclerotia, together with rising fungicide resistance, has reduced the durability of chemical control and exposed the limits of repeated external suppression. More effective management must instead act simultaneously on the pathogen, the host, and the surrounding soil microbiome. Three principles emerge from this review. Persistent inoculum must be targeted by disrupting the mechanisms underlying long-term survival. Host defense should be reinforced through systemic priming and biologically mediated resistance. At the same time, sustainable suppression depends on preserving the microbial complexity of the rhizosphere that supports antagonism and ecological stability. Together, these

principles support a multilayered strategy that reduces inoculum pressure, stabilizes crop performance, and improves resilience under variable environmental conditions. For sclerotia-forming pathogens, sustainable control is therefore more likely to come from integrated metabolic priming, microbial synergy, and microbiome-aware management than from further escalation of chemical inputs.

FUNDING

The authors acknowledge receiving funding from the Deanship of Scientific Research, Vice Presidency for Graduate Studies and Scientific Research at King Faisal University, Saudi Arabia, under Project No. KFU 263525, for supporting this research work.

ACKNOWLEDGEMENTS

The authors extend their appreciation to the Deanship of Research and Graduate Studies at King Khalid University for funding this work under grant number RGP2/419/47.

REFERENCES

Bandara, A.Y., Weerasooriya, D.K., Bradley, C.A., Allen, T.W., Esker, P.D., 2020. *Sclerotinia sclerotiorum (Lib.) de Bary: Biology and molecular traits of a cosmopolitan pathogen*. Frontiers in Plant Science, 11, 976.

Boland, G.J., and Hall, R., 1994. *Index of plant hosts of Sclerotinia sclerotiorum*. Canadian Journal of Plant Pathology, 16(2): 93-108.

Butler, M.J., Gardiner, R.B., Day, A.W., 2009. *Melanin synthesis by Sclerotinia sclerotiorum*. Mycologia, 101(3): 296-304.

Calvo, A.M., and Cary, J.W., 2015. *Association of polyketide synthesis with development and virulence in filamentous fungi*. Frontiers in Microbiology, 6, 297.

Cohen, S.D., 2022. *Estimating the Climate Niche of Sclerotinia sclerotiorum Using Maximum Entropy Modeling*. Journal of Fungi, 8(9), 966.

Cohen, S.D., 2023. *Estimating the climate niche of Sclerotinia sclerotiorum and Sclerotinia subarctica through species distribution modeling*. Journal of Fungi, 9(9), 892.

El-Debaiky, S.A., Amer, S.M., El-Shafay, A.A., Abou-Zeid, M., Mahmoud, Y.A.-G., 2026. *Integrated biosynthesized silver nanoparticles, chitosan, and Trichoderma asperelloides for managing onion white rot under greenhouse conditions*. Scientific Reports, 16, 18689. https://doi.org/10.1038/s41598-026-56842-6

El-Sharkawy, H.H.A., Rashad, Y.M., El-Blasy, S.S.A., Amin, B.H., Youssef, M.A.A., Hafez, M., Abd-ElGawad, A., Bourouah, M., Elsherbiny, E.A., Yousef, S.A., 2025. *Mycorrhizal symbiosis and application of vitamin B3-treated Trichoderma harzianum HE24 additively trigger immunity responses in faba bean plants against Rhizoctonia root rot and promote the plant growth and yield*. BMC Plant Biology, 25, 926. https://doi.org/10.1186/s12870-025-06950-8

Farag, F.M., Ghebrial, E.W.R., Mabrouk, O.I., Abou-Zeid, M.A., 2026. *Impact of endophytic bacterial and fungal isolates on tomato root rot under thermal stress*. Egyptian Journal of Phytopathology, 54(1): 103-128. DOI:10.21608/EJP.2026.454148.1173

Fouad, M.S., Ramadan, K.M.A., Mohamed, H.A., Abou-Zeid, M.A., Ibrahim, S.D., Munir, M., Bendary, E.S.A., Aboul Fotouh, M.M., Al Hashedi, S.A., AlShoaibi, A., Iqbal, Z., Ismail, A.M., El-Ghareeb, W.R., Najeeb, K.M.A., 2026. *Exploiting the biocontrol potential of Chaetomium globosum against Fusarium solani root rot in wheat: Climate-responsive phytohormonal regulation, genetic diversity, and sustainable management*. European Journal of Plant Pathology. Advance online publication. https://doi.org/10.1007/s10658-026-03242-1

Gupta, G.K., Sharma, S.K., Ramteke, R., 2012. *Biology, epidemiology and management of the pathogenic fungus Macrophomina phaseolina (Tassi) Goid with special reference to charcoal rot of soybean [Glycine max (L.) Merrill]*. Journal of Phytopathology, 160(4): 167-180. doi:10.1111/j.1439-0434.2012.01884.x

Hossain, M.M., Sultana, F., Li, W., Tran, L.-S.P., Mostofa, M.G., 2023. *Sclerotinia sclerotiorum (Lib.) de Bary: Insights into the pathogenomic features of a global pathogen*. Cells, 12(7), 1063. https://doi.org/10.3390/cells12071063

Huang, W., Mu, Y., Miao, Y., Li, J., Yu, H., Zhou, Y., Li, G., Ma, H., Xu, L., 2025. *The Velvet complex is essential for sclerotia formation and virulence in Sclerotinia sclerotiorum*. Journal of Fungi, 11(11), 786. https://doi.org/10.3390/jof11110786

Hussein, A.N., AL-Amery, S.M.H., Abdulabbas, H.S., AL-Kinani, M.A.A., Salman, H.S., 2024. *The effects of interaction between Trichoderma fungi and arbuscular mycorrhizal fungi on protect and improve the growth of plants*. GSC Biological and Pharmaceutical Sciences, 29(3): 276-285. https://doi.org/10.30574/gscbps.2024.29.3.0488

Jung, J., Gutierrez Burbano, A., Rutter, W.B., Ingram, T., Smith, D.L., Subramanian, S., 2024. *Triple*

interactions for induced systemic resistance in plants. Frontiers in Plant Science, 15, 1464710. https://doi.org/10.3389/fpls.2024.1464710

Khan, R.A.A., Najeeb, S., Hussain, S., Xie, B., Li, Y., 2020. *Bioactive secondary metabolites from Trichoderma spp. against phytopathogenic fungi*. Microorganisms, 8(6), 817. https://doi.org/10.3390/microorganisms8060817

Lee, G.H., Min, C.W., Wang, Y., Gupta, R., Kim, S.T., 2026. *Understanding the functional role of cell wall-degrading enzymes in Magnaporthe oryzae and their interactions with the rice immune system*. Plant Physiology and Biochemistry, 231, 111027.

Liang, Y., Xiong, W., Steinkellner, S., Feng, J., 2018. *Deficiency of the melanin biosynthesis genes SCD1 and THR1 affects sclerotial development and vegetative growth, but not pathogenicity, in Sclerotinia sclerotiorum*. Molecular Plant Pathology, 19(6): 1444-1453. https://doi.org/10.1111/mpp.12627

Mahmoud, E., Hussien, Z.N., Shehata, A.G.S., Marraiki, N., Almanzalawi, E., Alqahtani, T., Abou-Zeid, M., Kamhawy, M., 2025. *Induction and expression of systemic resistance to downy mildew disease in grapevine by chitosan*. Notulae Botanicae Horti Agrobotanici Cluj-Napoca, 53(1), 14265. https://doi.org/10.15835/nbha53114265

Mapfumo, P., Buthelezi, S., Archer, E., Swanevelder, D.Z.H., Wilken, P.M., Creux, N.M., 2024. *In-field climatic factors driving Sclerotinia head rot progression across different sunflower planting dates*. Plant Pathology, 73: 1112-1126. https://doi.org/10.1111/ppa.13873

Ordóñez-Valencia, C., Ferrera-Cerrato, R., Quintanar-Zúñiga, R.E., Flores-Ortiz, C.M., Márquez-Guzmán, G.J., Alarcón, A., Larsen, J., García-Barradas, O., 2015. *Morphological development of sclerotia by Sclerotinia sclerotiorum: A view from light and scanning electron microscopy*. Annals of Microbiology, 65(2): 765-770. doi:10.1007/s13213-014-0916-x

Pan, K.-Y., Liu, H.-H., Tseng, M.-N., Chang, H.-X., 2024. *Ca2+ affects the hyphal differentiation to sclerotia formation of Athelia rolfsii*. Microbiology Spectrum, 12(6), e00200-24. https://doi.org/10.1128/spectrum.00200-24

Petcu, V., Bubueanu, C., Casarica, A., Săvoiu, G., Stoica, R., Bazdoaca, C.M., Lazăr, D.A., Iordan, H.L., Horhocea, D., 2023. *Efficacy of Trichoderma harzianum and Bacillus subtilis as seed and vegetation application combined with integrated agroecology measures on maize*. Romanian Agricultural Research, 40: 439-448. https://doi.org/10.59665/rar4041

Rebollar-Alviter, A., Silva-Rojas, H.V., Fuentes-Aragón, D., Acosta-González, U., Martínez-Ruiz, M., Parra-Robles, B.E., 2020. *An emerging strawberry fungal disease associated with root rot, crown rot and leaf spot caused by Neopestalotiopsis rosae in Mexico*. Plant Disease, 104(8): 2054-2059. https://doi.org/10.1094/PDIS-11-19-2493-SC

Salazar-García, L.M., Ortega-Cuevas, R.I., Martínez-Álvarez, J.A., González-Hernández, S.E., Martínez-Álvarez, R.A., Mendoza-Olivares, D., Vázquez, M.Á., Flores-Martínez, A., Ponce-Noyola, P., 2022. *Melanin pathway determination in Sclerotium cepivorum Berk using spectrophotometric assays, inhibition compound, and protein validation*. Microbiology Research, 13(2): 152-166. https://doi.org/10.3390/microbiolres13020013

Shang, Q., Jiang, D., Xie, J., Cheng, J., Xiao, X., 2024. *The schizotrophic lifestyle of Sclerotinia sclerotiorum*. Molecular Plant Pathology, 25(2), e13423. doi:10.1111/mpp.13423

Silva, L.G., Camargo, R.C., Mascarin, G.M., Nunes, P.S.O., Dunlap, C.A., Bettiol, W., 2022. *Dual functionality of Trichoderma*: *Biocontrol of Sclerotinia sclerotiorum and biostimulant of cotton plants*. Frontiers in Plant Science, 13, 983127. https://doi.org/10.3389/fpls.2022.983127

Sousa Melo, B., Voltan, A.R., Arruda, W., Lopes, F.A.C., Georg, R.C., Ulhoa, C.J., 2019. *Morphological and molecular aspects of sclerotial development in the phytopathogenic fungus Sclerotinia sclerotiorum*. Microbiological Research, 229, 126326. https://doi.org/10.1016/j.micres.2019.126326

Talmo, N., and Ranjan, A., 2025. *Comparative insights into soybean and other oilseed crops' defense mechanisms against Sclerotinia sclerotiorum*. Frontiers in Plant Science, 16, 1616824.

Yousef, S.A.M., El-Metwally, M.M., El-Ghareeb, N.R., 2016. *Impact of antioxidants and micronutrients as fungicides alternatives in improvement the antagonism of Trichoderma spp*. Journal of Plant Protection and Pathology, 7(12): 803-808.

Yousef, S.A.M., Salem, S.H., El-Sharkawy, H.H.A., 2018. *Effect of some chemical inducers on antagonistic potential of Trichoderma harzianum against Rhizoctonia solani*. Journal of Plant Protection and Pathology, 9(8): 497-506.

Yousef, S.A.M., Ismail, I.M., El-Shishtawy, H.M., Elsherbiny, E.A., 2025a. *Enhancement of the biocontrol mechanisms of Trichoderma longibrachiatum combined with different supplements for controlling Sclerotinia sclerotiorum*. Microbial Pathogenesis, 204, 107595.

Yousef, S.A., Rashad, Y.M., Elsherbiny, E.A., El-Sharkawy, H.H.A., Shaltout, A.M., Ali, A.A., Deng, J.-X., 2025b. *First report of black root rot of lemon (Citrus limon) caused by Sordaria sclerogenia in Egypt*. New Disease Reports, 51(2), e70027. https://doi.org/10.1002/ndr2.70027

Yousef, S.A.M., Sobhy, S.E., Saleh, A., Hafez, E.E., 2025c. *Utilizing safe biotic and abiotic alternatives to control Botrytis californica and*

enhance strawberry quality. European Journal of Plant Pathology, 173(4).

Yousef, S.A.M., Al-Mutiry, M., Shehata, W.F., Almanzalawi, E.A., Alqahtani, T.M., Al-Zahrani, S.S., Al-Rashed, S., Mostafa, Y.S., Alamri, S.A., Abou-Zeid, M.A., 2026. *Morphogenesis of perithecia and rosette-like ascospores in Sordaria sclerogenia reveals virulence-associated metabolites and ultrastructural mechanisms of root invasion in lemon*. Physiological and Molecular Plant Pathology, 144, 103262. https://doi.org/10.1016/j.pmpp.2026.103262

Yousif, D.Y.M., 2019. *Evaluation of salicylic acid solution on fungus Botrytis cinerea that caused strawberry gray mold*. Plant Archives, 19(1): 229-238.

Zeilinger, S., Gruber, S., Bansal, R., Mukherjee, P.K., 2016. *Secondary metabolism in Trichoderma - Chemistry meets genomics*. Fungal Biology Reviews, 30: 74-90.

Ziedan, E.H., 2022. *Recent advances in the biological control of sclerotia-forming fungi*. Egyptian Journal of Phytopathology, 50(1): 45-60.