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Research Article

# Assessment of Fungicide Efficacy against White Mold Disease (*Sclerotinia sclerotiorum*) in Tomato

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**ABSTRACT**

White mold caused by *Sclerotinia sclerotiorum* is a destructive disease of tomato, leading to severe yield losses under protected and field conditions. Given the limited effectiveness of cultural practices alone, the present study aimed to evaluate the efficacy of commonly used fungicides against *S. sclerotiorum* under laboratory and greenhouse conditions to identify the most effective management options. Seven fungicides were first screened *in vitro* at four concentrations (25-100 µg $ml^{-1}$) to assess mycelial growth inhibition over time. Subsequently, the most promising fungicides were evaluated under greenhouse conditions through seed treatment and foliar application to determine their effects on disease incidence, disease severity, and disease control efficacy. In laboratory assays, azoxystrobin, propiconazole, carbendazim, and sulphur completely inhibited mycelial growth at all tested concentrations and observation intervals, whereas difenoconazole and tebuconazole + trifloxystrobin showed high but incomplete inhibition. Iprodione was consistently the least effective fungicide. Greenhouse experiments confirmed significant effects of fungicides, concentrations, and days after inoculation on disease development. Carbendazim emerged as the most effective fungicide in both seed treatment and foliar application, resulting in the lowest disease incidence and severity and the highest disease control efficacy, followed closely by propiconazole. Sulphur showed moderate effectiveness, while azoxystrobin provided comparatively lower and less consistent control under greenhouse conditions. Overall, carbendazim and propiconazole provided consistent, dose-dependent control of tomato white mold through both seed treatment and foliar application, supporting their optimized use in integrated management of *S. sclerotiorum*.

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## Introduction

Tomato (*Solanum lycopersicum* L.) is the second most widely cultivated vegetable and is regarded as an important cash and industrial crop worldwide (Akhtar et al., 2010). It plays a vital role in human nutrition due to its high nutritional value (Awan et al., 2012). Globally, tomato production is estimated at 186.11 million tons from 4.92 million hectares, while Pakistan contributes approximately 0.79 million tons from 0.067 million hectares (FAOSTAT, 2022). Tomato is susceptible to more than 200 diseases caused by fungi, bacteria, nematodes, and viruses. Major fungal diseases of tomato

include *Sclerotinia* rot, late blight, *Fusarium* wilt, *Fusarium* crown rot, and root rot (Ramyabharathi et al., 2012). Among these, white mold disease, also known as *Sclerotinia* stem and root rot, is one of the most destructive soil-borne diseases of tomato and is caused by *Sclerotinia sclerotiorum* (Lib.) de Bary. This pathogen is highly devastating, infecting susceptible tomato plants and causing yield losses exceeding 50% under favorable conditions (Heffer and Johnson, 2007; Gomaa et al., 2016). In Pakistan, *S. sclerotiorum* has been reported to infect several vegetable crops, including potato and cabbage (Alam et al., 2021; Zahid et al., 2022). The present study reports the occurrence of white mold disease in tomato caused by *S. sclerotiorum*.

*S. sclerotiorum* infects host plants through myceliogenic and carpogenic germination of sclerotia, with infection facilitated by the release of airborne ascospores. Disease development is favored by cool and moist environmental conditions, resulting in rotting of infected plant parts. Characteristic symptoms include the development of white, fluffy mycelium and black sclerotia on infected tissues. In processing tomato crops, myceliogenic germination of sclerotia is considered the primary source of infection, leading to rotting of aerial plant parts that come into contact with the soil (Purdy, 1979; Gao et al., 2014). The ability of sclerotia to survive in soil for more than four years poses a significant challenge for managing white mold disease (Fernando et al., 2004). Soil-borne pathogens are generally difficult to control; therefore, multiple management strategies are employed, including cultural practices, host resistance, fungicides, and biocides. However, cultural methods are often ineffective against *Sclerotinia* stem rot due to the pathogen's wide host range, survival as sclerotia, and infection via airborne ascospores. Resistance to white mold disease is limited because of the pathogen's broad host range and genetic diversity, and no commercially resistant tomato cultivars are currently available, making disease management particularly challenging (Mazumdar et al., 2021).

Fungicide application is one of the most effective approaches for the prevention and control of diseases caused by *S. sclerotiorum*. Fungicides remain the primary in-season management strategy by protecting flowers from infection initiated by airborne ascospores. In addition, fungicide application can significantly increase yield by reducing disease incidence in both susceptible and moderately resistant cultivars (Woodward et al., 2015). Fungicides are broadly classified into two categories: systemic and non-systemic (contact) fungicides. Non-systemic fungicides act preventively by inhibiting spore germination before mycelial growth and host infection. Since their introduction in the 1960s, systemic fungicides have largely replaced non-systemic fungicides due to their higher efficacy and broader spectrum of activity. Systemic fungicides, also known as curative or eradicant fungicides, inhibit or kill fungi after mycelial penetration, thereby preventing further infection and disease spread within the plant (Yuste and Gosttinear, 1999).

Several fungicide classes are commonly used for the management of *S. sclerotiorum*, including quinone outside inhibitors (QoIs), demethylation inhibitors (DMIs), methyl benzimidazole carbamates (MBCs), and succinate dehydrogenase inhibitors (SDHIs) (Peltier et al., 2012; Armenta et al., 2015; Liang et al., 2015; Di et al., 2016; Huzar-Novakowiski et al., 2017). These fungicides possess diverse chemical structures and modes of action for inhibiting the white mold pathogen. Effective fungicide-based management depends on proper timing of application to ensure optimal disease control. Therefore, growers must consider environmental conditions, disease pressure, and associated risks when planning management strategies. However, resistance to fungicides is emerging in pathogen populations due to the site-specific mode of action of many major fungicide classes (Derbyshire and Denton-Giles, 2016). *S. sclerotiorum* is generally considered to have a low risk of resistance development due to its homothallic nature, whereby a single ascospore can produce a sexually reproducing colony, and its limited asexual reproduction (Aldrich-Wolfe et al., 2015).

In the present study, the sensitivity of *S. sclerotiorum* isolates collected from tomato fields in Pakistan to different fungicides was evaluated, providing the first assessment of the response of local pathogen populations to major fungicide classes. Identification of effective fungicides against local isolates will assist farmers in selecting cost-effective treatments while reducing the unnecessary use of chemicals. Moreover, these findings contribute to the development of improved disease management strategies aimed at minimizing crop losses, enhancing tomato productivity, and increasing grower profitability, while establishing baseline information for the sustainable management of white mold disease in tomato.

## Materials and Methods

### Sample collection

Diseased tomato samples infected with white mold were collected during 2021 from the fields of the Vegetable Research Institute (AARI), University of Agriculture, Faisalabad (UAF), as well as from other open fields and plastic tunnels at various locations in Faisalabad. Samples exhibiting typical symptoms of white mold, including water-soaked lesions, white cottony mycelial growth, and the presence of sclerotia on leaves, stems, and fruits, were collected along with healthy samples and placed in brown paper bags. The samples were transported to the Plant Disease Diagnostic Laboratory, Department of Plant Pathology, University of Agriculture, Faisalabad, for fungal isolation. All samples were stored at 4°C in a Haier refrigerator (model HRF-420FLS) until further processing.

### Isolation and identification of the pathogen

The pathogen was isolated from infected leaves, stems, and fruits. Diseased tissues were cut into small segments and surface-sterilized with 0.1% sodium hypochlorite for 2-3 min, followed by two consecutive rinses with sterile distilled water to remove residual disinfectant. The sterilized tissues were dried on sterile filter paper and transferred aseptically to 9-cm Petri plates containing sterilized Potato Dextrose Agar (PDA) medium. The plates were incubated at 22 ± 2°C for 1-2 days. Emerging fungal colonies from infected lesions were sub-cultured onto fresh PDA plates and slants.

The fungus was purified using the hyphal tip technique. Identification of the purified pathogen was based on colony characteristics (mycelial growth, texture, and color) and sclerotial morphology, including time of formation (days after inoculation), growth pattern, shape, size, and number of sclerotia per plate, following the criteria described by Rathi et al. (2018). Molecular characterization was performed using Sanger sequencing, and the resulting sequence was submitted to the NCBI GenBank under accession number PQ656337.

### Pathogenicity test

Pathogenicity of the isolated fungus was confirmed under greenhouse conditions following Koch's postulates (Koch, 1882). Seeds of the susceptible tomato cultivar 'Nagina' were sown in earthen pots (30 cm diameter) filled with sterilized soil. Inoculum was prepared from seven-day-old PDA cultures of the isolated pathogen. One-month-old plants were inoculated by placing 5-mm mycelial discs onto artificially injured stem tissues. Control plants were inoculated with sterile PDA discs only. Disease symptoms were recorded 16 days after inoculation (DAI). The pathogen was re-isolated from symptomatic plants and identified based on symptom similarity and morphological characteristics, thereby confirming pathogenicity (Upadhyay et al., 2019).

### Inoculum preparation

Sclerotia were harvested from actively growing cultures of *S. sclerotiorum* on PDA plates. The collected sclerotia were surface-sterilized with 3% sodium hypochlorite, rinsed thoroughly with sterile distilled water, and dried on sterile filter paper. The dried sclerotia were cut into small pieces and aseptically transferred to PDA-filled Petri plates. After incubation at 22 ± 2°C for three days, newly formed sclerotia were collected, air-dried, and stored in paper bags at 4°C for future use.

For preparation of mycelial suspension, ten fully colonized, seven-day-old PDA plates (9 cm diameter) were homogenized in 1 L of sterile distilled water using a blender, following the method described by Ouhaibi-Ben Abdeljalil et al. (2016).

### Evaluation of fungicides against *S. sclerotiorum* under laboratory conditions

Seven fungicides were evaluated against *S. sclerotiorum* at four concentrations (25, 50, 75, and 100 μg $ml^{-1}$) using the poisoned food technique under laboratory conditions (Table 1). Stock solutions of each fungicide (1 g $ml^{-1}$) were prepared and subsequently diluted with sterile distilled water to obtain the required concentrations. Each concentration was added separately to 15 ml of sterilized PDA medium, poured into 9-cm Petri plates, and allowed to solidify.

The plates were inoculated at the center with a 5-mm mycelial disc taken from a seven-day-old culture of *S. sclerotiorum* and incubated at 22 ± 2°C. PDA plates without fungicide served as the control. The experiment consisted of seven treatments and one control, with three replications arranged in a Completely Randomized Design. Fungicidal efficacy was assessed by measuring mycelial radial growth and calculating percentage growth inhibition at 7, 14, and 21 days after inoculation (DAI) using the formula described by Sunder et al. (1995).

$$\% \text{ Growth inhibition} = \frac{X - Y}{X} \times 100$$

Where, X = Pathogen mycelial growth in control plates, Y = Pathogen mycelial growth in treated plates.

Table 1. List of fungicides used against *S. sclerotiorum*.

| Sr. No. | Codes | Commercial name | Active ingredients | Mode of action | Manufacturer |
|---|---|---|---|---|---|
| 1 | F1 | Amistar | Azoxystrobin | Systemic | Sygenta |
| 2 | F2 | Tilt | Propiconazole | Systemic | Sygenta |
| 3 | F3 | Bavistin | Carbendazim | Systemic | BASF |
| 4 | F4 | Nativo | Tebuconazole + Trifloxystrobin | Systemic | Bayer |
| 5 | F5 | Score | Difenoconazole | Systemic | Sygenta |
| 6 | F6 | Kumulus | Sulphur | Systemic | BASF |
| 7 | F7 | Rovral | Iprodione | Local systemic | FMC |

**Evaluation of fungicides against *S. sclerotiorum* under greenhouse conditions through seed and foliar treatments**

A greenhouse experiment was conducted to evaluate the efficacy of selected fungicides against *S. sclerotiorum* using the susceptible tomato variety 'Nagina'. The experiment was carried out in plastic pots (30 cm diameter) arranged in a Completely Randomized Design with three replications. Pots were surface-sterilized with a 10% bleach solution, filled with 3 kg of sterilized soil, and artificially inoculated with *S. sclerotiorum* using five sclerotia and 20 ml of mycelial suspension per pot.

Based on prior laboratory screening, four fungicides, Amistar (azoxystrobin), Tilt (propiconazole), Bavistin (carbendazim), and Kumulus (sulphur), were selected for evaluation as seed and foliar treatments. For seed treatment, seeds of the 'Nagina' variety were surface-sterilized with a 5% sodium hypochlorite solution for 2 min, followed by three rinses with sterile distilled water. The seeds were then soaked in fungicide solutions at concentrations of 25, 50, 75, and 100 µg $ml^{-1}$ for 15 min. After treatment, seeds were air-dried on blotter paper before sowing.

For foliar application, fungicides were applied as a single spray at 40 days after sowing (DAS) using the same concentrations (25, 50, 75, and 100 µg $ml^{-1}$). Sterilized water was used as the control treatment in both seed and foliar experiments.

Disease incidence, disease severity, and percent disease control were recorded at 16, 22, and 28 days after disease initiation in both treatment types. Disease incidence was calculated using the formula described by Wheeler (1969), while disease severity was assessed using the disease rating scale proposed by Grau et al. (1982), with slight modifications (Table 2). Percent disease control was calculated using the standard formula.

$$\% \text{ Disease incidence (PDI)} = \frac{\text{SIR}}{\text{NOP} - \text{MDR}} \times 100$$

Where, SIR = Sum of individual ratings, NOP = Number of observed plants, MDR = Maximum disease rating.

$$\% \text{ Disease severity (PDS)} = \frac{\Sigma\,(a \times b)}{N \times K} \times 100$$

Where, a = Number of infected leaves in each category, b = Numerical value of each category, N = Total number of examined leaves, K = Infection category at highest degree.

$$\% \text{ Disease control (PDC)} = \frac{\text{DIC} - \text{DIT}}{\text{DIC}} \times 100$$

Where, DIC = Disease in control, DIT = Disease in treatment.

**Statistical analysis**

The data recorded from the laboratory and greenhouse experiments were statistically analyzed using R software (R Core Team, 2021). A Completely Randomized Design was employed for both laboratory and greenhouse experiments. The effects of treatments were evaluated through analysis of variance (ANOVA), and both significant and non-significant effects were reported accordingly. Differences among treatment means were compared using Tukey's Honestly Significant Difference (HSD) test at a probability level of $P \leq 0.05$ (Steel et al., 1997).

Table 2. Disease severity rating scale for white mold in tomato.

| Grade | Description | Response |
|---|---|---|
| 0 | No visible symptoms | Resistant |
| 1 | 0.1-2cm lesion on stem | Moderately Resistant |
| 2 | 2-3cm lesion length on stem | Moderately Susceptible |
| 3 | >3cm lesion length on stem/completely death of plant | Susceptible |

## Results

### Evaluation of fungicides against *S. sclerotiorum* under laboratory conditions

The efficacy of seven fungicides at four concentrations (25, 50, 75, and 100 µg $ml^{-1}$) was evaluated against the pathogen *S. sclerotiorum* under laboratory conditions at 7, 14, and 21 days after inoculation (Table 1). Analysis of variance revealed that fungicides, concentrations, days, and their interactions had a significant effect on mycelial growth inhibition of *S. sclerotiorum* (Table 3).

Among the tested fungicides, azoxystrobin, propiconazole, carbendazim, and sulphur exhibited complete inhibition of mycelial growth. This was followed by difenoconazole, tebuconazole + trifloxystrobin, and iprodione, which showed mean mycelial growth of 1.4, 8.2, and 47.5 mm, corresponding to growth inhibition of 97.8, 89.2, and 30.1%, respectively, compared with the control (Figure 1, Table 4).

The interaction between fungicides and concentrations (F × C) showed that azoxystrobin, propiconazole, carbendazim, and sulphur were the most effective treatments, resulting in complete mycelial growth inhibition at all tested concentrations. In contrast, difenoconazole resulted in limited mycelial growth (1.6, 1.5, 1.4, and 1.3 mm) with growth inhibition of 97.8, 97.7, 97.7, and 98.1%, respectively. Tebuconazole + trifloxystrobin showed mycelial growth of 13.2, 6.9, 6.4, and 6.3 mm, corresponding to growth inhibition of 82.6, 90.9, 91.3, and 92.2%. Iprodione was the least effective, with mycelial growth of 56.1, 48.2, 43.7, and 41.9 mm and growth inhibition of 20.8, 29.1, 31.4, and 39%, respectively, compared with the control (Figure 2, Table 5).

The interaction between fungicides and days (F × D) further demonstrated that iprodione was the least effective fungicide, showing mycelial growth of 39.8, 46.8, and 55.8 mm and growth inhibition of 32.0, 30.1, and 28.1% at 7, 14, and 21 DAI, respectively. In contrast, azoxystrobin, propiconazole, carbendazim, and sulphur consistently resulted in complete inhibition of mycelial growth at all three observation intervals (7, 14, and 21 DAI) (Figure 3, Table 6).

Table 4. Effect of fungicides on mycelial growth inhibition of *S. sclerotiorum* under laboratory conditions.

| Sr. No. | Treatments | Mycelial growth inhibition (%) ± S.E. |
|---|---|---|
| 1 | F1 | 100.0 ± 0.0 a |
| 2 | F2 | 100.0 ± 0.0 a |
| 3 | F3 | 100.0 ± 0.0 a |
| 4 | F4 | 89.2 ± 2.1 c |
| 5 | F5 | 97.8 ± 0.1 b |
| 6 | F6 | 100.0 ± 0.0 a |
| 7 | F7 | 30.1 ± 1.4 d |
| 8 | Control | 0.0 ± 0.0 e |
| Tukey's HSD | | 0.7290025 |

Data represent the mean ± standard error (SE) of three replications per treatment. Treatments sharing the same letter are not significantly different at $p < 0.05$ according to ANOVA followed by Tukey's Honest Significant Difference (HSD) test. Fungicide codes: F1 = Azoxystrobin, F2 = Propiconazole, F3 = Carbendazim, F4 = Tebuconazole + Trifloxystrobin, F5 = Difenoconazole, F6 = Sulphur, F7 = Iprodione.

Table 3. Analysis of variance (ANOVA) for the efficacy of fungicides against *S. sclerotiorum* under laboratory conditions.

| SOV | df | MRG | Pr(>F) | MGI | Pr(>F) |
|---|---|---|---|---|---|
| | | MSS | | MSS | |
| Fungicides (F) | 7 | 25505 | <2e-16*** | 55693 | <2e-16*** |
| Concentrations (C) | 3 | 168 | <2e-16*** | 154 | <2e-16*** |
| Days (D) | 2 | 1195 | <2e-16*** | 345 | <2e-16*** |
| F*C | 21 | 56 | <2e-16*** | 75 | <2e-16*** |
| F*D | 14 | 293 | <2e-16*** | 271 | <2e-16*** |
| C*D | 6 | 13 | 1.42e-14*** | 23 | <2e-16*** |
| F*C*D | 42 | 11 | <2e-16*** | 26 | <2e-16*** |
| Residuals | 192 | 1 | | 1 | |

SOV = Source of variance, df = Degree of freedom, MRG = Mycelial radial growth, MGI = Mycelial growth inhibition, MSS = Mean sum of square, Significant codes = *** P<0.001.

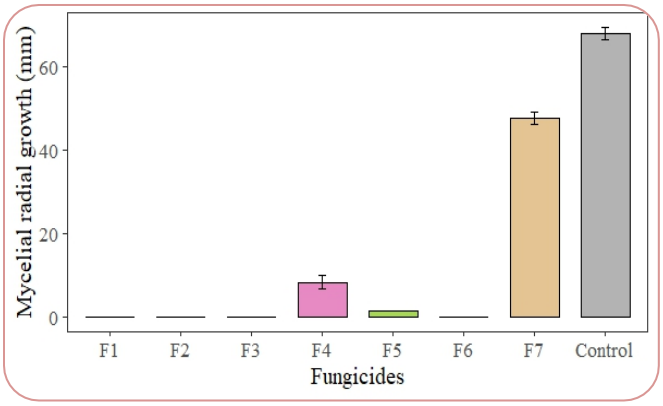


Figure 1. Effect of fungicides on the radial mycelial growth of *S. sclerotiorum* under laboratory conditions. Fungicide codes: F1 = Azoxystrobin, F2 = Propiconazole, F3 = Carbendazim, F4 = Tebuconazole + Trifloxystrobin, F5 = Difenoconazole, F6 = Sulphur, and F7 = Iprodione. Values represent the mean ± standard error of three replications.

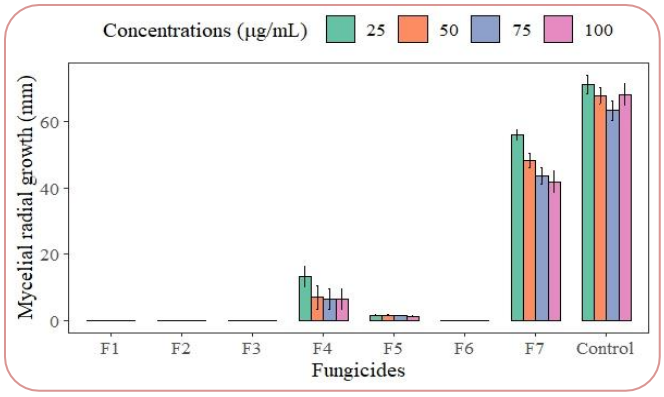


Figure 2. Effect of fungicides and their concentrations on the radial mycelial growth of *S. sclerotiorum* under laboratory conditions. Fungicide codes: F1 = Azoxystrobin, F2 = Propiconazole, F3 = Carbendazim, F4 = Tebuconazole + Trifloxystrobin, F5 = Difenoconazole, F6 = Sulphur, and F7 = Iprodione. Values represent the mean ± standard error of three replications.

Table 5. Interactive effect of fungicides and their concentrations (F × C) on mycelial growth inhibition of *S. sclerotiorum* under laboratory conditions.

| Treatments | Mycelial growth inhibition (%) ± S.E. | | | |
|---|---|---|---|---|
| | Concentrations (µg ml$^{-1}$) | | | |
| | 25 | 50 | 75 | 100 |
| F1 | 100.0 ± 0.0 a | 100.0 ± 0.0 a | 100.0 ± 0.0 a | 100.0 ± 0.0 a |
| F2 | 100.0 ± 0.0 a | 100.0 ± 0.0 a | 100.0 ± 0.0 a | 100.0 ± 0.0 a |
| F3 | 100.0 ± 0.0 a | 100.0 ± 0.0 a | 100.0 ± 0.0 a | 100.0 ± 0.0 a |
| F4 | 82.6 ± 3.9 d | 90.9 ± 4.5 c | 91.3 ± 4.4 c | 92.2 ± 3.9 c |
| F5 | 97.8 ± 0.1b | 97.7 ± 0.2 b | 97.7 ± 0.1b | 98.1 ± 0.1b |
| F6 | 100.0 ± 0.0 a | 100.0 ± 0.0 a | 100.0 ± 0.0 a | 100.0 ± 0.0 a |
| F7 | 20.8 ± 1.2 d | 29.1 ± 1.0 h | 31.4 ± 1.2 g | 39.0 ± 2.9 f |
| Control | 0.0 ± 0.0 i | 0.0 ± 0.0 i | 0.0 ± 0.0 i | 0.0 ± 0.0 i |
| Tukey's HSD | 1.826844 | | | |

Data represent mean values ± standard error (SE) of three replications per treatment. Treatments sharing the same letter are not significantly different at $p \leq 0.05$, as determined by analysis of variance (ANOVA) followed by Tukey's Honest Significant Difference (HSD) test. Fungicide codes: F1 = Azoxystrobin, F2 = Propiconazole, F3 = Carbendazim, F4 = Tebuconazole + Trifloxystrobin, F5 = Difenoconazole, F6 = Sulphur, F7 = Iprodione.

**Evaluation of fungicides against *S. sclerotiorum* under greenhouse conditions through seed treatment**

Four fungicides that showed superior performance under laboratory conditions, azoxystrobin, propiconazole, carbendazim, and sulphur, were evaluated under greenhouse conditions through seed treatment to manage white mold of tomato. Each fungicide was tested at four concentrations (25, 50, 75, and 100 µg ml$^{-1}$) to assess disease incidence, disease severity, and disease control efficacy.

Analysis of variance revealed that fungicides, concentrations, days after inoculation, and their interactions had a significant effect on disease development (Table 7). All treatments significantly reduced disease incidence and disease severity while increasing disease control efficacy compared with the control.

Among the tested fungicides, carbendazim resulted in the lowest disease incidence and disease severity (13.4% and 9.8%, respectively), followed by propiconazole (14.7% and 10.9%), sulphur (21.9% and 15.2%), and azoxystrobin (29.2% and 20.1%). Correspondingly, disease control efficacy was highest with carbendazim (72.3%), followed by propiconazole (70.0%), sulphur (54.2%), and azoxystrobin (40.2%) (Figure 4, Table 8).

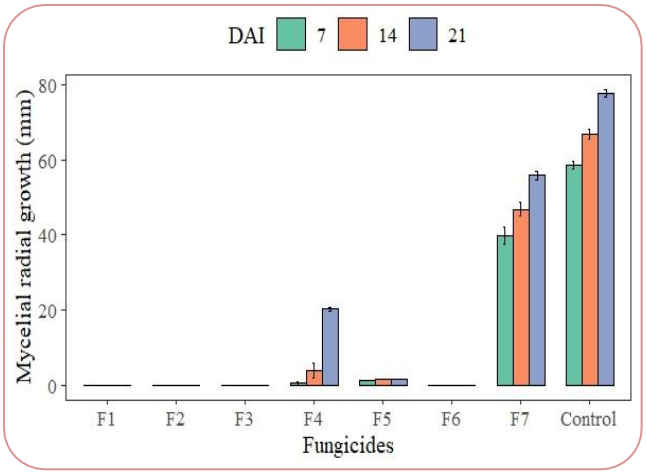


Figure 3. Effect of fungicides over time on the radial mycelial growth of *S. sclerotiorum* under laboratory conditions. Fungicide codes: F1 = Azoxystrobin, F2 = Propiconazole, F3 = Carbendazim, F4 = Tebuconazole + Trifloxystrobin, F5 = Difenoconazole, F6 = Sulphur, and F7 = Iprodione; DAI = days after inoculation. Each treatment represents the mean ± standard error of three replications.

The interaction between fungicides and concentrations (F × C) indicated that carbendazim was the most effective treatment, showing the lowest disease incidence (18.3%, 15.3%, 11.8%, and 8.1%) and disease severity (13.0%, 11.0%, 9.5%, and 5.7%) with increasing disease control efficacy (61.7%, 68.1%, 76.3%, and 83.2%) at 25, 50, 75, and 100 µg $ml^{-1}$, respectively. Propiconazole also performed effectively, resulting in disease incidence of 22.6%, 16.1%, 11.4%, and 8.7%, disease severity of 16.0%, 13.1%, 8.3%, and 6.4%, and disease control of 53.3%, 67.5%, 77.1%, and 81.9% at the corresponding concentrations.

Sulphur showed moderate efficacy, with disease incidence of 25.7%, 22.8%, 20.4%, and 18.8%, disease severity of 19.3%, 16.4%, 13.3%, and 11.7%, and disease control of 47.6%, 53.3%, 56.8%, and 59.1%. In contrast, azoxystrobin exhibited the highest disease incidence (33.8%, 30.6%, 27.7%, and 24.5%) and disease severity (24.2%, 20.9%, 18.8%, and 16.4%) with the lowest disease control efficacy (31.2%, 37.4%, 43.4%, and 48.6%) across the tested concentrations, compared with the control (Figure 5, Table 9).

The interaction between fungicides and days after inoculation (F × D) further demonstrated that azoxystrobin was the least effective treatment, recording the highest disease incidence (22.1%, 28.5%, and 36.8%) and disease severity (13.6%, 19.8%, and 26.8%) along with the lowest disease control efficacy (31.2%, 38.5%, and 50.8%). Conversely, carbendazim was the most effective fungicide, showing the lowest disease incidence (10.8%, 12.9%, and 16.4%), disease severity (5.8%, 9.0%, and 14.6%), and the highest disease control efficacy (66.6%, 72.3%, and 78.1%) at 16, 22, and 28 days after inoculation, respectively (Figure 6, Table 10).

Table 6. Interactive effect of fungicides and incubation days (F × D) on the inhibition of mycelial growth of *S. sclerotiorum* under laboratory conditions.

| Treatments | Mycelial growth inhibition (%) ± S.E. | | |
|---|---|---|---|
| | Days after inoculation | | |
| | 7 | 14 | 21 |
| F1 | 100.0 ± 0.0 a | 100.0 ± 0.0 a | 100.0 ± 0.0 a |
| F2 | 100.0 ± 0.0 a | 100.0 ± 0.0 a | 100.0 ± 0.0 a |
| F3 | 100.0 ± 0.0 a | 100.0 ± 0.0 a | 100.0 ± 0.0 a |
| F4 | 99.3 ± 0.7 ab | 94.5 ± 2.9 d | 73.9 ± 0.6 e |
| F5 | 97.6 ± 0.1 c | 97.8 ± 0.1 bc | 98.1 ± 0.1 bc |
| F6 | 100.0 ± 0.0 a | 100.0 ± 0.0 a | 100.0 ± 0.0 a |
| F7 | 32.0 ± 3.6 f | 30.1 ± 1.7 g | 28.1 ± 1.3 h |
| Control | 0.0 ± 0.0 i | 0.0 ± 0.0 i | 0.0 ± 0.0 i |
| Tukey's HSD | 1.520818 | | |

Mean values and standard errors of fungicide treatments (three Replications per treatment). Treatments sharing the same letter are not significantly different at $p < 0.05$ (ANOVA and Tukey's HSD Test). Fungicide Codes: F1 = Azoxystrobin, F2 = Propiconazole, F3 = Carbendazim, F4 = Tebuconazole + Trifloxystrobin, F5 = Difenoconazole, F6 = Sulphur, F7 = Iprodione; S.E. = standard error.

Table 7. Analysis of variance (ANOVA) for seed treatment with fungicides against *S. sclerotiorum* under greenhouse conditions.

| SOV | df | DI | Pr(>F) | DS | Pr(>F) | DC | Pr(>F) |
|---|---|---|---|---|---|---|---|
| | | MSS | | MSS | | MSS | |
| Fungicides (F) | 4 | 8468 | <2e-16*** | 5177 | <2e-16*** | 31278 | <2e-16*** |
| Concentrations (C) | 3 | 597 | <2e-16*** | 314 | <2e-16*** | 2127 | <2e-16*** |
| Days (D) | 2 | 3862 | <2e-16*** | 3102 | <2e-16*** | 2295 | <2e-16*** |
| F*C | 12 | 35 | <2e-16*** | 38 | <2e-16*** | 209 | <2e-16*** |
| F*D | 8 | 743 | <2e-16*** | 191 | <2e-16*** | 225 | <2e-16*** |
| C*D | 6 | 7 | 2.37e-07*** | 3 | 0.0239* | 98 | <2e-16*** |
| F*C*D | 24 | 4 | 2.27e-08*** | 6 | 6.32e-11*** | 29 | 2.46e-14*** |
| Residuals | 120 | 1 | | 1 | | 4 | |

SOV = Source of variance, df = Degree of freedom, DI = Disease incidence, DS = Disease severity, DC = Disease control, MSS = Mean sum of square, Significant codes = *** (P<0.001), * (P<0.05).

Table 8. Effect of fungicidal seed treatments on the control of *S. sclerotiorum* in greenhouse conditions.

| Sr. No. | Treatments | Disease control (%) ± S.E. |
|---|---|---|
| 1 | F1 | 40.2 ± 1.9 d |
| 2 | F2 | 70.0 ± 2.1 b |
| 3 | F3 | 72.3 ± 1.7 a |
| 4 | F4 | 54.2 ± 1.7 c |
| 5 | Control | 0.0 ± 0.0 e |
| Tukey's HSD | | 1.300584 |

Mean values and standard errors of three replications per treatment. Treatments sharing the same letter are not significantly different ($p < 0.05$) according to ANOVA and Tukey's HSD Test. Fungicide codes: F1 = Azoxystrobin, F2 = Propiconazole, F3 = Carbendazim, F4 = Sulphur; S.E. = standard error.

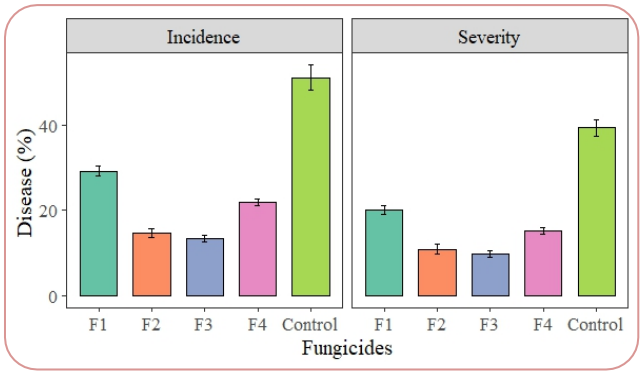


Figure 4. Effect of fungicide seed treatments on disease incidence and severity of *S. sclerotiorum* under greenhouse conditions. Fungicide codes: F1 = Azoxystrobin, F2 = Propiconazole, F3 = Carbendazim, and F4 = Sulphur. Values represent the mean ± standard error of three replications.

Table 9. Effect of fungicide seed treatments at different concentrations (F × C) on disease control of *S. sclerotiorum* under greenhouse conditions.

| Treatments | Disease control (%) ± S.E | | | |
|---|---|---|---|---|
| | Concentrations (µg $ml^{-1}$) | | | |
| | 25 | 50 | 75 | 100 |
| F1 | 31.2 ± 4.2 j | 37.4 ± 3.0 i | 43.4 ± 2.4 h | 48.6 ± 2.5 g |
| F2 | 53.3 ± 3.3 f | 67.5 ± 1.4 c | 77.1 ± 0.7 b | 81.9 ± 1.0 a |
| F3 | 61.7 ± 3.4 d | 68.1 ± 2.2 c | 76.3 ± 0.8 b | 83.2 ± 0.9 a |
| F4 | 47.6 ± 3.3 g | 53.3 ± 2.7 f | 56.8 ± 3.4 e | 59.1 ± 3.5 de |
| Control | 0.0 ± 0.0 k | 0.0 ± 0.0 k | 0.0 ± 0.0 k | 0.0 ± 0.0 k |
| Tukey's HSD | 3.404047 | | | |

Data are presented as mean values ± standard error of three replications per treatment. Treatments sharing the same letter are not significantly different at $p < 0.05$, based on ANOVA followed by Tukey's Honest Significant Difference test. Fungicide codes: F1 = Azoxystrobin, F2 = Propiconazole, F3 = Carbendazim, F4 = Sulphur; S.E. = standard error.

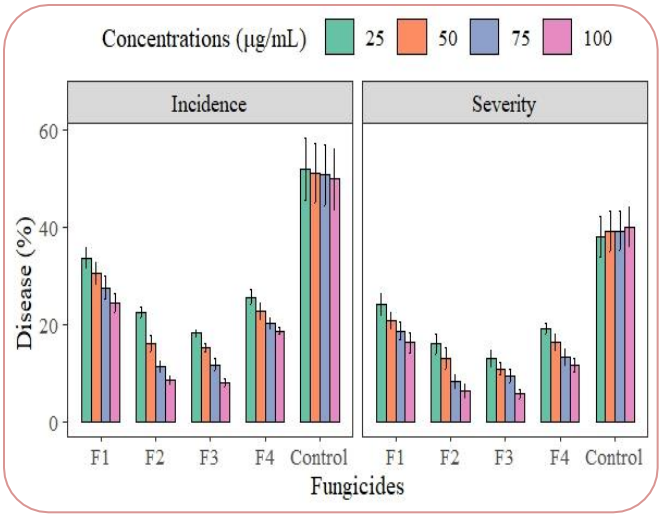


Figure 5. Effect of fungicide seed treatments at different concentrations on disease incidence and severity of *S. sclerotiorum* under greenhouse conditions. Fungicide codes: F1 = Azoxystrobin, F2 = Propiconazole, F3 = Carbendazim, and F4 = Sulphur. Each value represents the mean ± standard error of three replications.

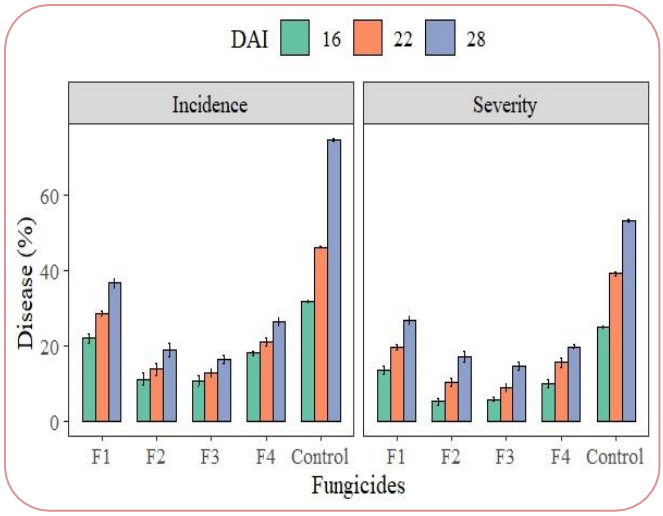


Figure 6. Effect of fungicide seed treatments over time on disease incidence and severity of *S. sclerotiorum* under greenhouse conditions. Fungicide codes: F1 = Azoxystrobin, F2 = Propiconazole, F3 = Carbendazim, and F4 = Sulphur; DAI = days after inoculation. Values represent the mean ± standard error of three replications.

Table 10. Effect of fungicide seed treatments across different days after inoculation (F × D) on disease control of *S. sclerotiorum* under greenhouse conditions.

| Treatments | Disease control (%) ± S.E | | |
|---|---|---|---|
| | Days after inoculation | | |
| | 16 | 22 | 28 |
| F1 | 31.2 ± 3.3 i | 38.5 ± 1.4 h | 50.8 ± 1.5 f |
| F2 | 65.1 ± 4.3 d | 70.2 ± 3.6 c | 74.6 ± 2.2 b |
| F3 | 66.6 ± 3.8 d | 72.3 ± 2.3 bc | 78.1 ± 1.4 a |
| F4 | 43.5 ± 1.2 g | 54.3 ± 2.0 e | 64.7 ± 1.4 d |
| Control | 0.0 ± 0.0 j | 0.0 ± 0.0 j | 0.0 ± 0.0 j |
| Tukey's HSD | 2.816833 | | |

Data represent the mean values ± standard error of three replications per treatment. Treatments sharing the same letter are not significantly different at $p < 0.05$, based on ANOVA followed by Tukey's Honest Significant Difference test. Fungicide codes: F1 = Azoxystrobin, F2 = Propiconazole, F3 = Carbendazim, F4 = Sulphur; S.E. = standard error.

**Evaluation of fungicides against *S. sclerotiorum* under greenhouse conditions through foliar application**

The effects of foliar fungicide application were assessed for tomato white mold disease under greenhouse conditions. Analysis of variance revealed that fungicides, concentrations, days after inoculation, and their interactions had significant effects on disease development (Table 11).

Among the tested fungicides, carbendazim was the most effective, resulting in the lowest disease incidence (17.7%) and disease severity (13.1%), followed by propiconazole (19.7% and 17.8%), sulphur (23.6% and 20.6%), and azoxystrobin (30.1% and 24.4%). Correspondingly, disease control efficacy was highest with carbendazim (64.2%), followed by propiconazole (61.0%), sulphur (52.1%), and azoxystrobin (39.5%) (Figure 7, Table 12).

The interaction between fungicides and concentrations (F × C) indicated that carbendazim consistently provided superior control, showing the lowest disease incidence (22.7, 18.3, 16.4, and 13.6%) and disease severity (16.4, 13.9, 11.7, and 10.3%) with corresponding disease control efficacies of 54.5, 63.9, 66.2, and 72.0% at 25, 50, 75, and 100 µg $ml^{-1}$, respectively. Propiconazole resulted in disease incidence of 24.2, 20.6, 18.4, and 15.4% and

disease severity of 21.4, 18.9, 16.7, and 14.2%, with disease control efficacies of 52.2, 60.2, 63.3, and 68.4%. Sulphur showed moderate efficacy, with disease incidence of 29.1, 24.3, 22.2, and 18.6%, disease severity of 24.5, 22.2, 19.8, and 16.0%, and disease control of 41.8, 51.9, 54.3, and 60.6%. In contrast, azoxystrobin was the least effective, exhibiting the highest disease incidence (36.6, 30.8, 27.7, and 25.1%) and disease severity (28.9, 25.7, 22.9, and 20.1%), along with the lowest disease control efficacies (26.1, 39.3, 44.5, and 48.2%) at the respective concentrations compared with the control treatment (Figure 8, Table 13).

The interaction between fungicides and days after inoculation (F × D) further demonstrated that azoxystrobin was the least effective treatment, with high disease incidence (22.8, 29.1, and 38.3%) and disease severity (17.2, 25.1, and 30.9%) and the lowest disease control efficacies (30.5, 38.2, and 49.9%). In contrast, carbendazim was the most effective in suppressing pathogen development, resulting in disease incidence of 14.2, 16.5, and 22.5% and disease severity of 6.4, 12.2, and 20.7%, with disease control efficacies of 56.9, 65.0, and 70.6% at 16, 22, and 28 days after inoculation, respectively (Figure 9, Table 14).

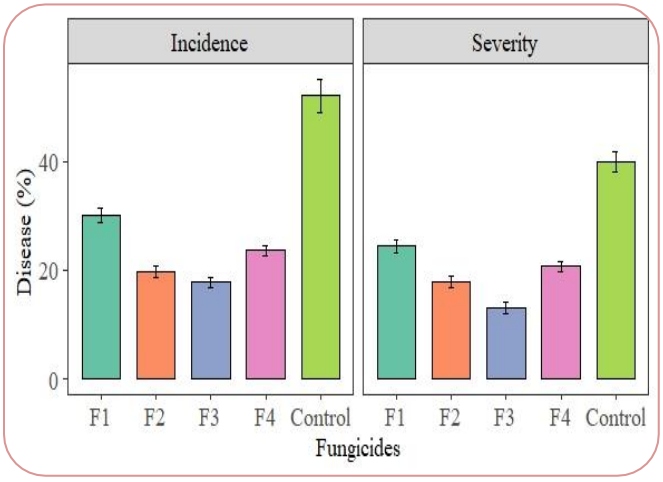


Figure 7. Effect of foliar application of fungicides on disease incidence and severity of *S. sclerotiorum* under greenhouse conditions. Fungicide codes: F1 = Azoxystrobin, F2 = Propiconazole, F3 = Carbendazim, and F4 = Sulphur. Each value represents the mean ± standard error of three replications.

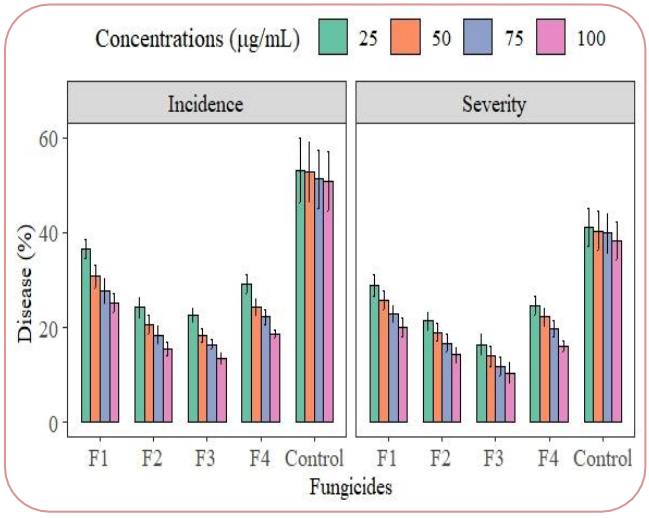


Figure 8. Effect of foliar fungicide treatments at different concentrations on disease incidence and severity of *S. sclerotiorum* under greenhouse conditions. Fungicide codes: F1 = Azoxystrobin, F2 = Propiconazole, F3 = Carbendazim, and F4 = Sulphur. Values represent the mean ± standard error of three replications.

Table 11. Analysis of variance (ANOVA) for fungicide foliar treatments against *S. sclerotiorum* under greenhouse conditions.

| SOV | df | DI | Pr(>F) | DS | Pr(>F) | DC | Pr(>F) |
|---|---|---|---|---|---|---|---|
| | | MSS | | MSS | | MSS | |
| Fungicides (F) | 4 | 6980 | <2e-16*** | 3766 | <2e-16*** | 24449 | <2e-16*** |
| Concentrations (C) | 3 | 577 | <2e-16*** | 366 | <2e-16*** | 1778 | <2e-16*** |
| Days (D) | 2 | 5003 | <2e-16*** | 3852 | <2e-16*** | 2217 | <2e-16*** |
| F*C | 12 | 21 | <2e-16*** | 11 | 2.91e-15*** | 126 | <2e-16*** |
| F*D | 8 | 668 | <2e-16*** | 148 | <2e-16*** | 209 | <2e-16*** |
| C*D | 6 | 13 | 2.79e-10*** | 4 | 0.000578*** | 77 | 7.56e-11*** |
| F*C*D | 24 | 3 | 0.000335*** | 3 | 1.56e-05*** | 19 | 4.55e-05*** |
| Residuals | 120 | 1 | | 1 | | 6 | |

SOV = Source of variance; df = Degree of freedom; DI = Disease incidence; DS = Disease severity; DC = Disease control; MSS = Mean sum of square; Significance codes: *** (P < 0.001).

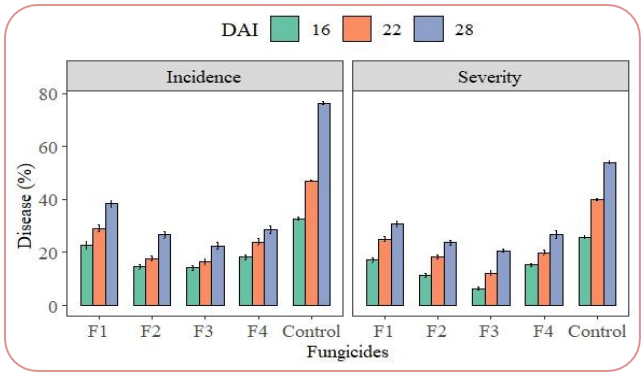


Figure 9. Effect of foliar application of fungicides on disease incidence and severity of *S. sclerotiorum* over time under greenhouse conditions. Fungicide codes: F1 = Azoxystrobin, F2 = Propiconazole, F3 = Carbendazim, and F4 = Sulphur; DAI = days after inoculation. Each treatment represents the mean ± standard error of three replications.

Table 12. Effect of foliar application of fungicides on the control of *S. sclerotiorum* under greenhouse conditions.

| Sr. No. | Treatments | Disease control (%) ± S.E. |
|---|---|---|
| 1 | F1 | 39.5 ± 2.1 d |
| 2 | F2 | 61.0 ± 1.3 b |
| 3 | F3 | 64.2 ± 1.5 a |
| 4 | F4 | 52.1 ± 1.8 c |
| 5 | Control | 0.0 ± 0.0 e |
| Tukey's HSD | | 1.628134 |

Data are presented as mean values ± standard error of three replications per treatment. Treatments sharing the same letter are not significantly different at $p < 0.05$ according to ANOVA followed by Tukey's Honest Significant Difference (HSD) test. Fungicide codes: F1 = Azoxystrobin, F2 = Propiconazole, F3 = Carbendazim, F4 = Sulphur; S.E. = standard error.

Table 13. Effect of foliar-applied fungicides at different concentrations (F × C interaction) on the control of *S. sclerotiorum* under greenhouse conditions.

| Treatments | Disease control (%) ± S.E | | | |
|---|---|---|---|---|
| | Concentrations (µg ml$^{-1}$) | | | |
| | 25 | 50 | 75 | 100 |
| F1 | 26.1 ± 5.1 j | 39.3 ± 2.6 i | 44.5 ± 2.1 gh | 48.2 ± 2.5 fg |
| F2 | 52.2 ± 2.7 ef | 60.2 ± 1.2 d | 63.3 ± 1.4 cd | 68.4 ± 1.4 ab |
| F3 | 54.5 ± 2.9 e | 63.9 ± 1.8 cd | 66.2 ± 2.1 bc | 72.0 ± 1.6 a |
| F4 | 41.8 ± 3.4 hi | 51.9 ± 2.3 ef | 54.3 ± 2.6 e | 60.6 ± 3.0 d |
| Control | 0.0 ± 0.0 k | 0.0 ± 0.0 k | 0.0 ± 0.0 k | 0.0 ± 0.0 k |
| Tukey's HSD | 4.26135 | | | |

Data represent the mean ± standard error of three replications per treatment. Treatments sharing the same letter are not significantly different at $p < 0.05$, based on ANOVA followed by Tukey's Honest Significant Difference (HSD) test. Fungicide codes are as follows: F1 = Azoxystrobin, F2 = Propiconazole, F3 = Carbendazim, F4 = Sulphur; S.E. = standard error.

Table 14. Effect of foliar fungicide treatments over time (F × D) on *S. sclerotiorum* disease control under greenhouse conditions.

| Treatments | Disease control (%) ± S.E | | |
|---|---|---|---|
| | Days after inoculation | | |
| | 16 | 22 | 28 |
| F1 | 30.5 ± 4.1 g | 38.2 ± 2.6 f | 49.9 ± 1.4 d |
| F2 | 55.6 ± 2.6 c | 62.3 ± 1.7 b | 65.1 ± 1.5 b |
| F3 | 56.9 ± 2.3 c | 65.0 ± 2.4 b | 70.6 ± 1.3 a |
| F4 | 44.5 ± 2.3 e | 49.3 ± 2.4 d | 62.6 ± 1.8 b |
| Control | 0.0 ± 0.0 h | 0.0 ± 0.0 h | 0.0 ± 0.0 h |
| Tukey's HSD | 3.526248 | | |

The data represent the mean values ± standard error of three replications per treatment. Treatments sharing the same letter are not significantly different at $p < 0.05$, based on ANOVA followed by Tukey's Honest Significant Difference (HSD) test. Fungicide codes: F1 = Azoxystrobin, F2 = Propiconazole, F3 = Carbendazim, F4 = Sulphur; S.E. = standard error.

**Discussion**

Tomato is one of the most important vegetable crops worldwide and serves as a major cash and industrial crop in many regions. It plays a vital role in providing a healthy diet due to its high nutritional value (Awan et al., 2012). However, tomato production is constrained by more than 200 diseases and pests, which cause both direct and indirect yield losses (Nowicki et al., 2013). Tomatoes are particularly vulnerable to several biotic stresses, including infections caused by fungal pathogens (Terna and Simon, 2017). Among these, white mold disease caused by *S. sclerotiorum* is a major and destructive disease of tomato (Heffer and Johnson, 2007). *Sclerotinia* diseases are difficult to manage using a single control strategy; therefore, integrated disease management approaches are widely adopted to minimize yield losses. These approaches include cultural practices, resistant cultivar selection, chemical control, and the use of biological antagonists (Peltier et al., 2012). Current management strategies for *S. sclerotiorum* rely heavily on fungicide applications (Chitrampalam et al., 2010; Bardin and Huang, 2001; Derbyshire and Denton-Giles, 2016). These fungicides primarily target aboveground infections caused by ascospores and provide effective disease suppression across a range of environmental conditions (Boland, 1997).

Considering the continued importance of fungicides in managing white mold disease, the present study was designed to evaluate the efficacy of seven fungicides (Azoxystrobin, Propiconazole, Carbendazim, Tebuconazole + Trifloxystrobin, Difenoconazole, Sulphur, and Iprodione) against *S. sclerotiorum* under laboratory and greenhouse conditions. Under laboratory conditions, Azoxystrobin, Propiconazole, Carbendazim, and Sulphur were the most effective fungicides, showing complete inhibition of mycelial growth. Difenoconazole also significantly suppressed pathogen growth, exhibiting minimal radial mycelial growth (1.4 mm) with a growth inhibition of 97.8%.

These findings are consistent with the results of Zamani (2021), who reported strong inhibition of *S. sclerotiorum* mycelial growth by Azoxystrobin. Similarly, the present results align with those of Zhang et al. (2018), who demonstrated the effectiveness of Propiconazole against the white mold pathogen in tomato. The findings are further corroborated by Hasna et al. (2022), who identified Carbendazim as one of the most effective fungicides against *S. sclerotiorum*. Comparable results were also reported by Prova et al. (2018), who observed significant inhibitory effects of Sulphur on *S. sclerotiorum*. Moreover, Ruan et al. (2023) reported strong *in vitro* inhibitory activity of Difenoconazole against *S. sclerotiorum*, supporting the present observations.

Based on laboratory performance, four fungicides (Azoxystrobin, Propiconazole, Carbendazim, and Sulphur) were further evaluated under greenhouse conditions using seed and foliar applications. Seed treatment with Carbendazim resulted in the lowest disease incidence (13.4%) and disease severity (9.8%), with the highest disease control (72.3%), followed by Propiconazole, which also significantly reduced disease incidence (14.7%) and severity (10.9%), achieving 70% disease control. Under foliar application, Carbendazim remained the most effective treatment, showing the lowest disease incidence (17.7%) and severity (13.1%) with maximum disease control (64.2%), followed by Propiconazole, which recorded disease incidence of 19.7%, disease severity of 17.8%, and disease control of 61%. Overall, seed treatment proved more effective than foliar application in managing tomato white mold caused by *S. sclerotiorum*.

These results are supported by Chaudhary et al. (2010) and Kumar and Lal (2024), who reported the effectiveness of Carbendazim applied as both seed and foliar treatments against *S. sclerotiorum*. Similarly, Zhang et al. (2023) demonstrated the efficacy of Carbendazim under field conditions. The effectiveness of Propiconazole as a seed and foliar treatment against *S. sclerotiorum* has also been reported by Upadhyay and Tiwari (2019). Furthermore, the superior performance of seed treatment over foliar application observed in this study is consistent with the findings of Sharma et al. (2016), who reported greater disease control and lower disease intensity through seed treatment in mustard affected by *Sclerotinia* rot.

Fungicides play a crucial role in protecting crops from plant diseases; however, their repeated and prolonged use can lead to the development of resistance in pathogens, resulting in economic losses for both farmers and the agrochemical industry (McDougall, 2010; Boyce, 2013). Effective fungicide resistance management is therefore essential for sustainable crop protection and includes early detection, quantification, and implementation of proactive and mitigation strategies. These strategies aim to reduce resistance pressure by limiting the continuous use of single-site mode-of-action

fungicides and integrating cultural practices, host resistance, and fungicides with multiple modes of action to delay resistance development and maintain long-term disease control (Gambhir et al., 2021). Understanding fungicide modes of action and resistance mechanisms at cellular, organismal, and population levels is also critical for effective resistance management (Yin et al., 2023).

Azoxystrobin is a systemic fungicide belonging to the methoxyacrylate group derived from naturally occurring strobilurins. It is a quinone outside inhibitor (QoI) fungicide that inhibits mitochondrial respiration by binding to the ubiquinol oxidation (Qo) site of the cytochrome $bc_1$ complex, thereby disrupting electron transport and preventing ATP synthesis. This mode of action ultimately inhibits spore germination and mycelial growth, conferring broad-spectrum activity against many fungal pathogens and oomycetes (Bartlett et al., 2002; Vincelli, 2002; Zhang et al., 2014; Andrade et al., 2022).

Propiconazole is a systemic demethylation inhibitor (DMI) fungicide that disrupts fungal cell membrane integrity by inhibiting sterol biosynthesis. Specifically, it blocks the demethylation step involved in ergosterol biosynthesis, an essential component for maintaining cell membrane stability, ultimately leading to fungal cell death (Brent and Hollomon, 1995; Li et al., 2020).

Carbendazim, a benzimidazole fungicide, is a systemic compound related to benomyl and thiophanate-methyl. It binds to β-tubulin, preventing microtubule assembly, which is essential for nuclear division and mycelial growth (Davidse, 1986; Davidse and Ishii, 1995; Yunlong et al., 2009). Benzimidazoles induce abnormalities in spore germination, germ tube elongation, cellular multiplication, and mycelial growth by inhibiting cell division (FRAC, 2025).

Sulphur is a non-systemic contact fungicide and an essential macronutrient that enhances plant defense mechanisms through sulfur-containing defense compounds (SDCs) such as glutathione, glucosinolates, and phytoalexins. These compounds regulate signal transduction pathways associated with plant hormones and reactive oxygen species, thereby strengthening plant defense responses and improving disease resistance (Kunstler et al., 2020; Sanders et al., 2025).

Overall, the present study demonstrates the effectiveness of fungicides in controlling *S. sclerotiorum*, supporting previous research findings. Fungicides remain an important tool for rapid and effective disease management, contributing to improved crop quality and yield. However, excessive reliance on chemical control poses environmental risks and threatens long-term sustainability. Persistent fungicide use can lead to resistance development, reduced efficacy over time, and accumulation of chemical residues that negatively affect soil microbial communities and biodiversity. These challenges highlight the need for judicious fungicide use, adherence to recommended doses, and integration with sustainable disease management strategies to preserve ecological balance.

## Conclusion

The present study provides evidence of *S. sclerotiorum* as the causal agent of white mold disease of tomato in district Faisalabad, Pakistan, and demonstrates the effectiveness of selected fungicides under laboratory and greenhouse conditions. Among the tested fungicides, Azoxystrobin, Propiconazole, Carbendazim, and Sulphur showed maximum inhibition of mycelial growth under laboratory conditions and resulted in the lowest disease incidence and severity, with the highest disease control under greenhouse conditions, particularly when applied as seed treatments. Therefore, these fungicides are recommended for effective management of tomato white mold disease, with emphasis on integrated and judicious application to ensure sustainable disease control.

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## Authors' Contributions

UR and AR conceptualized and designed the study. UR conducted the experimental work, collected the data, performed statistical analyses, and prepared the original draft of the manuscript. NAR and IK contributed to formal data analysis, critically reviewed the manuscript, and provided constructive input to improve its scientific quality, clarity, and presentation. All authors read and approved the final version of the manuscript.

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**Conflict of Interest**

The authors declare no conflict of interest.

**Sustainable Development Goals Targeted**

SDG 2: Zero Hunger

SDG 12: Responsible Consumption and Production

SDG 15: Life on Land

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