
# Comparative Evaluation of Chemical Resistance Inducers (Methyl Jasmonate, Salicylic Acid, Chitosan, Sodium Nitroprusside, and Calcium Carbide) for Managing Early Blight (*Alternaria solani*) in Tomato

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The present study was conducted to evaluate the efficacy of different resistance-inducing agents against fungal pathogens associated with diseased tomato plants collected from the National Agricultural Research Centre and PMAS-Arid Agriculture University, Rawalpindi. Two pathogens, *Alternaria solani* (causing early blight) and *Pythium* spp. (causing damping-off), were identified. Early blight incidence was recorded at 44% and 22% at the respective locations, while damping-off was observed only at PMAS-AAUR. Pathogenicity of *A. solani* was confirmed through Koch's postulates on tomato cultivar Reograndi and line-95017, with the latter exhibiting higher susceptibility and being selected for subsequent experiments. Resistance inducers, including methyl jasmonate (MeJA), salicylic acid (SA), chitosan (CH), sodium nitroprusside (SNP), and calcium carbide ($CaC_2$; ethylene donor), were applied before pathogen inoculation. Significant differences ($p \leq 0.05$) were observed among treatments for disease severity assessed 10 days after inoculation. MeJA treatment resulted in the lowest disease severity (14.15%), representing a ~68.5% reduction compared to the positive control (44.98%). However, the reduction achieved by MeJA was statistically at par with $CaC_2$ (16.58%) and CH (18.21%), indicating non-significant differences among these treatments. SNP also significantly reduced disease severity (23.80%), whereas SA showed comparatively lower efficacy (30.13%), remaining significantly higher than MeJA-treated plants but lower than the positive control. The results indicate that resistance inducers associated with jasmonic acid and ethylene signaling pathways were more effective against the necrotrophic pathogen *A. solani*, while SA-mediated resistance was comparatively less effective. The moderate efficacy of CH and SNP suggests their role in activating induced systemic resistance and basal defense mechanisms. Overall, the study demonstrates that resistance inducers can significantly reduce early blight severity and may be integrated into sustainable disease management strategies, provided that inducer selection aligns with pathogen biology.

**Keywords:** Tomato, *Alternaria Solani*, *Pythium* spp., Resistance Inducers, Early Blight, Induced Resistance

**Introduction:**

Solanum lycopersicum, or tomato, belongs to the Solanaceae family and is the second most important vegetable crop after potato. Tomatoes are produced on over 63.2 thousand hectares in Pakistan, which yields 599.7 thousand tonnes annually. The average yield of tomatoes in Pakistan is approximately 10.5 tonnes per hectare against 34.0 tonnes per hectare of modern agricultural systems of the world. In Pakistan, tomato has a low yield because of the number of production constraints, all of which are abiotic and biotic [1][2][3]. High temperature, drought, and salinity are some of the abiotic constraints [4]. Late blight (LB) and early blight (EB) disease also thrive in tomato plantations, particularly in the seedling stage, favorable environmental conditions (March–June) [5][6][7]. Viruses like tomato spotted wilt virus (TSWV) and tomato mosaic virus (ToMV) are also reported as biotic agents in Pakistan [8]. Several bacteria also cause losses in tomato crop, and these include *Ralstonia solanacearum*, *Xanthomonas campestris*, and *Pseudomonas syringae* [9][10]. Other researchers also mention root knot nematode (*Meloidogyne incognita*) in tomato plantations [11][12].

Fusarium wilt (*Fusarium oxysporum*) [13][14], Pythium spp [13] and damping off (*Rhizoctonia solani*) [14] are also reported as other tomato fungal diseases in Pakistan, along with LB and EB. Early blight (EB) caused by *Alternaria solani* (*A. solani*) falls under the belongs to the genus *Alternaria*. *A. solani* pathogen was dominant in areas where the rainfall was great, the humidity was high, and the temperature was quite high [5]. Semi-arid climates can also experience epidemics due to frequent and prolonged nightly dew. EB has a range of incidence between 49 and 91 percent in favorable conditions. Conidia germinate to form one or more germ tubes under free moisture or near-saturated humidity at a large temperature range (8–32 °C).

Cultural practices can be used to control EB to eliminate the infected parts of the plant since the pathogen survives over winter on infected debris of crops. Hence, a 3–4-year crop rotation eliminates the EB pathogen. But in intensive agriculture, it appears not to be a feasible possibility. All tomato breeding lines and commercially released cultivars reported so far show only moderate susceptibility to early blight (EB) [15][16]. As a result, the management of EB largely relies on the cultivation of relatively tolerant varieties such as Mountain Pride, Supreme, Gold, Fresh, and Belle, in combination with fungicidal applications. Commonly used fungicides for controlling the disease include mancozeb, copper oxychloride, chlorothalonil, and carbendazim [17].

However, the application of fungicides is not considered a sustainable solution due to the emergence of fungicide resistance in *Alternaria solani* and the associated adverse effects on the environment [17]. Alternatively, biocontrol agents can be used, including *Trichoderma* spp., *Pseudomonas* spp, *Bacillus pumilis* to control EB. Although biocontrol agents are relatively safe and environmentally safe alternatives, they are not without limitations. Limitations are variations in the environment, appropriate dosage, and even application of the biocontrol agent to safeguard the plant.

For this reason, plant defense researchers are increasingly exploring alternative strategies to stimulate resistance without causing environmental disturbance and that are less dependent on environmental conditions for effectiveness. A substantial body of literature has documented the role of signaling molecules such as salicylic acid (SA), methyl jasmonate (MeJA), ethylene (ET), and nitric oxide (NO) in regulating plant defense responses [18][19]. Although the interactions among these signaling pathways are complex, these molecules collectively contribute to strengthening plant resistance against a wide range of pathogens.

Systemic resistance in plants generally occurs in two forms: systemic acquired resistance (SAR) and induced systemic resistance (ISR). Salicylic acid and methyl jasmonate

are commonly associated with the activation of SAR, whereas ethylene is considered an important signaling component of ISR. In addition, plants are capable of recognizing conserved structural components of pathogens, such as bacterial flagellin and fungal chitin, which play a critical role in activating immune responses. These conserved molecules are known as pathogen-associated molecular patterns (PAMPs), and the defense response initiated upon their recognition is referred to as PAMP-triggered immunity (PTI) [20].

Another important signaling molecule produced in plants upon pathogen challenge is nitric oxide (NO), which interacts with hydrogen peroxide to restrict pathogen spread through a defense mechanism commonly known as the hypersensitive response [21]. Consequently, researchers have adopted a proactive strategy by exogenously applying key signaling molecules such as salicylic acid [22], chitosan [23], sodium nitroprusside [24] methyl jasmonate, and calcium carbide to enhance innate plant resistance. The activation of host defense responses by these inducers leads to stronger and more durable resistance against pathogen invasion [25]. This management strategy is particularly suitable for vegetable production systems, as these crops are largely consumed fresh or minimally processed, making fungicide residues unacceptable to both consumers and stakeholders [26][27].

Several efforts have been undertaken in the past to contain pathogens that incorporate physical elimination of the infected plants and cultivation of resistant varieties. Use of chemicals and application of bio-control agents and all these are pitfalls since they provide support externally. Thus, the current research is an attempt to observe the impact of exogenous use of inducers that trigger innate immunity in plants against fungi that destroy tomato seedlings.

**Novelty Statement:**

This study uniquely compares multiple resistance inducers (JA, SA, ET, and NO donors) within a single framework to elucidate their pathway-specific effectiveness against the necrotrophic pathogen *Alternaria solani*. It highlights the superior role of jasmonate- and ethylene-mediated defenses over salicylic acid in reducing disease severity. The findings provide a mechanism-based, sustainable alternative to fungicides for early blight management in tomato.

**Objectives:**

To isolate, identify, and confirm the pathogenicity of fungal pathogens associated with diseased tomato seedlings collected from Rawalpindi and Islamabad.

To quantitatively evaluate the efficacy of selected resistance inducers (methyl jasmonate, salicylic acid, chitosan, sodium nitroprusside, and calcium carbide) in reducing early blight severity caused by *Alternaria solani* under controlled conditions.

**Materials and Methods:**

The present study was conducted in the Department of Plant Pathology, Pir Mehr Ali Shah Arid Agriculture University, Rawalpindi (PMAS-AAUR). Tomato cultivar Reograndi and line 95017 were sown in plastic cups containing a growth medium composed of decomposed coconut dust, cow dung, sand, leaf litter, and soil mixed in a 2:1:1:1:1 ratio, respectively.

**Disease Samples**:

Collection of disease samples should be done by means of collection tubes or test tubes. Collection of Disease Samples. Disease samples should be collected using collection tubes or test tubes.

Tomato leaves showing distinctive symptoms of early blight (EB) and damping-off were collected from experimental fields and tunnels of NARC-Islamabad and PMAS-AAUR. Rhizosphere soil of infected plants was also taken. Pathogens were isolated from disease samples collected in sterile polythene bags and preserved at 4 °C in sterile polythene bags and preserving the samples at 4 °C. The occurrence of diseases was determined according to equation.

$$\text{Incidence of disease (\%)} = \frac{\text{Number of infected plants}}{\text{Total no. of plants}} \times 100$$

**Isolation and characterization of plant pathogenic fungi: isolation of *A. solani:***

Both infected and healthy plant tissues were excised into small segments (5–10 mm) and surface-sterilized by immersion in 1% Clorox (sodium hypochlorite) for 2 minutes to remove surface contaminants. To neutralize the residual effects of the disinfectant, the samples were rinsed five times with sterile distilled water and subsequently dried on sterile filter paper to remove excess moisture. Four tissue pieces were placed in Petri plates containing potato dextrose agar (PDA) and Czapek–Dox agar (CDA) supplemented with streptomycin (50 mg $L^{-1}$) and incubated at 25 ± 1 °C. After seven days, fungal growth emerging from the infected tissues was subcultured onto fresh CDA plates for further purification.

**Isolation of *Phythium spp*:**

Isolation of *Pythium spp.* was performed using the baiting technique, where small pieces of cucumber were placed in polythene bags containing infected soil, and a few drops of distilled water were added. The bags were incubated at 25 ± 1 °C for two days. *Pythium spp.* were cultured on corn meal agar (CMA) using the bait source. The Phythium spp mycelium was selected in the bait cucumber using an inoculating loop and placed at the centre of the Petri plate. The incubator at 25+1 o C was used to incubate petri plates over a period of seven days.

**Disease susceptibility, Pathogenicity, and Screening assay of tomato:**

The pathogenicity test was conducted on tomato line 95017 and cultivar Reograndi. Surface sterilization of tomato seeds of the two varieties was done by placing them in 2% sodium hypochlorite solution for two minutes, followed by washing using sterile distilled water. The conidial suspension was prepared by taking a ten-day culture of *A. solani* from a 10-day-old culture of *A. solani* at the rate of 5106 conidia/mL in sterile distilled water of a 10-day-old culture maintained with the assistance of a haemocytometer and combined with 0.01% Tween 80 solution. Tomato plants were inoculated at six weeks of age that were six weeks old with the help of a hand-run sprayer at the rate of 10-15 mL/plant, and maintenance of all the conditions conducive to tomato plants and the pathogen. Control plants were sprayed with sterile distilled water. All the test plants were incubated in polythene chambers at 20 o C and high relative humidity of 48 hours, and then uncovered and stored in a greenhouse. The experiment was conducted twice using five seedlings of each variety. The experimental plants were observed regularly, and after one week of inoculation, disease development was observed.

**Resistance induced using various inducers:**

The experiment followed a completely randomized design (CRD) with five replications, each replication comprising five plants. To ensure the reliability and reproducibility of the results, the entire experiment was conducted twice. Six-week-old plants of tomato line 95017 were used as the experimental material for evaluating resistance induction. Various resistance-inducing compounds were applied, including salicylic acid (SA), methyl jasmonate (MeJA), and chitosan (CH), while calcium carbide ($CaC_2$) and sodium nitroprusside (SNP) served as sources of ethylene (ET) and nitric oxide (NO), respectively. Before initiating the resistance induction trial, all inducers were evaluated to determine concentrations that did not cause phytotoxic effects. For the assessment of innate immune responses against a virulent pathogen, tomato plants treated with the resistance inducers were inoculated with the pathogen 48 hours after the application of the treatments.

**Fungal inoculum preparation and application:**

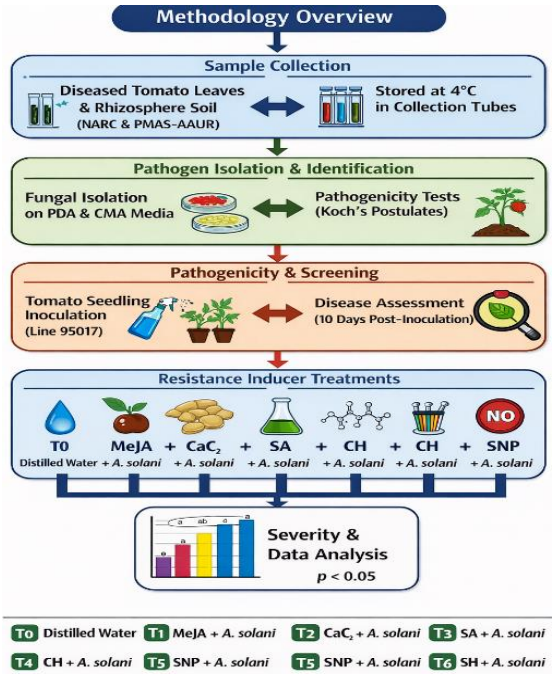
To prepare the spore suspension, a 10-day-old EB culture was grown and then harvested by teasing the fungal colony with a glass slide, and then combined with 0.01% Tween

80 solution. The suspension was mixed using a low-speed vortex. Suspension of spores was filtered using sterile cheesecloth to eliminate bigger portions of mycelia. The inoculum load was determined using a haemocytometer, and the plants were inoculated with 10–15 mL per plant of a spore suspension containing $5 \times 10^6$ conidia $mL^{-1}$. All inducer-treated plants were exogenously sprayed with the *Alternaria solani* spore suspension, whereas the negative control plants were sprayed only with sterile distilled water. Following inoculation, the plants were maintained in a polythene chamber at 20 °C and 95% relative humidity for 48 hours. Early blight (EB) severity on each treated plant was assessed using a 0–5 rating scale, where 0 indicated no visible leaf lesions, 1 represented up to 10% leaf area affected, 2 indicated 11–25% infection, 3 corresponded to 26–50% infection, 4 denoted 51–75% infection, and 5 represented 75–100% infection or complete leaf abscission. Disease symptoms were recorded 10 days after inoculation. Plants were classified as resistant when rated 0–1, moderately resistant when rated 2–3, and susceptible when rated 4–5. The disease ratings were subsequently converted into the percentage early blight index (PEBI) for each plant using the following formula.

$$\textbf{PEBI} = \frac{\textbf{Sum of all ratings}}{\textbf{no.of leaves sampled} \times \textbf{Maximum Disease scale}\ )} \times 100$$

**Treatments of Inducers and Fungal Inoculation:**

To=Distal water
T1=MeJA + *Alternaria solani*
T2=$CaC_2$ + *Alternaria solani*
T3= SA+*Alternaria solani*
T4=CH+ *Alternaria solani*
T5=SNP + *Alternaria solani*
T6=*Alternaita solani*




**Statistical Analysis:**

Analysis of variance (ANOVA) was performed by using SPSS 16.0 software. Post hoc tests were performed using the least significant difference (LSD) test, and a 5% level of significance was used ($\alpha = 0.05$).

**Results:**

As we sampled tomatoes of diseased plants, we observed tomato plants exhibiting

typical symptoms of EB on leaves that, in severe cases, would converge and result in the death of foliage Figure 1a. EB-infected tomato plants were also discovered not only at NARC but also in PMAS-arid agriculture. Nonetheless, the disease incidence is 66 in NARC and 22 in PMAS-AAUR. Nevertheless, we could only detect tomato plants with symptoms of damping off (54) in tomato plantations at NARC but not damping off in tomato plants at PMAS-AAUR Figure 1b; Table 1.

**Isolation and characterization of pathogens of infected tomato plants:**

In the present study, *Alternaria solani*, the causal agent of early blight (EB), exhibited black, circular colonies on potato dextrose agar (PDA) and dark brown growth on Czapek–Dox agar (CDA) after seven days of incubation at 25 °C Figure 2a. The fungus was slow-growing and required approximately seven days to completely cover the Petri plate. Microscopic examination showed that the conidiophores were solitary or arranged in small groups, straight or flexuous in form, and ranged in color from brown to dusky yellowish. The conidia were mostly solitary, variable in length, and typically contained both transverse and longitudinal septa. Conidia of diverse shapes were observed, including straight or ovoid forms with acuminate apices and long apical beaks, exhibiting brown to pale golden coloration. The colonies of *A. solani* displayed circular margins with a smooth surface Figure 2b. *Pythium spp.* did not grow on PDA but grew on CMA using the baiting method. Phythium spp grew mycelially on cucumber baits after 48 hrs which also grew successfully on CMA. The Phythium spp mycelium developed in a radial pattern that further spread to the entire petri plate. Whitish, fluffy, and cottony growth was observed Figure 2c and 2d was seen on CMA. Attempts to induce sporulation of *Pythium spp.* on artificial media, including rye meal agar (RMA), were unsuccessful.

**Tomato pants pathogenicity assay:**

Pathogenicity assay was also done to confirm the postulates of Koch, in addition to eliminating the majority of the most susceptible planting material between the tomato variety Reograndi and line-95017 to be used further in the resistance induction experiment. Our assay established the pathogenicity of EB on Riogrande and line-95017; however, relative to Riogrande, line-95017 exhibited a higher level of susceptibility Figure 3a and 3b. Re-isolation and re-inoculation on plants and media confirmed the postulates of Koch, as features of Tomato plants, and such mycelial growth had symptoms of septate hyphae as well as cross-sectional and peaked conidia on media plates.

**Influence of inducers in the tomato plant against EB:**

Tomato plants exogenously treated with resistance inducers, including methyl jasmonate (MeJA), calcium carbide ($CaC_2$) as an ethylene (ET) donor, salicylic acid (SA), chitosan (CH), and sodium nitroprusside (SNP) as a nitric oxide (NO) donor, were subsequently inoculated with early blight (EB) spores using a foliar spray method. In general, plants inoculated with *Alternaria solani* alone did not exhibit visible symptoms until 5–6 days post-inoculation, whereas plants pre-treated with resistance inducers required approximately 10 days to develop symptoms Figure 4a, Figure 4b. These observations indicate that disease severity, expressed as the number and size of blight lesions per leaf, decreased over time in treated plants, while lesion development progressively increased in untreated plants.

Disease severity data recorded 10 days after inoculation revealed that MeJA treatment was the most effective, resulting in the lowest percentage early blight index (PEBI) of 14.15% compared with the positive control (44.98%). A PEBI value of 14.15% corresponded to the moderately resistant category, whereas the value of 44.98% for the positive control fell within the susceptible category. Plants treated with $CaC_2$ (ET donor) and CH also showed statistically non-significant differences compared with MeJA, but exhibited significantly lower disease severity than SA-treated plants and the positive control. Plants treated with SNP (NO donor)

displayed disease severity levels comparable to CH, showing non-significant differences between these two treatments, but significant differences when compared with MeJA, $CaC_2$, SA, and the positive control.

Although SA treatment resulted in some enhancement of resistance compared to the positive control, the effect was not significant, as the PEBI value of 30.13% fell within disease rating scale of 3, indicating that the plants remained susceptible to further pathogen attack. Overall, pre-treatment of tomato plants with signaling molecules such as MeJA, $CaC_2$, SNP, and CH generally enhanced disease tolerance but failed to induce a fully resistant phenotype. In contrast, SA treatment showed limited potential in improving tolerance, and SA-treated plants remained vulnerable to EB infection Figure 5; Table 2.


**Figure 1.** Tomato plants showing symptoms of disease. (a) Tomato plants showing concentric rings on the leaves of EB. (b) Uprooted tomato plants with damaged roots showing damping-off symptoms.

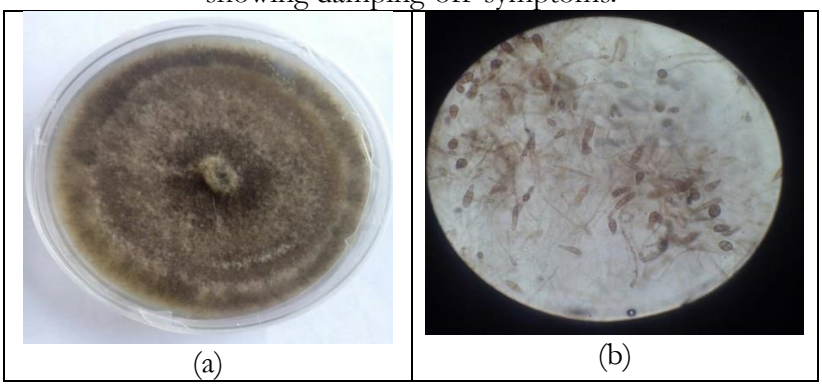



**Figure 2.** *Alternaria solani* growing on CDA media. (a) Dark brown concentric *A. solani* culture on a CDA plate. (b) *A. solani* cross-sectional and beaked conidiophores

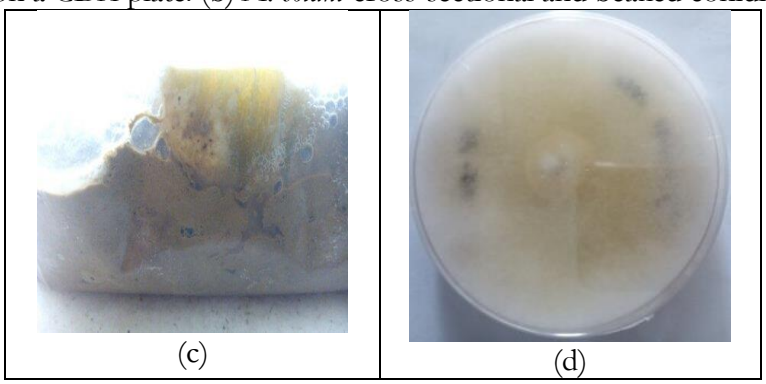
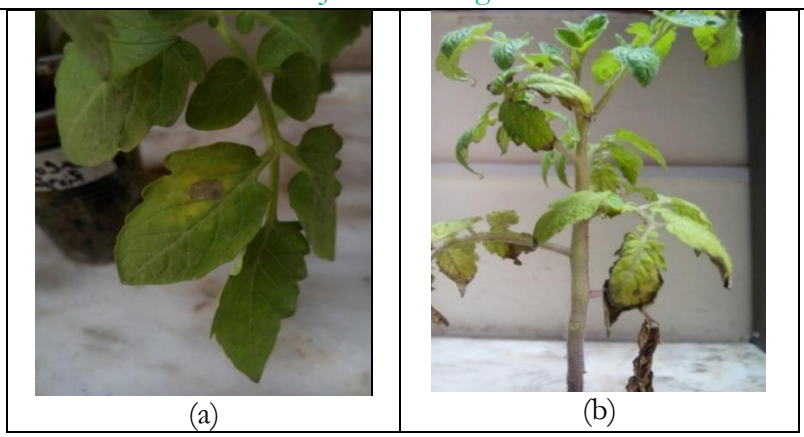



**Figure 2.** Isolation of *Phythium spp* (c) *Phythium spp* growing on cucumber slices (d) White cottony growth of *Phythium spp* on CMA media



(a) (b)

**Figure 3.** Pathogenicity assay on tomato plants after 10 days of post-inoculation with EB (a) Symptoms of EB on Riogrande (b) Symptoms on line-95017


(a) (b)

**Figure 4.** EB symptoms shown by inducer-treated and untreated plants after 10 days of *A. solani* inoculation (a) Inducer Un-treated Plant (b) Inducer Treated Plant

**Table 1.** Disease Incidence (%) of early blight and damping-off in tomato fields and tunnel

| Locality | Early Blight | Damping off |
|---|---|---|
| NARC | 66% | 54% |
| PMAS | 22% | 0% |

**Table 2.** Means (%) disease severity and standard deviation of EB on inducer-treated and untreated plants of tomato line-95017 after 10 days of post-inoculation

| Inducers | Mean | Std. Deviation |
|---|---|---|
| Methyl Jasmonate (MeJA) | 14.15 (d) | 1.92423 |
| Calcium carbide ($CaC_2$) | 16.58 (d) | 3.48131 |
| Chitosan (CH) | 18.21 (cd) | 5.46540 |
| Sodium nitroprusside (SNP) | 23.80 (c) | 4.67031 |
| Salicylic acid (SA) | 30.13 (b) | 4.06059 |
| Positive control | 44.98 (a) | 3.04562 |

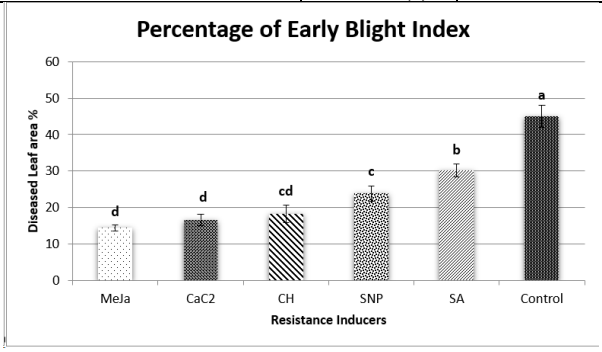



**Figure 5.** Diseased leaf area in tomato plants after 10 days of inoculation with EB

**Discussion:**

Plant resistance in the face of pathogenic attack operates at multiple layers of defense. The first barrier is typically *constitutive physical resistance*, including cuticle and wax layers, trichomes, and stomatal regulation, which inherently protect plants against a broad spectrum of microbes. This non-host resistance is pervasive and stable, providing a baseline immunity that prevents the majority of pathogens from establishing infection [28]. Constitutive physical structures thereby act independently of specific recognition events to prevent pathogen ingress.

At the molecular level, conserved microbial components such as bacterial flagellin and fungal cell-wall chitin function as pathogen/microbe-associated molecular patterns (PAMPs/MAMPs) that are perceived by pattern recognition receptors (PRRs) on the plant cell surface [29]. This recognition elicits PAMP-triggered immunity (PTI), an innate immune response that activates downstream signaling events—including influxes of $Ca^{2+}$, generation of reactive oxygen species (ROS), and MAP kinase cascades—leading to transcriptional reprogramming of defense genes [28][30]. PTI represents a basal resistance that is broad in scope but often insufficient to stop highly specialized pathogens.

To evade this first line of defense, many pathogens deploy effectors—proteins delivered into host cells via structures such as type III secretion systems (T3SS) or specialized infection structures like appressoria. Effectors can suppress PTI or manipulate host physiology to facilitate colonization. Plants counter these effectors through intracellular nucleotide-binding leucine-rich repeat (NLR) receptors, resulting in effector-triggered immunity (ETI), which typically leads to a stronger resistance response, including localized cell death (hypersensitive response) and systemic responses. Together, PTI and ETI form a multilayered immune architecture that limits pathogen success.

Downstream of PTI and ETI, signaling molecules such as salicylic acid (SA), jasmonic acid (JA), ethylene (ET), nitric oxide (NO), and ROS coordinate defense outputs and hormonal crosstalk. SA is mainly associated with resistance against biotrophs and systemic acquired resistance (SAR), whereas JA/ET pathways are critical for resistance against necrotrophic pathogens such as *Alternaria solani*. The SA–JA/ET interplay can be antagonistic or synergistic depending on the context and pathogen lifestyle.

Plants also possess PR (pathogenesis-related) proteins that accumulate in response to infection and contribute antimicrobial activity, as well as defense priming mechanisms that result in faster and more robust responses upon secondary challenge. Priming can be induced through environmental cues, beneficial microbes, or chemical elicitors, forming an enhanced state of readiness without inducing full defense responses until infection occurs.

**Findings of the Present Research:**

The current study was designed to observe the impact of exogenous application (priming) of the susceptible tomato line-95017 before challenge with *A. solani*. We report results consistent with previous work showing that environmental conditions typical of the *kharif* season (20–25°C and high moisture) are conducive to early blight (EB) development. EB is a polycyclic foliar disease, and inoculum levels remain high through conducive seasons, increasing disease pressure, especially in tender seedlings [31].

Seedling resistance is often controlled by single, environment-insensitive genes that confer early protection and are generally favored over adult-plant resistance, which may be conditional on environmental cues. In field sampling, damping-off due to *Pythium* spp. was observed at NARC but not at PMAS-AAUR, likely due to differences in inoculum presence and irrigation conditions that influence *Pythium* zoospore dissemination [6]. Our inability to sporulate *Pythium* spp. in vitro aligns with documented challenges in artificial cultivation of this pathogen.

In pathogenicity assays, variety Reograndi showed delayed EB symptom progression

compared to line-95017, indicating that genetic differences influence disease expression. Various inducers were applied, including MeJA, SA, ET donor ($CaC_2$), NO donor (SNP), and chitosan (CH). MeJA (JA pathway) significantly reduced susceptibility—approximately threefold less than positive controls—suggesting effective induction of defense responses aligned with ISR against necrotrophs and CH also showed enhanced tolerance compared to controls, which is consistent with the understanding that SA-mediated pathways that SA-mediated pathways are less impactful on necrotrophic pathogen resistance.

Inducers not only initiate systemic resistance but also elevate basal immune states (PTI), priming cells for future attacks [32][33]. Although NO triggers hypersensitive responses predominantly effective against biotrophs, its efficacy against necrotrophs like *A. solani* is limited [33]. Chitosan similarly acted as an effective inducer, corroborating prior findings in tomato challenged with other necrotrophic pathogens.

These results reinforce that induced resistance and defense priming represent environmentally safer and robust strategies for crop protection relative to conventional chemicals, which carry ecological risks. Despite decades of research, achieving complete immunity remains elusive due to pathogen adaptability; however, induced resistance offers a complementary tool, especially when applied prophylactically.

**Conclusion:**

The present study demonstrated that tomato defense against *Alternaria solani* operates through multiple layers, from constitutive physical barriers to inducible biochemical pathways. Seedling resistance, controlled by genetic factors, provided a reliable defense that persisted throughout plant development. Exogenous application of inducers, particularly MeJA and ET donors, effectively primed tomato plants, reducing disease severity by enhancing systemic and basal defenses. Chitosan also showed significant potential as a resistance inducer, whereas SA and NO were comparatively less effective against the necrotrophic *A. solani*. These findings reinforce the utility of defense priming as a sustainable and environmentally safe strategy to enhance crop resistance, complementing genetic resistance and biological control.

**Future perspective:**

Future efforts should focus on integrating genetic resistance with optimized inducer treatments, leveraging advances in genomics, gene editing, and multi-omics approaches to understand and enhance host-pathogen interactions. Additionally, large-scale field validation of inducers, exploration of novel bioactive elicitors, and the development of integrated disease management programs will be essential to provide sustainable, durable, and practical control of early blight in tomato under diverse environmental conditions.

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