Journal of Applied and Natural Science
15(1), 100 - 106 (2023)
ISSN : 0974-9411 (Print), 2231-5209 (Online)
journals.ansfoundation.org

Research Article

Screening of tomato (*Solanum lycopersicon* L.) genotypes by inducing systemic resistance against early blight disease caused by *Alternaria solani*

Rex B*
Department of Plant Pathology, SRM College of Agricultural Sciences, SRMIST, Chengalpattu - 603201 (Tamil Nadu), India
Gopu B
Department of Horticulture, SRM College of Agricultural Sciences, SRMIST, Chengalpattu - 603201 (Tamil Nadu), India
Vinothini N
Department of Seed Science & Technology, SRM College of Agricultural Sciences, SRMIST, Chengalpattu - 603201 (Tamil Nadu), India
Prabhu S
Department of Plant Protection, Tamil Nadu Agricultural University, Coimbatore - 641003 (Tamil Nadu), India

*Corresponding author. Email: rexpatho@gmail.com

Article Info
https://doi.org/10.31018/jans.v15i1.4285
Received: December 11, 2022
Revised: January 27, 2023
Accepted: February 1, 2023

How to Cite
Rex, B. *et al.* (2023). Screening of tomato (*Solanum lycopersicon* L.) genotypes by inducing systemic resistance against early blight disease caused by *Alternaria solani*. *Journal of Applied and Natural Science*, 15(1), 100 - 106. https://doi.org/10.31018/jans.v15i1.4285

Abstract
Early blight of tomato (*Solanum lycopersicon* L.) incited by *Alternaria solani* is highly destructive disease in the world. Environmental factors significantly impact early blight epidemics, leading to the loss of up to 78 per cent of tomato production. Twenty tomato genotypes were used in this study to identify the early blight resistant and susceptible genotypes selected to represent a range of reactions when screened under field conditions. The tomato plants were evaluated for early blight disease by using Per cent Disease Index (PDI). Pusa Uphar (20.18%) and Sankaranti (20.18%) showed resistance to early blight disease among the twenty genotypes. Anaka Kerala (61.25%), Arka Vikas (61.76%), Pusa Rohini (53.65%), Ashoka (50.60%) and Paiyur1 (56.08%) genotypes were found highly susceptible based on early blight disease intensity. Spore inoculation of *A. solani* was sprayed into tomato plants, it was discovered that the number of defense-inducing compounds *viz.,* total phenols, peroxidase (PO), polyphenol oxidase (PPO) and phenylalanine ammonia lyase (PAL) has increased. Among the genotypes, Pusa Uphar (T_2) and Sankaranti (T_1) genotypes showed a high level of defense enzyme production. After tomato plants were exposed to pathogens through artificial inoculation, the activity of these defensive enzymes and compounds was highly induced in the resistant and sensitive germplasm than in the control.

Keywords: *Alternaria solani,* Genotypes, Induced Systemic Resistance, Resistance, Susceptible

INTRODUCTION

After potato, tomato (*S. lycopersicon L.)* is the second-most valuable solanaceous vegetable crop grown for both domestic and international trade. It originates from South America and over 150 countries produced about 182 million tonnes of tomato in 2018 on an area of 4.76 million hectares. Since tomatoes are the richest source of antioxidants (lycopene pigment) and vitamins (A and C), they are essential to a balanced diet (Rasool *et al*., 2021). It is a significant commercial crop and a popular consistent vegetable among consumers due to its high nutritional content. Minerals, vitamins, organic acids, necessary amino acids, and dietary fibre are all abundant in tomato. The area planted with tomato has gradually increased, although production has fluctuated due to several diseases and insect pest damage. (Sahu *et al*., 2013).

Balanchard (1992) reported that fungi, bacteria, viruses, nematodes, and other abiotic agents are causing numerous diseases in tomatoes. One of the most devastating diseases of tomato in the world is target spot

disease, commonly known as early blight incited by *Alternaria solani* (Abada *et al*., 2008). *It* significantly reduces both the quantity and quality of fruit supply. Under favourable conditions, the soil-inhabiting and airborne causative organism cause illness to occur on leaves, stems, petioles, twigs, and fruits. This causes defoliation, twig drying out and premature fruit drop, which reduces fruit yield by 50 to 86 per cent. It is most prevalent in regions of high dew, rain, and relative humidity (Datar and Mayee, 1981). When the season begins with plenty of moisture or regular showers followed by warm and dry weather that is unfavourable for the host and aids in rapid disease growth, the disease spreads widely and becomes serious, inflicting significant economic loss to the growers (Shoaib *et al.,* 2020). The usage of agrochemicals is the principal method to control early blight disease. However, these chemical fungicides need to be reduced to comply with the global trend toward environmentally safe plant disease management in sustainable agriculture. Therefore, the only approach to creating cultivars of tomato resistant to early blight is to identify and use genetic resources resistant to *A. solani* through the use of proper breeding techniques (Rex *et al*., 2019). The most common approach for determining tomato genotypes' resistance to early blight is field evaluation following inoculation of the pathogen isolates (Farooq *et al*., 2019).

Induced Systemic Resistance (ISR) enhances multiple potential defense mechanism in plants by increasing higher activity of b-1,3-glucanases, chitinases, and peroxidases and also accretion of antimicrobial low molecular weight substances such as protective biopolymers formation like lignin and phytoalexins, hydroxy proline rich glycoprotein and callose (Shoaib *et al*., 2020). Pathogenesis-related proteins (PRs), such as chitinases, which break down the cell wall and result in cell lysis, are additional defence enzymes.

A pathogen is introduced to plants in a way that increases their ability to defend themselves against a wide range of infections. Induced resistance may offer an alternate method of plant protection, particularly for issues that other fungicides cannot adequately control (Daroodi et al., 2021). The present investigation was intended to evaluate different tomato genotypes against *Alternaria solani* causing early blight disease under field conditions.

MATERIALS AND METHODS

In the field trial, thirty days old seedlings of twenty tomato genotypes *viz.,* Pusa Rub, Pusa Sadabaha, Sankranti, Anaka Kerala, Arka Abha, Punjab Chuhara, Akshaya, Pusa Rohini, Ashoka, Arka Meghali, CO1, Pusa Gourav, PKM1, CO2, Pusa Sheetal, CO3, Pusa Uphar, Arka Vikas, Paiyur 1 and VR-20 were transplanted in four rows with four replications of each treatment and spacing of 60cm between rows and 50cm between plants was maintained. On Potato Dextrose Agar (PDA) medium, the pathogen *A. solani* that causes early blight disease was isolated from infected tomato leaves. The plates were incubated for 10-15 days at 25° ± 2°C. The plants were challenged with the pathogen *A. solani* ($5x10^6$ spores/ml) to all tomato genotypes after 45 days of transplanting to initiate the screening process with a sprayer. The uninoculated plants were considered as a control. The disease intensity was assessed on a 0-9 scale (TNAU, 1980).

Per cent Disease Index (PDI) was calculated using McKinney (1923) formula *viz.,*

PDI = (Sum of all numerical ratings/Total number of leaves observed) x (100 / Maximum grade in the score chart) ……Eq. 1

Disease reaction classes for early blight infection based on percent disease severity in tomato given by Singh *et al*. (2011) were followed (Table 1).

The twenty tomato genotypes were screened against the early blight disease by above formula. The Per cent Disease Index and disease reaction were graded by using the above conditions based on disease severity in tomato plants.

For enzyme assay, two tomato genotypes from each disease reaction were selected *viz*., Sankranti (T_1), Pusa Uphar (T_2), PKM1 (T_3), Punjab Chuhara (T_4), Arka Vikas (T_5), Paiyur 1 (T_6), CO2 (T_7), Pusa Rubi (T_8) and Control (T_9) during tomato genotype screening against early blight disease (Table 3). The tomato leaves were taken after one day of inoculation with *A. solani* and the uninoculated plant was considered as control. Induction of defence-related enzymes were identified in selected tomato genotypes challenged with *A. solani*.

Resistance mechanism

Assay of peroxidase (PO)

A pre-cooled pestle and mortar was used to ground one gram of fresh leaf tissue in one ml of 0.1 M phosphate buffer pH 7.0. The homogenate was centrifuged for 15 minutes at 15,000 rpm at 4° C and the supernatant solution was used as enzyme source. The reaction mixture includes 0.1 ml of enzyme extract, 1.5 ml of 0.05 M pyrogallol and 0.5 ml of 1 % Hydrogen peroxide. At ambient temperature (28 ± 2˚C) the reaction mixture's change in absorbance was recorded at 420 nm every 30 seconds for three minutes. The prepared boiled enzyme served as a control. The change in absorbance of the reaction mixture $min^{-1}g^{-1}$ of a leaf was used to express the enzyme activity (Hammerschmidt and Kuc, 1995).

Assay of poly phenol oxidase (PPO)

One g of leaf sample (fresh) was ground in 1 ml of 0.1 M $Na_3 PO_4$ buffer (pH 6.5). The homogenate was cen-

Table 1. Disease scale for early blight disease on tomato leaves

Disease rating	Symptom severity	PDI range (%)	Disease reaction
0	No visible symptoms	0	Immune (I)
1	50 per cent leaf area infected	0.01 - 10.00	Highly resistant (HR)
3	25 per cent leaf area infected	10.01 - 25.00	Resistant (R)
5	50 per cent leaf area infected	25.01 - 40.00	Moderately resistant (MR) / Moderately susceptible (MS)
7	75 per cent leaf area infected	40.01 - 60.00	Susceptible (S)
9	The entire plant has blighted leaves and is dead	> 60	Highly susceptible (HS)

trifuged for 15 minutes at 15,000 rpm at 4˚ C and the supernatant was used as the enzyme source. The reaction mixture combination includes 1.5 ml of 0.1 M Na_3 PO_4 buffer pH 6.5 and 0.1 ml of the enzyme extract. The reaction started with the addition of 0.2 ml of catechol (0.01M). The enzyme activity was expressed as a change in absorbance (at 495 nm at 30 seconds intervals for three minutes) $min^{-1}g^{-1}$ of the leaf (Mayer *et al.*, 1965).

Total phenols

10 ml of 80 per cent methanol was added to 1 g of the fresh leaf sample after it had been ground in a pestle and mortar. The homogenate was centrifuged for 20 minutes at 10,000 rpm. The supernatant solution was dried by evaporation and the residue materials was dissolved in 5 ml of distilled water. From this solution, 0.2 ml was taken and the volume was made up to 3 ml with distilled water. After that, the reagent 0.25 ml of (1N) Folin-Ciocalteau was added. After a period of 3 minutes, 1 ml of 20 per cent Na_2 CO_3 was added and mixed thoroughly. Then the tubes were placed in boiling water for 60 seconds and cooled. At 725 nm, the absorbance was calculated against a blank for the reagent. The total phenol activity was expressed as µg of catechol g^{-1} of plant tissue (Zieslin and Ben Zaken, 1993).

Assay of phenylalanine ammonia lyase (PAL)

500 mg of the leaf was homogenized in five ml of cold 25 mM borate HCl buffer (pH 8.8) containing 5 mM mercaptoethanol, 0.4 ml per litre. The homogenate was centrifuged for 15 minutes at 15,000 rpm and the supernatant was utilized. The mixture assay consists of 0.5 ml borate buffer, 0.2 ml of enzyme extract and 1.3 ml water. With the addition of one ml of 12 mML-Phenylalanine the reaction was started. At 32 °C, the reaction mixture was incubated for an hour. The reaction was stopped by the addition of 0.5 ml of 2N HCl. A blank was run in which phenylalanine was added after adding 2N HCl. The absorbance was measured at 290 nm. The enzyme activity was expressed as µ mol of trans-cinnamic acid/ minute/ g of the leaf (Dickerson *et al*., 1984).

Statistical analysis

The means of the treatments were compared using Duncan's Multiple Range Test (DMRT) after the data had been statistically analyzed (Gomez and Gomez, 1984). The International Rice Research Institute Biometrics Units, Philippines, produced the IRRI-Stat version 92-a software programme, which was used for the analysis.

RESULTS AND DISCUSSION

The twenty tomato genotypes were evaluated for resistance to early blight disease by testing the host with *A. solani*. The genotypes reacted to early blight disease based on the disease severity. The germplasm reaction was documented using the Per cent Disease Index (PDI) formula. The results of the screening revealed that no tested genotype was disease-free.

The germplasm Pusa Uphar (20.18 PDI) and Sankaranti (21.32 PDI) showed resistance to early blight disease compared to other genotypes. The moderately resistant / moderately susceptible reacted based on the disease severity was observed in the germplasm Pusa Gourav (26.80 PDI), Pusa Rubi (28.61 PDI), Akshaya (30.31 PDI), VR-1 (32.10 PDI), CO2 (35.45 PDI) and Arka Abha (36.08 PDI). While the susceptible germplasms were Arka Meghali (38.12 PDI), Pusa Sadabahar (40.74 PDI), CO1 (42.16 PDI), PKM1 (45.65 PDI), Punjab Chubara (47.23 PDI) and CO3 (48.12 PDI). Maximum disease severity was observed in highly susceptible varieties of Anaka Kerala (61.25 PDI), Arka Vikas (61.76 PDI), Pusa Rohoini (53.65 PDI), Ashoka (50.60 PDI) and Paiyur 1 (56.08 PDI) (Table 2).

Similarly, the resistant tomato genotypes has been identified by different genotype evolution in subtropical condition (Vijaya Mahanttesha *et al*., 2012). The response of the plant against *A. solani* was measured by observing the symptoms developed after inoculation. These results were similar to the earlier investigation, in which it was found that no tomato genotype, wild accession, or breeding line was totally immune to early blight (Foolad and Ashrafi, 2015). In this study, based on PDI two genotypes were categorised as resistant; six were moderately susceptible/ moderately susceptible, six were susceptible and six were highly suscepti-

ble to tomato early blight infection. These disease reactions were used in tomato varietal screening against early blight disease. (Shoaib *et al.*, 2020).

The use of resistant genotypes is considered a cost-effective and long-lasting disease control strategy. According to Gondal *et al*. (2012), developing resistant cultivars is the best way to combat disease. Significant work has been focused on identifying sources of resistant tomatoes under environmental conditions by evaluating tomato genotypes against *A. solani*. (Akhtar *et al*., 2019; Sales *et al*., 2011).

The induction of peroxidise, poly phenol oxidase, PAL and total phenol activity in tomato plants tested with *A. solani* was estimated and the result is presented in Table 3. The results showed that PO, PAL, PPO, and phenol activity were observed in all treatments, including the control. In addition, it was observed that the activity of PO, PPO and PAL slowly increased from the third day onwards and attained maximum level on the fifth day after injection and the level dropped in all the treatments during the seventh day (Fig. 1).

Among the treatments, (T_1) Sankranti exhibited a significantly higher level of PO activity in absorbance /min/g of leaf tissue (1.38), followed by (T_2) Pusa Uphar which recorded 0.98 changes in absorbance/min/g of leaf tissue, CO2 (T_7) recorded 2.07, Pusa Rubi (T_8) recorded 0.59, PKM1 (T_3) showed 0.55, Punjab Chuhara (T_4) observed 0.39, Paiyur1 (T_6) and Arka Vikas (T_5) recorded 0.31 and 0.23 changes in absorbance /min/g of leaf tissue. However, a very low level of changes in absorbance /min/g of leaf tissue was witnessed in control (0.20) (Table 3).

When tomato plants were treated with *A. solani*, a dramatic elevation in PPO activity was seen. (T_1) Sankranti had the highest (1.36) accumulation of PPO followed by (T_2) Pusa Uphar (1.03), Pusa Rubi (T_8) (0.51), CO2 (T_7) (0.46) level of changes in absorbance/ min/g of leaf tissue. Plants showed the least activity in PKM1 (T_3) (0.22), Paiyur1 (T_6) (0.20), Punjab Chuhara (T_4) (0.14) and Arka Vikas (T_5) (0.11) level of changes in absorbance/ min/g of leaf tissue. However, a very low level of µ mol of trans cinnamic acid / min /g of leaf tissue was observed in the control treatment (0.08) (Table 3).

Phenylalanine ammonia lyase activity in tomato plants was estimated. The result showed the highest activity in with 5.12 and 4.28 changes in µ mol of trans cinnamic acid /min/g of leaf tissue of (T_2) Pusa Uphar and (T_1) Sankranti. The result of other treatments showed 2.46, 2.35, 1.31, 1.08, 0.78 and 0.13 changes in µ mol of trans-cinnamic activity in Pusa Rubi (T_8) (0.51), CO2 (T_7), Punjab Chuhara (T_4), PKM1 (T_3), Arka Vikas (T_5) and Paiyur1 (T_6). The uninoculated control plants recorded 0.06 changes in µ mol of trans-cinnamic acid / min/g of leaf tissue (Table 3).

The phenol content was induced in tomato plants challenged with pathogen *A. solani.* The findings of this investigation showed that the phenolic content was raised in all the treatments. Among the treatments, (T_1) Sankranti showed a significantly increased level of changes in catechol equivalents /g of leaf tissue (2.08) followed by (T_2) Pusa Uphar (1.96), Pusa Rubi (T_8) (1.63), CO2 (T_7) (1.48), Punjab Chuhara ($_{T4}$) (1.39), Paiyur1 (T_6) (1.27), PKM1 (T_3) (1.21) and Arka Vikas (T_5) (1.10) µ g of catechol / g of tomato leaf tissue. The control showed a minimal level of phenol content (0.82 µ g of catechol / g of leaf tissue) which was uninoculat-

Table 2. Evaluation of tomato genotypes against early blight disease

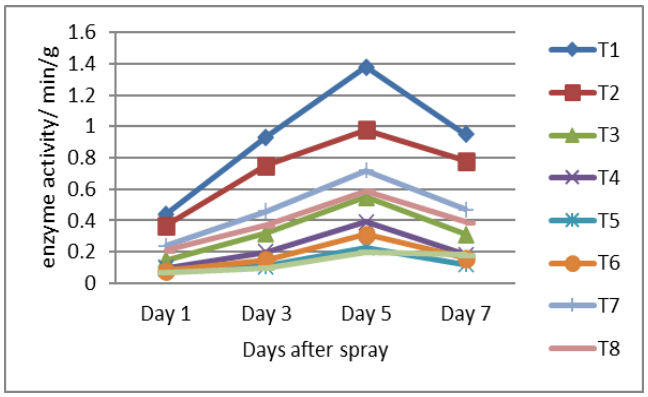
S. No.	Name of genotype	PDI (%)	Host reaction
1.	Pusa Rubi	28.61^{c} (31.53)	MR/MS
2.	Pusa Sadabaha	40.74^{h} (38.82)	S
3.	Sankranti	21.32^{a} (27.59)	R
4.	Anaka Kerala	61.25^{no} (51.42)	HS
5.	Arka Abha	36.08^{f} (37.11)	MR/MS
6.	Punjab Chuhara	47.23^{ij} (43.40)	S
7.	Akshaya	30.31^{d} (34.05)	MR/MS
8.	Pusa Rohini	53.65^{l} (47.92)	HS
9.	Ashoka	50.60^{k} (45.29)	HS
10.	Arka Meghali	38.12^{g} (38.02)	S
11.	CO1	42.16^{h} (40.32)	S
12.	Pusa Gourav	26.80^{b} (31.47)	MR/MS
13.	PKM1	45.65^{i} (42.14)	S
14.	CO2	35.45^{f} (37.34)	MR/MS
15.	Pusa Sheetal	61.76^{o} (51.65)	HS
16.	CO3	48.12^{j} (44.39)	S
17.	Pusa Uphar	20.18^{a} (27.16)	R
18.	Arka Vikas	59.75^{n} (51.92)	HS
19.	Paiyur 1	56.08^{m} (47.59)	HS
20.	VR-20	32.10^{e} (34.06)	MR/MS
SEd		0.515	-
CD (P=0.05)		1.043	-

*MR/MS – Moderately resistant /moderately susceptible; * S- Susceptible; *R- Resistant; *HS- Highly susceptible; *HR- Highly resistant

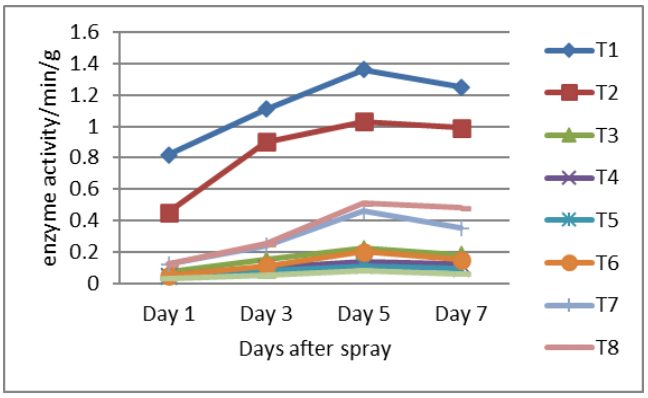
Table 3. Induction of PO, PPO, PAL and phenol activity in tomato germplasm challenged with *A. solani* causing early blight disease

Tr. No.	Genotypes	Disease reaction	PO (enzyme activity/ min/g)	Per cent Increase over control	PPO (enzyme Activity /min/g)	Per cent Increase over control	PAL (µ mol of t- Cinnamic acid/ min/g)	Per cent Increase over control	Total Phenol (µg of catechol /g)	Per cent In-crease over control
T1	Sankranti	R	1.38±0.35^{a}	5.90	1.36±1.12^{a}	16.00	4.28±0.94^{b}	70.33	2.08±0.64^{a}	1.53
T2	Pusa Uphar	R	0.98±0.41^{b}	3.90	1.03±0.98^{b}	11.87	5.12±0.79^{a}	84.33	1.96±0.78^{b}	1.39
T3	PKM1	S	0.55±0.80^{e}	1.75	0.22±0.87^{e}	1.75	1.08±1.00^{f}	17.00	1.21±0.61^{f}	0.47
T4	Punjab Chuhara	S	0.39±0.39^{f}	0.95	0.14±0.74^{f}	0.75	1.31±0.64^{e}	20.83	1.39±0.86^{e}	0.69
T5	Arka Vikas	HS	0.23±0.61^{h}	0.15	0.11±0.62^{h}	0.37	0.13±0.56^{h}	1.16	1.10±0.63^{g}	0.34
T6	Paiyur 1	HS	0.31±0.56^{g}	0.55	0.20±0.89^{f}	1.50	0.78±0.64^{g}	12.00	1.27±0.38^{f}	0.54
T7	CO2	MR/MS	0.72±0.64^{c}	2.60	0.46±1.01^{d}	4.75	2.35±0.36^{d}	38.16	1.48±0.47^{d}	0.80
T8	Pusa Rubi	MR/MS	0.59±0.78^{d}	1.95	0.51±0.66^{c}	5.37	2.46±0.78^{c}	40.00	1.63±0.48^{c}	0.98
T9	Control	-	0.20±0.55^{i}	-	0.08±0.52^{i}	-	0.06±0.90^{i}	-	0.82±0.51^{h}	-
SEd			0.03	0.10	0.04	0.16	0.05	0.87	0.08	0.05
C.D (P=0.05)			0.08	0.23	0.10	0.34	0.12	1.86	0.18	0.10

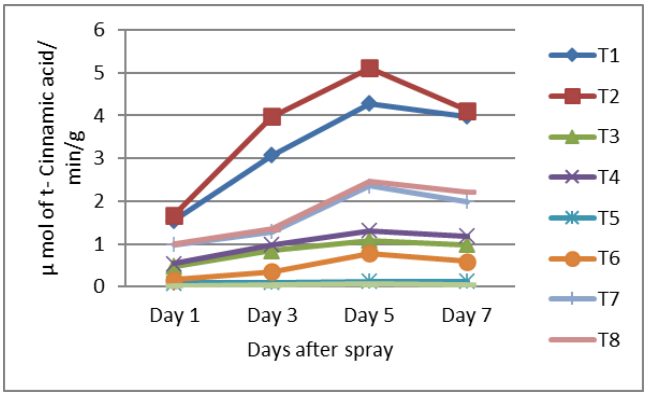
*Mean of three replications; ± represents SE of mean; Means in a column followed by same superscript are not significantly different by Duncan's Multiple Range Test at P (0.05)



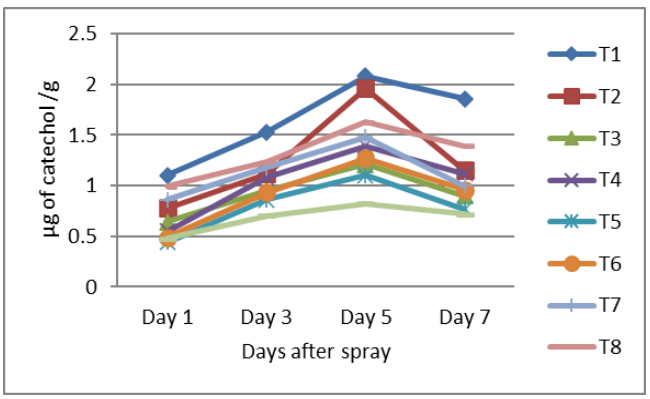
a) *Changes in PO activity*



b) *Changes in PPO activity*



c) *Changes in PAL activity*



d) *Changes in Phenol activity*

Fig 1. *Induction of PO, PPO, PAL and Phenol activity in tomato genotypes challenged with A. solani*

ed with the pathogen (Table 3).

Tomato genotypes (T_1) Sankranti and (T_2) Pusa Uphar showed a high level of defense enzymes after the plants were inoculated with *A. solani.* Similarly, Mahalakshmi *et al*. (2020) demonstrated earlier induction and increased levels of defense enzymes, *viz.,* PO, PPO, PAL and catalase, in tomato plants after inoculation of *Rhizophora apiculate.* The findings of greater enzymatic activity of PO, PPO, and chitinase cause more phenolic synthesis and accumulation, which may prevent the pathogen from spreading into the healthy ones (Anand *et al*., 2009; Gogoi *et al*., 2001). Induction of defense enzymes was found to be very low in uninoculated plants compared to the inoculated plants against early blight disease. Likewise, Kalim *et al.* (2000) reported that compared to untreated plants, plants raised with inoculation of *A. solani* had increased specific PO, PPO, and PAL and decreased specific catalase activities in their leaves. Therefore, the greater levels of total phenol and the elevated activity of PO, PPO, and PAL may have contributed to host resistance (Chen *et al*., 2000).

Conclusion

In the present study, among the twenty genotypes screened against *A. solani* under artificial inoculated conditions, two genotypes *viz.*, Sankaranti and Pusa Uphar were resistant to early blight disease. However, the stress by *A. solani* in resistant and moderately susceptible genotypes was less and even accelerated quantity of PAL, PO, and PPO activity could counter the toxic effect. The quantity of PO, PPO, and PAL activity required to counter the toxic effect due to the stress caused by *A. solani* in resistant and moderately susceptible genotypes. Hence, highly resistant genotypes with better horticultural traits are still needed to counter this threat.

Conflict of interest

The authors declare that they have no conflict of interest.

REFERENCES

1. Abada, K. A. Mostafa, S. H. & Hillal Mervat, R. (2008). Effect of some chemical salts on suppressing the infection by early blight disease of tomato. *Egypt. J. Appl. Sci*. 23 (20), 47-58.
2. Akhtar, K. P. Ullah, N. Saleem, M. Y. Iqbal, Q. Asghar, M. & Khan, A. R. (2019). Evaluation of tomato genotypes for early blight disease resistance caused by *Alternaria solani* in Pakistan. *Journal of Plant Pathology*, 101(4), 1159-1170. doi.org/10.1007/s42161-019-00304-8
3. Anand, T. Bhaskaran, R. Raguchander, T. Samiyappan, R. Prakasam, V. & Gopalakrishnan, C. (2009). Defence responses of chilli fruits to *Colletotrichum capsici* and *Alternaria alternata*. *Biol*. *Plant*, 53 (3), 553-559. doi.org/10.1007/s10535-009-0100-5
4. Balanchard, D. (1992). A colour atlas of tomato diseases. *Wolfe Pub. Ltd., Brook House*, London, p.298.
5. Chen, C. Belanger, R.R. Benhamou, N. & Paulitz, T. (2000). Defence enzymes induced in cucumber roots by treatment with plant growth promoting rhizobacteria (PGPR) and *Pythium aphanidermatum. Physiological and Molecular Plant Pathology,* 56, 13-23. doi.org/10.1006/pmpp.1999.0243
6. Daroodi, Z. Taheri, P. & Tarighi, S. (2021). Endophytic fungus *Acrophialophora jodhpurensis* induced resistance against tomato early blight via interplay of reactive oxygen species, iron and antioxidants. *Physiological and Molecular Plant Pathology*, *115*, 101681. doi.org/10.1016/j.pmpp.2021.101681
7. Datar, V. V. & Mayee, C. D. (1981). Assessment of loss in tomato yield due to early blight, *Indian Phytopath.* 34, 191-195.
8. Dickerson, D.P. Pascholati, S.F. Hagerman, A.E. Butler, L.G. & Nicholson, R.L. (1984) Phenylalanine ammonia-lyase and hydroxy cinnamate: CoA ligase in maize mesocotyls inoculated with *Helminthosporium maydis* or *Helminthosporium carbonum. Physiological Plant Pathology* 25, 111–123. doi.org/10.1016/0048-4059(84)90050-X
9. Farooq, S. Jat, R. R. Gupta, A. Singh, R. Majeed, M. Nabi, S. U. & Shah, O. S. (2019). Germplasms/genotypes/lines/cultivars of tomato against early blight (*Alternaria solani*) in field conditions.
10. Foolad, M.R. Ashrafi, H. (2015). Characterization of early blight resistance in a recombinant inbred line population of tomato: I. heritability and trait correlations. *Adv. Stud. Biol*. 7, 131–148. doi.org/10.12988/asb.2015.41162
11. Gogoi, R. Singh, D.V. & Srivastava, K.D. (2001). Phenols as a biochemical basis of resistance in wheat against Karnal bunt. *Plant Pathol*., 50:470-6. doi.org/10.1046/j.1365-3059.2001.00583.x
12. Gomez, K.A. & Gomez, A.A. (1984). Statistical procedures for agricultural research. John Wiley and Sons, New York, USA
13. Gondal, A.S. Ijaz, M. Riaz, K. & Khan, A.R. (2012). Effect of Different Doses of Fungicide (Mancozeb) against *Alternaria* Leaf Blight of Tomato in Tunnel. *J Plant Pathol Microb* 3, 3. doi.org/10.4172/2157-7471.1000125.
14. Hammerschmidt, R. & Kuc, A. (1995) Induced resistance to disease in plants. Kluwer Academic Publishers, Dordrecht L. The Netherlands: 182.
15. Kalim, S. Luthra, Y.P. Gandhi, S.K. (2000). Influence of Bavistin seed treatment on morphophysiological and biochemical parameters of cowpea roots susceptible to Rhizoctonia species. *J Mycol Plant Pathol.,* 30(3), 375-379.
16. Mahalakshmi, G. Vengadeshkumar, L. Rajamohan, K. Sanjaygandhi, S. & Sharmila, A. M. (2020). Leaf extract of *Rhizophora apiculata* as a potential bio-inducer of early blight disease resistance in tomato plant. *Nov Res Microbiol J*, *4*(2), 714. doi.org/10.21608/NRMJ.2020.84020.
17. Mayer, A.M. Harel, E & Shaul, R.B. (1965). Assay of catechol oxidase a critical comparison of methods. *Phytochemistry*, 5, 783-789. doi.org/10.1016/S0031-9422(00)83660-2.
18. Mckinney, H.H. (1923). A new system of grading plant diseases. *J. agric. Res.,* 26**,** 195-218.

19. Rasool, M. Akhter, A. Soja, G. & Haider, M. S. (2021). Role of biochar, compost and plant growth promoting rhizobacteria in the management of tomato early blight disease. *Scientific Reports*, 11(1), 1-16. doi.org/10.1038/s41598-021-85633-4.
20. Rex, B. (2021). Management of Early Blight of Tomato Caused by *Alternaria solani* using Phytobiocides. *Biopestic. Int.,* 17(1), 97-102
21. Rex, B. Prabhu, S. & Kumar, J. S. (2019). Antifungal efficacies of plant extracts against *Alternaria solani* (Ellis and Martin) jones and grout under *in vitro* condition. *Annals of Phytomedicine*, *8* (1), 1-5. doi.org/10.21276/ap.20 19. 8.1.19.
22. Sahu, D.K. Khare, C.P. & Patel, R. (2013). Seasonal occurrence of tomato diseases and survey of early blight in major tomato-growing regions of Raipur district. *The Ecoscan* vol. 4: 153-157.
23. Sales, J.R. Nunes, G. Michereff, S.J. Pereira, E.W.L. & Guimaraes, I.M. (2011). Reaction of families and lines of melon to powdery mildew. *Hort Brasileira.* 29, 382-386. doi.org/10.1590/S0102-05362011000300021.
24. Shoaib, A. Iqbal, J. & Khan, K. A. (2020). Evaluation of phenotypic, physiological and biochemical attributes connected with resistance in tomato against *Alternaria solani. Acta physiologiae plantarum*, 42(5), 1-17. doi.org/10.1007/s11738-020-03076-2.
25. Singh, P.C. Kumar, R. Singh, M. Rai, A. Singh, M.C. & Rai, M. (2011). Identification of resistant sources against early blight disease of tomato. *Indian J Hort*., 68:516–521
26. TNAU- (Tamil Nadu Agricultural University). (1980). Agriteck portel**.** Coimbatore, India.
27. Vijaya Mahanttesha, S.R. Rawal, R.D. Ramaswamy, G.R. (2012). Field evaluation of tomato genotypes for resistance to *Alternaria solani*. *Trends in Biosciences*, 5, 294.
28. Ziesline, N. & Ben-Zaken, R (1993). Peroxidase activity and presence of phenolic substances in peduncles of rose flowers. *Plant Physiol. Biochem.*, 107, 39-50.