



RESEARCH ARTICLE

Identification of novel source and inheritance of resistance to bacterial wilt (*Ralstonia solanacearum*) in tomato (*Solanum lycopersicum* L.) for sustainable disease management

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Abstract

Bacterial wilt caused by *Ralstonia solanacearum* is a destructive disease severely restricting tomato (*Solanum lycopersicum* L.) productivity in tropical and subtropical regions. The purpose of this study was to identify novel sources of resistance and explicate the inheritance pattern of bacterial wilt resistance in tomato. A total of 59 genotypes, including open-pollinated lines, hybrids and breeding accessions, were subjected to screening experiments during two consecutive seasons (2022–2023 and 2023–2024) under controlled net house conditions at Centurion University of Technology and Management and natural epiphytotic field conditions at Indian Institute of Horticultural Research - Central Horticultural Experiment Station, Bhubaneswar, India. Disease severity was evaluated using percent disease index (PDI) and area under disease progress curve (AUDPC). The open-pollinated cultivar 'Utkal Kumari (BT-10)' exhibited consistent high resistance, with significantly lower AUDPC values (<150 vs >1800 in susceptible checks) and high survival rates (90 %) across environments. Among others, tomato cultivars Arka Samrat, Arka Abhed and JK Desi, demonstrated significant resistance while genotypes such as Pusa Ruby and EW815 were consistently highly susceptible. A segregating F₂ population derived from a cross between resistant 'BT-10' and susceptible 'Pusa Ruby' exhibited continuous variation in disease response. Chi-square analysis revealed significant deviation from a 3:1 Mendelian segregation ratio ($\chi^2 = 72.30$, $P < 0.01$), indicating a quantitative mode of inheritance. These findings highlight the potential of Utkal Kumari and allied resistant lines as donors for resistance breeding and emphasize the need to integrate robust phenotypic screening with molecular approaches for durable and strategic bacterial wilt management in tomato.

Keywords: area under disease progress curve; disease screening; germplasm evaluation; quantitative resistance; segregating population; sustainable disease management

Introduction

Tomato (*Solanum lycopersicum* L.) is one of the most economically important horticultural crops worldwide. Originally native to South America, tomatoes are now grown globally and essential to many culinary dishes. The fruit varies in size, shape and colour, with colours ranging from deep red, yellow, orange, green and even purple. Tomatoes are rich in vitamins A, C and K, as well as minerals like potassium and manganese. They are also a good source of antioxidants, including lycopene, which has been linked to a reduced risk of prostate cancer (1). Although tomato is valued for its agricultural, industrial and economic significance, its cultivation in tropical and subtropical regions faces serious constraints from bacterial wilt, a devastating vascular disease caused by the *R. solanacearum* species complex (2).

In India, tomato bacterial wilt is a major constraint to tomato production, particularly in the states of Karnataka, Andhra Pradesh, Tamil Nadu, Odisha and Maharashtra (3). The disease is notorious for its widespread prevalence, high virulence and ability to cause up to 90 % yield losses under favourable environmental conditions making bacterial wilt one of the most intractable production constraints in Asia, including India (4–7). Bacterial wilt symptoms manifest at all stages of plant growth. However, symptoms are particularly severe during the flowering and fruiting phases. The pathogen invades the host through the roots, rapidly colonises the xylem vessels, disrupts water transport and ultimately leads to plant wilting and death (8).

The complexity of managing bacterial wilt stems from the pathogen's extensive genetic and pathogenic diversity. *R. solanacearum* has been classified into 4 major phylotypes: Phylotype I (Asia), II (Americas), III (Africa) and IV (Indonesia) with

further sub-groupings such as IIA and IIB (9). While phylotype IIB strains are particularly aggressive on potatoes (10), strains from all four phylotypes can infect solanaceous crops, including tomato (11, 12). This diversity complicates control strategies, as different strains may vary significantly in their pathogenicity and host range.

Resistance breeding has long been recognised as the most sustainable and eco-friendly strategy to manage bacterial wilt in tomato. Previous studies have reported various modes of genetic control for bacterial wilt resistance. In some genotypes, notably Hawaii 7996 and Hawaii 7998, resistance is governed by a single dominant gene (13, 14), while in other cases, resistance appears to be polygenic or governed by multiple quantitative trait loci (15). Some researchers have also reported resistance being conditioned by single or multiple recessive genes (16–23). In India, research has largely concentrated on southern and northern states, with comparatively limited systematic efforts in the eastern region despite its vulnerability due to warm, humid conditions favourable for bacterial wilt epidemics. The expression and durability of resistance are also affected by environmental conditions, variation of pathogen strains and genotype $\times$ environment interactions.

Despite various advancements, resistance to bacterial wilt in tomato is reported to be complex, ranging from single-gene control in some accessions to polygenic, quantitative inheritance in others. However, information on regionally adapted germplasm, particularly under natural epiphytotic conditions, remains limited. Moreover, the stability of resistance across environments and the genetic basis of tolerance in local cultivars have not been adequately explored. Therefore, the present study was undertaken to systematically screen a diverse panel of tomato germplasm under both controlled and field conditions in eastern India, identify novel and stable sources of resistance and assess the inheritance pattern of resistance using segregating populations derived from contrasting parents. We hypothesised that regionally adapted tomato germplasm from eastern India harbours novel and stable sources of resistance capable of withstanding natural epiphytotic pressure and that resistance derived from the cultivar 'Utkal Kumari' is governed by a quantitative, polygenic inheritance pattern rather than single-gene control. By integrating dual-environment phenotyping with inheritance analysis, this work provides insights into the durability of bacterial wilt resistance in regionally adapted tomato germplasm and highlights potential donors for future breeding strategies for sustainable management of bacterial wilt pathogens in Tomato.

Materials and Methods

Plant materials

A total of 59 tomato germplasm was collected, comprising 37 accession lines, 14 F_1 hybrids, 7 open-pollinated (OP) cultivars and 1 local landrace. These were sourced from ICAR-National Bureau of Plant Genome Research, New Delhi, ICAR-Indian Institute of Vegetable Research, Varanasi, Orissa University of Agriculture and Technology, Bhubaneswar and local collections. Two sets of experimentations were conducted simultaneously, one at the net houses of Centurion University of Technology and Management, Bhubaneswar, Odisha and the other at the sick plot of Indian Institute of Horticultural Research - Central Horticultural Experiment Station, Bhubaneswar, Odisha. The seeds were sown in 2 sets using 50-cavity germination trays (measuring 54 cm $\times$ 26 cm $\times$ 4.5 cm; L $\times$ W $\times$ H), filled with a sterilised potting mixture consisting of cocopeat

and vermicompost in an 80:20 ratio. Pusa Ruby and Arka Samrat tomato varieties were used as susceptible and resistant checks, respectively. To prevent non-target microbial interference, the trays were sterilised prior to sowing to eliminate fungal and bacterial contaminants and subsequently covered with mulching sheets to facilitate germination.

After germination, the trays were transferred to a greenhouse maintained under a 16/8 hr day/night cycle. The temperature ranged between 26 °C and 29 °C, with relative humidity levels between 81.5 % and 84.1 %. Seedlings were watered regularly whenever the topsoil appeared dry. A nutrient solution of NPK (19:19:19) at 2 g/L was applied weekly. Once the seedlings reached approximately 20 cm in height with 8–12 leaves (around 4 to 5 weeks old), they were transplanted into plastic pots and allowed to acclimate for a week before being evaluated for resistance to *R. solanacearum* under both field and controlled conditions. For the controlled experiments, one set of plants was water-deprived for 24 hr prior to inoculation. For field screening, the other set was transplanted into the sick plot. All plants were carefully monitored for disease development and progression.

Preparation of bacterial inoculum

The bacterial wilt pathogen *R. solanacearum* was procured from the Indian Type Culture Collection (ITCC), New Delhi, under the accession number BI0016. The culture was revived on CPG-TZC agar medium and maintained under standard conditions. The streaked plates were incubated at 27 °C for 48 hr. A single virulent bacterial colony, characterised by a fluidal texture and a pink center, was selected and transferred into freshly prepared nutrient broth using an inoculation loop. The bacterial culture was incubated in a shaker incubator for 42 hr at 160 rpm to promote growth. Following incubation, the culture was centrifuged to separate the bacterial cells from the nutrient medium. The supernatant was discarded and the bacterial pellet was resuspended in sterile distilled water to prepare the inoculum for screening experiments. The bacterial concentration was adjusted to approximately 10^8 cfu/mL, corresponding to an optical density (OD) of 1.35 at 600 nm.

Molecular detection of *Ralstonia solanacearum*

Genomic DNA was extracted from bacterial isolates using the Allprep Bacterial/Fungal DNA/RNA/Protein Kit (Qiagen, Germany), following the manufacturer's protocol. For preliminary molecular confirmation of the pathogen, the 16S rDNA region was subjected to polymerase chain reaction (PCR) amplification using the universal diagnostic primer pair OLI1, Y1 and Y2, as described by Seal et al. (1993) (24) (Supplementary Table 1). The thermal cycling conditions included an initial denaturation at 96 °C for 5 min, followed by 35 cycles of denaturation at 94 °C for 20 sec, annealing at 68 °C for 20 sec and extension at 72 °C for 30 sec. A final extension was performed at 72 °C for 10 min. The resulting PCR products were separated on a 1.5 % (w/v) agarose gel prepared in 1 $\times$ TAE buffer, stained with ethidium bromide and visualised using a gel documentation system.

Phenotypic screening of germplasm and disease assessment

Thirty plants per cultivar were selected for both controlled and field screening experiments. For screening under controlled conditions, root incisions were made approximately 2 cm away from the stem using sterile scissors to facilitate bacterial entry through the roots. Inoculation was carried out by drenching the soil around the root zone with 10 mL of bacterial suspension ($OD_{600} = 1.35$; $\sim 10^8$ cfu/mL) using a measuring tube. For field-based screening, the sick plot was

maintained at a bacterial concentration of approximately 10^8 cfu/mL by directly sprinkling a diluted inoculum into the soil. Disease progression was monitored in both conditions. Two well-established cultivars, Arka Samrat and Pusa Ruby, were included as resistant and susceptible checks, respectively (Supplementary Fig. 1). Following inoculation, the plants were watered on alternate days and the development of bacterial wilt symptoms was carefully observed. Disease progression was assessed on the 6th, 9th, 12th and 15th days post-inoculation by calculating the percentage of wilted leaves per plant. Each plant was evaluated using a rating scale as described by various literatures (25–27) (Supplementary Table 2). The disease scale was determined based on visual observation of the degree of wilting. The average value of disease severity for ten plants was calculated. The percentage of disease incidence (PDI) was recorded by the following Eqn.

$$\text{PDI \%} = \frac{\text{Number of dead plants due to bacterial wilt disease}}{\text{Total number of plants transplanted}} \times 100 \quad (\text{Eqn. 1})$$

The PDI score was assigned on a scale of 0 to 5 based on the PDI % observed for all the plants screened (Supplementary Table 3). The presence of *R. solanacearum* was further confirmed by performing ooze test.

Generation and screening of the mapping population

Following the identification of resistant and susceptible parental lines, cross-hybridisation was performed between the contrasting genotypes to generate segregating populations, specifically F₁ and F₂ generations. These populations were developed to facilitate the genetic dissection of bacterial wilt resistance. The resulting progenies were subjected to phenotypic screening under both controlled (glasshouse) and field conditions, utilising a standardised artificial inoculation method previously described. This dual-environment evaluation allowed for a robust assessment of disease responses across environments and enhanced the reliability of resistance classification. The inheritance pattern of bacterial wilt resistance was subsequently analysed based on the phenotypic performance of the segregating generations.

Statistical analysis

A comprehensive suite of statistical analyses was conducted to evaluate the disease severity and inheritance patterns. The area under disease progress curve (AUDPC) was calculated to quantify disease progression across time points for each genotype under both screening conditions following Eqn (28).

$$\text{AUDPC} = \sum_{i=1}^{n-1} \frac{(y_i + y_{i+1})}{2} (t_{i+1} - t_i) \quad (\text{Eqn. 2})$$

where y = disease severity at time t and n = number of observations.

To assess the relationship between controlled and field conditions, correlation analysis was performed, revealing the consistency of genotype performance across environments. A two-way analysis of variance (ANOVA) was conducted to determine the statistical significance of differences in PDI values across genotypes and conditions (29). Finally, for the F₂ population, a Chi-square (χ^2) test was applied to assess the goodness-of-fit to a 3:1 Mendelian segregation ratio (susceptible: resistant), aiming to infer the underlying genetic architecture (30, 31).

Results and Discussion

Phylogeny determination and molecular identification of *Ralstonia solanacearum*

Bacterial wilt infestation was confirmed through verification of rapid and irreversible wilting of leaves (Fig. 1a). While the leaf stayed green, they curled inwards and drooped during the hotter part of the day. The ooze test analysis showed milky white strands or clouds of bacterial ooze exuding from the cut end of the plant that confirmed *Ralstonia* infection (Fig. 1b). Steak plates of ooze water on CPG - TZC agar medium showed virulent bacterial colony, characterized by a fluidal texture and a pink centre (Fig. 1c). The PCR amplification of the 16S rDNA region using the OLI1/Y2 primer pair yielded a ~350 bp diagnostic band in all tested samples, confirming the strain identity as *R. solanacearum* (Fig. 1d). Similar morphological and molecular diagnostic methods have been widely used for pathogen validation in bacterial wilt studies (24). These confirmations established pathogen purity and validated the reliability of downstream resistance screening against the specific virulence profile prevalent in the region.

Evaluation of germplasm for disease reaction against bacterial wilt disease

Two-way analysis of variance exhibited significant differences among the 59 genotypes ($F = 23.52$, $p < 0.0001$), indicating significant genetic variance for bacterial wilt resistance. However, no substantial effect was observed amongst the controlled and the field environments ($F = 2.52$, $p = 0.118$), inferring that genotype performance was largely consistent across testing conditions (Table 1). Despite this statistical stability, notable differences were observed in disease progression dynamics. In the field, PDI was higher (19.54 %) at 6 days after inoculation (DAI) compared to the controlled screening (7.9 %), indicating earlier disease onset under natural

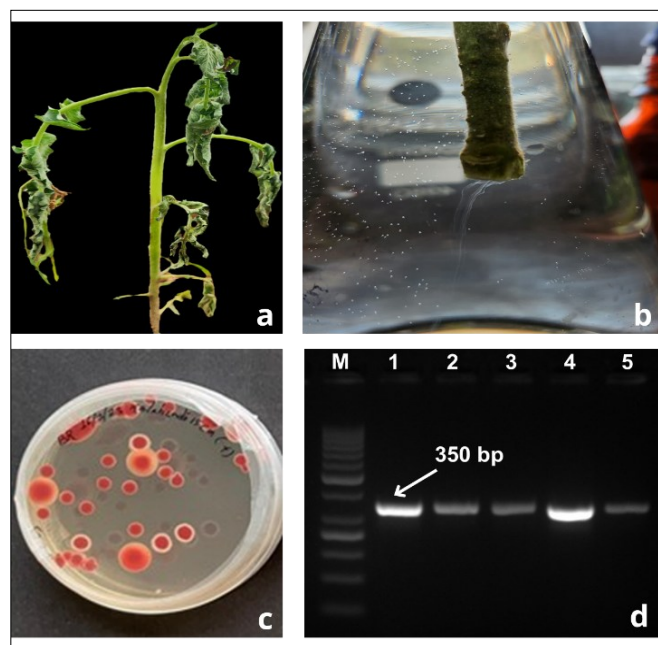


Fig. 1. Identification and characterization of bacterial wilt pathogen *Ralstonia solanacearum*. a: Wilted plant sample at Sick plot, IIHR-CHES, Bhubaneswar, Odisha; b: Bacterial ooze streaming from cut stem confirming infection (ooze test); c: *Ralstonia solanacearum* colonies on TZC medium showing characteristic fluidal, irregular white colonies with red centers; d: Gel electrophoresis showing 16S rDNA PCR amplification of *Ralstonia solanacearum* isolates using OLI1/Y2 primer. M: 50 bp ladder; Lanes 1–5: amplified ~300 bp bands confirming pathogen identity.

Table 1. Analysis of variance for the distribution over percentage of infection by *Ralstonia solanacearum* in tomato genotypes under field and controlled conditions

Source of variation	df	Sum of squares	Mean squares	F
Between Rows	58	94286.11	1625.62	23.52
Within Columns	1	174.29	174.29	2.52
Residual	58	4008.71	69.12	
Total	117	98469.11		

conditions. Visual wilting was corroborated by typical ooze tests confirming the presence of *R. solanacearum* in symptomatic plants (Fig. 2 a–f). The peak incidence occurred at 9 DAI in both environments (Fig. 2 g). Further, a comparative analysis of PDI and AUDPC revealed a significant difference between the susceptible check (Pusa Ruby) and resistant check (Utkal Kumari) cultivars (Fig. 2 h, i). These results highlight a more hostile and sustained disease pressure in the field, likely due to complex soil microbiome interactions. This widespread pathogen aggressiveness supports previous reports from Chhattisgarh (32), Tamil Nadu (33, 34), Himachal Pradesh (35, 36) and Tripura (37), which underscore the need for location-specific field screening to capture diverse environmental interactions that controlled experiments might overlook.

Identification of resistance sources and comparison with previous studies

Significant variability was observed among the genotypes, with disease incidence ranging from 0 % to 100 % (Table 2). In the controlled environment, genotypes like Arka Samrat, Arka Rakshak and Utkal lines (e.g., Utkal Kumari/ BT – 10) exhibited strong resistance (PDI 0–30 %). Notably, the open pollinated cultivar ‘Utkal Kumari’ demonstrated consistent high resistance across both environments (Fig 3). In contrast, the check cultivar ‘Pusa Ruby’ and hybrid ‘EW815’ consistently recorded >90 % PDI, categorising them as highly susceptible. These findings align with previous researchers (27), who reported high resistance in the ‘Arka’ series. However, our study extends this by identifying ‘Utkal Kumari’ as a stable donor for Eastern India, addressing a crucial gap in a region where yield losses often exceed 70 % (1, 38, 39). While other researchers classified resistance using a qualitative scale, our study utilised standardised inoculum ($OD_{600} = 1.35$, $\sim 10^8$ CFU/mL) and quantitative AUDPC assessment, providing a more precise resolution of resistance levels that corroborated stable resistance in ~25 % of the screened genotypes (6).

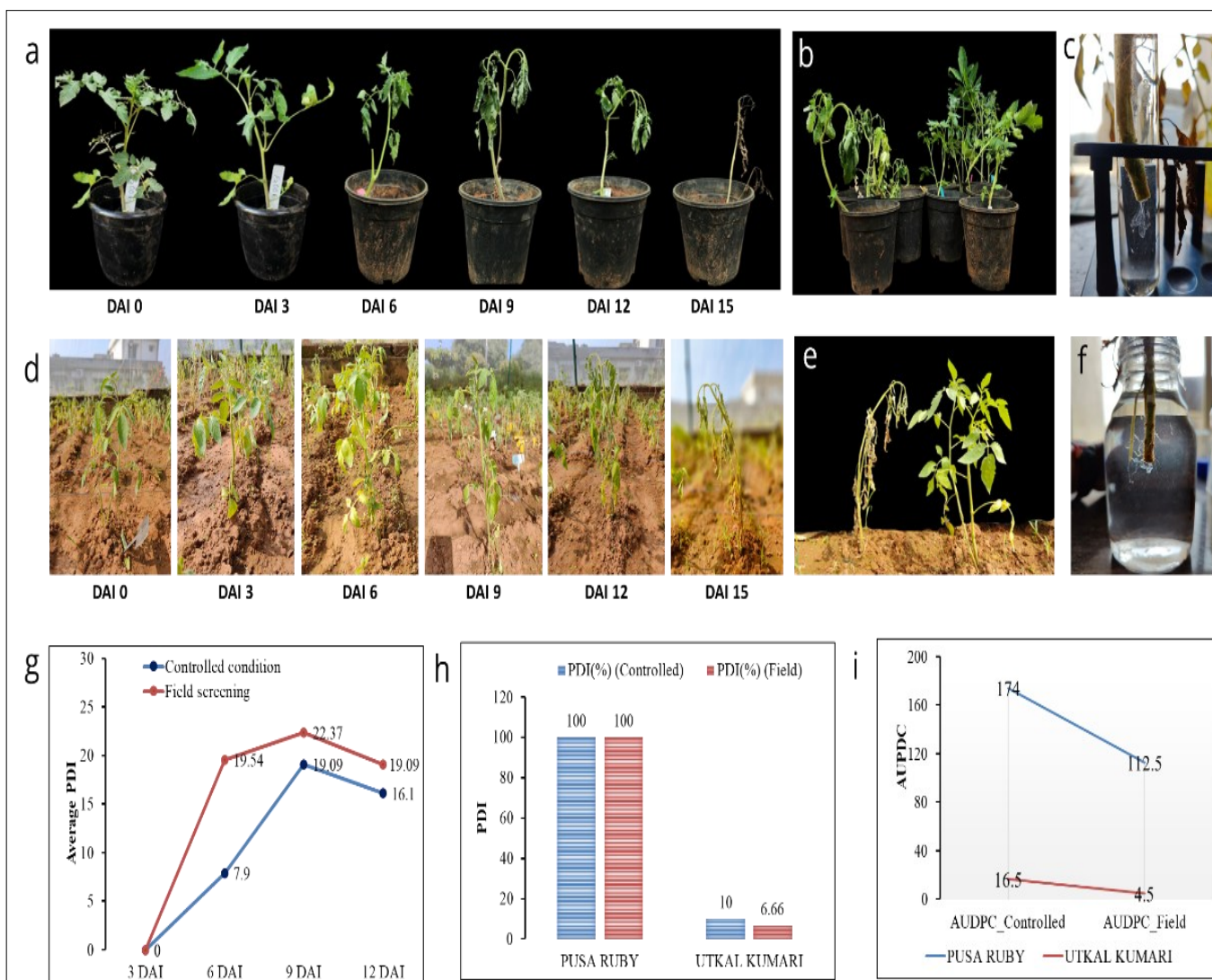
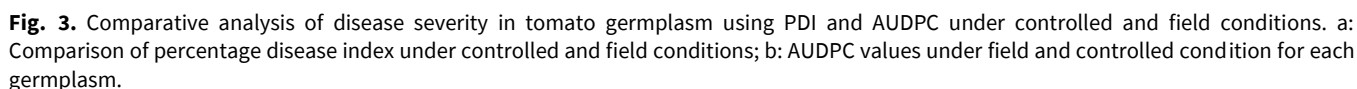


Fig. 2. Comparative evaluation of tomato genotypes for bacterial wilt resistance under controlled and field conditions. a: Disease prognosis under controlled conditions; b: Plants showing contrasting disease reaction (susceptible / resistant) under controlled conditions; c: Ooze test confirming infection in controlled conditions; d: Disease prognosis under field experimentations; e: Plants showing contrasting disease reaction (susceptible / resistant) under field experimentations; f: Ooze test confirming infection in field conditions; g: Average PDI comparison between ‘Utkal Kumari’ and ‘Pusa Ruby’; h: PDI comparison between ‘Utkal Kumari’ and ‘Pusa Ruby’; i: AUDPC comparison between ‘Utkal Kumari’ and ‘Pusa Ruby’.

Table 2. Phenotypic assessment of tomato germplasms and percent disease index under controlled and field conditions

Accession number / Variety name	Name of the institution / manufacturer	In controlled conditions			In Field Condition		
		PDI (%)	Scoring	Disease Reaction	PDI (%)	Scoring	Disease Reaction
EC- 615039	ICAR- NBPGR, Hyderabad	76.66	4	Susceptible	73.33	4	Susceptible
EC- 617055	ICAR- NBPGR, Hyderabad	70	4	Susceptible	66.66	4	Susceptible
EC- 617057	ICAR- NBPGR, Hyderabad	23.33	2	Moderately resistant	26.66	2	Moderately resistant
EC- 631325	ICAR- NBPGR, Hyderabad	46.66	3	Moderately susceptible	53.33	3	Moderately susceptible
EC- 631349	ICAR- NBPGR, Hyderabad	70	4	Susceptible	80	4	Susceptible
EC- 631351	ICAR- NBPGR, Hyderabad	90	5	Highly susceptible	100	5	Highly susceptible
EC- 631357	ICAR- NBPGR, Hyderabad	63.33	4	Susceptible	60	3	Moderately susceptible
EC- 631359	ICAR- NBPGR, Hyderabad	43.33	3	Moderately susceptible	33.33	2	Moderately resistant
EC- 631378	ICAR- NBPGR, Hyderabad	36.66	2	Moderately resistant	20	1	Highly resistant
EC- 631384	ICAR- NBPGR, Hyderabad	63.33	4	Susceptible	73.33	4	Susceptible
EC- 631386	ICAR- NBPGR, Hyderabad	63.33	4	Susceptible	80	4	Susceptible
EC- 631394	ICAR- NBPGR, Hyderabad	63.33	4	Susceptible	60	3	Moderately susceptible
EC- 631399	ICAR- NBPGR, Hyderabad	43.33	3	Moderately susceptible	40	2	Moderately resistant
EC- 631406	ICAR- NBPGR, Hyderabad	46.66	3	Moderately susceptible	46.66	3	Moderately susceptible
EC- 631411	ICAR- NBPGR, Hyderabad	76.66	4	Susceptible	86.66	5	Highly susceptible
EC- 631415	ICAR- NBPGR, Hyderabad	36.66	2	Moderately resistant	33.33	2	Moderately resistant
EC- 631431	ICAR- NBPGR, Hyderabad	63.33	4	Susceptible	80	4	Susceptible
EC- 631447	ICAR- NBPGR, Hyderabad	26.66	2	Moderately resistant	26.66	2	Moderately resistant
EC- 631957	ICAR- NBPGR, Hyderabad	76.66	4	Susceptible	86.66	5	Highly susceptible
EC- 632936	ICAR- NBPGR, Hyderabad	86.66	5	Highly susceptible	100	5	Highly susceptible
EC- 632944	ICAR- NBPGR, Hyderabad	96.66	5	Highly susceptible	100	5	Highly susceptible
EC- 635520	ICAR- NBPGR, Hyderabad	76.66	4	Susceptible	93.33	5	Highly susceptible
EC- 635525	ICAR- NBPGR, Hyderabad	70	4	Susceptible	73.33	4	Susceptible
EC- 635526	ICAR- NBPGR, Hyderabad	86.66	5	Highly susceptible	100	5	Highly susceptible
EC- 635530	ICAR- NBPGR, Hyderabad	46.66	3	Moderately susceptible	40	2	Moderately resistant
EC- 636877	ICAR- NBPGR, Hyderabad	30	2	Moderately resistant	20	1	Highly resistant
EC- 638522	ICAR- NBPGR, Hyderabad	93.33	5	Highly susceptible	100	5	Highly susceptible
EC- 687416	ICAR- NBPGR, Hyderabad	90	5	Highly susceptible	100	5	Highly susceptible
EC- 687422	ICAR- NBPGR, Hyderabad	70	4	Susceptible	93.33	5	Highly susceptible
EC- 687423	ICAR- NBPGR, Hyderabad	90	5	Highly susceptible	100	5	Highly susceptible
EC- 688516	ICAR- NBPGR, Hyderabad	70	4	Susceptible	100	5	Highly susceptible
EC- 690989	ICAR- NBPGR, Hyderabad	93.33	5	Highly susceptible	100	5	Highly susceptible
EC- 690990	ICAR- NBPGR, Hyderabad	86.66	5	Highly susceptible	100	5	Highly susceptible
EC- 692338	ICAR- NBPGR, Hyderabad	93.33	5	Highly susceptible	100	5	Highly susceptible
EC- 692341	ICAR- NBPGR, Hyderabad	93.33	5	Highly susceptible	100	5	Highly susceptible
EC- 615075	ICAR- NBPGR, Hyderabad	53.33	3	Moderately susceptible	66.66	4	Susceptible
EC- 615074	ICAR- NBPGR, Hyderabad	63.33	4	Susceptible	60	3	Moderately susceptible
Arka Apeksha F ₁	ICAR-IIHR, Bangalore	30	2	Moderately resistant	26.66	2	Moderately resistant
Arka Samrat F ₁	ICAR-IIHR, Bangalore	6.66	1	Highly resistant	0	0	Immune
Arka Rakshak F ₁	ICAR-IIHR, Bangalore	33.33	2	Moderately resistant	13.33	1	Highly resistant
Arka Abhed F ₁	ICAR-IIHR, Bangalore	16.66	1	Highly resistant	6.66	1	Highly resistant
BBS001	Locally Sourced	43.33	3	Moderately susceptible	20	1	Highly resistant
Pusa Ruby	PAHUJA SEEDS (Locally Sourced)	100	5	Highly susceptible	100	5	Highly susceptible
Pusa Early Dwarf	PAHUJA SEEDS (Locally Sourced)	56.66	3	Moderately susceptible	40	2	Moderately resistant
S-22	VIGEN SEEDS (Locally Sourced)	63.33	4	Susceptible	60	3	Moderately susceptible
Neil (To-7022) Hybrid	SYNGENTA (Locally Sourced)	23.33	2	Moderately resistant	26.66	2	Moderately resistant
JK Desi F ₁ Hybrid	JK SEEDS (Locally Sourced)	33.33	2	Moderately resistant	20	1	Highly resistant
EZ-9003 (F ₁ Hybrid)	ENZA ZADEN (Locally Sourced)	33.33	2	Moderately resistant	20	1	Highly resistant
Priya-6636 (F ₁ Hybrid)	CLAUSE VEGETABLE SEEDS (Locally Sourced)	63.33	4	Susceptible	73.33	4	Susceptible
Sonia (Nth-2591) (F ₁ Hybrid)	NUZIVEEDA SEEDS (Locally Sourced)	56.66	3	Moderately susceptible	53.33	3	Moderately susceptible
Bhagya (Nth-301) (F ₁ Hybrid)	NUZIVEEDA SEEDS (Locally Sourced)	60	3	Moderately susceptible	73.33	4	Susceptible
EW815 (F ₁ Hybrid)	EAST WEST SEEDS (Locally Sourced)	83.33	5	Highly susceptible	100	5	Highly susceptible
NTH-671 (F ₁ Hybrid)	NATH SEEDS (Locally Sourced)	63.33	4	Susceptible	86.66	5	Highly susceptible
Bahubali (KSP-1306) (F ₁ Hybrid)	KALASH SEEDS (Locally Sourced)	70	4	Susceptible	93.33	5	Highly susceptible
Utkal Pallavi (BT- 1)	OUAT, BBSR	23.33	2	Moderately resistant	20	1	Highly resistant
Utkal Deepti (BT- 2)	OUAT, BBSR	30	2	Moderately resistant	26.66	2	Moderately resistant
Utkal Kumari (BT- 10)	OUAT, BBSR	10	1	Highly resistant	6.66	1	Highly resistant
Utkal Raja (BT- 20-2-1)	OUAT, BBSR	36.66	2	Moderately resistant	20	1	Highly resistant
Utkal Pragyan (BT- 116-3-2)	OUAT, BBSR	30	2	Moderately resistant	20	1	Highly resistant



Despite demonstrating robust phenotypic resistance, the genotype ‘Utkal Kumari’ could not be validated using several known molecular markers allied with *R. solanacearum* resistance. The PCR amplification using SSR20, SCU176-534 (SCAR), RsR6-5 (CAPS) and RsR12-1 (dCAPS) markers failed to produce the expected bands in ‘Utkal Kumari’ (40–42). The absence of amplification suggests that these markers, which are linked to major resistance genes like *BWR-12* or *Bwr-6*, may not be universally applicable across all resistant germplasm. This implies that ‘Utkal Kumari’ likely possesses novel resistance loci distinctive from those in widely characterised donors. Consequently, future molecular dissection through QTL mapping or GWAS will be essential to uncover these unique regions.

To explicate the genetic control of this potential novel resistance, F1 and F2 populations derived from ‘Utkal Kumar’ x ‘Pusa Ruby’ were evaluated. The F1 hybrids displayed intermediate resistance trait. The F2 population (n = 270) exhibited a continuous frequency distribution for disease severity rather than distinct classes. Survival declined steadily from 100 % to 53 % by 12 DAI, contrasting with the rapid mortality of the single gene hypothesis (Table 3). Instead, the

Conclusion

The study identified Utkal Kumari (BT-10) as a highly resilient, stable donor for bacterial wilt resistance in Eastern India. Phenotypic screenings and molecular validation confirmed that its resistance mechanism is distinct from widely characterized genetic stocks, as traditional markers linked to major resistance loci (*Bwr-6* and *Bwr-12*) failed to amplify. Genetic dissection of the segregating F2 population demonstrated a continuous variation in disease response and a significant deviation from Mendelian ratios, confirming a quantitative, polygenic mode of inheritance rather than single-gene control. These findings offer a strategic pathway to manage the destructive *Ralstonia solanacearum* pathogen in

Generation	Observed phenotype		Expected ratio (R:S)	X ² value	df	p value
	Resistant	Susceptible				
Pusa Ruby (S)	-	43	All S	-	-	-
Utkal Kumari (R)	47	-	All R	-	-	-
F ₁ (R × S)	3	46	1:2:1	-	-	-
F ₂ [(R × S) selfing]	142	128	3:1	72.3	1	0.01

tropical and subtropical regions. By establishing the multi-genic nature of 'Utkal Kumari', this work provides a valuable foundation for future genomic mapping, marker discovery, and breeding programs aimed at deploying durable field resistance to secure stable tomato productivity.

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Authors' contributions

DD and RM conceived and designed the study, conducted the germplasm screening and analyzed the data. SD, AKA, BPN and GCA assisted with the experimental setup and data collection. DD, RKJ and RM were involved in the statistical analysis and interpretation of the results. All authors read and approved the final manuscript.

Compliance with ethical standards

Conflict of interest: Authors do not have any conflict of interests to declare.

Ethical issues: None

Declaration of generative AI and AI-assisted technologies in the writing process

The authors declare the use of large language model (LLM) tools (Grammarly Pro) for language enhancement, proofreading, and clarity improvement during manuscript preparation. These tools were used strictly to refine grammar and readability without altering the scientific integrity of the work. The authors take full responsibility for the content, interpretation, and conclusions presented in this manuscript.

References

- Denny T. Plant pathogenic *Ralstonia* species. In: Plant-Associated Bacteria. Dordrecht: Kluwer Academic Publishers; 2006. https://doi.org/10.1007/978-1-4020-4538-7_16
- Hayward AC. Biology and epidemiology of bacterial wilt caused by *Pseudomonas solanacearum*. Annu Rev Phytopathol. 1991;29(1):65–87. <https://doi.org/10.1146/annurev.py.29.090191.00043>
- Srinivas C, Sujatha K, Shivashankar S. Status of tomato bacterial wilt in India: an overview. J Plant Prot Res. 2014;54(3):219–23.
- Yabuuchi E, Kosako Y, Yano I, Hotta H, Nishiuchi Y. Transfer of two *Burkholderia* and an *Alcaligenes* species to *Ralstonia* gen. Nov.: Proposal of *Ralstonia pickettii* (Ralston, Palleroni and Doudoroff 1973) comb. Nov., *Ralstonia solanacearum* (Smith 1896) comb. Nov. and *Ralstonia eutropha* (Davis 1969) comb. Nov. Microbiol Immunol. 1995;39(11):897–904. <https://doi.org/10.1111/j.1348-0421.1995.tb03275.x>
- Artal RB, Gopalakrishnan C, Thippeswamy B. An efficient inoculation method to screen tomato, brinjal and chilli entries for bacterial wilt resistance. Pest Manag Hort Ecosyst. 2012;18(1):70–3.
- Aslam MN, Mukhtar T, Hussain MA, Raheel M. Assessment of resistance to bacterial wilt incited by *Ralstonia solanacearum* in tomato germplasm. J Plant Dis Prot. 2017;124(6):585–90. <https://doi.org/10.1007/s41348-017-0100-1>
- Acharya B, Dutta S, Dutta S, Chattopadhyay A. Breeding tomato for simultaneous improvement of processing quality, fruit yield and dual disease tolerance. Int J Veg Sci. 2018;24(5):407–23. <https://doi.org/10.1080/19315260.2018.1427648>
- Genin S. Molecular traits controlling host range and adaptation to plants in *Ralstonia solanacearum*. New Phytol. 2010;187(4):920–28. <https://doi.org/10.1111/j.1469-8137.2010.03397.x>
- Fegan M, Prior P. Bacterial wilt disease and the *Ralstonia solanacearum* species complex. In: Allen C, Prior P, Hayward AC, editors. Bacterial Wilt Disease and the *Ralstonia solanacearum* Species Complex. St. Paul, MN: APS Press; 2005.
- Janse J, Beld D, Van Den H. Introduction to Europe of *Ralstonia solanacearum* biovar 2, race 3 in *Pelargonium zonale* cuttings. J Plant Pathol. 2004;86:147–55.
- Cellier G, Prior P. Deciphering phenotypic diversity of *Ralstonia solanacearum* strains pathogenic to potato. Phytopathology. 2010;100(11):1250–61. <https://doi.org/10.1094/phyto-02-10-0059>
- Lebeau A, Daunay M-C, Fray A, Palloix A, Wang J-F, Dintinger J, et al. Bacterial wilt resistance in tomato, pepper and eggplant: Genetic resources respond to diverse strains in the *Ralstonia solanacearum* species complex. Phytopathology. 2011;101(1):154–65. <https://doi.org/10.1094/phyto-02-10-0048>
- Scott JW, Somodi GC, Jones JB. Bacterial spot resistance is not associated with bacterial wilt resistance in tomato. Proc Fla State Hort Soc. 1988;101:390–92.
- Grimault V, Prior P. A monogenic dominant resistance of tomato to bacterial wilt in Hawaii 7996 is associated with plant colonization by *Pseudomonas solanacearum*. J Phytopathol. 1995;143:349–52. <https://doi.org/10.1111/j.1439-0434.1995.tb00274.x>
- Ishihara T, Mitsuura I, Takahashi H, Nakaho K. Transcriptome analysis of quantitative resistance-specific response upon *Ralstonia solanacearum* infection in tomato. PLoS One. 2012;7(10):e46763. <https://doi.org/10.1371/journal.pone.0046763>
- Villareal RL, Lai SH. Reaction of three tomato cultivars, their F1's and three-way crosses to two isolates of bacterial wilt (*Pseudomonas solanacearum*). Rep Tomato Genet Coop. 1978;28:22–23.
- Tikoo SK, Anand N, Krishna R. Presence of two independent genetic systems for resistance to bacterial wilt (*Pseudomonas solanacearum*). In: Proceedings of the 15th International Congress of Genetics. New Delhi, India; 1983. p. 22–23.
- Monma S. Resistance to bacterial wilt in tomato cultivars. Bull Natl Res Inst Veg Ornament Plants Tea. 1993;6:1–12.
- Thakur AK, Kohli UK, Kumar M. Inheritance of resistance to bacterial wilt in tomato. Indian J Genet. 2004;64:79–80.
- Kim B, Hwang IS, Lee HJ, Lee JM, Seo E, Choi D, et al. Identification of a molecular marker tightly linked to bacterial wilt resistance in tomato by genome-wide SNP analysis. Theor Appl Genet. 2018;131(5):1017–30. <https://doi.org/10.1007/s00122-018-3054-1>
- Shin IS, Hsu J-C, Huang S-M, Chen J-R, Wang J-F, Hanson P, et al. Construction of a single nucleotide polymorphism marker-based QTL map and validation of resistance loci to bacterial wilt caused by *Ralstonia solanacearum* species complex in tomato. Euphytica. 2020;216(3):54. <https://doi.org/10.1007/s10681-020-2576-1>
- Nguyen TT, Le NT, Sim S-C. Genome-wide association study and marker development for bacterial wilt resistance in tomato (*Solanum lycopersicum* L.). Sci Hortic. 2021;289:110418. <https://doi.org/10.1016/j.scienta.2021.110418>
- Siddique MI, Silverman E, Louws F, Panthee DR. Quantitative trait loci mapping for bacterial wilt resistance and plant height in tomatoes. Plants. 2024;13(6):876. <https://doi.org/10.3390/plants13060876>
- Seal SE. Differentiation of *Pseudomonas solanacearum*, *Pseudomonas syzygii*, *Pseudomonas pickettii* and the blood disease

- bacterium by partial 16S rRNA sequencing: construction of oligonucleotide primers for sensitive detection by PCR. *Microbiology*. 1993;139(7):1587–94. <https://doi.org/10.1099/00221287-139-7-1587>
25. Winstead NN, Kelman A. Inoculation techniques for evaluating resistance to *Pseudomonas solanacearum*. *Phytopathology*. 1952;42:628–34.
 26. Aslam MN, Mukhtar T, Ashfaq M, Hussain MA. Evaluation of chili germplasm for resistance to bacterial wilt caused by *Ralstonia solanacearum*. *Australas Plant Pathol*. 2017;46(3):289–92. <https://doi.org/10.1007/s13313-017-0491-2>
 27. Kumar M, Srinivasa V, Kumari M. Screening of tomato line/varieties for bacterial wilt (*Ralstonia solanacearum*) resistance in hill zone of Karnataka, India. *Int J Curr Microbiol App Sci*. 2018;7:1451–55.
 28. Campbell CL, Madden LV. Introduction to plant disease epidemiology. New York: Wiley-Interscience; 1990.
 29. Gomez KA, Gomez AA. Statistical procedures for agricultural research. 2nd ed. Nashville, TN: John Wiley & Sons; 1984.
 30. Mather K. Statistical Analysis in Biology. London, England: Chapman and Hall; 1972.
 31. Mather K, Jinks JL. Introduction to Biometrical Genetics. London: Chapman and Hall; 1982. <https://doi.org/10.1007/978-1-4899-3406-2>
 32. Tiwari JK. Screening of tomato genotypes against bacterial wilt (*Ralstonia solanacearum*) under field condition for Chhattisgarh. *Glob J Biosci Biotechnol*. 2012;1(2):224–27.
 33. Balamurugan A, Kumar A, Muthamilan M, Sakthivel K, Vibhuti M, Ashajothi M, et al. Outbreak of tomato wilt caused by *Ralstonia solanacearum* in Tamil Nadu, India and elucidation of its genetic relationship using multilocus sequence typing (MLST). *Eur J Plant Pathol*. 2018;151(3):831–39. <https://doi.org/10.1007/s10658-017-1414-3>
 34. Balamurugan A, Muthamilan M, Kamalakannan A, Shanthi A, Arumugam T. Characterization of *Ralstonia solanacearum* causing bacterial wilt disease of tomato in Coimbatore district of Tamil Nadu, India. *Int J Curr Microbiol Appl Sci*. 2020;9(2):3010–16. <https://doi.org/10.20546/ijcmas.2020.902.345>
 35. Saini M, Sagar V, Gupta M, Sharma SK, Saini R. Identification, characterization and genetic diversity of *Ralstonia pseudosolanacearum* causing bacterial wilt of tomato in Himachal Pradesh, India. *Physiol Mol Plant Pathol*. 2025;138:102684. <https://doi.org/10.1016/j.pmpp.2025.102684>
 36. Saini M, Gupta M, Sagar V, Chauhan A, Saini R, Kumar G. Molecular identification and management of *Ralstonia pseudosolanacearum* (Phylotype I) causing bacterial wilt of tomato using copper oxide nanoparticles in Himachal Pradesh, India. *Crop Prot*. 2025;191:107152. <https://doi.org/10.1016/j.cropro.2025.107152>
 37. Dutta P, Rahman B. Varietal screening of tomato against bacterial wilt disease under subtropical humid climate of Tripura. *Int J Farm Sci*. 2012;2(2):40–43.
 38. Sharma JP, Kumar S, Singh D. Integrated management of bacterial wilt of tomato caused by *Ralstonia solanacearum*. *Indian J Agric Sci*. 2016;86(7):877–83. <https://doi.org/10.56093/ijas.v86i7.59855>
 39. Singh D. Characterization of biovar/races of *Ralstonia solanacearum*, the incitant of bacterial wilt in solanaceous crops. *Indian Phytopathol*. 2010;63(3):261–65.
 40. Kumar S, Ramanjini Gowda PH, Saikia B, Debbarma J, Velmurugan N, Chikkaputtaiah C. Screening of tomato genotypes against bacterial wilt (*Ralstonia solanacearum*) and validation of resistance linked DNA markers. *Australas Plant Pathol*. 2018;47(4):365–74. <https://doi.org/10.1007/s13313-018-0567-7>
 41. Dheemanth TL, Nazeem PA, Sadhan Kumar PG, Mathew SK, Amaranatha Reddy M. Validation of SSR markers for imparting disease resistance in tomato (*Solanum lycopersicum* L.). *Int J Curr Microbiol Appl Sci*. 2018;7(1):1513–22. <https://doi.org/10.20546/ijcmas.2018.701.184>
 42. Abebe AM, Choi J, Kim Y, Oh C-S, Yeom I, Nou I-S, et al. Development of diagnostic molecular markers for marker-assisted breeding against bacterial wilt in tomato. *Breed Sci*. 2020;70(4):462–73. <https://doi.org/10.1270/jsbbs.20027>

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