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Advancements in unraveling and enhancing bacterial wilt resistance in Solanaceous crops

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Abstract

Ralstonia solanacearum significantly threatens Solanaceous crops, necessitating efficient genetic control strategies. With advancements in genomics, untapped resources for disease resistance are expected to be identified soon. Intensified research in remote hybridization and somatic cell fusion is crucial to enhance resistance to bacterial wilt. The application of effectors could enable high-throughput methodologies for bacterial wilt resistance identification and promote screening in wild species. For difficult-to-identify receptors, resistant varieties could be developed by incorporating resistance genes from *Arabidopsis thaliana* and other Solanaceous plants. The use of genome-editing techniques and the completion of whole-genome sequencing for key Solanaceous crops should expedite resistance gene cloning. Methods such as Resistance gene enrichment sequencing (RenSeq) could expedite receptor identification, promising a future where *R. solanacearum*-resistant crop development is within reach.

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Introduction

Ralstonia solanacearum, a soil-borne pathogen, is responsible for bacterial wilt (BW) disease affecting 200 plant species in over 50 families, including crucial Solanaceous crops like potato, tomato, pepper, eggplant, and tobacco, as well as model plants, such as *Arabidopsis thaliana* and *Medicago truncatula*[1]. As such, it is ranked as the world's second most impactful plant pathogenic bacteria in terms of scientific and economic significance[1]. After infecting the host, *R. solanacearum* enters the plant's vascular system, resulting in wilting and, ultimately, death, thus preparing it for the next transmission cycle[1]. Water is a vital condition for its survival, particularly in agricultural irrigation environments[2]. Remarkably, under sterile water conditions in labs, *R. solanacearum* can survive without a decrease in pathogenicity for over four years[2], indicating its robust survival capabilities and partly explaining its global dissemination.

The extensive genetic diversity within *R. solanacearum*, stemming from factors such as geographical distribution, host range, and pathogenicity, led to the application of the term 'species complex' for this organism. This '*R. solanacearum* species complex' (RSSC) consists of four phylotypes, each predominantly originating from different global regions[3]. Phylotype I largely originates from Asia, phylotype II from America, phylotype III from Africa and surrounding islands in the Indian Ocean, and phylotype IV from Indonesia, Japan, and Australia[3]. Recent studies have redefined these phylotypes into separate species, including *R. solanacearum* (phylotype II), *R. pseudosolanacearum* (phylotypes I and III), and an array of *R. syzygii* (phylotype IV)[4,5]. Furthermore, a new sub-clade, referred to as phylotype IIC, has been identified within phylotype II[6]. The phylotypes of RSSC are not related to the host preference, reflecting the genetic diversity and difficulty of prevention and control of bacterial wilt[7].

The advent of high-throughput sequencing technology has accelerated our understanding of *R. solanacearum*'s genetics. The first complete genome sequence of this pathogen was published in 2002[8]. Currently, the NCBI database archives a total of 325 *R. solanacearum* genomes, although the hosts for 58 of these strains remain unidentified (Supplemental Table S1). Significantly, the predominant hosts among the identified strains are *Solanum lycopersicum* (tomato), *Solanum tuberosum* (potato), and *Capsicum annuum* (pepper) (Supplemental Table S1). These plants have given rise to 70, 69, and 23 strains respectively, indicating their key role as hosts for *R. solanacearum*. This substantial genomic repository has streamlined the process of identifying genes related to pathogenicity and comprehending their pathogenic mechanisms. This wealth of information forms an essential foundation for the development of disease-resistant Solanaceous varieties.

Identification and development of resistance resources for Solanaceous crops against bacterial wilt

Various Solanaceous crops and their closely related species present a diverse set of plant accessions with varied degrees of

disease resistance against *R. solanacearum* strains. For example, accessions of tomato (*S. lycopersicum* Hawaii 7996, TML46, CLN1463 and R3034), eggplant (*Solanum melongena* Ceylan SM 164, SM6, Surya, and AG91-25), pepper (*C. annuum* CA8 and MC4), potato (*S. chacoense* and *S. sparsipilum*), and tobacco (*Nicotiana tabacum*, *N. benthamiana*, and *N. glutinosa*) are resistant to BW[9–11]. The utilization of somatic hybridization has significantly enhanced BW resistance, especially in eggplant and potato cultivars (Table 1). Resistant somatic hybrids, generated through the fusion of different *Solanum* species, exhibit improved BW resistance, showcasing somatic hybridization's potential in enhancing resistance traits.

Resistant somatic hybrids in eggplants were successfully created *via* electrical fusion between *S. melongena* and *S. aethiopicum*[12]. Furthermore, their hybrids exhibited enhanced resistance to BW and displayed superior fertility compared to conventional eggplant varieties[13]. In another notable development, a somatic hybrid line (27-14) was produced through protoplast fusion involving *Solanum integrifolium*, a commonly used rootstock in eggplant cultivation, and *Solanum violaceum*, a wild species exhibiting BW tolerance. This hybrid exhibited increased resistance to BW[14]. Similarly, hybrids derived from *S. melongena* and *Solanum torvum* demonstrated commendable resistance to BW[15]. In an innovative approach, hybrids originating from UV-irradiated cotyledonary protoplasts of *S. integrifolium* and iodoacetamide-treated protoplasts of *S. sanitwongsei* were not only vigorous and capable of bearing viable seeds but also exhibited a morphology intermediate to the parent species. This suggests their potential as eggplant rootstock candidates[16].

In the pursuit of enhancing BW resistance in potatoes, breeders have identified and integrated resistance traits from various tuber-bearing *Solanums* into breeding programs. Despite the endosperm balance number incompatibility system restricting a direct cross between *S. commersonii* and *S. tuberosum*[17], innovative approaches such as somatic hybridization have enabled successful circumvention of this barrier. For instance, somatic hybrids between *S. tuberosum* and *S. commersonii* have overcome sexual incompatibilities, yielding disease-resistant lines compatible with *S. tuberosum*[18]. Moreover, resistant lines from their hybrids have been successfully bred with *S. tuberosum* varieties for amplified BW resistance[19]. Subsequent studies have undertaken an in-depth analysis of the behavior of *S. commersonii* chromosomes and the colonization patterns of *R. solanacearum* in the introgression lines[20,21]. In a similar vein, resistance traits from other *Solanum* species have been introduced into *S. tuberosum* through somatic hybridization. Notably, dihaploid potato and *Solanum phureja* hybrids have demonstrated increased BW resistance[22]. Remarkably, *S. tuberosum* and *S. stenotomum* hybrids have retained their parent's resistance level even after five years of *in vitro* maintenance[23]. Further, somatic hybrids originating from the protoplast fusion between *S. tuberosum* and *S. chacoense* exhibited enhanced BW resistance, marked by the presence of three specific *S. chacoense* simple sequence repeat (SSR) alleles linked to resistance[24,25]. An impressive achievement was the successful transfer of BW resistance from *S. melongena* cv. 508.3 to potato AC142-01 *via* interspecific symmetric protoplast fusion, resulting in resistant somatic hybrids dominant in potato parent nuclear genomes[26]. In the same lab, researchers also carried out asymmetric protoplast fusion and fused UV-treated protoplasts of the same resistant eggplant variety 508.3 with protoplasts of another susceptible potato variety to obtain 32 somatic hybrids, revealing the introgression of alien chromosome fragments and suggesting potential markers, emk03O04, emi04P17, and emd13E02a, associated with bacterial wilt resistance[27]. Later, researchers in the same lab successfully identified a gene, smPGH1, and seven BW-linked SSRs in somatic hybrids of potato and eggplant, providing valuable genetic resources for improving bacterial wilt resistance in cultivated potato through genome composition and transcriptome analysis[28].

In conclusion, developing additional resistant germplasm is essential to meet the co-evolutionary challenges posed by the pathogen and the diverse demands for improved agronomic traits. Broadening the scope of BW resistance through hybrid creation between resistant and susceptible varieties offers a promising approach to safeguard Solanaceous crops. Furthermore, ploidy manipulation through sexual hybridization could present viable alternatives to surpass sexual barriers, as exemplified by *S. commersonii*. By doubling its chromosome number, a 4x line of *S. commersonii* was crossed with 2x genotypes, generating triploid progeny. These triploids, producing 2n eggs, were crossed with *S. tuberosum* in 3x × 4x configurations, yielding offspring with a near-pentaploid chromosome number[29]. Significantly, seven out of 26 near-pentaploid sexual hybrids between *S. commersonii* and cultivated *S. tuberosum* displayed *S. commersonii*-like resistance to BW, with notable inhibition of bacterial growth in the plant's aerial part[30]. Subsequent research efforts by various groups have also accomplished the successful transfer of resistance from *S. commersonii* to *S. tuberosum*[31,32]. The ongoing advancement of such resistant germplasm is indispensable for effectively combating the evolving challenges from pathogens and meeting the diverse demands for improved agronomic traits.

Table 1. Production of somatic hybrids through protoplast fusion for bacterial wilt resistance in Solanaceous crops.

Crop	Parents	Reference
Eggplant	*S. melongena* × *S. aethiopicum*	[12]
	S. melongena × *S. aethiopicum*	[13]
	S. integrifolium × *S. violaceum*	[14]
	S. melongena × *S. torvum*	[15]
	S. integrifolium × *S. sanitwongsei*	[16]
Potato	*S. tuberosum* × *S. commersonii*	[18]
	S. tuberosum × *S. phureja*	[22]
	S. tuberosum × *S. commersonnii*	[19]
	S. tuberosum × *S. stenotomum*	[23]
	S. tuberosum × *S. chacoense*	[24]
Eggplant to potato	*S. melongena* × *S. tuberosum*	[26]

Receptors play a pivotal role in augmenting resistance against bacterial wilt

Plants employ sophisticated systems, including pattern-recognition receptors (PRRs) and nucleotide-binding leucine-rich repeat domains (NLRs), to perceive and defend against diverse microbial molecules. Positioned on the plasma membrane, PRRs operate as extracellular receptors that discern pathogen-associated molecular patterns (PAMPs). Conversely, NLRs, which reside within the cell, act as intracellular receptors

to sense proteins that pathogens directly inject into plant cells[33]. These mechanisms, broadly considered as invasion patterns, initiate a sequence of immune responses, strengthening the plant's defenses[34].

In *A. thaliana*, various PRRs involved in bacterial recognition have been identified, including FLS2, EFR, XPS1, RLP1, RLP32, LYM1/3, and LORE[33]. However, recognition of the elongation factor Tu (EF-Tu) by the Receptor-Like Kinase (RLK) EFR is limited to the Brassicaceae family[35,36]. Intriguingly, Solanaceous plants, lacking EFR, are incapable of recognizing EF-Tu from *R. solanacearum*. Yet, transgenic introduction of Arabidopsis EFR in tomato and potato enhances disease resistance to *R. solanacearum* infection, demonstrating that interfamily transfer of PRRs can extend recognition of bacterial PAMPs, potentially providing durable disease resistance[31,35,37–39]. Moreover, the recent discovery of an *R. solanacearum* csp22 peptide (csp22Rsol) has been found to initiate immune responses in *N. benthamiana* and tomato, but not in *A. thaliana*[40]. Remarkably, csp22Rsol treatment boosted resistance to BW in tomato. Even more, transgenic *A. thaliana* plants expressing the tomato csp22 receptor (SlCORE) acquired the ability to respond to csp22Rsol and developed greater resistance to *R. solanacearum* infection[40].

The main virulence determinant of RSSC bacteria is the type III secretion system (T3SS), a 'molecular syringe' that allows the translocation of several type III effector proteins (T3Es) directly into the host cell[41]. Termed *Ralstonia* Injected Proteins (Rips), these T3Es can be detected by Nucleotide-binding Leucine-rich Repeat proteins (NLRs) as avirulence effectors, thereby triggering resistance to BW. In *A. thaliana*, extensive molecular studies have identified the major resistance gene RRS1, encoding a Toll/Interleukin-1 Receptor-Nucleotide Binding Site-Leucine-Rich Repeat (TIR-NBS-LRR) resistance protein that interacts directly with the avirulence effector PopP2[42–44]. In addition, RRS1 requires the RD19 gene-encoded Cys protease to mediate resistance to the phylotype I strain GMI1000[45]. Moreover, the RRS1 gene collaborates with the RPS4 gene to enhance resistance to strains of both *Pseudomonas* and RSSC carrying AvrRps4 and PopP2 effectors, respectively[46,47].

Within Solanaceous crops, the identification of intracellular receptors remains relatively limited. The major gene ERs1 has been discovered in eggplant *via* a map-based cloning method[48]. Utilizing a candidate gene approach, RE-bw, an intracellular receptor encompassing both NB-ACR and WRKY domains, was located within eggplant and proven to confer resistance against BW[49]. Using a multiplexed NbNLR-VIGS library, the RRS-Y (RESISTANCE TO RALSTONIA SOLANACEA RUM RIPY) was identified in *N. benthamiana*[50]. Further, specific Rips have been detected as avirulence factors in Solanaceous plants, which can be recognized by known NLR proteins against other pathogens. As an example, RipB, a homolog of *Xanthomonas* XopQ, is recognized by the *N. benthamiana* NLR protein Roq1, signifying it as an avirulence factor in *N. benthamiana*[51]. Both RipE1 and RipBN can be recognized by Ptr1, thereby conferring resistance to BW in *N. benthamiana* and *S. lycopersicoides*, respectively[52–54]. Additional effectors, including RipA5, RipH2, RipP1, RipP2, RipX, RipAA, RipAT, RipAV, RipAX2, and RipBI, have been observed to trigger cell death in Solanaceous plants[55]. Moreover, a comparative genomic analysis of *R. solanacearum* strains HA4-1 and HZAU091 led to the identification of four candidate avirulence effectors in HA4-1 that trigger immunity in wild potatoes[56,57]. As research advances, the discovery of further intracellular receptors can be optimistically anticipated.

Utilizing T3Es as molecular probes to investigate host targets involved in plant immunity

As a model pathogen for root and vascular diseases, *R. solanacearum* contains a great quantity of functionally characterized T3Es. The T3Es have evolved sufficiently to adapt to the plant immune system over a long period of natural evolution, making them indispensable molecular probes in plant immunity studies. A pan-effectome of 140 *R. solanacearum* strains has been created, comprising 102 known T3Es and 16 putative ones[58]. Recently, the novel T3E RS_T3E_Hyp9 was identified and renamed as RipBT, shown to promote *R. solanacearum* infection in potatoes[59]. Roughly half of these effectors have been characterized to varying degrees, with nine having identified host targets (Table 2[58]). For instance, RipAB targets TGA transcription factors to disrupt SA signaling and suppress plant immunity[60]. RipAC inhibits MAPK-mediated SGT1 phosphorylation and targets the plant E3 ubiquitin ligase PUB4 to repress immunity[61,62]. RipAC also targets a quantitative susceptibility factor BWS1 to regulate the SGT1-dependent immune response[63]. RipAK can inhibit host catalases and the oligomerization and enzymatic activity of pyruvate decarboxylases to promote disease[64,65]. RipAS diminishes the nucleolar accumulation of StTOPP6, contributing to virulence in potato[66]. RipAY degrades glutathione, inhibits the RipE1-triggered immune responses, and thus suppresses the immune response[67–69]. RipD targets VAMP721/722 to promote disease[50]. RipI is a multifunctional effector that modulates plant metabolism and immunity. RipI enhances gamma-aminobutyric acid (GABA) accumulation by promoting calmodulin binding to glutamate decarboxylases (GADs), which contribute to virulence in tomato and Arabidopsis[70]. However, RipI also interacts with the transcription factor bHLH93, triggering a defense response in *N. benthamiana*[71]. RipTAL targets a 17-bp sequence upstream of arginine decarboxylase (ADC) genes, inhibiting the growth of *R. solanacearum* niche competitors in tomato[72,73]. Lastly, RipX suppresses the expression of mitochondrial atpA, inducing a defense response in *N. benthamiana*[74]. These identified host targets represent a wealth of genetic resources for breeding resistance to BW.

Identification of quantitative trait loci (QTL) and development of molecular markers for resistance breeding to *Ralstonia solanacearum*

Assisting in resistance breeding to *R. solanacearum*, marker-assisted selection (MAS) significantly improves breeding efficiency for oligogenic or polygenic resistance within Solanaceous crops. Notably, the Solanaceae family's genetic diversity offers multiple sources of BW resistance, extensively studied in tomatoes (Table 3). A case in point is the Hawaii 7996 tomato cultivar, which has demonstrated exceptional resistance against BW, achieving an impressive 97% survival rate across 12 field trials conducted in 11 countries spanning Asia, America, Australia, and the Indian Ocean region[75]. This resilience makes Hawaii 7996 a stable source of resistance and an ideal resistant parent for the creation of the interspecific population

Table 2. Rips with identified host targets in Solanaceous crops.

Rips	Anonation	Target genes	Target plants	References
RipAB	(PopB), harboring protein	TGA transcription factors	*Solanum tuberosum; Solanum lycopersicum; Arabidopsis thaliana.*	[60]
RipAC	(PopC), LRR domain	SGT1; PUB4; BWS1	*Arabidopsis thaliana; Solanum lycopersicum; Nicotiana benthamiana*	[61–63]
RipAK		CATs; PDCs	*Nicotiana tabacum; Arabidopsis thaliana; Solanum lycopersicum.*	[64,65]
RipAS		TOPP6	*Solanum tuberosum*	[66]
RipAY		TRX-h	*Arabidopsis thaliana; Nicotiana benthamiana.*	[67–69]
RipB	Inosine-uridine nucleoside N-ribohydrolase	Roq1	*Nicotiana benthamiana*	
RipBN	cysteine protease, AvrRPt2 family	Ptr1	*Solanum lycopersicoides*	[52,53]
RipD		VAMP721/722	*Arabidopsis thaliana*	[66]
RipE1		Ptr1	*Nicotiana benthamiana*	[54]
RipI		bHLH93 transcription factor; plant calmodulin and GADs	*Solanum lycopersicum; Arabidopsis thaliana*	[70,71]
RipP2	Acetyltransferase	bHLH94 transcription factor	*Arabidopsis thaliana*	[44]
RipTAL	Transcription Activator-Like protein	bHLH95 transcription factor	*Solanum lycopersicoides*	[72,73]
RipX	(PopA), Harpin	bHLH96 transcription factor	*Nicotiana benthamiana*	[74]
RipY	Ankyrin Repeats	bHLH97 transcription factor	*Nicotiana benthamiana*	[50]

Hawaii7996 × Wva700, designed for resistance mapping studies. Pioneering research discovered a genetic locus on chromosome 12 exhibiting robust resistance against a certain *R. solanacearum* strain, which alongside another locus on chromosome 6, contributed significantly to the control of resistance[76]. The marker TG564 on chromosome 12 emerged as the primary association with resistance, accounting for a substantial proportion of the genetic variation[76]. Subsequent research pinpointed four quantitative trait loci (QTLs)-*Bwr-3*, *Bwr-4*, *Bwr-6*, and *Bwr-8*-that accounted for 3.2 to 29.8% of the phenotypic variation, with *Bwr-6* and *Bwr-3* persistently detected in both cool and hot seasons, while *Bwr-4* and *Bwr-8* were only detected during the hot season, implicating environmental factors in resistance manifestation[77]. The study underscored the importance of *Bwr-6* and *Bwr-3* in resistance to *R. solanacearum* race 3-phylotype II and suggested a potential overlap with resistance QTLs against other strains[77]. Further studies conducted on recombinant inbred lines identified *Bwr-12* and *Bwr-6* as principal contributors to resistance, wherein *Bwr-12* controlling 17.9%–56.1% of total resistance variation, and *Bwr-6* accounting for 11.5%–22.2% of phenotypic variation, with lines containing resistance alleles from both loci exhibiting the least disease incidence[78]. Thus, these findings reinforce the polygenic nature of tomato resistance to BW and the significance of *Bwr-6* and *Bwr-12* in conferring resistance.

Beyond the aforementioned loci, the development of supplementary molecular markers has led to significant advancements. For instance, the SCAR marker (SCU176-534) was associated with BW resistance in the Hawaii 7996 line, as identified through bulked segregant analysis (BSA) and rapid amplified polymorphic DNA (RAPD) techniques[79]. This marker showed promise for accelerating the selection of resistant lines in breeding efforts involving Hawaii 7996. Separate investigations revealed 5,259 non-synonymous single nucleotide polymorphisms (SNPs) between seven BW-resistant tomato varieties and two susceptible counterparts, mainly located on chromosomes 6 and 12. Notably, the SNP marker KHU-1, located in gene *Solyc12g009690.1*, encoding a putative leucine-rich repeat (LRR) receptor-like protein and potentially linked to the *Bwr-12* QTL, effectively differentiated resistant from susceptible tomato varieties[80]. Further developments include the creation of two CAPS markers, RsR6-5 and RsR12-1 on chromosomes 6 and 12, respectively. These markers proved effective in distinguishing between resistant and susceptible tomato varieties to BW[81]. A comprehensive study mapped a genetic chart using 1604 SNP markers, locating seven QTLs linked to BW resistance on chromosomes 6 and 12 within the 'Hawaii 7996' tomato line[82]. By phenotyping 80 BC3F3 near-isogenic lines (NILs), this study verified the specific effects of *Bwr-6.1*, *Bwr-6.3*, and *Bwr-12* on disease severity after exposure to two different BW strains across two seasons[82]. In another study involving a cross between the resistant cultivar T51A and the susceptible cultivar T9230, a BSA applied to an F2 population identified two markers, TSCARAAT/CGA and TSCARAAG/CAT, using PCR-based amplified fragment length polymorphism (AFLP) techniques. These markers, converted into co-dominant SCAR markers, were found on the opposite side of TRSR-1[83]. Moreover, an analysis of resistance segregation in two populations and whole-genome sequence data from six BW-resistant and nine BW-susceptible tomato lines suggested possible roles of loci other than *Bwr-6* and *Bwr-12* in conferring resistance[84]. This investigation revealed 27,046 unique SNPs and 5,975 indels in the resistant lines, implicating 385 genes. Among these, a significant variant on chromosome 3, marked by Bwr3.2dCAPS in the *Asc* gene, was strongly associated with resistance[84].

Eggplant, exhibiting potential resistance to all phylotypes of *R. solanacearum* (RSSC), serves as an intriguing subject for studying BW resistance. Recent research on the resistant breeding line AG91-25, derived from *S. melongena* and *S. aethiopicum*, yielded promising results[85]. In this context, a recombinant inbred lines (RILs) population, derived from a cross between AG91-25 and a susceptible parent (line MM738), was phenotyped with phylotype I strains[48]. Utilizing AFLP, SSR, and SRAP markers, researchers generated an intraspecific map with 119 molecular markers across 18 linkage groups. This led to the identification of a unique monogenic resistance locus, *ERs1*, in crop RSSC resistance[48]. When exposed to four additional RSSC strains representing phylotypes I, IIA, IIB, and III, this

Table 3. Quantitative trait loci (QTLs) and molecular markers linked to the resistance loci to bacterial wilt in Solanaceous crops.

Crop	Locus	Chromosome	Marker ID	Marker type	Reference
Tomato	–	12	TG564	RFLP	[76]
	Bwr-3, Bwr-4, Bwr-6 and Bwr-8		–	RFLP	[77]
	–	–	TSCARAAT/CGA, TSCARAAG/CAT	SCAR	[83]
	Bwr-6, Bwr-12	6, 12		SSR	[78]
			SCU176-534	SCAR	[79]
	Bwr-12	12	KHU-1	SNP	[80]
	Bwr-6	6	–	SNP	[80]
	–	–	SCU176-534	SCAR	[79]
	Bwr-6, near Bwr-12	6, 12	RsR6-5, RsR12-1	CAPS	[81]
	Bwr-6.1, Bwr-6.3 and Bwr-12	6, 12	–	SNP	[82]
	Bwr-3		Bwr3.2dCAPS	SNP	[84]
Eggplant	ERs1	9	–	AFLP, SSR, and SRAP	[48]
	EBWR9(ERs1), EBWR14, EBWR2	9, 5, 2	–	SNP	[86]
	–	3, 6	–	SNP	[87]
	–	–	emh21J12, emf01K16	SSR	[88]
	–	–	emb01D10, emh11I06, emh02E08, SSR–46	SSR	[89]
Potato	qBWR-1, qBWR-2, qBWR-3, qBWR-4, and qBWR-5	1, 3, 7, 10 and 11	–	SNP	[90]
	PBWR-6b	6	–	SNP	[91]
	PBWR-6b	6	Rbw6-1	allele-specific	[92]
Pepper	Bw1	1	CAMS451	SSR	[93]
	qRRs-10.1	10	–	SNP	[93]
	Bwr6w-7.2, Bwr6w-8.1, Bwr6w-9.1, Bwr6w-9.2, and Bwr6w-10.1, Bwr6w-5.1, Bwr6w-6.1, and Bwr6w-7.1	5, 6, 7, 8, 9, 10	C07_224926788-HRM, C08_134064617-HRM, C09_3486004-HRM, C10_232244800-HRM, C05_224016474-HRM, and C07_115436147-HRM	HRM	[95]
Tobacco	qBWR3a, qBWR-3b, qBWR-5a and qBWR-5b	–	PT20275 and PT30229	SSR	[97]
	qBWR17a	–	–	SSR	[98]

population showed one major phylotype-specific QTL, EBWR9 (which coincided with the previously identified ERs1), and two broad-spectrum QTLs, EBWR14 and EBWR2[86]. Notably, EBWR14 and EBWR2, located on chromosomes 2 and 5, offered partial resistance to strains of phylotypes I, IIA, III and strains of phylotypes IIA and III, respectively[86]. Additional studies on 123 doubled haploid lines, bred from a susceptible eggplant line (MM738) and a resistant counterpart (EG203), resulted in the mapping of 10 and three resistance QTLs for phylotypes I and III, respectively[87]. Interestingly, the most reliable QTLs were found on chromosomes 3 and 6, with the one on chromosome 6 resonating with the broad-spectrum resistance QTL *Bwr-6* observed in tomatoes[87]. Screening of six elite eggplant genotypes in a field setting identified three—CARI-1, IIHR-7, and IIHR-500A—as resistant to BW. This led to the identification of two SSR markers, emh21J12 and emf01K16, associated with this resistance[88]. Subsequently, BSA was performed in two F2 populations exhibiting BW resistance, derived from crosses between resistant lines (CARI-1 and IIHR -7) and susceptible lines (Rampur Local and Arka Kushmakar IIHR-108). The SSR markers emb01D10, emh11I06, emh02E08, and SSR-46 co-segregated with resistant and susceptible genotypes of the two F2 populations and were linked to BW resistance loci[89].

In the realm of potato research, a crossbreeding effort was undertaken between a resistant diploid line, 10-03-30, and a susceptible diploid line, F1-1. This yielded a diploid F1 population of 94 genotypes, a kind of two-way pseudo-testcross[90]. From this population, five QTLs (*qBWR-1* to *-5*) were identified through QTL analysis. These QTLs were located on potato chromosomes 1, 3, 7, 10, and 11, accounting for 9.3%–18.4% of the phenotypic variance. Of particular note was that the alleles for *qBWR-2* to *-4* were resistant, whereas those for *qBWR-1* and *qBWR-5* were susceptible[90]. Subsequent investigation uncovered 10 resistance QTLs in an F1 population, which was derived from a cross between a resistant haploid line from Saikai 35 and a susceptible diploid line[91]. Among these, QTL *PBWR-6b* was the most effective, originating from the resistant parent and located on potato chromosome 6. In a later study, a resistance allele was identified and an allele-specific molecular marker (Rbw6-1) for *PBWR-6b* was developed[92]. This discovery marked a significant advance in our understanding of potato resistance to BW.

In the domain of pepper research, Mimura et al. employed a double haploid mapping population derived from 'California Wonder' (susceptible) and 'LS2341' (resistant) to probe pepper's resistance to BW. This study successfully generated a linkage map encompassing 15 groups through the application of SSRs and AFLP. A significant QTL, *Bw1*, was discovered on pepper chromosome 1 (P1), accounting for 33% of the resistance attributed to 'LS2341'. This QTL was mapped using the SSR marker CAMS451[93]. More recently, Du et al. shed light on the dynamics of bioluminescent *R. solanacearum* colonization through an examination using reciprocal grafts of a resistant line (BVRC 1) and a susceptible line (BVRC 25). They pinpointed a key QTL (*qRRs-10.1*) on chromosome 10, hosting several resistance and defense-related genes, which plays a significant role

in BW resistance[94]. Additionally, Lee et al. identified five QTLs (*Bwr6w-7.2*, *Bwr6w-8.1*, *Bwr6w-9.1*, *Bwr6w-9.2*, and *Bwr6w-10.1*) conferring resistance to a moderately pathogenic 'HS' isolate. In contrast, three QTLs (*Bwr6w-5.1*, *Bwr6w-6.1*, and *Bwr6w-7.1*) were found to resist a highly pathogenic 'HWA' isolate of *R. solanacearum* in two F2 populations derived from the highly resistant pepper cultivar 'Konesian hot'. Within the same study, six high-resolution melting (HRM) markers linked to these QTLs were also developed[95].

In the realm of tobacco research, four QTL mapping studies have been conducted for tobacco bacterial wilt (TBW) resistance across bi-parental and diverse genetic populations, utilizing SSR and AFLP markers. An AFLP analysis identified a significant QTL linked with 15 markers, accounting for over 30% of the resistance variance, within the resistant variety W6 and susceptible variety Michinoku 1, thus yielding 117 useful DNA markers[96]. In an examination of the F2:3 and F2:4 progeny resulting from crosses between wilt-resistant breeding lines (Enshu and Yanyan97) and a susceptible line (TI448A), Qian et al. uncovered four QTLs (*qBWR3a*, *qBWR-3b*, *qBWR-5a*, and *qBWR-5b*) in linkage groups 3 and 5[97]. The closely linked markers PT20275 and PT30229, detected in both crosses, offer a valuable tool for the selection of resistant plants[97]. In another significant study, a major QTL (*qBWR17a*) was identified that accounted for 30% of the phenotypic variation, providing a noteworthy advantage for MAS in TBW resistance breeding[98]. A distinct investigation into 'K346' tobacco's resistance to BW associated three QTLs with resistance, explaining 50.3% of the observed variation[99]. Furthermore, a pioneering study identified 142 quantitative trait nucleotides (QTNs) that account for a substantial portion of the phenotypic variance for TBW resistance, with 38 of these QTNs being stable across varied environments and methodologies[100]. This research, marking the first identification of QTNs and superior alleles for the breeding of TBW-resistant tobacco varieties, also suggested the five most effective cross combinations for resistance and highlighted 52 potential candidate genes. These insights are invaluable for future studies in genetic architecture, marker-assisted selection, and functional genomics of TBW resistance, aiming to increase crop productivity. As a result, these discoveries offer instrumental tools for MAS in the breeding program to enhance resistance to BW in Solanaceous crops.

Conclusions

R. solanacearum presents a considerable threat to Solanaceous crops, and the development of effective genetic control strategies remains a pressing priority. Emerging advancements in genomics, relating to both pathogens and host plants, offer the exciting potential to discover previously unrecognized resources for disease resistance in the near future. As such, intensifying our research efforts in remote hybridization and somatic cell fusion is critical, aiming to increase success rates and create a collection of Solanaceous germplasm with strong resistance to BW. The application of effectors could play a key role in implementing high-throughput methodologies for identifying BW resistance[101]. This strategy may also stimulate resistance screening in wild species, thereby enhancing the selection of disease-resistant materials and receptor identification. When identifying receptors in certain Solanaceous plants proves challenging, we could consider the development of resistant varieties by integrating resistance genes from *A. thaliana* and other Solanaceous plants. Moreover, genome-editing technologies present promising avenues for manipulating host target genes. The successful completion of whole-genome sequencing for key Solanaceous crops, including potato, tomato, eggplant, pepper, and tobacco (referenced at https://solgenomics.net), is set to expedite the cloning process for resistance genes against BW. Furthermore, the adoption of recent methodological advancements, such as Resistance gene enrichment sequencing (RenSeq)[102], could facilitate quicker receptor identification within Solanaceous crops. These combined efforts give rise to the promising future of *R. solanacearum*-resistant crop development, transforming it from a distant goal into an imminent reality.

Author Contributions

The authors confirm contribution to the paper as follows: study conception and design: study conception and design: Du J, Wang B, Chen H, Song B; data collection: Du J, Wang B, Huang M, Chen X, Nie L, Wang T; draft manuscript preparation: Du J, Wang B, Huang M, Chen X, Chen H, Song B. All authors reviewed the results and approved the final version of the manuscript.

Data availability

All data supporting the findings of this research are available within the paper and within its supplementary data.

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Conflict of interest

The authors declare that they have no conflict of interest.

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References

1. Mansfield J, Genin S, Magori S, Citovsky V, Sriariyanum M, et al. 2012. Top 10 plant pathogenic bacteria in molecular plant pathology. *Molecular Plant Pathology* 13:614–29
2. Álvarez B, López MM, Biosca EG. 2008. Survival strategies and pathogenicity of *Ralstonia solanacearum* phylotype II subjected to prolonged starvation in environmental water microcosms. *Microbiology* 154:3590–98
3. Fegan M, Prior P. 2005. How complex is the *Ralstonia solanacearum* species complex? In *Bacterial Wilt Disease and the Ralstonia Solanacearum Species Complex*, eds. Allen C, Prior P, Hayward AC. St. Paul, MN: APS Press. pp. 449–61.
4. Safni I, Cleenwerck I, De Vos P, Fegan M, Sly L, et al. 2014. Polyphasic taxonomic revision of the *Ralstonia solanacearum*

species complex: proposal to emend the descriptions of *Ralstonia solanacearum* and *Ralstonia syzygii* and reclassify current *R. syzygii* strains as *Ralstonia syzygii* subsp. *syzygii* subsp. nov., *R. solanacearum* phylotype IV strains as *Ralstonia syzygii* subsp. *indonesiensis* subsp. nov., banana blood disease bacterium strains as *Ralstonia syzygii* subsp. *celebesensis* subsp. nov. and *R. solanacearum* phylotype I and III strains as *Ralstonia pseudosolanacearum* sp. nov. *International Journal of Systematic and Evolutionary Microbiology* 64:3087–103

5. Prior P, Ailloud F, Dalsing BL, Remenant B, Sanchez B, et al. 2016. Genomic and proteomic evidence supporting the division of the plant pathogen *Ralstonia solanacearum* into three species. *BMC Genomics* 17:90
6. Sharma P, Johnson MA, Mazloom R, Allen C, Heath LS, et al. 2022. Meta-analysis of the *Ralstonia solanacearum* species complex (RSSC) based on comparative evolutionary genomics and reverse ecology. *Microbial Genomics* 8:000791
7. Lebeau A, Daunay MC, Frary A, Palloix A, Wang J, et al. 2011. Bacterial wilt resistance in tomato, pepper, and eggplant: genetic resources respond to diverse strains in the *Ralstonia solanacearum* species complex. *Phytopathology* 101:154–65
8. Salanoubat M, Genin S, Artiguenave F, Gouzy J, Mangenot S, et al. 2002. Genome sequence of the plant pathogen *Ralstonia solanacearum*. *Nature* 415:497–502
9. Lewis Ivey ML, Jimenez Madrid AM, Daunay MC, Shah DA. 2021. Evaluation of tomato, eggplant and pepper accessions for resistance to *Ralstonia solanacearum* species complex (RSSC) strains from Louisiana. *European Journal of Plant Pathology* 159:279–93
10. Poueymiro M, Cunnac S, Barberis P, Deslandes L, Peeters N, et al. 2009. Two type III secretion system effectors from *Ralstonia solanacearum* GMI1000 determine host-range specificity on tobacco. *Molecular Plant-Microbe Interactions* 22:538–50
11. Hawkes JG. 1994. Origins of cultivated potatoes and species relationships. In *Potato Genetics*, eds. Bradshaw J, MacKay G. Wallingford, UK: CAB INTERNATIONAL. pp. 3–42
12. Daunay MC, Chaput MH, Sihachakr D, Allot M, Vedel F, et al. 1993. Production and characterization of fertile somatic hybrids of eggplant (*Solanum melongena* L.) with *Solanum aethiopicum* L. *Theoretical and Applied Genetics* 85:841–50
13. Collonnier C, Mulya K, Fock I, Mariska I, Servaes A, et al. 2001. Source of resistance against *Ralstonia solanacearum* in fertile somatic hybrids of eggplant (*Solanum melongena* L.) with *Solanum aethiopicum* L. *Plant Science* 160:301–13
14. Tamura N, Murata Y, Mukaihara T. 2002. A somatic hybrid between *Solanum integrifolium* and *Solanum violaceum* that is resistant to bacterial wilt caused by *Ralstonia solanacearum*. *Plant Cell Reports* 21:353–58
15. Collonnier C, Fock I, Mariska I, Servaes A, Vedel F, et al. 2003. GISH confirmation of somatic hybrids between *Solanum melongena* and *S. torvum*: assessment of resistance to both fungal and bacterial wilts. *Plant Physiology and Biochemistry* 41:459–70
16. Iwamoto Y, Hirai M, Ohmido N, Fukui K, Ezura H. 2007. Fertile somatic hybrids between *Solanum integrifolium* and *S. sanitwongsei* (syn. *S. kurzii*) as candidates for bacterial wilt-resistant rootstock of eggplant. *Plant Biotechnology* 24:179–84
17. Johnston SA, Hanneman RE. 1980. Support of the endosperm balance number hypothesis utilizing some tuber-bearing *Solanum* species. *American Potato Journal* 57:7–14
18. Laferriere LT, Helgeson JP, Allen C. 1999. Fertile *Solanum tuberosum+S. commersonii* somatic hybrids as sources of resistance to bacterial wilt caused by *Ralstonia solanacearum*. *Theoretical and Applied Genetics* 98:1272–78
19. Kim-Lee H, Moon JS, Hong YJ, Kim MS, Cho HM. 2005. Bacterial wilt resistance in the progenies of the fusion hybrids between haploid of potato and *Solanum commersonii*. *American Journal of Potato Research* 82:129–37
20. Gaiero P, Mazzella C, Vilaró F, Speranza P, de Jong H. 2017. Pairing analysis and in situ Hybridisation reveal autopolyploid-like behaviour in *Solanum commersonii* × *S. tuberosum* (potato) interspecific hybrids. *Euphytica* 213:137
21. Ferreira V, Pianzzola MJ, Vilaró FL, Galván GA, Tondo ML, et al. 2017. Interspecific potato breeding lines display differential colonization patterns and induced defense responses after *Ralstonia solanacearum* infection. *Frontiers in Plant Science* 8:1424
22. Fock I, Collonnier C, Purwito A, Luisetti J, Souvannavong V, et al. 2000. Resistance to bacterial wilt in somatic hybrids between *Solanum tuberosum* and *Solanum phureja*. *Plant Science* 160:165–76
23. Fock I, Collonnier C, Lavergne D, Vaniet S, Ambroise A, et al. 2007. Evaluation of somatic hybrids of potato with *Solanum stenotomum* after a long-term *in vitro* conservation. *Plant Physiology and Biochemistry* 45:209–15
24. Chen L, Guo X, Xie C, He L, Cai X, et al. 2013. Nuclear and cytoplasmic genome components of *Solanum tuberosum* + *S. chacoense* somatic hybrids and three SSR alleles related to bacterial wilt resistance. *Theoretical and Applied Genetics* 126:1861–72
25. Cai X, Liu J, Xie C. 2004. Mesophyll protoplast fusion of *Solanum tuberosum* and *Solanum chacoense* and their somatic hybrid analysis. *Acta Horticulturae Sinica* 31:623–26
26. Yu Y, Ye W, He L, Cai X, Liu T, et al. 2013. Introgression of bacterial wilt resistance from eggplant to potato via protoplast fusion and genome components of the hybrids. *Plant Cell Reports* 32:1687–701
27. Liu T, Yu Y, Cai X, Tu W, Xie C, et al. 2016. Introgression of bacterial wilt resistance from *Solanum melongena* to *S. tuberosum* through asymmetric protoplast fusion. *Plant Cell, Tissue and Organ Culture (PCTOC)* 125:433–43
28. Wang H, Cheng Z, Wang B, Dong J, Ye W, et al. 2020. Combining genome composition and differential gene expression analyses reveals that *SmPGH1* contributes to bacterial wilt resistance in somatic hybrids. *Plant Cell Reports* 39:1235–48
29. Carputo D, Barone A, Cardi T, Sebastiano A, Frusciante L, et al. 1997. Endosperm balance number manipulation for direct *in vivo* germplasm introgression to potato from a sexually isolated relative (*Solanum commersonii* Dun.). *Proceedings of the National Academy of Sciences of the United States of America* 94:12013–17
30. Carputo D, Aversano R, Barone A, Di Matteo A, Iorizzo M, et al. 2009. Resistance to *Ralstonia solanacearum* of sexual hybrids between *Solanum commersonii* and *S. tuberosum*. *American Journal of Potato Research* 86:196–202
31. Boschi F, Schvartzman C, Murchio S, Ferreira V, Siri MI, et al. 2017. Enhanced bacterial wilt resistance in potato through expression of *Arabidopsis* EFR and introgression of quantitative resistance from *Solanum commersonii*. *Frontiers in Plant Science* 8:1642
32. González M, Galván G, Siri MI, Borges A, Vilaró F. 2022. Resistance to bacterial wilt in *Solanum commersonii* Dun. *Agrociencia Uruguay* 26:e1092
33. Ngou BPM, Ding P, Jones JDG. 2022. Thirty years of resistance: zig-zag through the plant immune system. *The Plant Cell* 34:1447–78
34. Cook DE, Mesarich CH, Thomma BPHJ. 2015. Understanding plant immunity as a surveillance system to detect invasion. *Annual Review of Phytopathology* 53:541–63
35. Lacombe S, Rougon-Cardoso A, Sherwood E, Peeters N, Dahlbeck D, et al. 2010. Interfamily transfer of a plant pattern-recognition receptor confers broad-spectrum bacterial resistance. *Nature Biotechnology* 28:365–69
36. Zipfel C, Kunze G, Chinchilla D, Caniard A, Jones JDG, et al. 2006. Perception of the bacterial PAMP EF-Tu by the receptor EFR restricts *Agrobacterium*-mediated transformation. *Cell* 125:749–60
37. Kunwar S, Iriarte F, Fan Q, Evaristo da Silva E, Ritchie L, et al. 2018. Transgenic expression of *EFR* and *Bs2* genes for field management of bacterial wilt and bacterial spot of tomato. *Phytopathology* 108:1402–11

38. Fort S, Ferreira V, Murchio S, Schvartzman C, Galván GA, et al. 2020. Potato plants transformed with the Arabidopsis EF-Tu receptor (EFR) show restricted pathogen colonization and enhanced bacterial wilt resistance under conditions resembling natural field infections. *Agrociencia Uruguay* 24:e413
39. Dalla-Rizza M, Schvartzman C, Murchio S, Berrueta C, Boschi F, et al. 2022. Field performance of resistant potato genotypes transformed with the EFR receptor from *Arabidopsis thaliana* in the absence of bacterial wilt (*Ralstonia solanacearum*). *The Plant Pathology Journal* 38:239–47
40. Wei Y, Caceres-Moreno C, Jimenez-Gongora T, Wang K, Sang Y, et al. 2018. The *Ralstonia solanacearum* csp22 peptide, but not flagellin-derived peptides, is perceived by plants from the *Solanaceae* family. *Plant Biotechnology Journal* 16:1349–62
41. Coll NS, Valls M. 2013. Current knowledge on the *Ralstonia solanacearum* type III secretion system. *Microbial Biotechnology* 6:614–20
42. Deslandes L, Olivier J, Theulières F, Hirsch J, Feng D, et al. 2002. Resistance to *Ralstonia solanacearum* in *Arabidopsis thaliana* is conferred by the recessive *RRS1-R* gene, a member of a novel family of resistance genes. *Proceedings of the National Academy of Sciences of the United States of America* 99:2404–9
43. Lahaye T. 2004. Illuminating the molecular basis of gene-for-gene resistance; *Arabidopsis thaliana RRS1-R* and its interaction with *Ralstonia solanacearum popP2*. *Trends in Plant Science* 9:1–4
44. Deslandes L, Olivier J, Peeters N, Feng D, Khounlotham M, et al. 2003. Physical interaction between RRS1-R, a protein conferring resistance to bacterial wilt, and PopP2, a type III effector targeted to the plant nucleus. *Proceedings of the National Academy of Sciences of the United States of America* 100:8024–29
45. Bernoux M, Timmers T, Jauneau A, Brière C, de Wit PJGM, et al. 2008. RD19, an *Arabidopsis* cysteine protease required for RRS1-R-mediated resistance, is relocalized to the nucleus by the *Ralstonia solanacearum* PopP2 effector. *The Plant Cell* 20:2252–64
46. Narusaka M, Shirasu K, Noutoshi Y, Kubo Y, Shiraishi T, et al. 2009. *RRS1* and *RPS4* provide a dual *Resistance*-gene system against fungal and bacterial pathogens. *The Plant Journal* 60:218–26
47. Sohn KH, Segonzac C, Rallapalli G, Sarris PF, Woo JY, et al. 2014. The nuclear immune receptor *RPS4* is required for $RRS1^{SLH1}$-dependent constitutive defense activation in *Arabidopsis thaliana*. *PLoS Genetics* 10:e1004655
48. Lebeau A, Gouy M, Daunay MC, Wicker E, Chiroleu F, et al. 2013. Genetic mapping of a major dominant gene for resistance to *Ralstonia solanacearum* in eggplant. *Theoretical and Applied Genetics* 126:143–58
49. Xiao X, Cao B, Li G, Lei J, Chen Q, et al. 2015. Functional characterization of a putative bacterial wilt resistance gene (*RE-bw*) in eggplant. *Plant Molecular Biology Reporter* 33:1058–73
50. Kim B, Yu W, Kim H, Dong Q, Choi S, et al. 2023. A plasma membrane nucleotide-binding leucine-rich receptor mediates the recognition of the *Ralstonia pseudosolanacearum* effector RipY in *Nicotiana benthamiana*. *Plant Communications*100640
51. Nakano M, Mukaihara T. 2019. The type III effector RipB from *Ralstonia solanacearum* RS1000 acts as a major avirulence factor in *Nicotiana benthamiana* and other *Nicotiana* species. *Molecular Plant Pathology* 20:1237–51
52. Mazo-Molina C, Mainiero S, Hind SR, Kraus CM, Vachev M, et al. 2019. The *Ptr1* locus of *Solanum lycopersicoides* confers resistance to race 1 strains of *Pseudomonas syringae* pv. *tomato* and to *Ralstonia pseudosolanacearum* by recognizing the type III effectors AvrRpt2 and RipBN. *Molecular Plant-Microbe Interactions* 32:949–60
53. Mazo-Molina C, Mainiero S, Haefner BJ, Bednarek R, Zhang J, et al. 2020. *Ptr1* evolved convergently with *RPS2* and *Mr5* to mediate recognition of AvrRpt2 in diverse solanaceous species. *The Plant Journal* 103:1433–45
54. Kim B, Kim I, Yu W, Li M, Kim H, et al. 2023. The *Ralstonia pseudosolanacearum* effector RipE1 is recognized at the plasma membrane by *NbPtr1*, the *Nicotiana benthamiana* homologue of *Pseudomonas tomato race 1*. *Molecular Plant Pathology* 24:1312–18
55. Jayaraman J, Segonzac C, Cho H, Jung G, Sohn KH. 2016. Effector-assisted breeding for bacterial wilt resistance in horticultural crops. *Horticulture, Environment, and Biotechnology* 57:415–23
56. Tan X, Qiu H, Li F, Cheng D, Zheng X, et al. 2019. Complete genome sequence of sequevar 14M *Ralstonia solanacearum* strain HA4-1 reveals novel type III effectors acquired through horizontal gene transfer. *Frontiers in Microbiology* 10:1893
57. Huang M, Tan X, Song B, Wang Y, Cheng D, et al. 2023. Comparative genomic analysis of *Ralstonia solanacearum* reveals candidate avirulence effectors in HA4-1 triggering wild potato immunity. *Frontiers in Plant Science* 14:1075042
58. Landry D, González-Fuente M, Deslandes L, Peeters N. 2020. The large, diverse, and robust arsenal of *Ralstonia solanacearum* type III effectors and their in planta functions. *Molecular Plant Pathology* 21:1377–88
59. Qiu H, Wang B, Huang M, Sun X, Yu L, et al. 2023. A novel effector RipBT contributes to *Ralstonia solanacearum* virulence on potato. *Molecular Plant Pathology* 24:947–60
60. Qi P, Huang M, Hu X, Zhang Y, Wang Y, et al. 2022. A *Ralstonia solanacearum* effector targets TGA transcription factors to subvert salicylic acid signaling. *The Plant Cell* 34:1666–83
61. Yu G, Xian L, Xue H, Yu W, Rufian JS, et al. 2020. A bacterial effector protein prevents MAPK-mediated phosphorylation of SGT1 to suppress plant immunity. *PLoS Pathogens* 16:e1008933
62. Yu G, Derkacheva M, Rufian JS, Brillada C, Kowarschik K, et al. 2022. The *Arabidopsis* E3 ubiquitin ligase PUB4 regulates BIK1 and is targeted by a bacterial type-III effector. *The EMBO Journal* 41:e107257
63. Demirjian C, Razavi N, Yu G, Mayjonade B, Zhang L, et al. 2023. An atypical *NLR* gene confers bacterial wilt susceptibility in *Arabidopsis*. *Plant Communications* 4:100607
64. Sun Y, Li P, Deng M, Shen D, Dai G, et al. 2017. The *Ralstonia solanacearum* effector RipAK suppresses plant hypersensitive response by inhibiting the activity of host catalases. *Cellular Microbiology* 19:e12736
65. Wang Y, Zhao A, Morcillo RJL, Yu G, Xue H, et al. 2021. A bacterial effector protein uncovers a plant metabolic pathway involved in tolerance to bacterial wilt disease. *Molecular Plant* 14:1281–96
66. Wang B, He W, Huang M, Feng J, Li Y, et al. 2023. *Ralstonia solanacearum* type III effector RipAS associates with potato type one protein phosphatase StTOPP6 to promote bacterial wilt. *Horticulture Research* 10:uhad087
67. Mukaihara T, Hatanaka T, Nakano M, Oda K. 2016. *Ralstonia solanacearum* type III effector RipAY is a glutathione-degrading enzyme that is activated by plant cytosolic thioredoxins and suppresses plant immunity. *mBio* 7:e00359–16
68. Sang Y, Wang Y, Ni H, Cazalé AC, She YM, et al. 2018. The *Ralstonia solanacearum* type III effector RipAY targets plant redox regulators to suppress immune responses. *Molecular Plant Pathology* 19:129–42
69. Sang Y, Yu W, Zhuang H, Wei Y, Derevnina L, et al. 2020. Intra-strain elicitation and suppression of plant immunity by *Ralstonia solanacearum* type-III effectors in *Nicotiana benthamiana*. *Plant Communications* 1:100025
70. Xian L, Yu G, Wei Y, Rufian JS, Li Y, et al. 2020. A bacterial effector protein hijacks plant metabolism to support pathogen nutrition. *Cell Host & Microbe* 28:548–57
71. Zhuo T, Wang X, Chen Z, Cui H, Zeng Y, et al. 2020. The *Ralstonia solanacearum* effector RipI induces a defence reaction by interacting with the bHLH93 transcription factor in *Nicotiana benthamiana*. *Molecular Plant Pathology* 21:999–1004

72. de Lange O, Schreiber T, Schandry N, Radeck J, Braun KH, et al. 2013. Breaking the DNA-binding code of *Ralstonia solanacearum* TAL effectors provides new possibilities to generate plant resistance genes against bacterial wilt disease. *New Phytologist* 199:773–86
73. Wu D, von Roepenack-Lahaye E, Buntru M, de Lange O, Schandry N, et al. 2019. A plant pathogen type III effector protein subverts translational regulation to boost host polyamine levels. *Cell Host & Microbe* 26:638–49
74. Sun T, Wu W, Wu H, Rou W, Zhou Y, et al. 2020. *Ralstonia solanacearum* elicitor RipX induces defense reaction by suppressing the mitochondrial *atpA* Gene in host plant. *International Journal of Molecular Sciences* 21:2000
75. Wang J, Hanson P, Barnes JA. 1998. Worldwide evaluation of an international set of resistance sources to bacterial wilt in tomato. In *Bacterial Wilt Disease*, eds. Prior P, Allen C, Elphinstone J. Berlin, Heidelberg: Springer. pp. 269–75. https://doi.org/10.1007/978-3-662-03592-4_39
76. Wang J, Olivier J, Thoquet P, Mangin B, Sauviac L, et al. 2000. Resistance of tomato line hawaii7996 to *Ralstonia solanacearum* Pss4 in Taiwan is controlled mainly by a major strain-specific locus. *Molecular Plant-Microbe Interactions* 13:6–13
77. Carmeille A, Caranta C, Dintinger J, Prior P, Luisetti J, et al. 2006. Identification of QTLs for *Ralstonia solanacearum* race 3-phylotype II resistance in tomato. *Theoretical and Applied Genetics* 113:110–21
78. Wang J, Ho FI, Truong HTH, Huang SM, Balatero CH, et al. 2013. Identification of major QTLs associated with stable resistance of tomato cultivar 'Hawaii 7996' to *Ralstonia solanacearum*. *Euphytica* 190:241–52
79. Truong HTH, Kim S, Tran HN, Nguyen TTT, Nguyen LT, et al. 2015. Development of a SCAR marker linked to bacterial wilt (*Ralstonia solanacearum*) resistance in tomato line Hawaii 7996 using bulked-segregant analysis. *Horticulture, Environment, and Biotechnology* 56:506–15
80. Kim B, Hwang IS, Lee HJ, Lee JM, Seo E, et al. 2018. Identification of a molecular marker tightly linked to bacterial wilt resistance in tomato by genome-wide SNP analysis. *Theoretical and Applied Genetics* 131:1017–30
81. Abebe AM, Choi J, Kim Y, Oh CS, Yeam I, et al. 2020. Development of diagnostic molecular markers for marker-assisted breeding against bacterial wilt in tomato. *Breeding Science* 70:462–73
82. Shin IS, Hsu JC, Huang SM, Chen JR, Wang JF, et al. 2020. 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* 216:54
83. Miao L, Shou S, Cai J, Jiang F, Zhu Z, et al. 2009. Identification of two AFLP markers linked to bacterial wilt resistance in tomato and conversion to SCAR markers. *Molecular Biology Reports* 36:479–86
84. Barchenger DW, Hsu YM, Ou JY, Lin YP, Lin YC, et al. 2022. Whole genome resequencing and complementation tests reveal candidate loci contributing to bacterial wilt (*Ralstonia* sp.) resistance in tomato. *Scientific Reports* 12:8374
85. Ano G, Hebert Y, Prior P, Messiaen CM. 1991. A new source of resistance to bacterial wilt of eggplants obtained from a cross: *Solanum aethiopicum* L ×*Solanum melongena* L. *Agronomie* 11:555–60
86. Salgon S, Jourda C, Sauvage C, Daunay MC, Reynaud B, et al. 2017. Eggplant resistance to the *Ralstonia solanacearum* species complex involves both broad-spectrum and strain-specific quantitative trait loci. *Frontiers in Plant Science* 8:828
87. Salgon S, Raynal M, Lebon S, Baptiste JM, Daunay MC, et al. 2018. Genotyping by sequencing highlights a polygenic resistance to *Ralstonia pseudosolanacearum* in eggplant (*Solanum melongena* L.). *International Journal of Molecular Sciences* 19:357
88. Khapte PS, Singh TH, Lakshmana Reddy DC. 2018. Screening of elite eggplant (*Solanum melongena*) genotypes for bacterial wilt (*Ralstonia solanacearum*) in field conditions and their genetic association by using SSR markers. *The Indian Journal of Agricultural Sciences* 88:1502–9
89. Pandiyaraj P, Singh TH, Reddy KM, Sadashiva AT, Gopalakrishnan C, et al. 2019. Molecular markers linked to bacterial wilt (*Ralstonia solanacearum*) resistance gene loci in eggplant (*Solanum melongena* L.). *Crop Protection* 124:104822
90. Habe I, Miyatake K, Nunome T, Yamasaki M, Hayashi T. 2019. QTL analysis of resistance to bacterial wilt caused by *Ralstonia solanacearum* in potato. *Breeding Science* 69:592–600
91. Habe I, Miyatake K. 2022. Identification and characterization of resistance quantitative trait loci against bacterial wilt caused by the *Ralstonia solanacearum* species complex in potato. *Molecular Breeding* 42:50
92. Habe I, Sakamoto Y, Matsumoto K. 2023. The development and efficient utilization of molecular markers for the major quantitative trait locus of bacterial wilt resistance in potato. *Euphytica* 219:68
93. Mimura Y, Kageyama T, Minamiyama Y, Hirai M. 2009. QTL analysis for resistance to *Ralstonia solanacearum* in *Capsicum* accession 'LS2341'. *Journal of the Japanese Society for Horticultural Science* 78:307–13
94. Du H, Wen C, Zhang X, Xu X, Yang J, et al. 2019. Identification of a major QTL (*qRRs-10.1*) that confers resistance to *Ralstonia solanacearum* in pepper (*Capsicum annuum*) using SLAF-BSA and QTL mapping. *International Journal of Molecular Sciences* 20:5887
95. Lee S, Chakma N, Joung S, Lee JM, Lee J. 2022. QTL mapping for resistance to bacterial wilt caused by two isolates of *Ralstonia solanacearum* in chili pepper (*Capsicum annuum* L.). *Plants* 11:1551
96. Nishi T, Tajima T, Noguchi S, Ajisaka H, Negishi H. 2003. Identification of DNA markers of tobacco linked to bacterial wilt resistance. *Theoretical and Applied Genetics* 106:765–70
97. Qian Y, Wang X, Wang D, Zhang L, Zu C, et al. 2013. The detection of QTLs controlling bacterial wilt resistance in tobacco (*N. tabacum* L.). *Euphytica* 192:259–66
98. Lan T, Zheng S, Yang L, Wu S, Wang B, et al. 2014. Mapping of quantitative trait loci conferring resistance to bacterial wilt in tobacco (*Nicotiana tabacum* L.). *Plant Breeding* 133:672–77
99. Drake-Stowe K, Bakaher N, Goepfert S, Philippon B, Mark R, et al. 2017. Multiple disease resistance loci affect soilborne disease resistance in tobacco (*Nicotiana tabacum*). *Phytopathology* 107:1055–61
100. Lai R, Ikram M, Li R, Xia Y, Yuan Q, et al. 2021. Identification of novel quantitative trait nucleotides and candidate genes for bacterial wilt resistance in tobacco (*Nicotiana tabacum* L.) using genotyping-by-sequencing and multi-locus genome-wide association studies. *Frontiers in Plant Science* 12:744175
101. Du J, Rietman H, Vleeshouwers VGAA. 2014. Agroinfiltration and PVX agroinfection in potato and *Nicotiana benthamiana*. *Journal of Visualized Experiments* 83:e50971
102. Jupe F, Witek K, Verweij W, Śliwka J, Pritchard L, et al. 2013. Resistance gene enrichment sequencing (RenSeq) enables reannotation of the NB-LRR gene family from sequenced plant genomes and rapid mapping of resistance loci in segregating populations. *The Plant Journal* 76:530–44