Identification and characterization of resistance quantitative trait loci against bacterial wilt caused by the Ralstonia solanacearum species complex in potato

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Research Article

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Abstract

Bacterial wilt (BW) caused by the *Ralstonia solanacearum* species complex (RSSC) represents one of the most serious diseases affecting potato cultivation. The development of BW-resistant cultivars represents the most efficient strategy to control this disease. The resistance-related quantitative trail loci (QTLs) in plants against different RSSC strains have not been studied extensively. Therefore, we performed QTL analysis for evaluating BW resistance using a diploid population derived from *Solanum phureja*, *Solanum chacoense*, and *Solanum tuberosum*. Plants cultivated *in vitro* were inoculated with different strains (phylotype I/biovar 3, phylotype I/biovar 4, and phylotype IV/biovar 2A) and incubated at 24°C or 28°C under controlled conditions. Interval mapping was performed for the disease indexes using the resistant parent-derived map consisting of 1,476 single-nucleotide polymorphism (SNP) markers and the susceptible parent-derived map consisting of 2,663 SNP markers. We identified five major and five minor resistance QTLs on potato chromosomes 1, 3, 5, 6, 7, 10, and 11. The major QTLs *qBWR-3* and *qBWR-7* conferred stable resistance against *Ralstonia pseudosolanacearum* (phylotype I) and *Ralstonia syzygii* (phylotype IV), while *qBWR-6b* was a strain-specific major resistance QTL against phylotype I/biovar 3 and was more effective at a relatively lower temperature. Therefore, we suggest that broad-spectrum QTLs and strain-specific QTLs can be combined to develop the most effective BW-resistant cultivars for particular areas.

Introduction

Solanum tuberosum L. (potato) is cultivated worldwide and is the most important *Solanaceae* crop (Liu et al. 2016); over 462 million metric tons were produced in 2019 (Food and Agriculture Organization of the United Nations 2021). However, its cultivation is frequently limited by pests and diseases, including bacterial wilt (BW), which represents one of the most serious and widespread bacterial diseases in the tropics, subtropics, and warm temperate regions worldwide (Hayward 1985).

BW is caused by the *Ralstonia solanacearum* species complex (RSSC), which mainly enters plants via roots and then colonizes the xylem vessels and spreads through the vascular system of plants. RSSC infection causes typical wilting symptoms, leading to rapid death of the host plant. RSSC has been reported to infect over 250 plant species including many cash crops and major food crops, such as banana, potato, tomato, and eggplant (Peeters et al. 2013). Recently, it has been ranked second in the list of the most scientifically/economically important bacterial pathogens (Mansfield et al. 2012).

RSSC is classified into five races based on host range or six biovars based on their ability to produce acid using various disaccharides and sugar alcohols (Buddenhagen and Kelman 1964; Denny 2006). Based on molecular analysis, RSSC is classified into four monophyletic groups called phylotypes (Gillings and Fahy 1994). A probable geographical origin represents an attribute of each phylotype; phylotypes I, II, III, and IV have been considered to originate from Asia, the Americas, Africa, and Indonesia and Australia, respectively (Fegan and Prior 2005; Wicker et al. 2012). Recently, RSSC has been suggested to comprise three species: *R. solanacearum* including phylotype II, *Ralstonia pseudosolanacearum* including phylotypes I and III, and *Ralstonia syzygii* including the former *R. solanacearum* phylotype and the closely related pathogen *R. syzygii* (Safni et al. 2014). Although genomic, proteomic, and functional phenotypic analyses support this classification (Prior et al. 2016), the ecological and evolutionary relationships of these species remain unknown.

Strategies to control BW, such as crop rotation, elimination of weeds that represent alternative hosts, and biological control, are insufficient and the disease continues to cause major profit loss (Huet 2014). In addition, chemical-based control using chloropicrin has an adverse impact on the environment and its use is undesirable. The development of BW-resistant cultivars is cost-effective and environmentally friendly; however, only a few factors that confer BW resistance have been identified (Laferriere et al. 1999). BW resistance has been found in cultivated diploid species and closely related wild species (Thurston and Lozaro 1968; Sequeira and Rowe 1969; French and De Lindo 1982; Laferriere et al. 1999; Fock et al. 2000, 2001; Kim-Lee et al. 2005; Carputo et al. 2009; Chen et al. 2013). The cultivated diploid species *Solanum phureja* is often used as a source of BW resistance factors (Sequeira and Rowe 1969; Watanabe et al. 1992; French et al. 1998). The *S. phureja*-derived breeding clone Saikai 35 has a high level of BW resistance (Mori et al. 2012), from which a BW-resistant cultivar Nagasaki Kogane has been derived (Sakamoto et al. 2017).

BW resistance is controlled by multiple genes (Elphinstone 1994; Rowe and Sequeira 1970; Sequeira 1979). Two major quantitative trait loci (QTLs) *bwr-12* and *bwr-6* and several minor QTLs have been identified in the tomato cultivar *Solanum lycopersicum* Hawaii 7996. *bwr-12* confers partial resistance to the phylotype I strain and *bwr-6* confers partial resistance to both phylotype I and II strains or broad-spectrum resistance (Thoquet et al. 1996a, 1996b; Wang et al. 2000, 2013; Carmeille et al. 2006). In potatoes, BW resistance against race 1/biovar 3 strains has been found in somatic hybrids of *S. tuberosum* + *Solanum chacoense*, and the resistance QTLs have been identified in chromosomes 2 and 9 (Chen et al. 2013). However, resistance QTLs against different strains have not been studied extensively.

We previously identified resistance QTLs against the phylotype Ⅱ/biovar 4 strain on chromosomes 1, 3, 7, 10, and 11 using a diploid mapping population consisting of *S. tuberosum*, *S. chacoense*, and *S. phureja* (Habe et al. 2019). In this study, we assayed the same diploid population using different strains (phylotypes I and IV or biovars 3, 4, and 2A) at different incubation temperatures (24°C and 28°C) after inoculation and performed QTL analysis.

Materials And Methods

Plant materials

Saikai 35 is a breeding clone highly resistant to BW (Mori et al. 2012). From Saikai 35, a resistant haploid clone (10-03-30) was obtained via parthenogenesis by crossing the pollen of a haploid inducer *S. phureja* 460 (= IvP 35). This resistant parent (RP) was crossed as the female parent with a susceptible diploid clone F_1-1 (SP) as the male parent, which generated 94 F_1 plants grown *in vitro* using Murashige and Skoog (MS) medium (Murashige and Skoog 1962). The F_1 population (Habe et al. 2019) was previously characterized for BW resistance to the strain MAFF327001 (phylotype I/ biovar 4).

Inoculation and disease resistance analysis

The *in vitro* inoculation test (Habe 2018) was performed to evaluate resistance in the F_1 plants. The *in vitro* screening medium, containing 30 mL vermiculite and 20 mL MS liquid medium in a glass tube (40 mm × 130 mm), was sterilized via autoclaving. The plants cultivated *in vitro* were cut at nodes below the third or fourth leaf from the apex. The cut stems were transplanted into an *in vitro* screening medium and incubated in a growth chamber for two weeks to promote rooting. The light-dark cycle was 16 h light at 3000–4000 lux and 8 h dark. The incubation temperature was 18°C.

The RSSC strains MAFF327001 (phylotype I/biovar 4), MAFF327095 (phylotype IV/biovar 2A), and MAFF327142 (phylotype I/ biovar 3) isolated from potato (Horita et al. 2010) were used in this study for the inoculation test (Table 1). The strains were cultured at 30 ºC in 2,3,5-triphenyltetrazolium chloride solid medium (Kelman 1954). White fluid-containing colonies were transferred to casamino acid-peptone-glucose medium (Hendrick and Sequeira 1984). The inoculum cell concentration was determined by measuring the optical density at 600 nm and adjusted to 10^8 colony-forming units/mL in sterile water. The bacterial suspension (1 mL) was poured into each screening medium. Nine or ten plantlets per genotype were used as one replicate, and three replicates were tested for BW resistance. After inoculation, one set was incubated at 24°C and the other set was incubated at 28°C.

Table 1
QTLs detected by CIM analysis for the resistance to the *R. solanacearum* species complex in the F_1 population

BW strain	Temperature	QTL[1)]	Detected map	Chr	Position (cM)	Position of maximum LOD (cM)	Maximum LOD score	Explained variance (%)
MAFF327142 (Phylotype I/ biovar 3)	24 C°	*qBWR-6b*	R map	6	10.8–23.7	21.7	14.17	40.5
		qBWR-10b	S map	10	53.2–56.6	55.6	4.32	14.5
	28 C°	*qBWR-6b*	R map	6	13.8–23.7	21.7	5.51	17.6
		qBWR-6a	S map	6	0.0	0.0	3.93	13.6
MAFF327001 (Phylotype I/ biovar 4)	24 C°	*qBWR-7*	R map	7	12.2–27.2	25.3	6.86	20.5
	28 C°	*qBWR-1b*	S map	1	79.0–79.1	79.1	3.83	11.4
		qBWR-3	S map	3	13.8–17.0	15.0	4.30	13.0
		qBWR-5	S map	5	54.7–55.7	54.7	3.76	11.2
		qBWR-7	R map	7	15.2–27.2	25.3	6.64	21.9
		qBWR-10a	S map	10	6.6–10.9	8.8	4.93	15.1
MAFF327095 (Phylotype IV/ biovar 2A)	28 C°	*qBWR-1a*	S map	1	74.6–75.7	74.7	4.91	15.1
		qBWR-7	R map	7	25.2–26.3	25.3	4.39	15.3
		qBWR-11	S map	11	32.6–33.6	32.6	3.94	12.4

[1)] Detected by a permutation test (1,000 permutations) at a 0.01 level

The resistance level is represented as the disease index (DI) measured 20 d after inoculation using a 0–4 scale based on the extent of stem wilting: 0 (no symptoms), 1 (up to 25% stem wilting), 2 (26–50%), 3 (51–75%), and 4 (76–100%) (Habe 2018).

QTL analysis

A genetic map (Habe et al. 2019) was previously constructed using single-nucleotide polymorphism (SNP) markers. Since both diploid parents were highly heterozygous, the segregating population was considered a two-way pseudo testcross population (Grattapaglia and Sederoff 1994) and the parental maps were constructed. For RP, 1,476 heterozygous SNP loci were mapped, while for SP, 2,663 heterozygous SNP loci were mapped on 12 chromosomes (Supplemental Table 1). QTL Cartographer version 2.5 (Wang et al. 2005) was used to perform composite interval mapping (CIM; Zeng 1994), which was specifically designed to reduce background noise that can affect QTL detection; CIM was performed on a backcross design by regarding the F_1 population as a backcross population. Parameters of the analysis were set for model 6 with a window size of 2 cM and 0.05 probabilities for “into” and “out”. A LOD threshold for QTL detection was obtained via permutation tests using 1,000 repetitions to control for a genome-wide error rate of 1%. Since the distributions of DIs from the resistance tests using MAFF327001 at 28°C and MAFF327095 at 24°C were slightly distorted from normal distributions, an additional QTL analysis was performed on all data using an R/qtl package (Broman et al. 2003) of R software (R Core Team 2017) which performs nonparametric QTL mapping. The function “scanone” with model=“np” and step = 1 cM was used for nonparametric interval mapping, which is an extension of the Kruskal–Wallis test (Kruskal and Wallis 1952; Kruglyak and Lander 1995). A 5% LOD score threshold was determined using a permutation test (1,000 permutations). The interval estimate of genetic factor location was calculated using the “lodint” function, which computes the interval position corresponding to 1.0-LOD support intervals; the “expandtomarkers” argument determines the nearest flanking markers of the interval’s higher limits. QTL analyses were performed separately for each of the two parental linkage maps. Linkage maps and QTL positions were drawn using MapChart 2.30 (Voorrips 2002).

Statistical analysis

All statistical analyses, excluding QTL analysis, were performed using Rcmdr package (Fox 2005) and EZR package (Kanda 2013) of R version 3.3.3. (R Core Team 2017). Phenotypic correlations between variables were estimated using the Spearman’s rank coefficient for each trial. The Mann–Whitney U test was performed to analyze the mean DIs of the F_1 population on the allele differences of the markers at the nearest locus of each QTL.

Results

Evaluation of BW resistance

The 94 plants in the F_1 population were evaluated under six treatments: three strains (phylotype I/biovar 4, phylotype IV/biovar 2A, and phylotype I/biovar 3) at two incubation temperatures (24°C or 28°C). The DIs of RP 10-03-30 varied from 0.00–0.73, while those of SP F_1-1 ranged from 2.00–3.47, indicating a clear difference between the parents in all treatments (Fig. 1). The DIs of F_1 plants varied consistently between susceptible and resistant plants in all treatments, with the mean DIs ranging from 1.21–2.88 (Fig. 1), and were all positively correlated between treatments (r = 0.25–0.61, P < 0.05)(Supplemental Table 2). Incubation at 28°C was associated with

relatively higher DIs for all strains, and phenotypes with higher and lower DIs than those of SP and RP, respectively (transgressive segregation) were observed in all treatments. Particularly, the DIs against the strain MAFF327142 (phylotype I/biovar 4) changed drastically between temperatures: the distribution was skewed toward relatively lower DIs at 24°C, whereas its was skewed toward relatively higher DIs at 28°C. Thus, the resistance in the F_1 population against MAFF327142 varied greatly depending on the incubation temperature.

QTL detection

Since the F_1 population exhibited both normal and skewed distributions at different treatments, CIM and nonparametric interval mapping were performed for the DIs of the F_1 population using the RP map consisting of 1,476 SNP markers and the SP map consisting of 2,663 SNP markers. The LOD thresholds were determined using permutation tests with 1000 repetitions for the 1% significance level in CIM and 5% significance level in nonparametric interval mapping analysis. The CIM analysis identified ten QTLs on seven chromosomes (*qBWR-1a*, *qBWR-1b*, *qBWR-3*, *qBWR-5*, *qBWR-6a*, *qBWR-6b*, *qBWR-7*, *qBWR-10a*, *qBWR-10b*, and *qBWR-11*) (Table 1). Nonparametric interval mapping analysis revealed four QTLs on three chromosomes (*qBWR-3*, *qBWR-6a*, *qBWR-6b*, and *qBWR-7*): three of them were detected in the same treatments via the two analyses, whereas *qBWR*-3 was detected against MAFF327001 (phylotype I/biovar 4) at 28°C via CIM analysis and against MAFF327095 (phylotype IV/biovar 2A) at 28°C via nonparametric interval mapping analysis (Table 2). The locations of these QTLs are schematically shown in Fig. 2.

Table 2
Genetic factors detected by a nonparametric QTL mapping method for the resistance to the *R. solanacearum* species complex in the F_1 population

BW strain	Temperature	QTL[1)]	Detected map	Chr	Position (cM)[2)]	Position of maximum LOD (cM)	Maximum LOD score
MAFF327142 (Phylotype I/ biovar 3)	24 C°	*qBWR-6b*	R map	6	10.8–33.6	28.0	8.66
	28 C°	*qBWR-6b*	R map	6	10.8–42.0	21.7	3.42
		qBWR-6a	S map	6	0.0–26.4	2.0	2.59
MAFF327001 (Phylotype I/biovar 4)	24 C°	*qBWR-7*	R map	7	12.2–31.1	27.8	4.82
	28 C°	*qBWR-7*	R map	7	12.2–65.5	28.9	3.17
MAFF327095 (Phylotype IV/biovar 2A)	28 C°	*qBWR-3*	S map	3	5.4–36.4	18.8	2.91

[1)]Detected by a permutation test (1,000 permutations) at a 0.05 level

[2)]Positions were indicated by the 1.0-LOD interval

Function of the detected QTLs

The mean DIs for two genotypes (AA or AB since the population was treated as a pseudo testcross population) in the nearest SNP locus to the QTL were compared (Table 3). All QTLs showed significant resistance effects, although to varying degrees, on at least one strain. *qBWR-6b*, located at 21.7 cM in chromosome 6, considerably contributed to the resistance to MAFF327142 (phylotype I/biovar 3) alone at both 24°C and 28°C (explaining 40.5% and 17.6% of the variances, respectively). *qBWR-6a* and *qBWR-10b* contributed to the resistance against this strain at 28°C and 24°C, respectively. *qBWR-3* located at 15.0 cM in chromosome 3 considerably contributed to resistance against MAFF327142 at 24°C, while *qBWR-7*, located at 25.3 cM in chromosome 7, contributed to resistance against this strain at 28°C. Furthermore, *qBWR-3* and *qBWR-7* conferred resistance against MAFF327001 (phylotype I/biovar 4) and MAFF327095 (phylotype IV/biovar 2A) at both temperatures. The other five QTLs *qBWR-1a*, *qBWR-1b*, *qBWR-5*, *qBWR-10a*, and *qBWR-11* conferred resistance to a minor extent and were more effective at 28°C than at 24°C (Table 3).

Table 3
Mean DIs in BW resistant vs susceptible genotypes

QTL	SNP[1)]	MAFF327142 (Phylotype I/biovar 3)		MAFF327001 (Phylotype I/biovar 4)		MAFF327095 (Phylotype IV/biovar 2A)	
		24 C°	28 C°	24 C°	28 C°	24 C°	28 C°
qBWR-1a	c2_4943	1.25 vs 1.47	2.67 vs 3.07*	1.29 vs 1.40	1.53 vs 1.71	1.40 vs 1.46	1.69 vs 2.29**
qBWR-1b	c2_37816	1.19 vs 1.46	2.64 vs 3.05*	1.30 vs 1.41	1.51vs 1.71	1.41 vs 1.45	1.68 vs 2.31**
qBWR-3	c2_50637	1.10 vs 1.75**	2.76 vs 3.05	1.15 vs 1.64**	1.45 vs 1.88**	1.23 vs 1.74**	1.80 vs 2.32**
qBWR-5	c2_10291	1.15 vs 1.47	2.83 vs 2.91	1.26 vs 1.44	1.36 vs 1.82**	1.39 vs 1.49	1.90 vs 2.12
qBWR-6a	c2_55554	0.98 vs 1.63*	2.48 vs 3.15***	1.29 vs 1.39	1.45 vs 1.74*	1.26 vs 1.55	1.89 vs 2.08
qBWR-6b	c1_12696	0.73 vs 2.19***	2.53 vs 3.31***	1.32 vs 1.38	1.59 vs 1.68	1.46 vs 1.39	2.01 vs 1.99
qBWR-7	c2_4555	1.11 vs 1.57	2.56 vs 3.16**	0.98 vs 1.73***	1.31 vs 1.90***	1.23 vs 1.65**	1.74 vs 2.29**
qBWR-10a	c2_32779	1.27 vs 1.45	2.72 vs 3.05	1.24 vs 1.46	1.45 vs 1.80*	1.38 vs 1.49	2.00 vs 2.03
qBWR-10b	c2_22699	0.90 vs 1.70***	2.75 vs 2.96	1.32 vs 1.40	1.51 vs 1.71	1.41 vs 1.45	1.76 vs 2.24*
qBWR-11	c1_7668	1.29 vs 1.54	2.77 vs 3.22*	1.27 vs 1.61	1.53 vs 1.93*	1.32 vs 1.77*	1.88 vs 2.47**

Significance levels between resistant and susceptible genotypes were tested by Mann-Whitney U-test; *0.05, **0.01, ***0.001

[1)]SNP identity was given without the prefixed identity “solcap_snp_”

Discussion

Polygenic segregation of BW resistance in the hybrid population

BW resistance is controlled by multiple genes in potato plants (Elphinstone 1994; Rowe and Sequeira 1970; Sequeira 1979) and is greatly influenced by environmental conditions such as temperature and soil moisture (Tung et al. 1990a, b). Different strains show resistance to different extents (French and De Lindo 1982; Katayama and Kimura 1984; Tung et al. 1990a; Suga et al. 2013). Thus, the resistance was evaluated against three strains using an *in vitro* assay method (Habe 2018) under controlled environmental conditions at 24°C and 28°C. The RP and SP plants showed stable resistance and susceptibility against all the strains used, including phylotype I/biovar 3, phylotype I/biovar 4, and phylotype IV/biovar 2A. The resistance levels in the hybrid population varied consistently, confirming that the resistance was polygenically controlled, and the resistance was positively correlated among all six treatments, indicating that the pathogenicity was similar between phylotypes I and IV in potato plants. This was in agreement with previous findings that indicate no difference in virulence between phylotypes I and IV and between phylotypes II and III in potato cultivars (Habe et al. 2016; Sharma et al. 2021). Although Suga et al. (2013) reported that phylotype IV is more virulent than phylotype I, the classification of phylotypes may not correlate with the degree of virulence as suggested for tomato, eggplant, and pepper plants (Lebeau et al. 2011).

Segregation of multiple resistance QTLs in the hybrid population

CIM and non-parametric QTL mapping were performed to evaluate BW resistance using a hybrid population, which identified five major QTLs (*qBWR-3*, *qBWR-6a*, *qBWR-6b*, *qBWR-7*, and *qBWR-10b*) and five minor QTLs (*qBWR-1a*, *qBWR-1b*, *qBWR-5*, *qBWR-10a*, and *qBWR-11*). Only QTLs conferring heterozygous resistance in either one of the parents could be segregated and mapped in the population. Thus, the ten QTLs were likely the minimal ones that could be detected using this population. The combined segregation resulted in transgressive segregation, where hybrid plants with higher levels of resistance than that in RP and those with lower levels of susceptibility than that in SP were obtained. The resistance-related QTL alleles were derived from both parents.

Resistance specificity to strains and temperatures

We found strain-specific and temperature-dependent QTLs (*qBWR-6a*, *qBWR-6b*, and *qBWR-10b*) and strain-non-specific and broad-spectrum resistance QTLs (*qBWR-3* and *qBWR-7*). *qBWR-6a*, *qBWR-6b*, and *qBWR-10b* considerably contributed to the resistance to MAFF327142 (phylotype I/biovar 3), in which the latter two were more effective at 24°C. The distributions of the DIs in the F_1 population were skewed toward relatively lower DIs at 24°C and toward higher DIs at 28°C, which was likely due to the effect of *qBWR-6b* (Fig. 1ab). *qBWR-6b* was considered to be derived from RP 10-03-30, a haploid clone of Saikai 35 (Habe et al. 2019), which was originally derived from *S. phureja* (Mori et al. 2012). *S. phureja* is a well-known source of BW-resistant factors, and the resistance is strain-specific and sensitive to high temperatures (Sequeira and Rowe 1969; Sequeira 1979; Ciampi and Sequeira 1980; French and De Lindo 1982). The strain-specific resistance of *S. phureja* appeared to be simply inherited in few cases (Elphinstone 1994). Therefore, we suggest that *qBWR-6b* was derived from *S. phureja* and functions as a simply-inherited, major QTL at low temperatures. *qBWR-3* and *qBWR-7* showed stable resistance to all strains at low and high temperatures, irrespective of different phylotypes and biovars. These QTLs may be effective under diverse environmental conditions and highly desired in breeding BW-resistant cultivars.

Reliability of the BW resistance QTLs

Chen et al. (2013) identified *S. chacoense*-derived BW resistance QTLs against the race 1/biovar 3 strain on chromosomes 2 and 9. The SP used in our study was F_1-1, an interspecific hybrid between *S. chacoense* and *S. phureja* (Hosaka and Hanneman 1998). However, we did not identify any QTLs on chromosomes 2 and 9, indicating that the source of resistance factors for all the QTLs detected in our study was *S. phureja*. In our previous study using the same F_1 population and the same inoculum (MAFF327001, phylotype I/ biovar 4) at 28°C, five QTLs were identified on chromosomes 1, 3, 7, 10, and 11 (Habe et al. 2019). When their locations were compared, the previously identified QTLs *qBWR-1*, *qBWR-2*, *qBWR-3*, *qBWR-4*, and *qBWR-5* correspond to the QTLs identified in the present study: *qBWR-1b*, *qBWR-3*, *qBWR-7*, *qBWR-10a*, and *qBWR-11*, respectively. The QTL *qBWR-5* on chromosome 5 which showed a minor contribution to resistance was newly found, and *qBWR-11* was not significant in this study (Table 1). Repeated resistance assays may increase or decrease certain genetic variances, affecting significance levels of the QTLs. Here, difficulty in the BW resistance evaluation was featured again, and importance of the major-effect QTLs is emphasized.

Universal resistance QTLs

BW resistance QTLs have been identified in chromosomes 3, 4, 6, 8, 10, 11, and 12 in tomato plants (Thoquet et al. 1996a, 1996b; Mangin et al. 1999; Carmeille et al. 2006; Wang et al. 2000, 2013) and in chromosomes 1, 2, 3, 4, 5, 6, 7, 8, and 9 in eggplants (Mimura et al. 2012; Lebeau et al. 2013; Salgon et al. 2017, 2018). Comparison of the physical location in each chromosome indicates that the strain-specific resistance QTL *qBWR-6b* is likely to be colocalized with tomato QTL (*Bwr-6*) and eggplant QTL (*ERPR6*) on chromosome 6 (Fig. 3b). However, both *Bwr-6* and *ERPR6* confer resistance against phylotypes I and II (Carmeille et al. 2006; Wang et al. 2013; Salgon et al. 2018; Shin et al. 2020), whereas the resistance of *qBWR-6b* is limited to phylotype I/biovar 3. The mapping position of *Bwr-6* slightly varies depending on different inoculums and field conditions (Wang et al. 2013), which was similarly observed for *ERPR6* (Salgon et al. 2018). These findings indicate that strain-specific single-locus resistance genes are clustered on the same chromosome (Meyers et al. 1998; Andolfo et al. 2013), which superficially made *Bwr-6* and *ERPR6* broad-spectrum resistance genes (Salgon et al. 2018). For *Bwr-6* in tomato, 18 candidate genes have been proposed (Kim et al. 2018; Shin et al. 2020; Abebe et al. 2020).

A tomato-derived QTL (*Bwr-3*) and two eggplant-derived QTLs (*ERPR3a* and *ERPR3b*) have been reported on chromosome 3 (Thoquet et al. 1996b; Carmeille et al. 2006; Wang et al. 2013; Salgon et al. 2018). *Bwr-3* and *ERPR3b* are colocalized and may include the same locus (Salgon et al. 2018), while the potato-derived QTL *qBWR-3* is likely colocalized with *ERPR3a* (Fig. 3a). Like *qBWR-3*, *ERPR3a* is a strain-non-specific, broad-spectrum QTL (Salgon et al. 2018). The nearest SNP locus to *qBWR-3* (solcap_snp_c2_50637) is located in the receptor-like kinase gene (PGSC0003DMG400016685). This gene may represent one of candidate genes for BW resistance because a leucine-rich repeat receptor-like kinase gene (*ERECTA*) is involved in BW resistance in *Arabidopsis thaliana* (Godiard et al. 2003).

The broad-spectrum resistance QTL *qBWR-7* was detected in a span between 12.2 and 27.2 cM or between 10.9 and 39.2 Mb near the centromere, where recombination is less likely to occur, and comprised 23 SNP loci at the peak position (25.3 cM) (Table 1). The long arm of chromosome 7 in potato plants harbors a resistance gene hotspot containing *Rpi1* and *Rpi2* against *Phytophthora infestans* and *Gro1-4* against *Globodera rostochiensis* (Ballvora et al. 1995; Kuhl et al. 2001; Paal et al. 2004; Ruggieri et al. 2014; Yan et al. 2017); however, *qBWR-7* is considered to be excluded in this hot spot. Since the effect of resistance of *qBWR-7* is slightly higher than that of *qBWR-3*, additional fine mapping is desired to determine the accurate location and to develop molecular markers.

Conclusion

RSSC strains have spread worldwide and show a wide host range (Hayward 1985; Peeters et al. 2013). Potatoes are infected by all four phylotypes of this species complex. Therefore, BW-resistant varieties are sought after that show resistance to these four phylotypes. The phylotype-specific resistance has been reported in *S. phureja* (Suga et al. 2013), which emphasizes the need for phylotype-specific breeding (Horita et al. 2014). However, in tomato and eggplant, both phylotype-specific and non-specific, broad-spectrum resistance QTLs have been identified. We identified five major and five minor resistance QTLs, which included both strain-specific and broad-spectrum resistance QTLs. The major QTLs *qBWR-3* and *qBWR-7* showed stable resistance against *R. pseudosolanacearum* (phylotype I) and *R. syzygii* (phylotype IV), which are major phylotypes in Asia (Fegan and Prior 2005; Wicker et al. 2012). *qBWR-6b* is a strain-specific major resistance QTL against phylotype I/biovar 3 and can be effectively used in relatively cool area because the resistance conferred was more effective at a relatively lower temperature. Therefore, we suggest that broad-spectrum QTLs and strain-specific QTLs can be combined to develop the most efficient BW-resistant cultivars in particular areas.

Declarations

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Author Contributions

All authors contributed to the study design. Material preparation, data collection and analysis were performed by Ippei Habe. The first draft of the manuscript was written by Ippei Habe and all authors commented on previous versions of the manuscript. All authors approved the final manuscript.

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Ethics approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

Competing Interests

The authors declare no competing interests.

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Figures

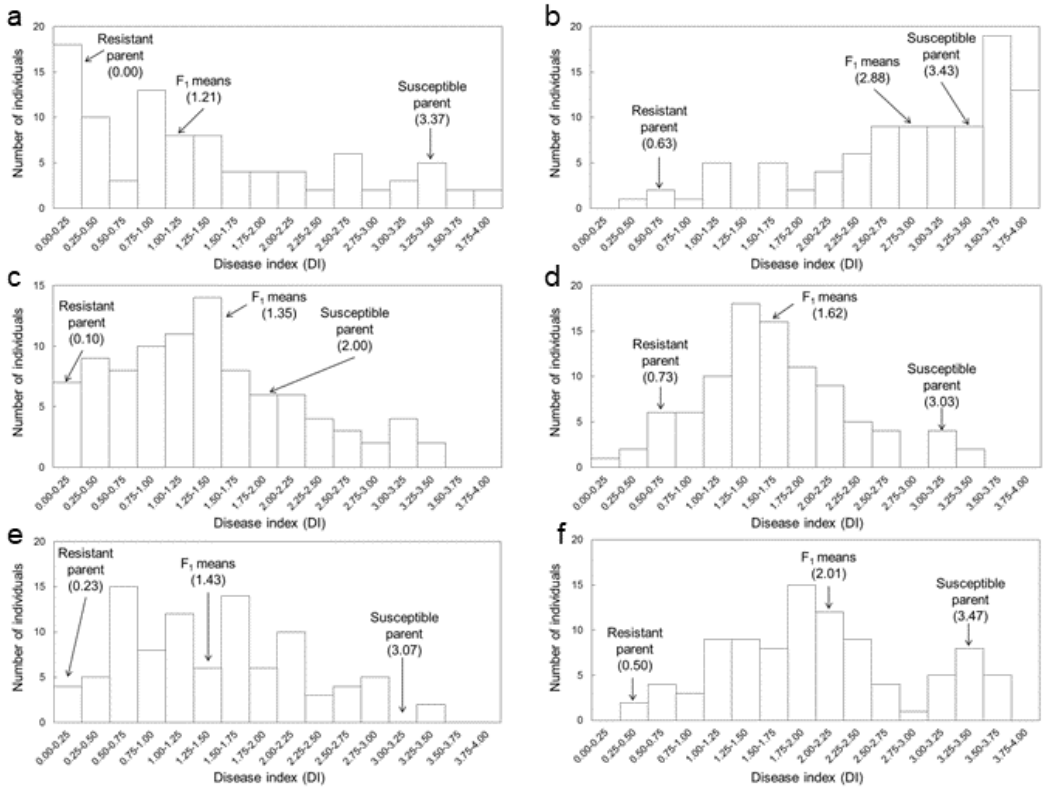

Figure 1

The DIs in the F_1 population inoculated with *Ralstonia solanacearum* species complex strains MAFF327142 (phylotype I/biovar 3) (**a, b**), MAFF327001 (phylotype I/biovar 4) (**c, d**), or MAFF327095 (phylotype IV/biovar 2A) (**e, f**) and incubated at 24 ℃ (**a, c, e**) or 28 ℃ (**b, d, f**). DI, disease index

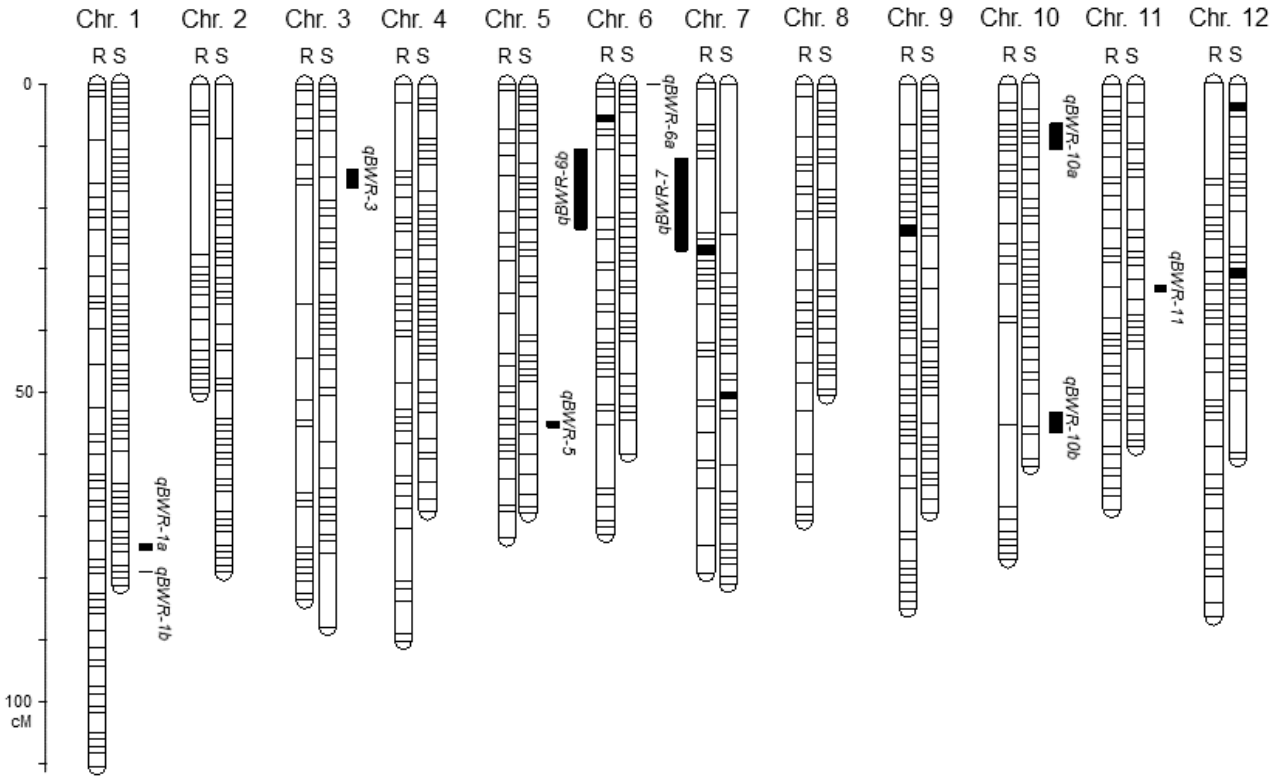

Figure 2

Locations of the BW resistance QTLs on SNP-based genetic maps for the resistant parent chromosomes (R) and the susceptible parent chromosomes (S). QTL-position bars are shown on the genetic map based on the results of the Composite interval mapping (Table 1). Lines on chromosomes indicate loci on the genetic maps. BW, bacteria wilting; QTL, quantitative trait locus; SNP, single-nucleotide polymorphism

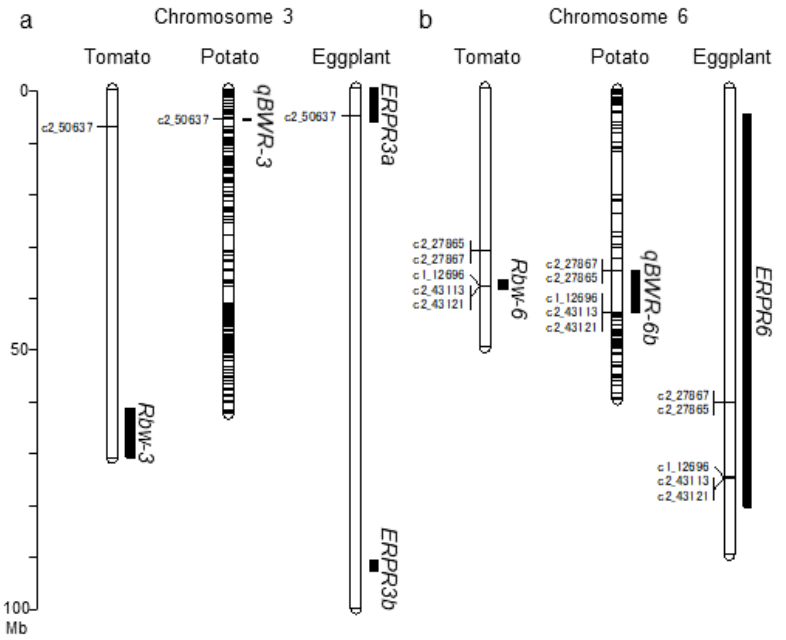

Figure 3

Comparisons of BW resistance QTLs among potato, tomato, and eggplant on chromosomes 3 (**a**) and 6 (**b**). SNP identity was given without the prefixed identity "solcap_snp_". The physical positions of SNPs in the *qBWR-3* and *qBWR-6b* regions in CIM analysis are presented in the potato DM v4.03 genome (Sharma et al. 2013), the tomato SL2.50 genome (https://solgenomics.net), and the eggplant HQ-1315 genome (Wei et al. 2020) using the BLASTN program in the Galaxy/NAAC (https://galaxy.dna.affrc.go.jp/) (Zhang et al. 2000), with a cut-off value of 1×10^{-15}. The physical locations of *Bwr-3*, *Bwr-6*, *ERPR3a*, *ERPR3b*, and *ERPR6* are shown based on Salgon et al. (2018). BW, bacterial wilt; QTL, quantitative trait locus; SNP, single-nucleotide polymorphism

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