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Novel QTL associated with *Rhizoctonia solani* Kühn resistance identified in two table beet × sugar beet $F_{2:3}$ populations using a new table beet reference genome

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Abstract

The necrotrophic fungus *Rhizoctonia solani* Kühn is a major concern for table beet (*Beta vulgaris* L. ssp. *vulgaris*) producers across the United States causing upward of 75% losses in severe instances. Thus far, there have been minimal efforts to incorporate host resistance to *R. solani* in table beet germplasm. To investigate the genetic control of *R. solani* resistance in table beet, we developed two mapping populations. Parents of the two populations were a *Rhizoctonia*-susceptible table beet inbred W357A and a resistant sugar beet germplasm FC709-2 (sugar beet resistance population, SBRP) and a *Rhizoctonia*-resistant table beet inbred W364B and a susceptible sugar beet inbred FC901/C817 (table beet resistance population, TBRP). In Spring 2020, $F_{2:3}$ families were evaluated for response to artificial inoculation with *R. solani* AG 2-2 IIIB isolate R1 in replicated greenhouse experiments. This work also represents the first use of the W357B table beet reference genome, utilized here to align genotyping-by-sequencing reads to identify polymorphic markers. Using interval linkage mapping, we identified one quantitative trait locus (QTL) in each of the two populations, each accounting for 30% of the phenotypic variation. The QTL in both the SBRP and TBRP were found on chromosome 2 and contained several putative resistance genes in annotations of the *Beta vulgaris* and *Arabidopsis thaliana* genomes. This is the first report of a QTL on chromosome 2 for resistance to *R. solani* in *B. vulgaris* ssp. *vulgaris* and the first identification of QTL for disease resistance in table beet. The newly developed table beet reference genome and markers identified in this study may be of value for marker-assisted selection in breeding for resistance to *R. solani* in both sugar beet and table beet breeding programs.

Abbreviations: AG, Anastamosis group; CMS, Cytoplasmic male sterility; DNA, Deoxyribonucleic acid; GBS, Genotyping by sequencing; LOD, logarithm of the odds; MAF, Minor allele frequency; MAS, Marker assisted selection; QTL, Quantitative trait locus or loci; RSB, Resistant sugar beet; SBRP, Sugar beet resistance population; SG, Subgroup; SNP, Single nucleotide polymorphism; SSB, Susceptible sugar beet; TBRP, Table beet resistance population; UWBRC, University of Wisconsin Bioinformatics Resource Center.

1 | INTRODUCTION

Rhizoctonia solani Kühn attacks table beets (*Beta vulgaris* L. ssp. *vulgaris*) at both the seedling and mature stages (Sneh et al., 1996). Disease caused by *R. solani* in the adult stages is referred to as Rhizoctonia root and crown rot, or sometimes pocket rot (Pethybridge et al., 2018; Windels et al., 2009). Infection by the fungus results in lesions of dry black tissue that render roots unharvestable and lead to major losses during processing (Abawi et al., 1986; Natti, 1953; Pethybridge et al., 2018). *R. solani* has a wide host range, with 12 different anastomosis groups (AG) described according to their ability to fuse hyphae with one another (Ogoshi, 1985). Some of the AG are subdivided into subgroups (SG) based on pathogenic, biochemical, and genetic characteristics (Ogoshi, 1996). AG 2-2 causes root rot in beets (Naito et al., 1978), and within AG 2-2, SGs IIIB (Büttner et al., 2002; Strausbaugh et al., 2011; Watanabe & Matsuda, 1966) and IV (Bolton et al., 2010; Engelkes & Windels, 1996) are virulent on table beet and its close relative the sugar beet (also *Beta vulgaris* L. ssp. vulgaris). Different SGs within AG 2-2 has been found to cause disease on other typical crops in rotation with beets, including: maize (Ohkura et al., 2009; Strausbaugh et al., 2011; Sumner & Bell, 1982), snap bean, cucumber, southern pea, lima bean (Sumner & Bell, 1982), soybean (Engelkes & Windels, 1996; Sumner & Bell, 1982), pinto bean, broad bean, and navy bean (Engelkes & Windels, 1996).

R. solani forms survival structures known as sclerotia and is capable of surviving in the soil and plant debris saprophytically for many years (Abawi et al., 1986; Cubeta & Vilgalys, 1997). The wide host range of *R. solani* in combination with its ability to survive long periods in soil and organic material makes control of the fungus difficult. Disease management strategies include crop rotation with nonhost crops (Abawi et al., 1986; Hecker & Ruppel, 1976; Pethybridge et al., 2018), proper weed management (Harveson, 2003), seed treatments, in-season fungicide applications (Pethybridge et al., 2018), and cultural practices (Schneider et al., 1982).

Table beet has historically been grown in low-input cropping systems (Goldman & Navazio, 2003), where host resistance is critical for reducing losses, especially when managing challenging diseases like *Rhizoctonia*. Host resistance is also a cornerstone of pest management in organic cropping systems where controls are limited to cultivar selection and cultural practices (van Bruggen et al., 2016). Consumer demand and public policy concerns have increased the acreage of vegetable production with minimal or no synthetic pesticides (Goldman & Navazio, 2003), further driving the need for table beets resistant to *R. solani*. There are very few available cultivars with partial resistance to *R. solani* including 'Solo,' 'Shiraz,' 'Rubra,' 'Kestrel' (Pethybridge et al., 2018), and 'Pacemaker III' (Goldman, 1996; Pethybridge et al., 2018). In greenhouse screens conducted in 2018, Pacemaker III and Solo displayed mean diseased tissue percentages of 27.1% and 33.7%, respectively, compared to 6.6% in FC709-2, a highly resistant sugar beet germplasm (Wigg & Goldman, 2020). Given this lack of strong resistance, a major goal of our table beet breeding program is to incorporate disease resistance into inbred breeding lines.

Core Ideas

- Table beets resistant to *Rhizoctonia solani* have not been identified, but sugar beet has resistance.
- We used two populations developed from crosses between sugar beet and table beet and identified two new QTL.
- These QTL were both located on chromosome 2 in a new table beet reference genome.

Table beet is biennial, requiring two growing seasons to flower and produce seed (Ford-Lloyd, 1995; Goldman & Navazio, 2003). To shorten the time needed to go from seed to seed, our table beet breeding program utilizes both field and greenhouse environments (Goldman & Navazio, 2003). This biennial lifecycle of table beet increases the importance of efficient selection. An important technique that has improved the efficiency of selection in numerous crops is marker-assisted selection (MAS). Numerous studies have identified markers associated with traits in sugar beet including root elongation and glucose and fructose content (Stevanato et al., 2010), sucrose content and quality (Schneider et al., 2002), cytoplasmic male sterility (CMS) (Hjerdin-Panagopoulos et al., 2002; Honma et al., 2014; Moritani et al., 2013), post-winter bolting resistance (Pfeiffer et al., 2014), yield (Schwegler et al., 2014), and resistances to beet diseases including: Rhizomania (Beet Necrotic Yellow Vein Virus; BNYVV) (Barzen et al., 1997; Gidner et al., 2005; Grimmer et al., 2007a; Lein et al., 2007; Scholten et al., 1999), beet yellows virus (BYV) (Grimmer et al., 2008), powdery mildew (*Erysiphe polygoni* DC.) (Grimmer et al., 2007b; Janssen et al., 2003), Aphanomyces root rot (*Aphanomyces cochlioides* Drechsler) (Taguchi et al., 2009, 2010), root-knot nematode (*Meloidogyne* spp.) (Bakooie et al., 2015; Weiland & Yu, 2003), and Cercospora leaf spot (*Cercospora beticola* Sacc.) (Nilsson et al., 1999; Schäfer-Pregl et al., 1999; Setiawan et al., 2000; Taguchi et al., 2011). MAS has already been utilized in sugar beet to select for resistance to some diseases as well as for reproductive traits such as CMS (Moritani et al., 2013); however, use of the technology for host resistance has not yet been reported in table beet, and thus far, only a single marker–quantitative trait loci (QTL) association for geosmin concentration has been reported in table beet (Hanson et al., 2021).

Host resistance to *R. solani* has been identified in sugar beet, which is a major contributor to domestic sugar production (Holmquist et al., 2021; McGrath & Panella, 2019). Gaskill (1968) used mass and recurrent selection to increase resistance to *R. solani*, and many resistant sugar beet lines have since been released (Halloin et al., 2000; Hecker & Gaskill, 1972; Hecker & Ruppel, 1977a, 1985, 1988, 1991; Panella et al., 2015). Genetic studies done by Hecker & Ruppel (1975) showed that at least two loci, with two or three alleles, together with modifying genes, are responsible for resistance to *R. solani* in sugar beet and estimated a broad sense heritability for this trait of 0.65. Through reciprocal crosses, they also demonstrated a lack of maternal or male sterile cytoplasm (CMS) effects on Rhizoctonia infection (Hecker & Ruppel, 1976). It is presently not known if sources of resistance to *R. solani* in these studies are present in table beet. A later study by Lein et al. (2008) identified three QTL associated with *R. solani* resistance in a $F_{2:3}$ population developed from a cross between resistant and susceptible sugar beet parents. These loci on chromosomes 4, 5, and 7 were estimated to together explain 71% of the total phenotypic variation (Lein et al., 2008); however, the population size of 95 families may result in an over-estimation of the amount of variation explained.

Several linkage maps have been developed for sugar beet (Barzen et al., 1992, 1995; Pillen et al., 1992, 1993; Schondelmaier et al., 1995, 1996; Uphoff & Wricke, 1995; Wagner & Wricke, 1991; Wagner et al., 1992). In 2007, a genetic map developed from a sugar beet × table beet cross was used to help assemble a physical genome (McGrath et al., 2007). In 2014, the first reference genome sequence for sugar beet, RefBeet, was published (Dohm et al., 2014). Improvements in sequencing technologies have improved genome contiguity by using long-read sequencing (in particular, Pacific Biosciences) and longer-range scaffolding technologies (such as HiC). These improvements facilitated the development of a genome assembly for sugar beet inbred EL10 (McGrath et al., 2020). Further improvements to EL10.1 were incorporated and resulted in EL10.2 (McGrath et al., 2020). In some of his work with other *Beta* crops, J. M. McGrath also self-pollinated the table beet inbred, W357B, for several generations (Galewski & McGrath, 2020). W357B is a round-rooted inbred released by the University of Wisconsin table beet breeding program and has been widely used as a parent for commercial hybrid table beet seed production (Goldman, 1996). Following the additional inbreeding generations, K. M. Dorn developed a chromosome-scale assembly for W357B (Dorn, 2022). The EL10.2 and W357B reference assemblies facilitated the construction of linkage maps used in this study (Dorn, 2022; McGrath et al., 2020).

We adapted the sugar beet screening methods described by Hecker and Ruppel (1977b) to create a controlled environment screen for *R. solani* in table beet that was conducted in a greenhouse. This screen has since been used to evaluate commercial cultivars, inbreds, and Plant Introductions of table beet (Wigg & Goldman, 2020). The objective of this study was to utilize this screening method to identify regions of the table beet genome associated with resistance to *R. solani* and identify markers to be used for MAS in table beet breeding programs.

2 | MATERIALS AND METHODS

2.1 | Development of mapping populations

Two mapping populations were developed in this study. Parents of the first were the table beet inbred W357A and a *Rhizoctonia*-resistant sugar beet FC709-2 (Panella, 1999). Parents of the second were the table beet inbred W364B and a *Rhizoctonia*-susceptible sugar beet FC901/C817 (Gaskill et al., 1967). Hereafter, these populations will be referred to by their source of resistance: W357A × FC709-2 as the sugar beet resistance population (SBRP) and W364B × FC901/C817 as the table beet resistance population (TBRP). FC709-2 and FC901/C817 are used as resistant and susceptible controls, respectively, in sugar beet breeding programs, and we adopted their use as controls in our controlled environment screens (Fenwick et al., 2018; Gaskill et al., 1967; Panella, 1999; Panella & Hanson, 2001; Wigg & Goldman, 2020). W357A was expected to exhibit susceptibility to *R. solani*, while W364B has been observed to perform similarly to the resistant sugar beet control in pilot screens (Wigg & Goldman, 2020), hence its pairing with the susceptible sugar beet as parents of the TBRP. We made the initial crosses of the parents in the greenhouse in Spring 2017.

In our table beet breeding program, seed is planted in the field in late spring, and at the end of summer, plants are harvested, topped, and the roots stored in a cooler at 4 ˚C for approximately 12 weeks to vernalize. This vernalization period promotes the switch from vegetative to reproductive growth when roots are planted in the greenhouse in early December (Benjamin et al., 1997). Pollinations are conducted in the greenhouse in the late winter/early spring, and seed is produced and harvested in time to be planted by late spring.

F_1 seed was harvested and grown in the field at Arlington, Wisconsin, in Summer 2017. Since the seed parent in the TBRP was a self-fertile maintainer line, we selected progeny with root phenotypes intermediate between sugar beet and table beet for continuation, in order to avoid selecting products of self-pollination. All F_1 plants in the SBRP were guaranteed to be true hybrids with no further selection because the seed parent carried sterile cytoplasm and recessive alleles at the nuclear restorer locus and was therefore male sterile. In August, F_1 roots were harvested and vernalized. In Spring 2018, those plants were self-pollinated. F_2 seed for each

population was harvested from a single F_1 plant and planted in Summer 2018 at Arlington, Wisconsin. F_2 roots were then harvested and vernalized, and in Spring 2019, 174 F_2 plants were self-pollinated (90 and 84 plants from the SBRP and TBRP, respectively), with the seed collected from each plant representing a $F_{2:3}$ family. Six plants were chosen at random from each family in each of the 68 families from the SBRP and 79 families from the TBRP in our controlled environment disease screens.

2.2 | Disease screens

Controlled environment disease screens of the $F_{2:3}$ families were completed in Winter 2019 and Spring 2020 in the Walnut Street Greenhouses at the University of Wisconsin-Madison. Screens were completed in accordance with the protocol outlined by Wigg & Goldman (2020). Briefly, we planted seeds from each family and transplanted plants into pots so that each pot contained a single plant. A 3:1 mix (by volume) of silty loam compost soil collected from Arlington Agricultural Research Station (Arlington, WI) and soilless medium (MetroMix; Sun Gro Horticulture, Agawam, MA) was used in 2780 cm^3 plastic pots. Sixteen-hour day lengths were provided using high-pressure sodium supplemental lighting (1000 µmol at bench height). Temperatures were maintained between 25 ˚C and 30 ˚C in air-conditioned greenhouses with forced-air heating, providing a conducive environment for *R. solani* disease development (Parmeter, 1970). Beets were watered and fertilized as needed for optimum plant growth. A 400 mg L^{-1} solution of 20N-4.4P-16.6K fertilizer with micronutrients (Peters Professional Peat-Lite Special; ICL Specialty Fertilizers, Dublin, OH) was used. *Bacillus thuringiensis* ssp. *israelensis* applications (Gnatrol WDG; Valent Biosciences Corporation, Libertyville, IL) were applied as needed to manage fungus gnats.

Fungus gnats are endemic in the greenhouse environment. This, along with the added attraction of fungus from the inoculation of *R. solani* in our experiment (Cloyd, 2010), meant fungus gnats were present in our experiments and we included their presence/absence in our analysis. When disease symptoms other than those caused by *R. solani* were present, we included their presence/absence in our analysis and referred to these as "other diseases."

2.3 | Experimental design

The $F_{2:3}$ families within each population were arranged in a completely randomized design that was replicated twice in time, hereafter referred to as an experimental run. The majority of the $F_{2:3}$ families (65% and 53% of the families in the SBRP and TBRP, respectively) were replicated three times in each experimental run, with some exceptions based on seed germination and emergence. In general, each $F_{2:3}$ family was replicated six times by six single plants across the two experimental runs (81% and 80% of the families in the SBRP and TBRP, respectively). Plants of three of the four parents in these two populations—W364B, FC709-2, and FC901/C817—were included in the screens as comparisons to the $F_{2:3}$ families. Data for W357A were collected from screens prior to this experiment (Wigg, unpublished data).

2.4 | Inoculum

R. solani AG 2-2-IIIB isolate R1 was used to inoculate beet plants (Nagendran et al., 2009). This isolate was originally collected from a sugar beet field in Colorado in the 1960s. Assays at Michigan State University concluded that R1 is a very aggressive isolate on beets (L. Hanson, personal communication; Nagendran et al., 2009). Following the protocol described by Naito et al. (1993), R1 was grown on autoclaved barley grains and then stored at 4–7 ˚C until use. Immediately prior to artificial inoculations, inoculum was roughly ground using a coffee grinder (FreshGrind, Hamilton Beach Brands, Glen Allen, VA). Each plant was inoculated by displacing a small amount of growth medium adjacent to the root 2–3 cm below the soil surface and depositing 0.6 g or ~0.6 mL of ground inoculum (Hecker & Ruppel, 1977b). The medium was smoothed back over the inoculum and plants were gently watered following inoculations.

Plants were artificially inoculated 8 weeks after planting. Non-inoculated controls were included in each replication. Disease evaluations were completed 3 weeks after inoculations as described by Wigg and Goldman (2020). Internal and external disease ratings for root symptoms on a scale from 0–5 were assigned to each root as follows: 0 = 0% diseased tissue, root surface clean with no visible lesions; 1 = 1%–10% diseased tissue, superficial, scattered non-active lesions; 2 = 11%–30% of root affected; 3 = 31%–60% of root affected; 4 = 61%–99% of root blackened with rot extending into interior; and 5 = root 100% rotted and foliage dead or dying (adapted from Campbell et al., 2014; Ruppel et al., 1979). Both internal and external symptoms caused by *Rhizoctonia solani* may be important in table beet. Beet processors may be able to remove outer root tissue layers during canning operations and in such cases may be able to remove external symptoms of the disease. Internal tissue damage likely renders the root unusable for processing.

2.5 | Statistical analyses

To account for the nonlinearity of the visual rating scale, the data were normalized for linear regression by converting

the internal and external disease ratings for each plant into a diseased tissue percentage based on the following: 0 = 0%; 1 = 5.5%; 2 = 20.5%; 3 = 45.5%; 4 = 80%; and 5 = 100%. The diseased tissue percentages for each of the ratings were determined based on the mean value within that rating. For example, a rating of 3 ranges from 31% to 60% diseased tissue, so the mean value would be 45.5%. To give each root a single diseased tissue percentage, those percentages were then weighted as described by the following equation:

$$(External\ diseased\ tissue\ percentage \times 0.25) + (Internal\ diseased\ tissue\ percentage \times 0.75) = Weighted\ average\ diseased\ tissue\ percentage$$

This weighted average emphasizes the importance of internal root quality because it is of greater importance for processing beets (A. Bennett, personal communication). External lesions closer to the surface can more easily be peeled away in processing with minimal loss (M. Badtke, personal communication).

R v3.6.2 statistical software (R Core Team, 2019) was used for all analyses (R code available from authors by request). Parameters to include in multiple linear regression models were selected using the Akaike Information Criterion through the "stepAIC" function in the MASS package (Venables & Ripley, 2002). Replicate and experimental run were not significant; therefore, replicates and experimental runs were combined for analysis. Linear models included family, presence of fungus gnats, and presence of other diseases as fixed effects. Analysis of variance (ANOVA) was conducted using the weighted average diseased tissue percentages to evaluate the significance of differences in the level of resistance among the families. Differences among families in the presence of *R. solani* were evaluated with the non-inoculated controls excluded from the dataset.

Broad-sense heritabilities for weighted average diseased tissue percentage were estimated for each population, with a focus on the genotype term, using the following:

$$h^2 = \frac{\sigma_G^2}{\sigma_G^2 + \sigma_\varepsilon^2}$$

where σ^2_G and σ^2_ε are the variance components for genotype and residual terms, respectively. The genotype variance is the overall variance of the $F_{2:3}$ families over both experimental runs. Variance components were estimated using the "lmer" function in the lme4 package (Bates et al., 2015).

2.6 | Tissue collection and DNA extraction

Whole leaf tissue from F_2 plants was collected in the greenhouse in Spring 2019. The tissue was stored at −80 ˚C until lyophilization. Approximately 1 cm^2 disks of lyophilized tissue were collected in a microtube plate (Collection Microtubes, Qiagen, Germantown, Md.). Plates were submitted to the University of Wisconsin-Madison Biotechnology Center (UWBC) for DNA extraction and sequencing. UWBC completed genomic DNA extraction using the QIAGEN DNeasy mericon 96 QIAcube HT Kit (Qiagen, Germantown, Md.). DNA was quantified using the Quant-iT™ PicoGreen® dsDNA kit (Life Technologies, Grand Island, NY).

2.7 | Genotyping by sequencing

Beet GBS libraries were prepared according to Elshire et al. (2011) with minimal modification. Briefly, restriction enzymes NsiI and BfaI (New England Biolabs, Ipswich, Mass.) were used to digest DNA. These restriction enzymes were selected based on prior optimization in *B. vulgaris* ssp. *vulgaris* (James Speer, UW-Madison Biotechnology Center, personal communication). Barcoded adaptors amenable to Illumina sequencing were added to DNA by ligation using T4 ligase (New England Biolabs, Ipswich, Mass.). Ninety-six adapter-ligated samples were pooled and amplified to provide library quantities amenable for sequencing, and adapter dimers were removed by SPRI bead purification. Quality and quantity of the finished libraries were assessed using the Agilent TapeStation (Agilent Technologies, Inc., Santa Clara, Calif.) and Qubit® dsDNA HS Assay Kit (Life Technologies, Grand Island, N.Y.), respectively. Libraries were sequenced on a NovaSeq6000 S2 platform (Illumina, Inc., San Diego).

2.8 | Sequence data processing and reference genome development

Quality control, sequence alignment, and SNP calling were completed on GBS data by the University of Wisconsin Bioinformatics Resource Center (UWBRC). Adapters, low-quality bases, and primers were trimmed from reads to obtain a Phred score of 20 via the trimming software Skewer (Jiang et al., 2014). Reads too short to be used were also discarded. A total of 467,109,727 high-quality reads passed quality control, with an average of 3,273,420 reads per sample. Sequences had between 39% and 40% GC content. Sequence quality scores for samples had a mean Phred score of 35.

The Tassel v2 GBS Pipeline (Glaubitz et al., 2014) and Bowtie2 alignment software (Langmead & Salzberg, 2012) were used to align demultiplexed 64 bp forward reads to a draft assembly for table beet, W357B (Dorn, 2022), and sugar beet reference genome, EL10.2-P01197_ID_57232 (McGrath et al., 2020), for the SBRP and TBRP, respectively. U.S. Department of Agriculture Agricultural Research Service (USDA-ARS) researchers created a chromosome-scale assembly for table beet inbred W357B using PacBio Hifi and

DoveTail Omni-C sequencing (K.M. Dorn, https://zenodo.org/record/5911852#.Ypd87uzMI6A). The PacBio HiFi reads were assembled with hifiasm and then scaffolded with the DoveTail HiRise pipeline (K.M. Dorn, personal communication). The assembly contains nine pseudomolecules with a total size of 724 Mb (K.M. Dorn, personal communication). The individual W357B plant used for sequencing was confirmed to be highly homozygous via kmer analysis of Illumina data (K.M. Dorn, personal communication). McGrath et al. (2020) used a combination of short- and long-read sequencing, physical/optical maps, genetic maps, and Hi-C chromatin confirmation capture to create the EL10 sugar beet genome assembly. The 540 Mb annotated EL10.1 assembly has 24,255 predicted protein coding regions with a mean of 2,559 coding regions per chromosome. Inversions associated with the assembly process itself were resolved, and scaffold placement improved to create EL10.2 (McGrath et al., 2020). Alignment rates of 97.3% and 94.8% were obtained for short-reads derived from the SBRP and TBRP with the assemblies for W357B and EL10.2, respectively.

The Tassel v2 Discovery and Production SNP Caller system was used to detect 179k and 202k unfiltered variants in the SBRP and TBRP, respectively. Variants were recorded in a variant call format (VCF) file. bcftools then was used for VCF file processing (Danecek et al., 2021). VCF files were filtered to include biallelic sites and minor allele frequency (MAF) > = 0.05. Additional filtering included sites where at least 75% of samples had a depth of at least four reads. Linkage disequilibrium (LD) pruning was performed to include only sites with pairwise $r^2 < 0.99$ within a 100 kb window. The remaining 39k and 35k high-quality SNPs in the SBRP and TBRP, respectively, were then used to construct genetic maps.

2.9 | Linkage map construction and QTL analysis

R v3.6.2 (R Core Team, 2019), and in particular the MapRtools (0.23; Endelman, 2021) and R/QTL (1.48-1; Broman et al., 2003) packages were used for analyses (R code available from authors by request). Genotypes were recoded according to the parental genotypes for each population. Genetic maps were calculated using the marker ordering provided by the reference genome, and pairwise recombination frequencies were estimated by leveraging the marker encoding described above. MapRTools was used to estimate map distances using the Kosambi mapping function (Kosambi, 1943). 19-point multiple regression was utilized, that is, bins of markers containing up to 19 markers were used in the logarithm of the odds (LOD) score-weighted least-squares regression. Markers with significant deviation from an expected 1:2:1 segregation ratio were removed ($P < 0.1$). Haley–Knott regression was used to perform interval mapping using the scanone function of R/QTL to generate LOD profiles. LOD score thresholds were determined using 1000 permutations ($P < .05$).

TABLE 1 Relationships between scaffolds and chromosomes of sugar beet RefBeet 1.2.2 assembly (chromosomes), the draft assembly for table beet W357B (scaffolds), and sugar beet EL10.2 assembly (scaffolds). RefBeet 1.2.2 numbering is reported in the results of this study

RefBeet 1.2.2	W357B	EL10.2
1	4	4
2	3	8
3	9	7
4	7	3
5	6	2
6	5	1
7	1	6
8	8	5
9	2	9

The MUMmer4 system (Marçais et al., 2018) was used to align scaffolds from each population to the RefBeet 1.2.2 chromosome ordering as described by Dohm et al. (2014). The scaffold-to-chromosome alignments are shown in Table 1 and Figures S2 and S3. The alignments to the RefBeet 1.2.2 ordering are described hereafter. The CrossMap program was used to convert genome coordinates between the W357B, EL10.2, and RefBeet 1.2.2 genome assemblies (Zhao et al., 2013).

2.10 | Gene model projections from RefBeet 1.2.2 to W357B assembly

Annotations from the *Beta vulgaris* RefBeet 1.2.2 (https://ftp.ncbi.nlm.nih.gov/genomes/all/GCF/000/511/025/GCF_000511025.2_RefBeet-1.2.2/) assembly (GCF_000511025.2_RefBeet-1.2.2_genomic.gff) were transferred by sequence homology using the software package Liftoff v1.6.3 (https://github.com/agshumate/Liftoff) using default parameters. Genome annotations (Type = Gene) in the SBRP QTL region on W357B chromosome 2 (Scaffold 3) between coordinates 56,099,742 bp and 62,113,791 bp were further interrogated to identify potential candidate genes based on predicted function.

3 | RESULTS

3.1 | Disease screening

ANOVAs for each population showed main effects of family, presence of fungus gnats, and presence of other diseases to be

TABLE 2 Analysis of variance for the weighted average diseased tissue percentage of the 68 and 79 *Beta vulgaris* subsp. *vulgaris* $F_{2:3}$ families of the sugar beet resistance population (SBRP) and the table beet resistance population (TBRP), respectively, in response to inoculation with *Rhizoctonia solani* AG 2-2 isolate R1 in a greenhouse screen evaluated in 2020. Parents of the SBRP were the table beet inbred W357A and FC709-2, a *Rhizoctonia*-resistant sugar beet. Parents of the TBRP were the table beet inbred W364B and FC901/C817, a *Rhizoctonia*-susceptible sugar beet

Source	SBRP			TBRP		
	df	Mean squares	Significance	df	Mean squares	Significance
Family	67	556	***	78	310	***
Fungus gnats	1	28438	***	1	12271	***
Other diseases	1	6571	***	1	2761	***
Family: fungus gnats	56	127	NS	53	90	NS
Family: other diseases	40	187	*	72	166	NS
Fungus gnats: other diseases	1	140	NS	1	984	**
Family: fungus gnats: other diseases	6	268	NS	9	33	NS
Residuals	212	128		222	124	

NS, *,**,*** Nonsignificant or significant at $P \leq 0.05$, 0.01, or 0.001, respectively.

highly significant ($P < 0.001$) for weighted average diseased tissue percentage (Table 2). The interaction between family and presence of other diseases was significant in the SBRP ($P < 0.05$). In the TBRP, the interaction between presence of fungus gnats and presence of other diseases was significant ($P < 0.01$).

In both populations, most inoculated plants exhibited weighted average diseased tissue ranging from 5% to 40% (Figures 1 and 2) compared to the non-inoculated checks. Non-inoculated checks showed no diseased tissue (data not shown).

Significant differences for weighted average diseased tissue percentage were observed among families within each F_2 population and between the parents of each population. In both the SBRP and TBRP, weighted average diseased tissue percentage was continuously distributed among families and displayed transgressive segregation (Figures 1 and 2). In the SBRP, weighted average diseased tissue percentage per family ranged from 2.0% to 42.9% (Figure 1). The mean of weighted average diseased tissue percentage for families in the SBRP was 23.8% with a standard deviation of 9.7%. The resistant sugar beet parent had a weighted average diseased tissue percentage of 12.17%. In our pilot screens, the table beet parent in this population, W357A, had a weighted average diseased tissue percentage of ~24.2%. In the TBRP, weighted average diseased tissue percentage per family ranged from 7.2% to 44.8% (Figure 2). The weighted average diseased tissue percentage for families in the TBRP was 22.5% with a standard deviation of 7.92%. The susceptible sugar beet parent and W364B had a weighted average diseased tissue percentage of 30.4% and 17.6%, respectively. Heritabilities for weighted average diseased tissue percentage in the SBRP and TBRP were 0.87% and 0.12%, respectively (Table 3).

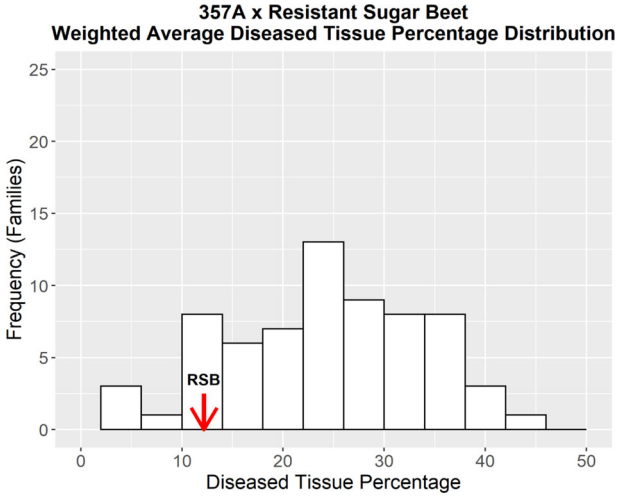

FIGURE 1 Distribution of family means for the weighted average diseased tissue percentage of the 68 *Beta vulgaris* subsp. *vulgaris* $F_{2:3}$ families of the sugar beet resistance population (SBRP) in response to inoculation with *Rhizoctonia solani* AG 2-2 isolate R1 in a greenhouse screen evaluated in 2020. Parents of the SBRP were the table beet inbred W357A and FC709-2, a *Rhizoctonia*-resistant sugar beet (RSB). RSB is annotated on the histogram. W357A was not included in this screen, and in pilot screens was assigned a 2.5 rating on a 0–7 scale, which translates to ≈24.2% weighted mean diseased tissue percentage in the current study (results not shown)

3.2 | Genetic linkage map

The Tassel v2 Discovery and Production SNP Caller system detected 179k and 202k unfiltered variants in the SBRP and TBRP, respectively. After filtering, 39K and 35k SNPs remained in the SBRP and TBRP, respectively. SNPs in the SBRP were ordered based on the W357B draft assembly.

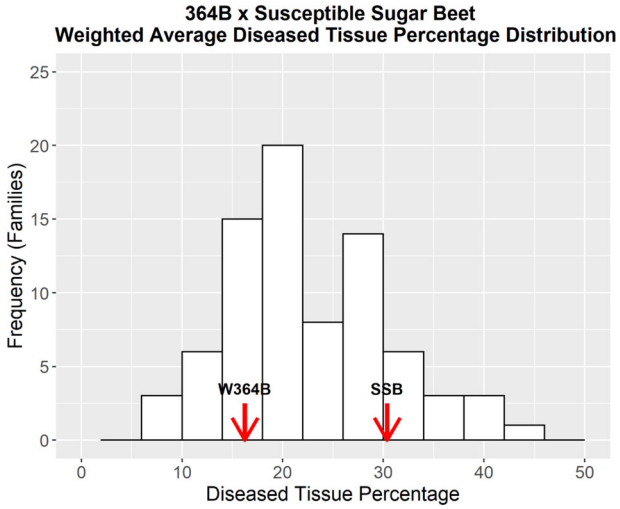

FIGURE 2 Distribution of family means for the weighted average diseased tissue percentage of the 79 *Beta vulgaris* subsp. *vulgaris* $F_{2:3}$ families of the table beet resistance population (TBRP) in response to inoculation with *Rhizoctonia solani* AG 2-2 isolate R1 in a greenhouse screen evaluated in 2020. Parents of the TBRP were the table beet inbred W364B and FC901/C817, a *Rhizoctonia* susceptible sugar beet (SSB), and are annotated on the histogram

TABLE 3 Variance components for weighted average diseased tissue percentage (WADTP) of the 68 and 79 *Beta vulgaris* subsp. *vulgaris* $F_{2:3}$ families of the sugar beet resistance population (SBRP) and the table beet resistance population (TBRP), respectively, in response to inoculation with *Rhizoctonia solani* AG 2-2 isolate R1 in a greenhouse screen evaluated in 2020

	SBRP WADTP	TBRP WADTP
Genotype (σ^2_G)	261.07	23.97
Error (σ^2_e)	40.72	169.72
Heritability (h^2), %	0.87	0.12

SNPs in the TBRP were ordered based on the EL10.2 sugar beet assembly (McGrath et al., 2020). SNPs were aligned to the nine table beet chromosomes to create a high-density linkage map containing 12,676 and 9,543 SNPs in the SBRP and TBRP, respectively. Scaffolds from each population were aligned to the chromosome ordering of RefBeet 1.2.2 (Dohm et al., 2014) using the MUMmer4 system (Marçais et al., 2018) (Table 1; Figure S4). Chromosomes reported in these results are the alignments with RefBeet 1.2.2.

In the SBRP, the number of markers per chromosome ranged from 968 on chromosome 2 to 1686 on chromosome 5. The nine chromosomes had an average of 1407 markers per chromosome. The average distance between markers ranged from 38,826 bp on chromosome 3 to 67,385 bp on chromosome 1. The overall average distance between markers across chromosomes was 51,089 bp.

In the TBRP, number of markers per chromosome ranged from 530 on chromosome 2 to 1420 on chromosome 3. The nine chromosomes had an average of 1058 markers per chromosome. The average distance between markers ranged from 40,193 bp on chromosome 3 to 106,444 bp on chromosome 2. The overall average distance between markers across chromosomes was 62,977 bp.

3.3 | QTL identification

Haley–Knott regression using the scanone function of r/QTL identified a QTL on RefBeet 1.2.2 chromosome 2 in both the SBRP and TBRP (Figures 3 and 4). The QTL on chromosome 2 was at position 60,853,362 bp in the SBRP and had an LOD score of 5.32 and Bayesian credible interval of 56,099,742 to 62,113,791 bp (Figure 5). The QTL identified in the TBRP on chromosome 2 at 31,756,598 bp had an LOD score of 6.31 with a Bayesian credible interval of 7,511,726 to 33,304,080 bp (Figure 6).

Dominance effects of the QTL on chromosome 2 in the TBRP were greater than those measured for the QTL on chromosome 2 of the SBRP (Figures 7 and 8). Additive and dominance parameters for the QTL were estimated in both populations (Table 4). The additive effect of the chromosome 2 QTL in the SBRP was 7.10. The dominance effect of the QTL in the SBRP was 6.42. The ratio of the additive to dominance effect was 0.90. The QTL in the TBRP had an additive effect of −5.16 and dominance effect of −4.86, resulting in an additive to dominance ratio of 0.94. The QTL on chromosome 2 in the SBRP explained 30.28% of the phenotypic variance, while 30.79% of phenotypic variance was explained by the QTL on chromosome 2 in the TBRP.

3.4 | Candidate gene prediction

Following genome position conversion using the CrossMap program (Zhao et al., 2013), the corresponding windows were searched for genes in RefBeet 1.2.2. and EL10.1. As the confidence interval for the QTL on chromosome 2 in the SBRP was considerably smaller than that of the QTL on chromosome 2 in the TBRP, we focused on identifying the candidate genes present within this genomic region (56,099,742–62,113,791 bp on W357 scaffold 3). Following genome position conversion through the CrossMap program (Zhao et al., 2013), the corresponding windows were searched for genes in RefBeet 1.2.2. (305,147–1,384,987 bp) and EL10.1 (48,728,559–54,645,150 bp).

The QTL on chromosome 2 in the SBRP contained a total of 95 genes in RefBeet 1.2.2. and 309 genes in EL10.1. Three of these genes in RefBeet contained putative leucine-rich repeat (LRR) motifs (Table 5). Within

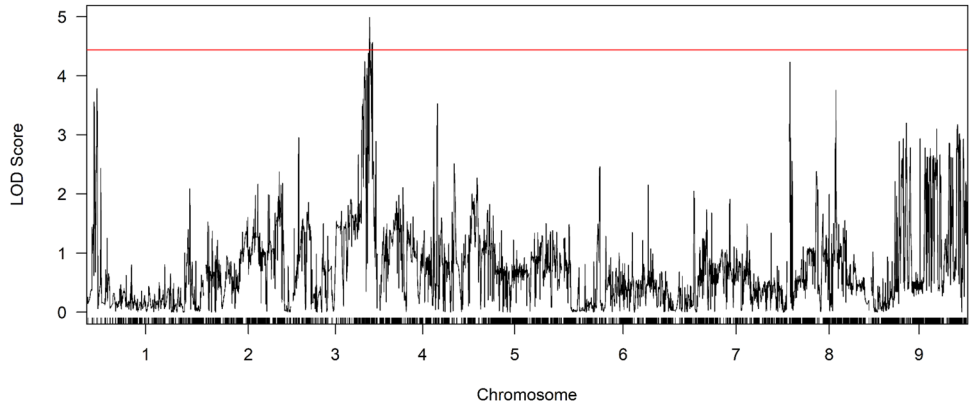

FIGURE 3 Logarithm of the odds (LOD) profile of the sugar beet resistance population (SBRP). Parents of the SBRP were the table beet inbred W357A and FC709-2, a *Rhizoctonia* resistant sugar beet (RSB). An LOD threshold of 4.44 was obtained through 1000 permutations. Scaffold 3 corresponds to chromosome 2

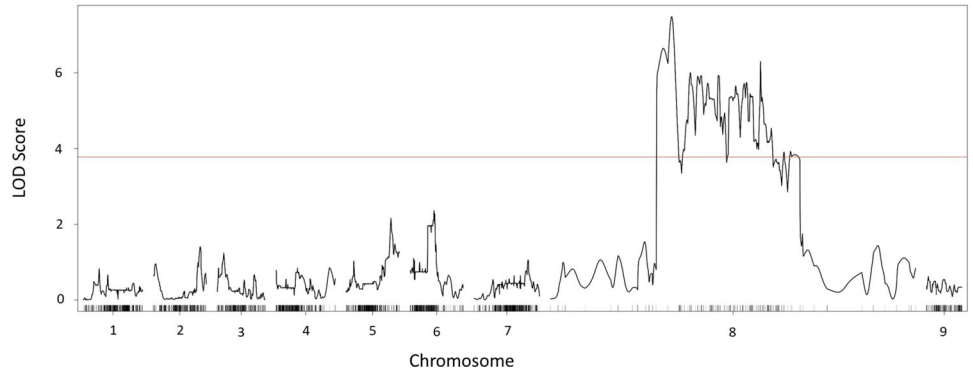

FIGURE 4 Logarithm of the odds (LOD) profile of the table beet resistance population (TBRP). Parents of the TBRP were the table beet inbred W364B and FC901/C817, a *Rhizoctonia* susceptible sugar beet (SSB). An LOD threshold of 3.69 was obtained through 1000 permutations. Scaffold 8 corresponds to chromosome 2

EL10.1, genes surrounding the QTL coded for polyphenol oxidase, pectinesterase, cystatin-C, a fungal trichothecene efflux pump, callose synthase, a toll-interleukin resistance (TIR) domain-containing protein, and interleukin-1 receptor-associated kinase 4 (IRAK4) (Table 5). We also examined the 522 gene models in the QTL found in SBRP using a BLAST search against *Arabidopsis thaliana*. Several *Arabidopsis* functional genes for disease resistance were associated with these QTL, including putative disease resistance proteins RGA3 and RGA4, isoform X1 and leaf rust disease resistance locus receptor-like kinases 1.4 and 2.4 isoform X1.

4 | DISCUSSION

Novel QTL associated with resistance *to R*. solani were identified in both the SBRP and TBRP. While both QTL may have utility in increasing resistance to *R. solani* in table beet populations, the resistance in the TBRP may be more readily introgressed. The resistant table beet parent, W364B, is already being used as a parent in hybrid table beet seed production. In contrast, the resistance exhibited in the SBRP is likely to introduce some degree of linkage drag from sugar beet. Table beet × sugar beet crosses often exhibit external russeting and increased crown size, both of which are not desirable in table beet markets. These traits are characteristic of sugar beet and can take upward of 10 generations to remove from table beet populations (Wang & Goldman, 1999). Therefore, unless flanking markers were utilized to precisely introgress the resistance from the SBRP, the resistance from the TBRP is likely to produce an acceptable product in fewer generations.

In addition to the genetic background of the QTL, size of the QTL must also be considered. The QTL on chromosome 2 reported in the SBRP is much smaller and can therefore more readily be used for MAS. In contrast, the QTL on chromosome 2 in the TBRP has a larger confidence interval. While the size of this QTL as reported here is certainly a function

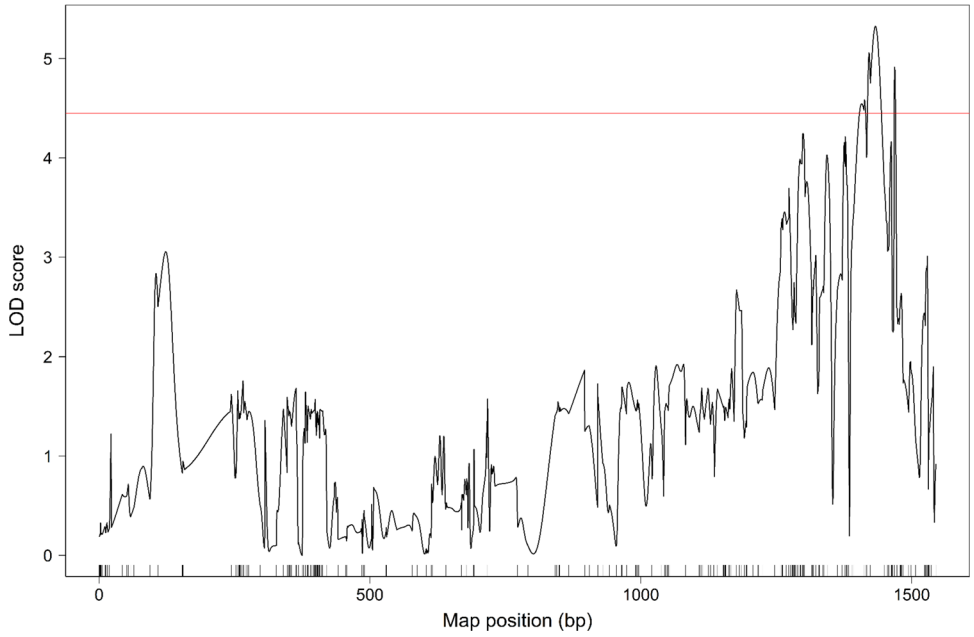

FIGURE 5 Logarithm of the odds (LOD) profile of chromosome 2 in the sugar beet resistance population (SBRP). Parents of the SBRP were the table beet inbred W357A and FC709-2, a *Rhizoctonia* resistant sugar beet (RSB). An LOD threshold of 4.44 was obtained through 1000 permutations

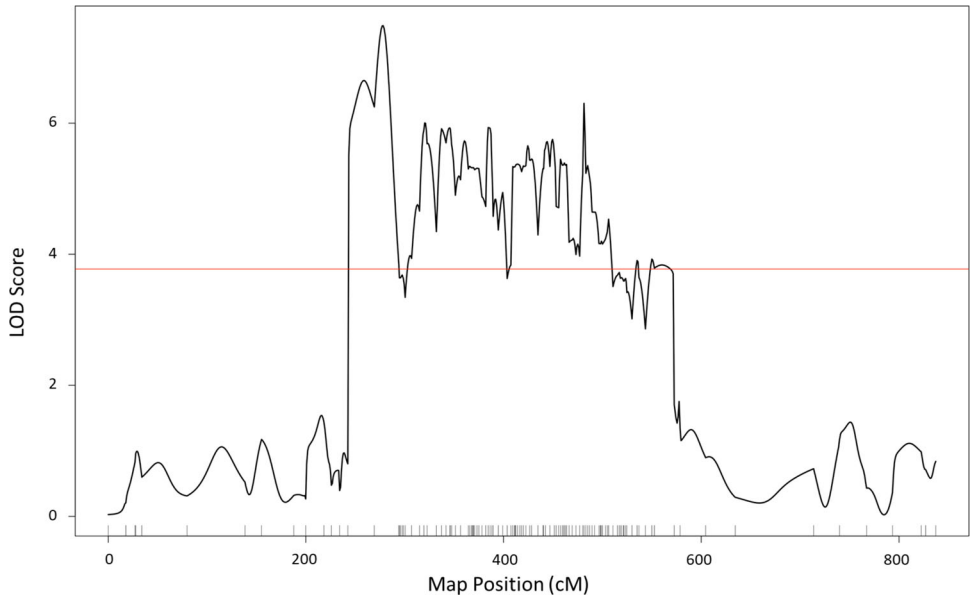

FIGURE 6 Logarithm of the odds (LOD) profile of chromosome 2 in the table beet resistance population (TBRP). Parents of the TBRP were the table beet inbred W364B and FC901/C817, a *Rhizoctonia* susceptible sugar beet (SSB). An LOD threshold of 3.69 was obtained through 1000 permutations

of the marker density and population size used in this study—and not necessarily a reflection of the true genetic size of the QTL—the immediate usefulness of this QTL will nonetheless be limited without additional fine mapping.

In the TBRP, a QTL was identified on chromosome 2 which also was found to have a large confidence interval. To investigate whether multiple QTL were present on chromosome 2, we assessed several linear models by fitting additional QTL within the confidence interval and dropping additional QTL from a linear model one at a time. Based on this analysis, we determined that only a single QTL was present in the TBRP ($P < 0.001$). Both populations used in this study were small

TABLE 4 Characterization of quantitative trait loci (QTL) for *Rhizoctonia* resistance in the sugar beet resistance population (SBRP) and the table beet resistance population (TBRP). Additive effects are here considered to be half the difference between the two homozygous genotypic classes, while dominance effects represent the value of the heterozygotic genotypic class, minus this additive effect

Population	Chromosome	QTL	Next marker	Bayesian credible interval (Mb)	Logarithm of the odds score	R^2%	Additive effect	Dominance effect	Level of dominance
SBRP	2	c3.loc1433	SCAFFOLD_3_60853362	56.1–62.1	5.32	30.28	7.10	6.42	0.90
TBRP	2	SCAFFOLD_8_31756598	SCAFFOLD_8_30917354	7.5–33.3	6.31	30.79	−5.16	−4.86	0.94

FIGURE 7 Effect plot for the quantitative trait loci on chromosome 2 of the sugar beet resistance population (SBRP). Parents of the SBRP were the table beet inbred W357A and FC709-2, a *Rhizoctonia* resistant sugar beet (RSB). The resistant sugar beet allele is represented by "A" and the susceptible table beet allele by "B"

relative to those typically used in linkage mapping studies. Smaller population sizes and mapping in the F_2 generation can lead to larger QTL sizes—compared to larger population sizes and mapping populations with more generations of selfing–since they both lead to relatively fewer recombination events. Populations such as recombinant inbred lines (RILs) that typically have undergone five to six selfing generations have more recombination events, leading to smaller haplotype blocks, increasing the resolution of QTL. Since both populations used in this study are comparable in size to one another, we did not expect one population to possess a QTL with a much smaller confidence interval than the other; however, this finding may be attributable to chance or residual heterozygosity in the families tested. Future studies may fine map these QTL using an increased population size, and possibly with increased number of self-pollinated generations. We are also aware that the sizes of both populations used in this study are relatively small, potentially leading to overestimation of QTL effects (Beavis, 1998). Nevertheless, the identification of these QTL should enable an opportunity for further fine mapping efforts in larger populations.

The results from this study revealed different QTL than those reported by Lein et al. (2008). In a sugar beet mapping population, they discovered three major QTL on chromosomes 4, 5, and 7, explaining 71% of phenotypic variation for resistance to *R. solani* in sugar beet (Lein et al., 2008). The differences in QTL location may be attributed to the different genetic backgrounds of the parents in these mapping populations. In the SBRP, parents were the table beet inbred W357A and FC709-2, a *Rhizoctonia*-resistant sugar beet. Parents of the TBRP were the table beet inbred W364B and FC901/C817, a *Rhizoctonia*-susceptible sugar beet. Both of

TABLE 5 Predicted candidate genes located within the confidence interval for the quantitative trait loci on chromosome 2 in the sugar beet resistance population

Reference assembly	Gene	Location	Protein
EL10.1	EL10Ac8g20134.1	49,610,675	PTHR31321:SF8–PECTINESTERASE 8-RELATED
EL10.1	EL10Ac8g20214.1	51,039,442	K13899–cystatin-C (CST3)
EL10.1	EL10Ac8g20220.1	51,147,986	PF00083//PF06609–sugar (and other) transporter (sugar tr)//fungal trichothecene efflux pump (TRI12) (TRI12)
EL10.1	EL10Ac8g20309.1	52,781,310	PTHR31008:SF2–TOLL-INTERLEUKIN-RESISTANCE (TIR) DOMAIN-CONTAINING PROTEIN
EL10.1	EL10Ac8g20317.1	52,962,326	K00422–polyphenol oxidase (E1.10.3.1)
EL10.1	EL10Ac8g20324.1	53,101,805	K00422–polyphenol oxidase (E1.10.3.1)
EL10.1	EL10Ac8g20326.1	53,147,827	K00422–polyphenol oxidase (E1.10.3.1)
EL10.1	EL10Ac8g20327.1	53,173,899	K00422–polyphenol oxidase (E1.10.3.1)
EL10.1	EL10Ac8g20344.1	53,474,535	K04733–interleukin-1 receptor-associated kinase 4 (IRAK4)
EL10.1	EL10Ac8g20382.1	54,128,645	PTHR12741//PTHR12741:SF23–LYST-INTERACTING PROTEIN LIP5 DOPAMINE RESPONSIVE PROTEIN DRG-1//CALLOSE SYNTHASE 11
RefBeet	LOC104903747	1,067,534	F-box/LRR-repeat protein At4g14103-like
RefBeet	LOC104903754	1,066,212	Putative F-box/LRR-repeat protein At3g58920
RefBeet	LOC104903789	1,139,922	Probable leucine-rich repeat receptor-like protein kinase At5g49770

the table beet inbreds used here were first released from the University of Wisconsin table beet breeding program in 1983 (Goldman, 1996). W357A was derived from W303, W217, and W187, and W364B was derived from W32 and "Red Pak." FC709-2 was the result of three cycles of selection for resistance to *R. solani* within sugar beet line 871016 and is a self-fertile, multigerm line with resistance to Fusarium yellows (*Fusarium oxysporum* f. sp. *betae*) and high sugar content (Panella, 1999). FC901/C817 was developed through an initial cross of US201 × curly top resistant material, followed by backcrosses to curly top resistant material (Gaskill et al., 1967). US201 was released in 1940 and reported as being a multigerm line with resistance to Fusarium yellows (Panella et al., 2015). In contrast, the mapping population D4 (DIE4 in Lein et al., 2007) used by Lein et al. (2008) was developed from a cross between a highly resistant line, 98–80019, and a susceptible pollinator, 98–99286. The D4 or DIE4 population was developed by the breeding company Strube-Dieckmann and was described as segregating for resistance to rhizomania and Rhizoctonia (Lein et al., 2007). It has been hypothesized that the resistance in the D4 population might have come from some of the early USDA sugar beet releases from Ft. Collins, Colorado (K. M. Dorn and J. M. McGrath, personal communication). However, the relationship between the early FC series, including FC701 to FC705 and FC709 is unknown. In addition, studies have shown large phenotypic variation within FC709-2 indicating the potential presence of multiple resistance genes (K. M. Dorn, personal communication). While FC709-2 was generally one of the most resistant accessions in our disease screens (Wigg & Goldman, 2020), it is entirely possible that given the absence of complete homozygosity within the germplasm, other studies using FC709-2 may identify other QTL associated with resistance.

We observed a large difference in the heritabilities of *Rhizoctonia solani* resistance between the SBRP and TBRP. Our calculation of heritability for the weighted average diseased tissue percentage made use of the genotypic variance based on the variance of F_3 families. This result reflects the fact that genotypic variance in the SBRP was tenfold higher than that of the TBRP and that residual variance in the TBRP was fourfold higher than that of the SBRP. The difference in genotypic variances between the two populations may be due to the genetic backgrounds of the parents in each population. Although both of the table beet parents come from the same breeding program, as do the two sugar beet parents, all of the parental lines used in these mapping populations have been selected for different criteria (Gaskill et al., 1967; Goldman, 1996; Panella, 1999). Additionally, the higher residual variance in the TBRP is partially due to an increased presence of

fungus gnats and other pathogens (73% and 55% of observations, respectively) present in the TBRP compared to that of the SBRP (53% and 61% of observations, respectively). This is not surprising since the two populations were screened in adjacent greenhouse bays and the TBRP was evaluated after the SBRP. In previous studies, we observed an increase of fungus gnats and other pathogens as the length of the screen increased (Wigg, unpublished data).

We identified a total of 13 candidate genes in the region surrounding the QTL on chromosome 2 of the SBRP. Some of these included leucine-rich repeats (LRR) and toll and interleukin receptor (TIR) proteins, which have been associated with host resistance to plant pathogens (see D. Jones & J. Jones, 1997; Ve et al., 2015 for review of LRR and TIR, respectively). In fact, the resistance gene analogue (RGA) closely linked to the *Rz1* gene responsible for resistance to Rhizomania contains a nucleotide-binding site and LRR protein (Lein et al., 2007). One of the genes we identified in the region surrounding the QTL on chromosome 2 of the SBRP (Table 5), the TIR-containing protein, EL10Ac8g20309, was first identified in a recent study of predicted resistance genes (Funk et al., 2018). Interestingly, prior to the work by Funk et al. (2018), TIR domains were not known to exist in *B. vulgaris*. Funk et al. (2018) also identified 231 nucleotide-binding (NB-ARC) loci in the EL10 genome assembly (Funk et al., 2018). Of these, eight were reported to be located on scaffold 8 of EL10 (chromosome 2 of RefBeet 1.2.2). These NB-ARC loci had homologs in *Arabidopsis thaliana* and *Solanum bulbocastanum* that were either described as disease resistance proteins or putative or probable disease resistance proteins.

QTL identified in the SBRP contained resistance gene sequences that were identified from a BLAST search of *Arabidopsis* as putative disease resistance proteins RGA3 and RGA4, isoform X1. Oladzad et al. (2019) found RGAs were associated with QTL conferring resistance to *Rhizoctonia solani* in common bean, suggesting a connection between these proteins and resistance to the *Rhizoctonia* pathogen.

We identified four genes encoding for polyphenol oxidase (PPO) in the regions surrounding the QTL on chromosome 2. Positive correlations between this enzyme and disease resistance in plants have been frequently observed (Mayer, 2006). Li and Steffens (2002) found that transgenic tomato plants overexpressing PPO had increased resistance to the bacterium *Pseudomonas syringae* pv. *tomato*. Similarly, Jia et al. (2016) discovered that overexpression of *FaPPO1* genes in strawberry delayed infection by gray mold (*Botrytis cinerea*). Indeed, Funk et al. (2018) identified an overlap of a PPO with an NB-ARC domain.

We also identified EL10Ac8g20134.1 near our QTL in the SBRP. This gene codes for a pectinesterase-related protein, which plays a role in the esterification of pectin. The activity of pectinesterase plays an important role in plant–pathogen interactions as it can make pectin more accessible to microbial pectic enzymes and increase the accessibility of cell wall components to other cell wall degrading enzymes (CWDE) (Lionetti et al., 2012). Relationships between pectinesterase or pectin methyl esterase levels and activity have been reported for *P.carotovorum*-resistant potatoes (Marty et al., 1997), tomatoes resistant to *Ralstonia solanacearum* (Wydra & Beri, 2006), and beans resistant to *Colletotrichum lindemuthianum* (Boudart et al., 1998).

Another gene near the QTL on chromosome 2 in the SBRP was EL10Ac8g20220.1, which encodes for a sugar transporter/fungal trichothecene efflux pump (TRI12). While there is some evidence that sugar transport proteins (STPs) may play a role in plant defenses against microbes (Yamada et al., 2016), there are no studies of the relationship of trichothecene efflux pumps in plants. Yamada et al. (2016) describe decreased amounts of apoplastic sugars in wild-type *Arabidopsis* plants inoculated with *P. syringae* pv. tomato compared to apoplastic sugar levels in the non-inoculated wild-type control plants and mutant *stp13* plants. Lower apoplastic sugar levels provide less nutrients for the bacteria. This indicates that STP13 is essential for control of sugar uptake, and this competition for resources is a defense mechanism in the plant (Yamada et al., 2016). Similar findings were reported in studies of wild-type *Arabidopsis* roots compared to loss-of-function *SWEET2* mutants under attack by *Pythium irregulare* (Chen et al., 2015) and wild-type sweet potato and *IbSWEET10*-overexpressing lines infected with *F. oxysporum* f. sp. *batatas* (Li et al., 2017). TRI12 was first described by Alexander et al. (1999) and is a trichothecene efflux pump from *F. sporotrichioides*.

Another protein found near the QTL in the SBRP is cystatin-C (CST3), which is encoded by EL10Ac8g20214.1. Cystatins inhibit cysteine proteases, which in turn degrade other proteins and play a role in plant growth and development, as well as in senescence and programmed cell death (reviewed in Grudkowska & Zagdańska, 2004). Pernas et al. (1999) reported that a purified cystatin from sweet chestnut inhibited fungal growth of *Botrytis cinerea*, *Colletotrichum graminicola*, and *Septoria nodorum*, suggesting that the cystatin plays a role in chestnut's defense against phytopathogens. Morphological changes, including hyphal shortening and wall thickening, and growth arrest of the fungi were also observed with increased concentrations of chestnut cystatin (Pernas et al., 1999). A recent study by Yu et al. (2017) investigated the effects of a cystatin in ramie (*Boehmeria nivea* L.), an important fiber crop in India, China, and other Pacific Rim and Southeast Asian countries, on several phytopathogenic fungi. Assays using the purified phytocystatin gene, *reBnCPI* (recombinant expressed cysteine protease inhibitor), demonstrated inhibited growth of the fungi tested, which included: *F. oxysporum*, *Alternaria aternata*, *B. cinerea*, and *Pythium vexans* (Yu et al., 2017). Studies of cystatin function in beet thus

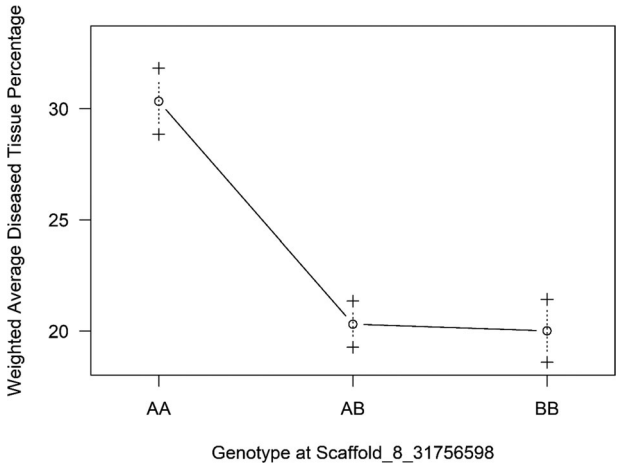

FIGURE 8 Effect plot for the quantitative trait loci on chromosome 2 of the table beet resistance population (TBRP). Parents of the TBRP were the table beet inbred W364B and FC901/C817, a *Rhizoctonia* susceptible sugar beet (SSB). The resistant table beet allele is represented by "B" and the susceptible sugar beet allele by "A"

far have focused on abiotic stresses (Wang et al., 2012). Wang et al. (2012) discovered increased transcription of cystatin in sugar beet line M14 under salt stress. When this gene was cloned and over-expressed in *Arabidopsis* plants, the transgenic plant exhibited improved salt tolerance compared to the wild-type (Wang et al., 2012).

Callose was first described as being rapidly deposited by plant cells in response to mechanical damage or fungal attack by Aist (1983). Since then, numerous studies have reported relationships between the presence of callose and increased resistance to plant pathogens (see Wang et al., 2021 for review). Ali et al. (2013) noted that in *Arabidopsis* plants overexpressing the transcription factor RAP2.6, callose deposition in the syncytia induced by *H. schachtii* was increased and lead to increased resistance to the beet cyst nematode. In a study of grapevines, Yu et al. (2016) discovered two callose synthase genes were upregulated when resistant grapevines were exposed to the downy mildew pathogen, *Plasmopara viticola*. Callose has also been described in the plant response to viruses. Li et al. (2012) reported that in observations of compatible and incompatible combinations of soybean and *Soybean mosaic virus*, compatible interactions had no callose deposited at plasmodesmata, while incompatible interactions had callose depositions at the plasmodesmata at the site of inoculation, and no viral RNA of coat protein was detected in the leaf above the inoculated one. This indicates that callose deposits restricted the movement of the virus to the cells of the inoculation site (Li et al., 2012). Given these findings, it is possible that the callose synthase gene near the QTL in the SBRP is playing a role in resistance to *R. solani* in beet.

Dominance effects were measured for the QTL on chromosome 2 in the TBRP (Figure 8). This is consistent with the report from Gaskill et al. (1970) describing FC702/3, one of the early *Rhizoctonia*-resistant lines of sugar beet from Ft. Collins, as having resistance that was almost completely dominant. Hecker & Ruppel (1976) further corroborated this with findings of partial dominance of crosses made between susceptible × resistant sugar beets. The dominance effects displayed by the QTL in the TBRP combined with the table beet genetic background of the resistance make using this QTL in improving host resistance a more promising endeavor. These QTL, combined with the plot-based heritability of 0.87 and 0.12 in the SBRP and TBRP, respectively, suggest the potential for MAS to improve resistance to *Rhizoctonia* in beet populations.

These QTL can also be used to screen germplasm for resistance. We screened a representative number of PIs, cultivars, and publicly available inbreds for resistance to *R. solani* in a greenhouse study (Wigg & Goldman, 2020). However, given the wide range of root phenotypic variation present across these accessions, PIs especially, it would be informative to screen those accessions and others in germplasm collections, such as the National Plant Germplasm System (NPGS), for the QTL reported in this study. Gaining a wider perspective of levels of resistance would provide avenues to broaden the genetic base of resistance in beet germplasm. Using MAS to introduce more genetic variation in resistance genes increases the resiliency of these plants to pathogens over time.

MAS is also helpful for the selection of traits that are impacted by environmental conditions. The disease triangle concept in plant pathology demonstrates the important role the environment plays in disease development (Stevens, 1960). If a suitable environment or sufficient inoculum is not provided, disease will not develop, leading to "disease escape" plants which may misinform disease screening studies (Agrios, 2005). Francis and Asher (2000) highlight the use of MAS for disease resistance breeding because no disease inoculations and screens are needed, so such escape plants are avoided entirely. In addition to disease escapes, Wigg and Goldman (2020) briefly discuss that the presence of fungus gnats and other diseases in the greenhouse screen environment increased the likelihood that an accession would have an increased mean diseased tissue percentage compared to that same accession without fungus gnats or other diseases present, potentially impacting disease evaluations of accessions. Thus, while heritability of environmentally sensitive traits can be quite low, the heritability of markers is by definition 100%. MAS also allows for selection that is more time and resource efficient. MAS can be used on young seedlings, which saves space, time, and money compared to labor-intensive 13-week-long greenhouse screens (Hecker & Ruppel, 1977b; Wigg & Goldman, 2020). In addition, the implementation of MAS in breeding programs will lead to more efficient selection for polygenic traits such as *Rhizoctonia* resistance. While the polygenic nature of *Rhizoctonia*

resistance in beet increases difficulty of resistance breeding, it also lessens the likelihood of the fungus overcoming that resistance. This can be compared to traits such as sugar beet cyst nematode (SBCN; *Heterodera schachtii* Schmidt) and Rhizomania resistance that are simply inherited (Lewellen et al., 1987; Stevanato et al., 2015). In the early 2000s, the first major resistance gene in sugar beet for Rhizomania, *Rz1*, was overcome (Liu et al., 2005; Panella et al., 2014). Fortunately, researchers had already identified other sources of resistance and were introgressing those into sugar beet germplasm (Panella et al., 2014).

Since MAS can be used to test for multiple genes simultaneously (Francis & Asher, 2000), stacking or pyramiding of resistance traits is enabled. Developing table beet lines with multiple disease resistance traits is important because there is not likely to be only one disease present in a given field. Not only that, but many diseases are synergistic (Hanson, 2010; Strausbaugh & Gillen, 2009; Strausbaugh et al., 2013). Using lines resistant to several diseases will help reduce disease overall both in the field and storage.

Mean genome sizes of 742 and 729 Mb/1C show similarity between table beet and sugar beet, respectively. However, genome-wide allele frequency data showed a clear distinction between sugar and table beet crop types, which agrees with selective breeding for the two crop types (Galewski & McGrath, 2020). Despite being the most divergent of beet crop types, sugar and table beet readily cross, forming fertile offspring. This suggests there is a small degree, if any, of chromosomal variation between sugar and table beet (Galewski & McGrath, 2020). In addition to the present study, Laurent et al. (2007) and McGrath et al. (2007) have created segregating populations from sugar × table beet crosses. Distortions in mapped chromosomes have been reported in both populations derived from sugar × sugar beet (Pillen et al., 1992; Schumacher et al., 1997; Weber et al., 1999) and sugar × table beet crosses (Laurent et al., 2007; McGrath et al., 2007). We did not detect particular patterns of segregation distortion in our intercrop crosses. In a sugar beet linkage map based on restriction fragment length polymorphisms (RFLP), Pillen et al. (1992) attributed distorted segregation to gametic selection of linked lethal loci. Wagner et al. (1992) also concluded that in a map developed using isozyme loci and morphological markers, most distortions were likely the result of gametic selection. In contrast, McGrath et al. (2007) concluded that genetic discordance between sugar and table beet resulted in segregation distortion in a linkage map made of amplified fragment length polymorphism (AFLP). Given the close relationship between table and sugar beet, it is reasonable to utilize resources developed by the sugar beet research community for improvement of table beet germplasm.

In addition to the traits introgressed from sugar beet, table beet genetics and breeding work have largely focused on shape and color. Numerous studies have investigated the inheritance of color in the crop (Goldman et al., 1996; Keller, 1936; Linde-Laursen, 1972; Watson & Gabelman, 1984; Watson & Goldman, 1997; Wolyn & Gabelman, 1989, 1990). More recently, researchers have investigated flavor characteristics, namely the earthy flavor imparted by the compound geosmin. In the first genetic mapping experiment in table beet, Hanson et al. (2021) used association analysis and selective genotyping to search for QTL associated with geosmin concentration. Large portions of chromosome 5 were significantly associated with the production of this volatile terpenoid (Hanson et al., 2021). These studies suggest MAS may be useful in improving table beet.

In addition to fine-mapping the QTL described, future studies may focus on using RNAseq in these populations to characterize differentially expressed genes (DEGs) in the absence and presence of the pathogen. This has proven to be a viable method in other crops. Researchers have identified DEGs related to the response of susceptible and resistant lines of rice to sheath blight caused by *R. solani* (Shi et al., 2020; Zhang et al., 2017). In sugar beet, Holmquist et al. (2021) identified three major latex encoding genes displaying increased transcriptional activity in lines with partial resistance to *R. solani* when inoculated with an isolate of AG 2-2-IIIB. Additional transcriptomic studies in beet and other crops will improve our understanding of the mechanism of resistance to the fungus. It will be informative to learn whether different gene products are associated with resistance in different populations. If so, these QTL could be "stacked" to provide a more robust resistance in germplasm.

These experiments identified two QTL associated with resistance to *R. solani* AG 2-2 IIIB that can be used to screen table beet germplasm. Once accessions with the markers have been identified, greenhouse screens can be used to obtain a more precise evaluation of the level of resistance.

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AUTHOR CONTRIBUTIONS

Katharina S. Wigg: Conceptualization; Data curation; Formal analysis; Investigation; Methodology; Project administration. Scott H. Brainard: Data curation; Formal analysis; Investigation; Methodology. Nicholas Metz: Data curation; Formal analysis; Investigation. Kevin M. Dorn: Data curation; Formal analysis; Investigation; Methodology. Irwin L. Goldman: Conceptualization; Formal analysis; Funding acquisition; Investigation; Methodology; Project administration.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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