Variability in Reaction to Root and Crown Rot Caused by *Rhizoctonia Solani* Among Table Beet Cultivars, Breeding Lines, and Plant Introductions in Controlled Environment Conditions

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Abstract. Table beet (*Beta vulgaris* ssp. *vulgaris*) is adversely affected by the soilborne fungus, *Rhizoctonia solani*, which greatly decreases root quality. There are no reports of breeding efforts designed to improve resistance to this fungus in table beet. A collection of table beet PIs, cultivars, and publicly available inbreds were characterized for their response to inoculation with *R. solani* in three replicated greenhouse experiments conducted between 2017 and 2019. An isolate of AG 2-2 IIIB was used to inoculate 8-week old plants, and both susceptible and resistant sugar beet genotypes were used for comparison. Plants were evaluated for internal and external disease characteristics 3 and 5 weeks postinoculation. Compared with uninoculated controls, PI accessions ranged from 2% to 44% infection, whereas cultivars and inbred lines ranged from 16% to 53% and 22% to 52%, respectively. No commercial cultivar outperformed the resistant sugar beet genotype; however, several PI accessions exhibited lower mean diseased tissue percentages than the resistant sugar beet. This suggests that resistance to *R. solani* may exist in table beet germplasm. PI accessions exhibited greater resistance on average than did cultivars. Inbred lines exhibited the least amount of variation. Ordinal regression allowed for prediction of infection response, which may be helpful in future selection efforts. ‘Badger Flame’, Ames 22164, PI 502294, PI 169015, ‘Long Season’, ‘Red Cloud’, and ‘Albina Vereduna’ were some of the most resistant accessions in our screens. The findings from this study can be used to inform decisions for breeding for host plant resistance in table beet.

Table beet (*Beta vulgaris* L. ssp. *vulgaris*) is a regionally important crop in the United States. Wisconsin leads U.S. production with more than 1600 ha devoted to production (U.S. Department of Agriculture, 2017). A significant detriment to table beet quality and yield is *Rhizoctonia* root and crown rot caused by the fungus *Rhizoctonia solani* Kühn (Abawi et al., 1986; Natti, 1953; Pethybridge et al., 2018), which renders table beet roots unmarketable. Currently, there are few chemical options for controlling this fungus in table beet production, and no reports of efforts to improve host plant resistance through breeding.

R. solani is a soilborne necrotrophic pathogen affecting many crop families. Within the *Rhizoctonia* complex, 12 anastomosis groups (AGs) have been described. These AGs distinguish genetically diverse isolates according to their affinity for hyphal fusion with one another (Ogoshi, 1985). Several AGs have been further divided into “intraspecific groups” (ISGs) (Ogoshi, 1987), or more appropriately “subgroups” (SGs) (Ogoshi, 1996) to categorize isolates by their biochemical, pathogenic, and genetic quali-

ties. AGs 1, 2-2, and 4 infect beets, with AG 2-2 causing root rot and AGs 1, 2-2 (Naito et al., 1978), and 4 infecting the crown and foliage (Naito et al., 1976). A study by Yamaguchi et al. (1977) confirmed that isolates of AG 2-2 caused severe disease on sugar beet roots compared with those of AG 1 and 4. AGs 2-2-IIIB (Büttner et al., 2002; Strausbaugh et al., 2011; Watanabe and Matsuda, 1966) and 2-2-IV (Bolton et al., 2010; Engelkes and Windels, 1996) are virulent on table beet and the closely related sugar beet (both *Beta vulgaris* L. ssp. *vulgaris*) and *Rhizoctonia* infections can cause severe yield losses in both the field and during processing (Gaskill, 1968; Hecker and Ruppel, 1977b; Ruppel et al., 1979; Stivers, n.d.). Sugar beet seedlings are susceptible to a wider range of AGs (Bolton et al., 2010; Rush et al., 1994; Windels and Nabben, 1989) compared with mature plants, resulting in damping off and stand reduction. In contrast, Windels and Nabben (1989) found that plants older than 8 weeks in their experiments were primarily infected by AG 2-2. This was corroborated by Bolton et al.

(2010) when they observed that out of a range of 15 tested AGs and SGs, only AG 2-2-IIIB and AG 2-2-IV caused significant disease in 10-week-old sugar beet plants suggesting that mature plants are less susceptible to some AGs.

R. solani can affect beet plants at two stages in the crop’s lifecycle: at the seedling stage and later in the growing season when plants are maturing (Sneh et al., 1996). Shortly after crop emergence, seedlings may damp-off caused by *R. solani* attacking the hypocotyl below the soil level. Severely diseased seedlings succumb, whereas mild cases result in less vigorous and misshapen mature roots (Sneh et al., 1996). The occurrence of *R. solani* on mature plants later in the growing season is often referred to as *Rhizoctonia* root and crown rot. In the case of table beets, the disease is commonly called pocket rot for the numerous empty “pockets” left behind in the field in the disease’s wake (Pethybridge et al., 2018; Windels et al., 2009). Plants exhibit aboveground symptoms including black lesions on petioles touching the soil surface followed by the wilting and death of older leaves (Windels et al., 2009). Underground symptoms include copious circular root lesions ranging from 2 to 25 mm in diameter (Windels et al., 2009). Lesions have a well-defined border and exhibit alternating light and dark colors in concentric bands (Windels et al., 2009). Internally, roots may contain mycelium-filled cankers easily distinguished from healthy root tissue (Sneh et al., 1996). The fungus overwinters and survives for many years in colonized plant tissue and sclerotia (Abawi et al., 1986; Cubeta and Vilgalys, 1997). Sumner and Bell (1994) observed disease caused by AG 2-2 in crop plants and low soil inoculum densities (average of 3.8 colony-forming units per 100 g 32 months after planting) of that same subgroup in the third year of a study investigating survival of *Rhizoctonia* spp. in a rotation of a corn–snap bean double crop, peanut monocrop, and corn–snap bean double crop.

Current strategies for management of *Rhizoctonia* root and crown rot in beet include crop rotation with nonhost plants (Abawi et al., 1986; Hecker and Ruppel, 1976; Pethybridge et al., 2018), proper weed management (Harveson, 2003), avoiding covering plant crowns with soil during cultivation (Schneider et al., 1982), using treated seed (Abawi et al., 1986), and applying fungicides during the growing season (Colquhoun et al., 2020; Pethybridge et al., 2018). Growers often implement a combination of these strategies in an integrated pest management approach.

In 1977, Hecker and Ruppel (1977b) suggested a 4- to 5-year rotation of sugar beet with cereals, alfalfa, or maize to control *R. solani*. Many beet-growing areas list crop rotation as a management practice (Cattanach et al., 1991; Colquhoun et al., 2020; Khan et al., 2020; Stivers, n.d.). Management of this pathogen through crop rotation, however, has proven to be difficult because of the wide range of plants affected by AG 2-2. Decreasing effectiveness of suppression in table beet–maize rotations in New York led

Ohkura et al. (2009) to investigate the situation. They found most of the isolates pathogenic on table beet tested were also pathogenic on maize. Isolates in AG 2-2, AG 5, and AG 11 were highly aggressive in the experiments (Ohkura et al., 2009). In 2011, Strausbaugh et al. observed that seven of the 18 AG 2-2 IIIB isolates from sugar beet tested in their study were capable of attacking maize and sugar beet. These findings supported previous work by Sumner and Bell (1982) in which AG 2-2 was discovered to induce lateral, brace, and crown rot in maize crops. In this study, AG 2-2 isolates from maize were highly virulent on other Georgian crops, including snap bean, lima bean, southernpea, juvenile soybean, and cucumber (Sumner and Bell, 1982). In spite of these findings, crop rotations are certainly not obsolete; without them, losses to the fungus would be much higher. In Nebraska, Schuster and Harris (1960) discovered 4 years of continuous sugar beets led to an average of 63% infected beets.

In-season fungicide applications are often used by growers to manage *Rhizoctonia* root and crown rot. Olaya et al. (1994) completed initial studies for chemical control of *R. solani* on table beet and since then several fungicides have been registered for use on beet (Arabiat and Khan, 2016). One of the most widely used fungicides, azoxystrobin, was labeled in 1999 and is effective for increasing stands and yields (Arabiat and Khan, 2016; Windels and Brantner, 2005) and controlling *Rhizoctonia* root and crown rot (Gugino et al., 2008; Kiewnick et al., 2001), but timing of application is crucial (Kirk et al., 2008; Stump et al., 2002). Fungicide applications can cost more than \$60

per hectare (M.F.R. Khan, personal communication). In addition to the challenge of timing applications and costs involved, the potential for *R. solani* populations to develop fungicide resistance is a threat to disease management (Jacobsen, 1997; Kiewnick et al., 2001). Several beet-growing areas have documented resistance in *Cercospora beticola* Sacc., another beet pathogen controlled by azoxystrobin (Abawi et al., 2014; Vaghefi et al., 2016) and resistance to azoxystrobin has been reported in other fungal pathogens affecting crops, including almond, grape, pistachio, strawberry, watermelon, and perennial ryegrass (Jiang et al., 2009; Ma et al., 2003; Stevenson et al., 2004; Vincelli and Dixon, 2002). Azoxystrobin-resistant isolates of *R. solani* AG 1-IA, the causal organism of rice sheath blight, were first reported in Louisiana in 2011, and studies showed that resistance had spread from the initial area reported and more than a third of the isolates collected exhibited resistance to the fungicide (Lunos, 2016; Olaya et al., 2012). Pethybridge et al. (2018) cautions the risk of azoxystrobin resistance development within *R. solani*. Arabiat and Khan (2016) investigated the sensitivity to fungicides of isolates of AG 2-2 collected from sugar beets before and after the registration and usage of fungicides to manage damping off and root and crown rot caused by *R. solani*. They found a mean half maximal effective concentration (EC_{50}) of $296.1 \mu\text{g}\cdot\text{mL}^{-1}$ for isolates after the registration and usage of azoxystrobin compared with an EC_{50} of $49.7 \mu\text{g}\cdot\text{mL}^{-1}$ before registration (Arabiat and Khan, 2016). Although azoxystrobin was still effective at the labeled rate in controlling *R. solani* in the greenhouse study, these results demonstrate the capability of *Rhizoctonia* populations to develop resistance to the fungicide.

A reduction in the effectiveness of crop rotation, the ability of *R. solani* to survive saprophytically in the soil for several years, and the threat of pathogen populations developing resistance to fungicides make developing host plant resistance critical to economically sustainable table beet production (Gaskill, 1968; Harveson et al., 2002; Pethybridge et al., 2018).

Efforts to breed *B. vulgaris* for resistance to *R. solani* have primarily focused on the close relative of the table beet, the sugar beet, by mass selection or recurrent field selection (Gaskill, 1968). Many *Rhizoctonia*-resistant sugar beet lines have been developed (Hallowin et al., 2000; Hecker and Gaskill, 1972; Hecker and Ruppel, 1977a, 1985, 1988, 1991; Panella et al., 2015a) and screening is actively done in sugar beet breeding programs (Panella et al., 2015b). Hecker and Ruppel (1975) determined that resistance in sugar beet is polygenic, involving at least two loci, each with two or three alleles, and including modifying genes. They estimated a broad sense heritability of ≈ 0.65 (Hecker and Ruppel, 1975). In conjunction with breeding for disease resistance, many publicly released *B. vulgaris* subsp. *vulgaris*

sugar beet lines and accessions in the National Plant Germplasm System (NPGS) have been evaluated for resistance to *Rhizoctonia* over the years (Hanson and Panella, 2007; Panella, 2000; Panella and Hanson, 2001; Panella and Ruppel, 1998; Panella et al., 2008).

The use of *Rhizoctonia*-resistant cultivars for sugar beet production is recommended. Harveson et al. (2002) recommended resistant cultivars as a practical and economical disease management approach, and in some cases the only effective approach (McGrath and Panella, 2019). Although resistance is available in sugar beet, it is incomplete and there is often decreased yield potential associated with resistant lines (Jacobsen et al., 2004). Planting resistant varieties may be less attractive, especially when they have shown responses to fungicide applications and still not achieved the yield of susceptible varieties with fungicide applications (Jacobsen, 2006).

One approach to breed for *R. solani* resistance in table beet germplasm might involve the introgression of resistance from sugar beet. However, progeny derived from crosses between sugar beet and table beet have exhibited external russetting and increased crown diameter (two distinctive characteristics of sugar beets) even after 10 generations of backcrossing (Wang and Goldman, 1999). These are undesirable qualities in the table beet industry. A different approach would be to identify resistance in existing table beet germplasm. At present, only 30% of NPGS table beet accessions have been evaluated for their responses to inoculation with *R. solani* [Germplasm Resources Information Network (GRIN), 2020].

Although resistance to *Rhizoctonia* root and crown rot has not been well-characterized in table beet, several cultivars have appeared on the market with putative resistance or tolerance. These include 'Shiraz', 'Rubra', 'Kestrel', 'Solo' (Pethybridge et al., 2018), and 'Pacemaker III' (Goldman, 1996; Pethybridge et al., 2018); however, these claims have not been experimentally verified. Despite these cultivars, little work has been accomplished in assessing reaction to *R. solani* in table beet and in breeding for host resistance. The goal of this study was to use controlled conditions in a greenhouse to evaluate the reaction of commercial cultivars, publicly available inbred table beet lines, and PIs from the U.S. NPGS to *R. solani*. We hypothesized that table beet accessions with different genetic backgrounds would respond differently to inoculation with *R. solani* AG 2-2.

Materials and Methods

Germplasm. A large pool of crop genetic diversity is available in the U.S. NPGS with more than 2000 *B. vulgaris* ssp. *vulgaris* accessions (GRIN, 2020). This includes both table beets used for processing and sugar beets for sugar production; however, only a small fraction, ≈ 200 unique accessions, are

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vegetable types. Accessions with the production descriptor “type” and designation of “table” were identified through GRIN. In Nov. 2017 there were 234 accessions fitting that description. Duplicate and unavailable accessions were removed, and the 94 remaining accessions were used in this investigation (Table 1). This screen will be referred to as the “PI Screen.” In addition to the PI Screen, 56 cultivars were identified from 23 commercial sources (Table 2). In addition, nine publicly available inbred lines released from the University of Wisconsin table beet breeding program were included, along with a recently released open-pollinated cultivar, Badger Flame, from the University of Wisconsin (Wisconsin Alumni Research Foundation, 2019). Efforts were made to obtain only nontreated seed, but several lines were available only with treated seed. ‘Carillon’, ‘Eagle’, and ‘Vulture’ were treated with thiram (Thiram 50WP, tetramethylthiuram disulfide; Bayer CropScience, Research Triangle Park, NC) and metalaxyl (Apron; Syngenta Crop Protection, LLC, Greensboro, NC). ‘Pacemaker III’ and ‘Bohan’ were treated with both thiram and metalaxyl as well as fludioxonil (Maxim4S; Syngenta Crop Protection, LLC). Of these three seed treatments, only fludioxonil is labeled to control seed decay, seedling damping off, and seedling blight caused by *R. solani*. Seed treatments are unlikely to interfere with fungal growth and infection by the time of inoculation (Jacobsen et al., 2004; Kataria and Gisi, 1996; Stump et al., 2004; Windels and Brantner, 2002). This screen is referred to as the “Commercial Screen.”

To validate the results from the PI and Commercial Screens and quantify the amount of variation within accessions relative to the greenhouse environment itself, a third screen was conducted. This screen included a subset of several accessions from both the PI and Commercial Screens, along with the doubled haploid sugar beet line KDH13 (Eujayl et al., 2016) (Table 3). This screen made use of increased replication compared with the PI and Commercial Screens and is referred to as the “Validation Screen.” In each of the three screens, the table beet inbred line W364 was included as a control because it was fairly resistant to *Rhizoctonia* in our pilot screens (K. Wigg and I. Goldman, unpublished data). The inbred line W364 is a commonly used parent for F₁ hybrid table beet cultivars (Goldman, 1996). Sugar beet inbred lines FC709-2 and FC901/C817 were also included as resistant and susceptible controls, respectively (Gaskill et al., 1967; Panella, 1999) [courtesy of Lee Panella, Crops Research Unit, U.S. Department of Agriculture–Agricultural Research Station (USDA-ARS)]. The Sugar Beet Research Unit in Ft. Collins, CO, uses FC709-2 (Panella, 1999) as a highly resistant control and FC901/C817 (Fenwick et al., 2018; Gaskill et al., 1967; Panella and Hanson, 2001) as a susceptible control in their *Rhizoctonia* screens. All germplasms, whether inbreds, PI accessions, or commer-

cial lines, are hereafter referred to as accessions.

Greenhouse culture. All screens were completed in the Walnut Street Greenhouses at the University of Wisconsin-Madison. Experiments were conducted from Nov. 2017 through Jan. 2019. A minimum of five seeds were sown into a 3:1 mix (by volume) of silty loam compost soil collected from West Madison Agricultural Research Station (Arlington, WI) and soilless medium (MetroMix; Sun Gro Horticulture, Agawam, MA) in 2780 cm³ plastic pots. Following germination and plant establishment, each pot was thinned to two plants per pot. High-pressure sodium supplemental lighting (1000 μ mol at bench height) provided 16-h daylengths. Air-conditioned greenhouses with forced-air heating were used to maintain temperatures between 25 and 30 °C to provide a conducive environment for *R. solani* disease development (Parmeter, 1970). Beets were watered as needed daily 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 applied as needed for optimum plant growth. *Bacillus thuringiensis* ssp. *israelensis* 35% to 45% (Gnatrol WDG; Valent Biosciences Corporation, Libertyville, IL) was applied as needed to manage fungus gnats.

Because of the endemic nature of fungus gnats in the greenhouse environment, along with the added attraction of fungus from the inoculation of *R. solani* in our experiment (Cloyd, 2010), 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.”

Experimental design. Each screen was replicated in time and conducted twice. The term replicate is used here to describe each experimental run but is also used to describe a complete set of treatments within a run. Following planting, pots were placed into a randomized complete block design with each block assigned to the 3- or 5-week postinoculation evaluation period. In both the PI and Commercial Screens, each accession was represented by eight plants across the two replicates and two plants in each evaluation period. Both the PI and Commercial Screens included FC901/C817, FC709-2, and W364B as noninoculated controls. In the Validation Screen, each accession was represented by 40 plants across the two replicates and 10 plants in each evaluation period. In addition to the aforementioned three controls, the Validation Screen also included W411B as a noninoculated control. W411B was included as a noninoculated control because it exhibited little variation and was susceptible in the Commercial Screen.

Inoculations. All inoculations were performed using *R. solani* isolate R-1 (AG-2-2-IIIB) obtained from Linda Hanson, USDA-ARS (Nagendran et al., 2009). The isolate was

grown on autoclaved barley grains to produce the inoculum and was stored at 4 to 7 °C until use according to Naito et al. (1993). The isolate used in this study was originally collected from an infected sugar beet root from Platteville, CO, in the 1960s. Tests conducted by Michigan State researchers have classified the isolate as highly virulent on *B. vulgaris* ssp. *vulgaris* (L. Hanson, personal communication; Nagendran et al., 2009). This barley-fungus inoculum was roughly ground using a coffee grinder (*FreshGrind*; Hamilton Beach Brands, Glen Allen, VA) immediately before artificial inoculation. Each plant was inoculated by displacing a small amount of growth medium adjacent to the root 2 to 3 cm below the soil surface and depositing 6 g or $\approx$ 6 mL of ground inoculum (Hecker and Ruppel, 1977b; A. Fenwick, personal communication). Growth medium was then smoothed back over the inoculum. Plants were gently watered immediately following inoculations and then watered daily.

Disease testing. Artificial inoculation took place 8 weeks after planting. Noninoculated controls were included in each block and replication. Plants were evaluated for disease at 3 and 5 weeks postinoculation. The timeline of this screen was in accordance with sugar beet screening methods (L. Panella, personal communication).

At the time of evaluation, plants were gently removed from the medium. The beet tops were separated from the roots at the crown using a knife, weighed, and then discarded. Roots were brushed off with a cloth, gently washed with tap water, dried with a cloth, and then weighed. A visual disease rating scale adapted from the visual scale described by Campbell et al. (2014) and first used by Ruppel et al. (1979) was used to assign an external and internal disease rating for each plant (Fig. 1). Following the external rating, each root was halved from the crown to the bottom. The tissue visible from this cut was used to assign an internal rating. Ratings ranged from 0 to 5, with a rating of 0 = 0% diseased tissue, root surface clean with no visible lesions; 1 = 1% to 10% diseased tissue, superficial, scattered nonactive lesions; 2 = 11% to 30% of root affected; 3 = 31% to 60% of root affected; 4 = 61% to 99% of root blackened with rot extending into interior; and 5 = root 100% rotted and foliage was dead or dying (adapted from Campbell et al., 2014). These groupings were chosen because the researcher could visually distinguish among them.

To normalize the data for linear regression, the internal and external disease ratings for each plant were converted 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

Table 1. Mean diseased tissue percentage, most likely internal disease ratings and probabilities, and disease progression categories of 97 *Beta vulgaris* subsp. *vulgaris* PI accessions in response to inoculation with *Rhizoctonia solani* AG 2-2 isolate R1 in a greenhouse screen evaluated in 2018.

Code ^z	Accession	Name	Origin ^y	Mean diseased tissue percentage ^x	First most likely internal DR ^w	Second most likely internal DR ^w	Probability of first most likely internal DR (SE) ^w	Probability of second most likely internal DR (SE) ^w	Disease progression ^v
68	Ames 22164	Long Smoothe Dark Red	US	1.9 a	0	1	0.56 (0.20, 0.91)	0.37 (0.15, 0.59)	SC
57	PI 502294	IDBBNR 5544	UZ	6.0 ab	1	2	0.47 (0.40, 0.53)	0.30 (0.12, 0.47)	SC
86	PI 169015	IDBBNR 5260	TK	6.4 ab	1	0	0.46 (0.37, 0.54)	0.37 (0.11, 0.64)	SC
73	PI 105335	Tzu Lo Pu Tou	China	6.6 ab	1	2	0.47 (0.42, 0.52)	0.27 (0.10, 0.45)	SC
47	PI 181717	IDBBNR 5359	LB	10.0 ab	1	2	0.46 (0.37, 0.54)	0.32 (0.15, 0.49)	SC
62	PI 205987	Egyptist Platrund W:S/51	SW	10.7 ab	1	0	0.47 (0.42, 0.53)	0.31 (0.07, 0.55)	SC
93	PI 148625	Chaghonda	Iran	10.9 ab	1	2	0.46 (0.37, 0.54)	0.32 (0.14, 0.50)	SC
53	PI 116906	Lablabu	AF	12.1 abc	1	2	0.40 (0.26, 0.54)	0.40 (0.26, 0.54)	SC
41	PI 323938	Avon Early	UK	12.2 abc	1	0	0.45 (0.36, 0.55)	0.38 (0.12, 0.63)	SC
65	NSL 28024	Extra Early	US	13.1 abcd	1	0	0.47 (0.41, 0.53)	0.31 (0.07, 0.56)	SC
89	PI 169028	IDBBNR 5272	TK	13.1 abcde	1	2	0.42 (0.29, 0.54)	0.38 (0.23, 0.53)	SC
46	PI 179175	IDBBNR 5347	Iraq	13.2 abcde	0	1	0.45 (0.18, 0.72)	0.43 (0.30, 0.55)	SS
94	PI 590606	Fordhook Giant	US	13.3 abcdef	1	2	0.41 (0.27, 0.54)	0.39 (0.25, 0.54)	SC
12	PI 612338	Detroit Type	DN	13.3 abcdef	1	2	0.43 (0.32, 0.55)	0.36 (0.20, 0.52)	SC
66	NSL 28020	Earlidark	US	13.3 abcdef	1	0	0.47 (0.43, 0.51)	0.26 (0.05, 0.47)	SC
45	PI 590617	Crosbys Egyptian	NI	13.3 abcdef	1	2	0.45 (0.36, 0.54)	0.33 (0.16, 0.50)	SS
22	PI 379097	Crveno	MC	13.4 abcdef	1	0	0.47 (0.43, 0.51)	0.26 (0.05, 0.46)	SC
77	PI 141919	IDBBNR 5214	NI	13.6 abcdef	1	2	0.45 (0.36, 0.54)	0.33 (0.16, 0.50)	SC
3	PI 177272	IDBBNR 5338	TK	14.2 abcdef	1	0	0.47 (0.43, 0.52)	0.28 (0.06, 0.49)	SC
59	PI 380754	IDBBNR 9582	Iran	14.4 abcdef	1	2	0.45 (0.36, 0.54)	0.33 (0.16, 0.50)	SS
37	PI 271439	IDBBNR 5435	India	14.7 abcdef	1	2	0.47 (0.42, 0.52)	0.27 (0.10, 0.45)	SC
7	W6 22192	POL 175453	PL	15.0 abcdef	1	2	0.42 (0.29, 0.54)	0.38 (0.24, 0.53)	SC
74	PI 109039	IDBBNR 5161	TK	15.2 abcdef	1	2	0.47 (0.41, 0.53)	0.29 (0.12, 0.46)	SS
42	PI 357351	Polsko	MC	15.7 abcdef	1	2	0.47 (0.40, 0.53)	0.29 (0.12, 0.47)	SS
75	PI 124528	Chakunda	India	15.9 abcdef	1	0	0.47 (0.43, 0.51)	0.26 (0.05, 0.47)	SC
16	PI 612334	Rodbeta Slowbolt	SW	16.7 abcdef	2	1	0.42 (0.30, 0.55)	0.37 (0.22, 0.53)	SS
51	W6 44532	Feuer Kugel	CN	17.0 abcdef	1	2	0.44 (0.34, 0.54)	0.35 (0.18, 0.51)	SS
25	PI 531261	Detroit	HN	17.1 abcdef	1	2	0.42 (0.30, 0.54)	0.38 (0.23, 0.53)	SS
26	PI 590593	Improved Early Egyptian	NI	17.5 abcdef	2	1	0.47 (0.41, 0.53)	0.28 (0.11, 0.45)	SC
39	PI 285591	Okragly Ciemnoczzerwony	PL	17.7 abcdef	1	2	0.45 (0.35, 0.54)	0.34 (0.17, 0.51)	SS
76	PI 140358	IDBBNR 5208	Iran	17.9 abcdef	1	2	0.47 (0.42, 0.52)	0.27 (0.10, 0.44)	SC
96	FC709-2 ^a	FC 709-2	US	18.2 abcdef	1	2	0.44 (0.36, 0.52)	0.35 (0.23, 0.47)	SC
92	PI 251512	Shogondak	Iran	18.2 abcdef	1	2	0.47 (0.43, 0.52)	0.25 (0.08, 0.42)	SC
24	PI 531260	Bordo	SU	18.4 abcdef	1	2	0.42 (0.29, 0.54)	0.38 (0.23, 0.53)	SS
50	W6 44531	Chioggia Barbietola	CN	18.6 abcdef	1	2	0.41 (0.29, 0.54)	0.39 (0.24, 0.53)	SC
48	PI 193458	IDBBNR 5372	ET	18.6 abcdef	1	2	0.46 (0.38, 0.54)	0.31 (0.14, 0.48)	SC
54	PI 177274	IDBBNR 5340	Syria	18.7 abcdef	1	2	0.47 (0.42, 0.52)	0.28 (0.11, 0.45)	SC
61	PI 269308	Egyptisk Plattgrund	SW	18.9 abcdef	2	1	0.41 (0.27, 0.54)	0.40 (0.26, 0.53)	SC
55	PI 181716	IDBBNR 5358	LB	19.5 abcdef	1	2	0.40 (0.26, 0.54)	0.40 (0.26, 0.54)	SC
81	PI 163178	Choghundur	India	19.7 abcdef	2	1	0.41 (0.28, 0.55)	0.39 (0.24, 0.53)	SS
80	PI 144675	IDBBNR 5229	Iran	20.1 abcdef	1	2	0.42 (0.29, 0.54)	0.38 (0.23, 0.53)	SC
11	PI 612339	Albina Vereduna	NT	20.2 abcdef	1	2	0.44 (0.34, 0.54)	0.34 (0.18, 0.51)	SS
13	PI 612337	Kogel	NT	20.4 abcdef	2	1	0.46 (0.38, 0.54)	0.32 (0.15, 0.48)	SC
28	PI 590600	Detroit Perfected	NI	20.4 abcdef	1	0	0.47 (0.43, 0.51)	0.26 (0.06, 0.47)	SC
1	PI 169032	IDBBNR 5276	TK	20.4 abcdef	1	2	0.44 (0.35, 0.54)	0.34 (0.18, 0.50)	SC
72	NSL 6611	Eclipse	US	20.4 abcdef	1	2	0.45 (0.37, 0.54)	0.32 (0.16, 0.49)	SC
95	FC901/C817 ^a	FC901/C817	US	20.5 abcdef	1	0	0.47 (0.43, 0.51)	0.29 (0.12, 0.46)	SC
67	Ames 8292	IDBBNR 9509	UK	20.5 abcdef	2	1	0.42 (0.29, 0.54)	0.38 (0.24, 0.53)	SC
71	NSL 6320	Winter Keeper	NI	20.5 abcdef	2	1	0.46 (0.39, 0.54)	0.31 (0.14, 0.47)	SC
32	PI 596529	Bordo 237	AR	21.1 abcdef	2	1	0.41 (0.27, 0.54)	0.39 (0.25, 0.54)	SS
49	PI 177275	Beledi	TK	21.2 abcdef	2	1	0.42 (0.29, 0.55)	0.38 (0.23, 0.53)	SC
87	PI 169019	IDBBNR 5264	TK	21.5 abcdef	1	2	0.43 (0.31, 0.54)	0.37 (0.21, 0.52)	SC
33	PI 612328	Bikores	NT	21.5 abcdef	2	1	0.47 (0.40, 0.54)	0.30 (0.13, 0.46)	SC
84	PI 164805	Choghundur	India	21.6 abcdef	2	3	0.47 (0.42, 0.53)	0.22 (0.10, 0.34)	SC
6	W6 22468	BV-00-01	SL	21.6 abcdef	1	2	0.44 (0.33, 0.54)	0.35 (0.19, 0.51)	SC
15	PI 612336	Albino White	CN	22.1 abcdef	1	2	0.45 (0.35, 0.54)	0.34 (0.17, 0.50)	SC
18	PI 612330	Replata	NT	22.1 abcdef	1	2	0.42 (0.30, 0.54)	0.37 (0.22, 0.53)	SS
83	PI 164659	IDBBNR 5245	India	22.1 abcdef	2	1	0.40 (0.27, 0.54)	0.40 (0.26, 0.54)	SS
88	PI 169022	IDBBNR 5267	TK	22.1 abcdef	2	1	0.41 (0.29, 0.54)	0.39 (0.24, 0.53)	SC
85	PI 164968	IDBBNR 5251	TK	22.3 abcdef	2	1	0.44 (0.32, 0.55)	0.36 (0.20, 0.52)	SC
23	PI 414934	IDBBNR 4621	SA	22.4 abcdef	2	1	0.44 (0.33, 0.55)	0.36 (0.20, 0.51)	SC
91	PI 222234	IDBBNR 5391	Iran	22.6 abcdef	1	2	0.46 (0.38, 0.54)	0.31 (0.14, 0.48)	SC
14	PI 612342	Rote Kugel	DE	23.1 abcdef	2	1	0.45 (0.35, 0.55)	0.34 (0.18, 0.50)	SS
63	Ames 14432	Sperlings Bluta Bordo-60	BG	23.1 abcdef	1	2	0.41 (0.28, 0.54)	0.39 (0.24, 0.53)	SC
90	PI 169029	Pancar	TK	23.9 abcdef	1	2	0.45 (0.37, 0.54)	0.32 (0.16, 0.49)	SC
8	W6 22162	POL 175450	PL	24.1 abcdef	2	1	0.43 (0.32, 0.55)	0.36 (0.21, 0.52)	SS

(Continued on next page)

Table 1. (Continued) Mean diseased tissue percentage, most likely internal disease ratings and probabilities, and disease progression categories of 97 *Beta vulgaris* subsp. *vulgaris* PI accessions in response to inoculation with *Rhizoctonia solani* AG 2-2 isolate R1 in a greenhouse screen evaluated in 2018.

Code ^z	Accession	Name	Origin ^y	Mean diseased tissue percentage ^x	First most likely internal DR ^w	Second most likely internal DR ^w	Probability of first most likely internal DR (SE) ^w	Probability of second most likely internal DR (SE) ^w	Disease progression ^v
38	PI 285590	Epipski Hoser	PL	24.3 abcdef	2	1	0.47 (0.41, 0.53)	0.28 (0.12, 0.45)	SS
40	PI 296129	IDBBNR 5459	US	25.3 abcdef	2	1	0.45 (0.36, 0.54)	0.33 (0.17, 0.49)	SC
60	PI 140353	IDBBNR 5203	Iran	25.4 abcdef	1	2	0.45 (0.36, 0.54)	0.34 (0.18, 0.50)	SC
52	PI 222233	Laboo	Iran	25.7 abcdef	2	1	0.41 (0.27, 0.54)	0.39 (0.25, 0.54)	SC
44	PI 357360	Ohridska Zolta	MC	25.8 abcdef	1	2	0.45 (0.35, 0.54)	0.34 (0.17, 0.50)	SS
69	NSL 28026	Gardeners Model	US	26.2 abcdef	2	1	0.46 (0.37, 0.54)	0.32 (0.16, 0.49)	SS
4	W6 44530	Golden Beet	CN	26.2 abcdef	2	1	0.48 (0.43, 0.52)	0.23 (0.07, 0.39)	SC
36	PI 269310	Spangsbjerg	SW	26.3 abcdef	2	1	0.46 (0.39, 0.54)	0.30 (0.14, 0.47)	SS
9	W6 22161	POL 175449	PL	26.5 abcdef	2	1	0.41 (0.27, 0.54)	0.39 (0.25, 0.53)	SC
21	PI 590635	Burpees Improved	NI	27.2 abcdef	2	1	0.46 (0.38, 0.54)	0.32 (0.15, 0.48)	SC
		Blood Turnip							
5	W6 44528	Bull's Blood	CN	27.3 abcdef	2	1	0.46 (0.38, 0.54)	0.32 (0.15, 0.48)	SS
64	Ames 22163	Iowa	US	27.5 abcdef	2	3	0.47 (0.40, 0.53)	0.23 (0.11, 0.35)	SS
2	PI 174059	IDBBNR 5310	TK	27.8 abcdef	2	1	0.44 (0.34, 0.55)	0.35 (0.19, 0.50)	SC
17	PI 612332	Egyptische Egy	NT	27.9 abcdef	2	1	0.47 (0.40, 0.53)	0.29 (0.13, 0.46)	SS
35	PI 269309	Good for	SW	28.1 abcdef	2	1	0.47 (0.41, 0.53)	0.28 (0.12, 0.45)	SC
		All Rikssort							
58	PI 357356	Okruglo	MC	28.8 abcdef	2	1	0.45 (0.36, 0.55)	0.33 (0.17, 0.49)	SS
20	PI 590627	Special Crosby	NI	28.8 abcdef	2	1	0.48 (0.43, 0.52)	0.24 (0.08, 0.40)	SC
31	PI 592989	Red Baron	US	29.0 abcdef	2	1	0.45 (0.36, 0.54)	0.33 (0.17, 0.49)	SC
79	PI 142820	Choghondar	Iran	29.1 abcdef	2	1	0.43 (0.31, 0.56)	0.37 (0.20, 0.53)	SC
29	PI 590636	Early Wonder/	NI	30.1 abcdef	2	1	0.48 (0.43, 0.53)	0.26 (0.09, 0.42)	SC
		Boston Crosby							
70	NSL 43404	Parma Globe	NI	30.3 abcdef	1	2	0.41 (0.28, 0.54)	0.39 (0.24, 0.54)	SC
78	PI 142810	Choghondar	Iran	30.6 abcdef	1	2	0.41 (0.28, 0.54)	0.39 (0.24, 0.54)	SC
43	PI 357357	Okrugla	MC	31.1 abcdef	2	1	0.44 (0.33, 0.55)	0.35 (0.19, 0.51)	SS
19	PI 612331	Detroit Vuurbal	NT	32.1 abcdef	2	1	0.44 (0.33, 0.55)	0.36 (0.20, 0.51)	QP
34	PI 612329	Regala	NT	32.6 bcdef	2	1	0.47 (0.42, 0.53)	0.27 (0.11, 0.44)	SC
10	PI 612340	Boltardy	NT	33.7 bcdef	2	3	0.47 (0.42, 0.53)	0.22 (0.10, 0.34)	SC
30	PI 590640	Detroit Dark	NI	34.3 bcdef	2	1	0.48 (0.43, 0.52)	0.26 (0.09, 0.42)	SS
		Red Garnet							
97	W364B ^s	W364B	US	40.5 cdef	1	2	0.44 (0.36, 0.52)	0.35 (0.24, 0.47)	SC
82	PI 164292	IDBBNR 5239	India	43.0 def	2	3	0.47 (0.42, 0.52)	0.22 (0.10, 0.34)	SC
27	PI 590595	Eastern Wonder	NI	43.6 ef	2	3	0.44 (0.33, 0.54)	0.28 (0.17, 0.39)	QP
56	PI 103042	Ken Ta Tsai	NI	43.8 f	2	1	0.47 (0.40, 0.54)	0.29 (0.13, 0.46)	SS

^zCode refers to the accessions in Supplemental Fig. 2 and Supplemental Table 2.

^yOrigin Code: US = United States, UK = United Kingdom, TK = Turkey, SW = Sweden, CN = Canada, PL = Poland, NT = Netherlands, MC = Macedonia, UZ = Uzbekistan, LB = Lebanon, HN = Hungary, AR = Armenia, BG = Bulgaria, AF = Afghanistan, DN = Denmark, SU = Soviet Union, ET = Ethiopia, SL = Sri Lanka, SA = South Africa, DE = Germany, NI = Not Included on Germplasm Resources Information Network.

^xHSD_{0.05} = 6.2. Different letters indicate significant differences as determined by Tukey pairwise comparisons at $\alpha = 0.05$.

^wDetermined through ordinal modeling. Predictions are based on combined data for fungus gnats, other diseases, and evaluation times. SE is the standard error of the probability of each most likely disease rating (DR).

^vSlopes between zero days postinoculation and the first evaluation period and the first evaluation period and second evaluation period were determined from spline regression and used to categorize accessions according to their disease progression behavior. Disease progression categories included the following: slow followed by spike (SS, Slope 1 ≤ 1.25 , Slope 2 > 1.25), quick followed by plateau (QP, Slope 1 > 1.25 , Slope 2 ≤ 1.25), quick constant (QC, Slope 1 > 1.25 , Slope 2 > 1.25), and slow constant (SC, Slope 1 ≤ 1.25 , Slope 2 ≤ 1.25).

^uFC709-2, resistant sugar beet control.

^tFC901/C817, susceptible sugar beet control.

^sW364B, table beet inbred control.

percentages were then weighted as described by the following equation:

$$\begin{aligned} & (\text{External Diseased Tissue Percentage} * 0.25) \\ & + (\text{Internal Diseased Tissue Percentage} * 0.75) \\ & = \text{Weighted Average Diseased Tissue Percentage} \end{aligned}$$

The weighted average was calculated to emphasize the internal root characteristics because those are of greater importance for processing beets (A. Bennett, Seneca Foods, personal communication). External lesions, which are closer to the root surface, can be peeled in processing without much loss of tissue (M. Badtke, Del Monte Foods, Inc., personal communication). Following the calculation of the weighted average diseased tissue percentage for one plant, the percentages for the two plants in a pot were averaged as subsamples, providing one averaged

weighted diseased tissue percentage for a single pot. These averages were then used for linear regression analysis and to calculate the area under the disease progression curve (AUDPC) (Madden et al., 2007). R code from Schandry (2017) was adapted to provide areas under the curve for each accession. Additional notes were collected regarding the presence of fungus gnat larvae and other diseases.

Statistical analysis. R statistical software (R 3.5.2; R Core Team, 2018) was used for all analyses (R code available from authors by request). Analysis of variance (ANOVA) was conducted using the “stats” package in R (R Core Team, 2018). Linear models were fitted using the “lmerTest” package (Kuznetsova et al., 2017). Models were fitted first for the Validation Screen because that experiment

included the highest number of samples per accession. The model for the Validation Screen included accession, inoculation status, evaluation time (3 and 5 weeks postinoculation), presence of fungus gnats, and presence of other diseases as fixed effects. Models were selected using the Akaike information criterion provided by the “stepAIC” function in the “MASS” package (Venables and Ripley, 2002) in combination with researcher discretion. ANOVAs were conducted for the weighted average diseased tissue percentages to evaluate the significance of differences in the level of resistance among the genotypes. Differences among accessions in the presence of *R. solani* were evaluated with the noninoculated controls excluded from the dataset. Following all significant ANOVAs ($P < 0.05$), Tukey's

Table 2. Mean diseased tissue percentage, most likely internal disease ratings and probabilities, and disease progression categories of 62 *Beta vulgaris* subsp. *vulgaris* commercial cultivars and publicly available inbred accessions in response to inoculation with *Rhizoctonia solani* AG 2-2 isolate R1 in a greenhouse screen evaluated in 2018.

Code ^z	Accession	Mean diseased tissue percentage ^y	First most likely internal DR ^x	Second most likely internal DR ^x	Probability of first most likely internal DR (SE) ^x	Probability of second most likely internal DR (SE) ^x	Disease progression ^w
64	FC709-2 ^v	6.6 a	1	0	0.49 (0.41, 0.57)	0.31 (0.06, 0.55)	SC
28	Long Season	16.3 ab	1	2	0.48 (0.37, 0.58)	0.32 (0.15, 0.49)	SC
12	Red Cloud	16.7 ab	1	2	0.41 (0.26, 0.57)	0.40 (0.26, 0.55)	SC
52	Albina Vereduna	19.2 ab	2	1	0.43 (0.31, 0.55)	0.38 (0.21, 0.55)	SC
65	W364B ^u	21.9 ab	1	0	0.47 (0.34, 0.59)	0.40 (0.14, 0.65)	SC
17	Bull's Blood	22.1 ab	1	2	0.46 (0.34, 0.58)	0.34 (0.18, 0.51)	SS
11	Boro	23.0 ab	2	3	0.47 (0.38, 0.55)	0.29 (0.14, 0.45)	SC
33	Perfected Detroit	23.0 ab	1	2	0.47 (0.37, 0.58)	0.32 (0.15, 0.49)	QP
10	Falcon	23.6 ab	1	2	0.49 (0.42, 0.57)	0.27 (0.10, 0.44)	SC
47	Carillon	23.9 ab	2	1	0.44 (0.33, 0.56)	0.37 (0.20, 0.53)	SC
45	Green Top Bunching	24.4 ab	2	1	0.46 (0.38, 0.55)	0.32 (0.15, 0.50)	SC
51	Sweetheart	25.5 ab	2	1	0.46 (0.37, 0.55)	0.33 (0.16, 0.50)	SC
61	Badger Flame	25.6 ab	2	1	0.46 (0.38, 0.55)	0.32 (0.14, 0.49)	SC
53	W443B	25.7 ab	2	1	0.48 (0.41, 0.54)	0.28 (0.09, 0.47)	QP
56	W427B	25.9 ab	2	1	0.48 (0.41, 0.54)	0.28 (0.10, 0.46)	SC
21	Babybeat	26.0 ab	2	1	0.48 (0.42, 0.53)	0.27 (0.10, 0.45)	SS
3	Bohan	26.1 ab	2	1	0.47 (0.40, 0.54)	0.30 (0.12, 0.48)	QP
63	FC901/C817 ^t	26.7 ab	1	2	0.48 (0.39, 0.58)	0.30 (0.12, 0.48)	SC
39	Pacemaker III	27.1 ab	2	1	0.41 (0.28, 0.55)	0.40 (0.24, 0.56)	SC
16	Early Wonder Tall Top	27.8 ab	1	2	0.46 (0.35, 0.58)	0.34 (0.18, 0.51)	SC
24	Chioggia Guardsmark	28.5 ab	1	2	0.47 (0.36, 0.58)	0.33 (0.17, 0.50)	SC
40	Eagle	28.5 ab	1	2	0.48 (0.40, 0.57)	0.30 (0.13, 0.47)	SC
15	Cylindra	29.2 ab	2	1	0.43 (0.30, 0.55)	0.38 (0.22, 0.55)	SS
18	Zeppo	29.6 ab	2	1	0.44 (0.33, 0.55)	0.36 (0.20, 0.53)	SS
46	Ruby Queen	29.7 ab	2	1	0.47 (0.40, 0.54)	0.31 (0.13, 0.48)	SS
37	Vulture	29.8 ab	1	2	0.46 (0.33, 0.58)	0.36 (0.19, 0.52)	QP
34	Boltardy	30.1 ab	2	1	0.43 (0.32, 0.55)	0.37 (0.21, 0.54)	QP
7	MacGregor's Favorite	31.8 ab	2	1	0.47 (0.41, 0.54)	0.29 (0.11, 0.47)	SS
20	Touchstone Gold	32.0 ab	1	2	0.41 (0.26, 0.56)	0.40 (0.26, 0.55)	QP
2	Soldier	32.0 ab	2	1	0.44 (0.33, 0.56)	0.36 (0.19, 0.53)	QP
4	Subeto	32.6 ab	2	1	0.47 (0.39, 0.55)	0.31 (0.13, 0.49)	SS
57	W446B ^s	33.0 ab	2	3	0.46 (0.33, 0.60)	0.30 (0.00, 0.60)	—
9	Flat of Egypt	33.5 ab	2	1	0.42 (0.29, 0.55)	0.40 (0.24, 0.56)	QP
50	Rhonda	33.5 ab	2	1	0.46 (0.36, 0.55)	0.33 (0.16, 0.51)	QP
43	Burpee's Red Ball	33.6 ab	1	2	0.46 (0.34, 0.57)	0.35 (0.20, 0.51)	QP
38	Solo	33.7 ab	2	1	0.47 (0.38, 0.55)	0.31 (0.14, 0.49)	QP
6	Pablo	34.0 ab	2	1	0.47 (0.38, 0.55)	0.31 (0.14, 0.49)	SS
25	Merlin	34.1 ab	2	3	0.47 (0.40, 0.54)	0.28 (0.13, 0.44)	QC
29	Letherman's Green Top	34.2 ab	2	1	0.44 (0.31, 0.56)	0.37 (0.21, 0.54)	QP
8	Lutz Green Leaf	34.5 ab	1	2	0.45 (0.32, 0.57)	0.36 (0.21, 0.52)	QP
14	Taurus	34.7 ab	2	3	0.47 (0.40, 0.54)	0.28 (0.12, 0.44)	SS
44	Albino	35.1 ab	2	1	0.43 (0.30, 0.55)	0.39 (0.22, 0.55)	QP
22	Moneta	35.6 ab	2	1	0.44 (0.33, 0.56)	0.36 (0.19, 0.53)	QP
30	Kestral	37.0 ab	2	1	0.48 (0.43, 0.53)	0.24 (0.07, 0.40)	QP
48	Detroit Dark Red	37.0 ab	2	1	0.44 (0.33, 0.56)	0.36 (0.20, 0.53)	QP
13	Detroit Supreme	37.9 abc	2	1	0.47 (0.40, 0.54)	0.30 (0.12, 0.47)	QP
35	Crosby Egyptian	38.8 abc	2	1	0.44 (0.34, 0.55)	0.36 (0.20, 0.52)	QP
27	White Detroit	38.9 abc	2	1	0.47 (0.39, 0.55)	0.31 (0.13, 0.48)	SS
54	W448B	39.3 abc	2	1	0.48 (0.41, 0.54)	0.28 (0.08, 0.48)	QP
26	First Crop	39.3 abc	2	1	0.47 (0.40, 0.54)	0.29 (0.12, 0.47)	QC
49	Red Ace	40.1 abc	2	3	0.48 (0.42, 0.53)	0.26 (0.10, 0.42)	QP
36	Paonazza D Egitto	40.4 abc	2	3	0.46 (0.37, 0.55)	0.31 (0.15, 0.46)	SS
23	Avalanche	41.3 abc	2	1	0.47 (0.41, 0.54)	0.29 (0.11, 0.47)	QC
41	Moulin Rouge	42.1 abc	2	3	0.48 (0.42, 0.53)	0.25 (0.10, 0.41)	QC
59	W424B	42.6 abc	2	3	0.47 (0.40, 0.54)	0.29 (0.13, 0.45)	QP
42	Burpee's Golden	43.5 abc	2	1	0.46 (0.36, 0.55)	0.33 (0.16, 0.51)	QC
32	Blankoma	49.0 abc	2	3	0.45 (0.35, 0.55)	0.32 (0.16, 0.49)	QC
5	Robin	50.2 abc	2	3	0.46 (0.36, 0.55)	0.32 (0.16, 0.47)	QC
55	W411B	51.2 abc	2	3	0.46 (0.35, 0.58)	0.30 (0.06, 0.54)	QC
1	Syngenta Golden	51.9 bc	3	2	0.43 (0.36, 0.51)	0.29 (0.12, 0.46)	SS
19	Boldor	53.2 bc	2	3	0.44 (0.33, 0.55)	0.34 (0.19, 0.49)	QC
60	W434B ^r	100.0 c	5	—	1.00 (1.00, 1.00)	—	—

^zCode refers to the accessions in the boxplots in Supplemental Fig. 3 and Supplemental Table 3.

^yHSD_{0.05} = 6.0. Different letters indicate significant differences as determined by Tukey pairwise comparisons at $\alpha = 0.05$.

^xDetermined through ordinal modeling. Predictions are based on combined data for fungus gnats, other diseases, and evaluation times. SE is the standard error of the probability of each most likely disease rating (DR).

^wSlopes between zero days postinoculation and the first evaluation period and the first evaluation period and second evaluation period were determined from spline regression and used to categorize accessions according to their disease progression behavior. Disease progression categories included the following: slow followed by spike (SS, Slope 1 ≤ 1.25 , Slope 2 > 1.25), quick followed by plateau (QP, Slope 1 > 1.25 , Slope 2 ≤ 1.25), quick constant (QC, Slope 1 > 1.25 , Slope 2 > 1.25), and slow constant (SC, Slope 1 ≤ 1.25 , Slope 2 ≤ 1.25).

^vFC709-2, resistant sugar beet control.

^uW364B, table beet inbred control.

^tFC901/C817, susceptible sugar beet control.

^sDisease progression category not provided as slopes could not be calculated, observations only at the second evaluation period.

^rSecond most likely internal disease rating, probability of second most likely internal disease rating, and disease progression category not provided due to only one observation.

Table 3. Mean diseased tissue percentage, most likely internal disease ratings and probabilities, and disease progression categories of 10 *Beta vulgaris* subsp. *vulgaris* commercial cultivars and publicly available accessions in response to inoculation with *Rhizoctonia solani* AG 2-2 isolate R1 in a greenhouse screen evaluated in 2019.

Code ^a	Accession	Mean diseased tissue percentage ^y	First most likely internal DR ^x	Second most likely internal DR ^x	Probability of first most likely internal DR (SE) ^x	Probability of second most likely internal DR (SE) ^x	Disease progression ^w
6	Badger Flame	29.4 a	2	1	0.37 (0.31, 0.42)	0.25 (0.18, 0.31)	SC
1	FC709-2 ^v	31.8 ab	2	1	0.37 (0.31, 0.42)	0.25 (0.19, 0.32)	SC
10	Detroit Supreme	36.9 abc	2	3	0.37 (0.32, 0.43)	0.26 (0.17, 0.35)	QP
7	Moneta	39.1 abc	2	3	0.37 (0.32, 0.43)	0.28 (0.19, 0.38)	QP
8	Soldier	42.2 bc	2	3	0.37 (0.32, 0.43)	0.29 (0.20, 0.39)	QC
4	W364B ^u	42.8 cd	2	1	0.35 (0.29, 0.42)	0.27 (0.21, 0.34)	QP
2	FC901/C817 ^t	45.6 cd	2	3	0.37 (0.31, 0.42)	0.25 (0.16, 0.33)	QC
9	Touchstone Gold	47.5 cd	2	3	0.37 (0.32, 0.42)	0.34 (0.24, 0.45)	QP
3	KDH13	53.5 d	3	2	0.42 (0.31, 0.52)	0.35 (0.30, 0.41)	QP
5	W411B	54.9 d	3	2	0.49 (0.36, 0.61)	0.31 (0.23, 0.40)	QC

^aCode refers to the accessions in Supplemental Fig. 1 and Supplemental Table 1.

^yHSD_{0.05} = 4.5. Different letters indicate significant differences as determined by Tukey pairwise comparisons at $\alpha = 0.05$.

^xDetermined through ordinal modeling. Predictions are based on combined data for fungus gnats, other diseases, and evaluation times. SE is the standard error of the probability of each most likely disease rating (DR).

^wSlopes between zero days postinoculation and the first evaluation period and the first evaluation period and second evaluation period were determined from spline regression and used to categorize accessions according to their disease progression behavior. Disease progression categories included the following: slow followed by spike (SS, Slope 1 ≤ 1.25 , Slope 2 > 1.25), quick followed by plateau (QP, Slope 1 > 1.25 , Slope 2 ≤ 1.25), quick constant (QC, Slope 1 > 1.25 , Slope 2 > 1.25), and slow constant (SC, Slope 1 ≤ 1.25 , Slope 2 ≤ 1.25).

^vFC709-2, resistant sugar beet control.

^uW364B, table beet inbred control.

^tFC901/C817, susceptible sugar beet control.

honestly significant difference test (at $\alpha = 0.05$) in “agricolae” was used to separate means (de Mendiburu, 2019). The Wilcoxon rank sum test for independent samples was used to validate the results of the PI and Commercial Screens. The weighted average diseased tissue percentages of the accessions in the Validation Screen were compared with their counterparts in the Commercial and PI Screens.

In spline regression models, AUDPC was included as the dependent variable to determine the slopes between zero days postinoculation to the first evaluation time and between the first and second evaluation periods. These slopes were then used to assign accessions to the following disease progression categories: slow followed by spike (Slope 1 ≤ 1.25 , Slope 2 > 1.25), quick followed by plateau (Slope 1 > 1.25 , Slope 2 ≤ 1.25), quick constant (Slope 1 > 1.25 , Slope 2 > 1.25), and slow constant (Slope 1 ≤ 1.25 , Slope 2 ≤ 1.25).

Linear regression has limitations when analyzing ordinal data such as those collected in these experiments. Liddell and Kruschke (2018) demonstrated that when ordinal variables are analyzed as metric, low rates of correct detection, inflated type I error rates, inversions of differences between groups, and distorted effect-size estimates can occur. Therefore, in addition to linear regression, we also conducted ordinal regression to analyze all data and predict the most likely disease rating for each accession. We used the “ordinal” package in R to fit ordinal models (Christensen, 2019) and a “cumulative” ordinal model as described by Bürkner and Vuorre (2019). Internal and external disease ratings were modeled separately to maintain the integrity of each rating. Because internal characteristics are of greater importance in the vegetable processing industry, we focus

on those in our presentation of the results. Because of constraints of ordinal modeling, subsamples were not averaged.

Results

Validation screen. Significant interactions were found for replicate (i.e., experimental run) with evaluation time, other diseases, and inoculation ($P < 0.001$, 0.05, and 0.05, respectively). However, replicate $\times$ accession interactions were not significant ($P > 0.05$). Therefore, replicate was excluded from the model and both replicates were combined for analysis. ANOVA showed main effects of accession, fungus gnats, other diseases, and inoculation to be highly significant for average weighted diseased tissue (Table 4).

Accession $\times$ other disease and accession $\times$ inoculation interactions were significant ($P < 0.01$ and 0.001, respectively).

Most inoculated plants exhibited diseased tissue ranging from 20% to 60% (Table 3, Supplemental Fig. 1) compared with the noninoculated checks in the Validation Screen. Noninoculated checks showed no to very little diseased tissue (less than 2%). The percentage of diseased tissue was higher in the 5-week postinoculation evaluation time period compared with the 3-week time period.

Ordinal regression predicted that, in the absence of fungus gnats and other diseases, many accessions were likely to have an internal disease rating of 1 or 2 (Table 3). In contrast, when fungus gnats and other diseases were present, these accessions were predicted to have an internal disease rating of a 2 or 3 (data not shown).

Ordinal models predicted the likelihood of accessions receiving a specific internal disease rating (Table 3). ‘Badger Flame’

and FC709-2 were the most resistant in the screen with mean diseased tissue percentages of 29 and 32, respectively. KDH13 and W411B were on the susceptible end, with mean diseased tissue percentages of 54 and 55, respectively. It is interesting to note that both ‘Badger Flame’ and FC709-2 were most likely to receive a 2 internal disease rating (both $P = 0.37$), followed by a 1 internal disease rating (both $P = 0.25$). On the other end, KDH13 and W411B were most likely to receive a 3 internal disease rating ($P = 0.42$ and 0.49, respectively), followed by a 2 internal disease rating ($P = 0.35$ and 0.31, respectively). Both of the most resistant accessions also demonstrated a disease progression that was slow constant (Table 3, Supplemental Fig. 1, Supplemental Table 1). The other eight accessions in this screen exhibited either a quick constant or quick followed by plateau disease progression.

PI screen. Replicate $\times$ accession interaction was not significant ($P = 0.95$), and therefore replicate was excluded from the model and replicates were combined for analysis. ANOVA demonstrated significant main effects contributing to the percentage of diseased tissue, including accession, fungus gnats, other diseases, evaluation time, and inoculation (Table 5).

In general, inoculated plants exhibited between 6% and 40% diseased tissue (Table 1, Supplemental Fig. 2) compared with no or very little diseased tissue (an average of 2%) in the noninoculated checks. The accessions in the PI Screen exhibited increased amounts of diseased tissue in the 5-week postinoculation evaluation period compared with the 3-week evaluation period.

Ordinal models predicted the likelihood of accessions receiving a specific internal disease rating (Table 1). Ames 22164 and PI 502294 were the most resistant in the

Fig. 1. Disease rating scale for *Beta vulgaris* subsp. *vulgaris* inoculated with *Rhizoctonia solani* AG 2-2 isolate R1. 0 = 0% diseased tissue, root surface clean with no visible lesions; 1 = 1% to 10% diseased tissue, superficial, scattered nonactive lesions; 2 = 11% to 30% of root affected; 3 = 31% to 60% of root affected; 4 = 61% to 99% of root blackened with rot extending into interior; and 5 = root 100% rotted and foliage is dead or dying.

Table 4. Analysis of variance for the average weighted diseased tissue percentage of 10 *Beta vulgaris* subsp. *vulgaris* commercial cultivars and publicly available accessions in response to inoculation with *Rhizoctonia solani* AG 2-2 isolate R1 in a greenhouse screen evaluated in 2019.

Source	df	Mean squares	Significance
Accession	9	1,312	***
Fungus gnats	1	17,799	***
Other diseases	1	3,035	***
Evaluation time	1	127	NS
Inoculation	1	11,080	***
Accession: fungus gnats	9	24	NS
Accession: other diseases	8	316	**
Accession: inoculation	2	1,438	***
Fungus gnats: other diseases	1	340	NS
Accession: fungus gnats: other diseases	4	122	NS
Residuals	169	103	

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

screen with mean diseased tissue percentages of 2 and 6, respectively. PI 590595 and PI 103042 were on the susceptible end, both with mean diseased tissue percentages of 44. Ames 22164 and PI 502294 were most likely to receive a 0 and 1 internal disease rating, respectively ($P = 0.56$ and 0.47 , respectively), followed by a 1 and 2 internal disease rating ($P = 0.37$ and 0.30 , respectively). On the other end, PI 590595 and PI 103042 were most likely to receive a 2 internal disease rating ($P = 0.44$ and 0.47 , respectively), followed by an internal disease rating of 3 and 1, respectively ($P = 0.28$ and 0.29 , respectively).

Predictions from ordinal modeling demonstrated the effect of fungus gnats and other diseases on the response of beets to inoculation with *R. solani*. In the presence of fungus gnats and other diseases, most accessions in the PI Screen were predicted to have an internal disease rating of 1 (Table 1). In contrast, when fungus gnats and other diseases were present, most accessions were predicted to have an internal disease rating of 2 (data not shown). Interactions between accession and fungus gnats and accession and inoculation were significant as well.

Many of the most resistant accessions in the PI Screen had mean diseased tissue percentages less than that of the resistant sugar beet check (Table 1, Supplemental Fig. 2). The 11 most resistant accessions also demonstrated a disease progression that was slow constant (Supplemental Fig. 2, Supplemental Table 2). Of 97 accessions, most at 67 had disease progressions that were slow constant, 28 were slow followed by spikes, and only two were quick followed by plateaus.

Commercial screen. Replicate $\times$ accession interactions were not significant ($P = 0.24$). We attributed replicate significance to the replicate $\times$ fungus gnat interaction ($P < 0.01$). Therefore, replicate was excluded from the model and both replicates were combined for analysis. ANOVA showed all main effects to be highly significant ($P < 0.01$) for average weighted diseased tissue (Table 6).

Most inoculated plants exhibited diseased tissue ranging from 16% to 50% (Table 2, Supplemental Fig. 3) compared with the noninoculated checks (an average of less than 1% diseased tissue) in the Commercial Screen. The percentage of diseased tissue was higher in the 5-week postinoculation

evaluation time period compared with the 3-week time period.

Ordinal models predicted the likelihood of accessions receiving a specific internal disease rating (Table 2). FC709-2 and Long Season were the most resistant in the screen with mean diseased tissue percentages of 7 and 16, respectively. Syngenta Golden, Boldor, and W434B were on the susceptible end, with mean diseased tissue percentages of 52, 53, and 100.0, respectively. Both FC709-2 and Long Season were most likely to receive a 1 internal disease rating ($P = 0.49$ and 0.48 , respectively), followed by an internal disease rating of 0 and 2, respectively ($P = 0.31$ and 0.32 , respectively). On the other end, Syngenta Golden, Boldor, and W434B were most likely to receive a 3, 2, and 5 internal disease rating, respectively ($P = 0.43$, 0.44 , 1.00 , respectively). The second most likely internal disease rating for Syngenta Golden and Boldor was a 2 and 3, respectively ($P = 0.29$ and 0.34 , respectively).

Ordinal regression predicted that, in the absence of fungus gnats and other diseases, many accessions were likely to have an internal disease rating of 1 (Table 2). In contrast, when fungus gnats and other diseases were present, these accessions were predicted to have an internal disease rating of a 2 or 3 (data not shown).

The most resistant accessions in the Commercial Screen had mean diseased tissue percentages similar to that of FC709-2 (Table 2, Supplemental Fig. 3). These resistant accessions also demonstrated a disease progression that was slow constant (Supplemental Fig. 3, Supplemental Table 3). Of 62 accessions, 17 had disease progressions that were slow constant, 12 slow followed by spikes, 22 quick followed by plateaus, and 9 quick constant.

Variance of mean diseased tissue percentage was 223.4, 263.9, and 300.4 across accessions for percent diseased tissue in the Validation, PI, and Commercial Screens,

Table 5. Analysis of variance for the average weighted diseased tissue percentage of 97 *Beta vulgaris* subsp. *vulgaris* PI accessions in response to inoculation with *Rhizoctonia solani* AG 2-2 isolate R1 in a greenhouse screen evaluated in 2018.

Source	df	Mean squares	Significance
Accession	96	254	***
Fungus gnats	1	39,659	***
Other diseases	1	4,817	***
Evaluation time	1	1,004	**
Inoculation	1	1,622	***
Accession: fungus gnats	83	156	**
Accession: other diseases	59	105	NS
Accession: inoculation	2	579	**
Fungus gnats: other diseases	1	543	*
Accession: fungus gnats: other diseases	1	71	NS
Residuals	153	93	

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

Table 6. Analysis of variance for the average weighted diseased tissue percentage of 62 *Beta vulgaris* subsp. *vulgaris* commercial cultivars and publicly available inbred accessions in response to inoculation with *Rhizoctonia solani* AG 2-2 isolate R1 in a greenhouse screen evaluated in 2018.

Source	df	Mean squares	Significance
Accession	61	534	***
Fungus gnats	1	13,865	***
Other diseases	1	2,882	***
Evaluation time	1	1,557	**
Inoculation	1	1,647	**
Accession: fungus gnats	51	190	NS
Accession: other diseases	31	196	NS
Accession: inoculation	2	113	NS
Fungus gnats: other diseases	1	32	NS
Residuals	104	162	

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

respectively, demonstrating consistency in the three screening experiments.

Discussion

The methods described for a controlled environment screen for *R. solani* in table beet provides a valuable assessment tool for table beet breeding programs. In our greenhouse experiments, we were able to create a screening environment that was repeatable and consistent. Using increased replications of accessions in the Validation Screen, we validated our findings from the PI and Commercial Screens. Even though each screen had a different mean diseased tissue percentage and range of values, the Validation Screen generally confirmed the relative performance of the two sugar beet control genotypes and a table beet inbred that were included in all three screens. Our screen was adapted from those conducted with sugar beet (Hecker and Ruppel, 1977b; L. Panella, personal communication; Ruppel et al., 1979). Many studies have demonstrated consistency between laboratory or greenhouse and field settings for sugar beet (Büttner et al., 2004; Campbell and Altman, 1976; Schneider et al., 1982; Scholten et al., 2001; Weiland et al., 1999), therefore, there is a high likelihood that our findings will be applicable in field environments for table beet.

We identified substantial variation in diseased tissue among all three types of accessions we evaluated, demonstrating a range of reaction to inoculation with *R. solani* AG 2-2 in table beet. These results are not particularly surprising given that there has been little

direct selection in table beet for resistance to *R. solani*. The two sugar beet accessions we included as controls performed comparably to the same or similar accessions in screens completed in other sugar beet breeding programs (Hanson, 2010; Panella, 1999; Strausbaugh et al., 2013a). In our and other screens, FC709-2 had less diseased tissue compared with FC901/C817 (Panella, 1999; Panella and Hanson, 2001; Strausbaugh et al., 2013a). We found some table beet accessions that performed as well as the resistant sugar beet check genotype, suggesting promise for breeding in table beet. Some of the top performing accessions included 'Badger Flame', 'Long Season', 'Red Cloud', 'Albina Vereduna', and numerous PIs. Many of these accessions had AUDPCs similar to or less than that of that of the resistant sugar beet check.

Many of the more resistant accessions in our experiments displayed a disease progression that was slow constant (Tables 1–3, Supplemental Figs. 1–3, Supplemental Tables 1–3). Accessions with higher mean diseased tissue percentages exhibited a disease progression that was slow followed by spike, quick followed by plateau, or quick constant. In the Commercial Screen especially, most of the accessions at the susceptible end displayed a quick constant disease progression. Although some variability may occur between the first and second evaluation periods due to individual plant differences as well as variability in response over time, the disease progression categories obtained from the slopes of the AUDPC curve may be useful for informing selection programs aimed at improving resistance. Selection for the slow

constant disease progression type may result in germplasm with greater resistance to *R. solani*. Planting of accessions with a slow constant disease progression may also provide more flexibility for harvest windows for growers.

We were also interested in determining the best method of measuring diseased tissue percentages using both external and internal observations. Using the ratio of 75 internal:25 external diseased tissue percentages, we found that most accessions ranged between a disease rating of 1 and 3. Using this weighted average, variances for percent diseased tissue were relatively consistent across the screens with variances of 223.4, 263.9, and 300.4 for mean disease tissue percentage in the Validation, PI, and Commercial Screens, respectively. However, had we focused on strictly external diseased tissue, most accessions would have ranged between a disease rating of 1 and 5 and we would have found variances of 444.3, 856.6, and 722.9 for mean disease tissue percentage across accessions in the Validation, PI, and Commercial Screens, respectively. If we had only considered internal diseased tissue, most accessions would have received a disease rating between 0 and 3 and mean disease tissue percentage variances were tighter at 236.8, 194.1, and 277.2 for the Validation, PI, and Commercial Screens, respectively. This finding indicates that there was more variation in the external root symptoms compared with their internal symptoms. Other studies have observed variation in plant parts in response to inoculation with *R. solani* AG 2-2 (Scholten et al., 2001). In greenhouse studies, Scholten et al. (2001) observed that leaf symptoms in sugar beet plants were widely variable compared with root symptoms and not correlated. In addition, rank changes among accessions occurred when either internal or external ratings were considered separately. This is because some accessions, like 'Eagle', had a large amount of diseased tissue on the root surface and very little diseased tissue internally. Other accessions, like 'Red Ace', had more extensive internal disease, with less externally. We focused on the internal symptoms in our studies because that is of greater importance in the table beet processing industry. In sugar beet experiments with *R. solani*, disease evaluations are typically done using root surface area (Ruppel et al., 1979; Scholten et al., 2001). Gaining a better understanding of the relationship between external and internal characteristics may allow for increased efficiency in disease evaluations. Further, the differences in internal and external variances suggest that genetic variation for external and internal disease reaction may be inherited separately in table beet.

We had expected the PI accessions to have the greatest amount of variation given that germplasm bank accessions typically exhibit large amounts of variation for performance traits (Frese et al., 2001; Panella and Lewellen, 2007). For example, of the 803 *B. vulgaris* subsp. *vulgaris* accessions classified

as sugar beet type previously screened for *R. solani* AG 2-2, more than 50 accessions were rated at a 3 or lower on a 0 to 9 or very resistant to highly susceptible disease rating scale (GRIN, 2020). Sugar beet cyst nematode screens of NPGS *Beta* germplasm have shown that a number of accessions were segregating for resistance to the nematode (Panella and Lewellen, 2007). Prebreeding populations of sugar beet derived from PIs also demonstrated segregation for reaction to the destructive oomycete, *Aphanomyces cochlioides* Dresch (Panella and Lewellen, 2007). Surprisingly, we observed larger variation among commercial cultivars than among PI accessions. This might be because some of the accessions in the Commercial Screen are open-pollinated (OP) or heirloom cultivars, whereas others are F₁ hybrids. Some examples of each of these are 'Soldier' and 'Ruby Queen' (OP/heirloom) and 'Red Ace' and 'Boldor' (hybrids) with variances in diseased tissue percentage of 106.6, 366.9, 271.8, and 472.6, respectively. Hybrids generally exhibited greater variation than heirloom and OP cultivars, whereas inbred lines exhibited the lowest amount of variation.

Some of the most resistant accessions in the PI Screen were commercial cultivars that had been contributed to the NPGS, but there were many other resistant accessions that were not cultivars. This provides additional evidence for the utility of the table beet germplasm in the NPGS for increasing resistance to *R. solani* AG 2-2 in table beet lines. However, the accessions in the Commercial Screen displayed a smaller increase in mean diseased tissue percentage from the 3- to 5-week postinoculation evaluation periods compared with a greater increase in mean diseased tissue percentage in the PI Screen. This finding indicates that many commercial accessions may be better able to resist the progression of disease, and may be an important consideration in breeding for disease resistance and disease management from a grower perspective.

The predictions provided by ordinal regression can be used to make better informed decisions when selecting germplasm for breeding and selecting varieties for production. Predictions can be used to estimate how much diseased tissue can be expected on an accession when *R. solani* is present. We observed significant interactions between accession $\times$ fungus gnats, accession $\times$ other diseases, and accession $\times$ inoculation. It is expected that accessions with different genetic backgrounds will respond differently to environmental factors. The role of environment and microclimate surrounding individual plants in the development of Rhizoctonia root and crown rot on sugar beet has been noted by Hecker and Ruppel (1977b). Others have examined the effects of temperature and moisture on *R. solani* (Bolton et al., 2010; Engelkes and Windels, 1994; Kirk et al., 2008; McKeen, 1949). Bolton et al. (2010) observed a positive correlation between root disease index and increased temperature and soil moisture.

We noted that when fungus gnats and other diseases were present, accessions were more likely to exhibit an increased percentage of diseased tissue. Such relationships or disease complexes for *R. solani* have been described with *Fusarium oxysporum* f. sp. *radis-betae* (Harveson and Rush, 1994; Jacobsen, 2006), *Pythium* spp. (Abawi et al., 1986), and *A. cochlioides* (Harveson and Rush, 1994; Harveson et al., 2002). In Michigan, sugar beets with resistance to *R. solani* were found to have more severe root rot when inoculated with both *R. solani* and *Rhizopus stolonifer* (Ehrenberg) Vuillemin than either fungus by itself (Hanson, 2010). This is especially noteworthy, because the growing conditions in this study were not conducive to disease production by *R. stolonifer* on its own. The relationship between *R. solani* and bacteria in root rot complexes was investigated by Strausbaugh and Gillen (2009). In field studies, fungal and bacterial rot only developed on plants inoculated with *R. solani* in the crown area, when no fungal infection was present, neither was bacterial rot (Strausbaugh and Gillen, 2009). Later experiments showed a doubled amount of root rot when both the bacteria *Leuconostoc mesenteroides* subsp. *dextranicum* (Beijerinck) Garvie and *R. solani* AG 2-2 IIIB were inoculated on sugar beets compared with *R. solani* alone and no rot when *L. mesenteroides* was inoculated alone, further demonstrating synergism between the bacteria and fungus (Strausbaugh et al., 2013b).

Interactions between fungi and insects may also be a factor in the response to *R. solani* and Hatcher (1995) provides an extensive review of relationships between pathogenic fungi, insects, and host plants. Increased levels of root damage and decreased alfalfa yield occurred when both clover root curculio, *Sitona hispidulus* (Fabricius), and a complex of soilborne root rot fungi were present compared with either pest alone (Godfrey and Yeagan, 1987). Caesar (2003) observed a synergistic relationship when *R. solani* and *F. oxysporum* were inoculated in combination with *Aphthona* spp., flea beetle, leading to increased damage on the roots on *Euphorbia esula* var. *virgata*. These synergistic relationships between *R. solani* and other plant pathogenic organisms are of great importance for management practices and breeding. There is a need for the development of beet lines with resistance to multiple diseases (Gaskill et al., 1970; Harveson and Rush, 1994; Jacobsen et al., 2004). Incorporating multiple disease resistances will be time-consuming, as resistance to several of the most destructive pathogens, including *Rhizoctonia* (Afanasiev and Sharp, 1961; Hecker and Ruppel, 1975) and *Aphanomyces* (Afanasiev and Sharp, 1961) is polygenic.

These experiments provide a good foundation for the study of resistance to *R. solani* in table beet. However, there are many areas that can be explored further to gain a better understanding of the nature of resistance in table beet, one of which is exploring possible

interactions between *R. solani* AG 2-2 isolates and table beet genotype. In our experiments, we used isolate R1 to inoculate the beets, because it is the isolate of choice for other sugar beet disease research programs (L. Hanson, personal communication). In early experiments, Herr (1970) inoculated eight sugar beet breeding lines developed by the USDA-ARS Crops Research Laboratory with three highly virulent Ohio *R. solani* isolates and discovered that the breeding lines were also resistant to Ohio isolates. Other experiments demonstrated no interactions between *R. solani* isolates from sugar beet roots and sugar beet lines (Ruppel, 1972). Hecker and Ruppel (1977b) tested six *R. solani* isolates on both resistant and susceptible sugar beets and found no significant interaction between fungal strain and sugar beet genotype. Hecker and Ruppel (1977b) also discussed experiments done by Sugimoto in Hokkaido, Japan, that confirmed similar levels of resistance exhibited by FC 701/2 and FC702/2 inoculated with two Hokkaido *R. solani* isolates. These two breeding lines exhibited the highest level of resistance in Sugimoto's field and greenhouse experiments (Hecker and Ruppel, 1977b). The applicability of resistance to Colorado isolates was again demonstrated by Windels et al. (1995) when six resistant lines were inoculated with Colorado *R. solani* AG 2-2 IIIB isolate R9 and four highly virulent Minnesota isolates in field experiments in both Colorado and Minnesota (Strausbaugh et al., 2011). However, in the 2000s some of these resistant sugar beet cultivars were not performing as expected in the U.S. Intermountain West (IMW) region. Greenhouse pathogenicity tests with 94 IMW region isolates on a susceptible sugar beet line led Strausbaugh et al. (2009) to recommend more study was needed for the isolates used for host resistance selection. On further investigation, Strausbaugh et al. (2013a) determined that although interactions with strains of *R. solani* AG 2-2 IIIB were not present with the susceptible and highly resistant checks (FC901/C817 and FC 709-2, respectively), sugar beet lines with intermediate levels of resistance had significant interactions with the nine isolates tested. This supports the necessity of carefully selecting isolates for screening in disease breeding nurseries. It would be interesting to compare how different isolates of *R. solani* AG 2-IIIB affect table beets. Collecting isolates from local fields would allow researchers to screen germplasm against the isolates most specific to their geographic region, allowing for local adaptation of germplasm.

To screen germplasm with *R. solani* on table beet plants while containing the pathogen, we conducted our experiments in greenhouses. Despite being in a controlled environment and making applications of *B. thuringiensis* ssp. *israelensis*, fungus gnats and symptoms atypical of *R. solani*, including brown and gray tissue and vascular discoloration, were present on the specimens.

Although other diseases are likely to be present in a field environment, their presence in our experiments made it more difficult to precisely measure the performance of germplasm in response to the fungus. Several studies have been conducted by inoculating seeds and seedlings in sugar beet (Bolton et al., 2010; Liu et al., 2019; Nagendran et al., 2009). In his discussion of breeding for resistance to *Rhizoctonia* in sugar beet, Gaskill (1968) mentions that resistance in mature sugar beets developed through selection was ineffective at preventing losses to *Rhizoctonia* in seedlings. This was later supported by Bolton et al. (2010) when several AGs and ISGs were capable of causing disease on sugar beet seedlings despite host resistance. A recent study by Liu et al. (2019) examined commercial cultivars that were susceptible, moderately resistant, and resistant to *R. solani* and found that no significant resistance to AG 2-2 IIIB was exhibited by the resistant cultivars until 4 and 5 weeks of age. In a study investigating host-pathogen interactions of *R. solani* and sugar beet seedlings, EL51, a line with resistance to *Rhizoctonia* crown and root rot, was discovered to have damping off resistance to AG 2-2 IIIB when inoculated 2 weeks after germination (Holloin et al., 2000; Nagendran et al., 2009). Another approach might be to directly inoculate portions of table beet roots in a laboratory environment. In studies of plant pathogens affecting leaves, such experiments are referred to as “detached leaf assays” (Nowakowska et al., 2014). Such procedures have also been referred to as “direct challenge assays” (Woodward and Pearce, 1988) and may allow for evaluation of more precise responses to *R. solani*. The results presented here demonstrate a 13-week greenhouse screening method to separate genotypes in their response to inoculation by *R. solani*, identify useful germplasm, and make progress toward breeding for host resistance in table beet.

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Supplemental Fig. 3. The response of 62 *Beta vulgaris* subsp. *vulgaris* commercial cultivars and publicly available inbred accessions to inoculation with *Rhizoctonia solani* AG 2-2 isolate R1 in a greenhouse screen. Accession codes are in Table 2. Slopes between zero days postinoculation and the first evaluation period and the first evaluation period and second evaluation period were determined from spline regression and used to categorize accessions according to their disease progression behavior. Disease progression categories included the following: slow followed by spike (Slope 1 $\leq$ 1.25, Slope 2 > 1.25), quick followed by plateau (Slope 1 > 1.25, Slope 2 $\leq$ 1.25), quick constant (Slope 1 > 1.25, Slope 2 > 1.25), and slow constant (Slope 1 $\leq$ 1.25, Slope 2 $\leq$ 1.25). [○]FC709-2, resistant sugar beet control; [●]FC901/C817, susceptible sugar beet control; [□]W364B, table beet inbred control.

Supplemental Table 1. The slopes predicted by spline regression for 10 *Beta vulgaris* subsp. *vulgaris* commercial cultivars and publicly available accessions to inoculation with *Rhizoctonia solani* AG 2-2 isolate R1. Accession codes are in Table 3. Slope 1 was calculated for the diseased tissue percentage between 0 and 3 weeks postinoculation. Slope 2 was calculated between 3 and 5 weeks postinoculation. Slopes were then used to assign each accession to a disease progression categories according to the following: slow followed by spike (Slope 1 $\leq$ 1.25, Slope 2 > 1.25), quick followed by plateau (Slope 1 > 1.25, Slope 2 $\leq$ 1.25), quick constant (Slope 1 > 1.25, Slope 2 > 1.25), and slow constant (Slope 1 $\leq$ 1.25, Slope 2 $\leq$ 1.25).

Accession code	Slope 1 (SE)	Slope 2 (SE)
1 ^z	1.23 (0.47)	0.91 (1.35)
2 ^y	1.69 (0.47)	1.61 (1.35)
3	2.30 (0.47)	0.61 (1.34)
4 ^x	2.06 (0.47)	-0.33 (1.35)
5	2.21 (0.47)	1.57 (1.55)
6	1.04 (0.47)	1.23 (0.56)
7	1.52 (0.47)	1.09 (1.35)
8	1.52 (0.47)	1.64 (1.35)
9	2.06 (0.47)	0.47 (1.35)
10	1.57 (0.47)	0.51 (1.35)

^zFC709-2, resistant sugar beet control.

^yFC901/C817, susceptible sugar beet control.

^xW364B, table beet inbred control.

Supplemental Table 2. The slopes predicted by spline regression for 97 *Beta vulgaris* subsp. *vulgaris* PI accessions to inoculation with *Rhizoctonia solani* AG 2-2 isolate R1. Accession codes are in Table 1. Slope 1 was calculated for the diseased tissue percentage between 0 and 3 weeks postinoculation. Slope 2 was calculated between 3 and 5 weeks postinoculation. Slopes were then used to assign each accession to a disease progression categories according to the following: slow followed by spike (Slope 1 $\leq$ 1.25, Slope 2 > 1.25), quick followed by plateau (Slope 1 > 1.25, Slope 2 $\leq$ 1.25), quick constant (Slope 1 > 1.25, Slope 2 > 1.25), and slow constant (Slope 1 $\leq$ 1.25, Slope 2 $\leq$ 1.25).

Accession code	Slope 1 (SE)	Slope 2 (SE)
1	0.47 (0.59)	1.06 (0.95)
2	1.25 (0.58)	-0.90 (2.34)
3	0.51 (0.57)	0.06 (2.33)
4	1.06 (0.57)	-0.33 (2.26)
5	0.22 (0.59)	3.08 (2.36)
6	0.51 (0.56)	1.22 (2.29)
7	0.51 (0.57)	0.19 (2.31)
8	0.31 (0.58)	2.39 (2.32)
9	0.65 (0.59)	1.11 (2.26)
10	1.11 (0.57)	0.40 (2.22)
11	0.27 (0.57)	1.85 (2.31)
12	0.23 (0.57)	0.95 (2.26)
13	0.67 (0.59)	0.42 (2.38)
14	0.32 (0.57)	2.09 (2.31)
15	0.51 (0.57)	1.28 (2.30)
16	0.18 (0.56)	1.75 (2.29)
17	0.46 (0.57)	2.29 (2.30)
18	0.24 (0.56)	2.33 (2.29)
19	1.31 (0.57)	-0.50 (2.42)
20	1.15 (0.57)	-0.38 (2.38)
21	0.83 (0.57)	0.79 (2.31)
22	0.27 (0.59)	0.94 (2.38)
23	0.70 (0.57)	0.50 (2.33)
24	0.21 (0.57)	1.51 (2.22)
25	0.15 (0.57)	2.20 (2.39)
26	0.58 (0.57)	0.22 (2.26)
27	1.78 (0.57)	-0.62 (2.38)
28	0.47 (0.57)	1.14 (2.33)
29	0.88 (0.57)	0.78 (2.26)
30	0.27 (0.59)	3.92 (2.36)
31	1.17 (0.59)	-0.22 (2.36)
32	0.18 (0.59)	1.94 (2.26)
33	0.70 (0.59)	0.46 (2.31)
34	1.06 (0.59)	0.60 (2.26)
35	0.54 (0.57)	1.73 (2.26)
36	0.45 (0.58)	1.71 (2.23)
37	0.28 (0.59)	0.99 (2.30)
38	0.13 (0.58)	3.05 (2.32)
39	0.20 (0.57)	1.61 (2.26)
40	0.49 (0.59)	1.48 (2.26)
41	0.14 (0.59)	1.28 (2.38)
42	0.11 (0.57)	1.64 (2.27)
43	0.27 (0.57)	3.08 (2.26)
44	0.30 (0.59)	2.32 (2.30)
45	0.10 (0.59)	1.43 (2.31)
46	0.13 (0.57)	1.40 (2.31)
47	0.10 (0.59)	0.87 (2.26)
48	0.47 (0.57)	0.80 (2.27)
49	0.38 (0.59)	1.45 (2.30)
50	0.54 (0.57)	0.47 (2.26)
51	0.23 (0.59)	1.59 (2.36)
52	0.94 (0.57)	0.09 (2.27)
53	0.34 (0.59)	0.46 (2.31)
54	0.38 (0.57)	1.05 (2.27)
55	0.71 (0.59)	0.18 (2.43)
56	0.83 (0.59)	3.17 (2.36)
57	0.14 (0.59)	0.30 (2.31)
58	0.56 (0.57)	2.36 (2.39)
59	0.11 (0.58)	1.63 (2.32)
60	0.88 (0.59)	0.38 (2.38)
61	0.33 (0.59)	1.30 (2.30)
62	0.09 (0.59)	1.10 (2.30)
63	0.75 (0.58)	0.40 (2.23)
64	0.55 (0.57)	2.08 (2.38)
65	0.10 (0.59)	1.24 (2.26)
66	0.20 (0.57)	1.35 (2.39)
67	0.42 (0.58)	1.06 (2.30)
68	0.06 (0.56)	0.02 (2.29)

(Continued on next page)

Supplemental Table 2. (Continued) The slopes predicted by spline regression for 97 *Beta vulgaris* subsp. *vulgaris* PI accessions to inoculation with *Rhizoctonia solani* AG 2-2 isolate R1. Accession codes are in Table 1. Slope 1 was calculated for the diseased tissue percentage between 0 and 3 weeks postinoculation. Slope 2 was calculated between 3 and 5 weeks postinoculation. Slopes were then used to assign each accession to a disease progression categories according to the following: slow followed by spike (Slope 1 $\leq$ 1.25, Slope 2 $>$ 1.25), quick followed by plateau (Slope 1 $>$ 1.25, Slope 2 $\leq$ 1.25), quick constant (Slope 1 $>$ 1.25, Slope 2 $>$ 1.25), and slow constant (Slope 1 $\leq$ 1.25, Slope 2 $\leq$ 1.25).

Accession code	Slope 1 (SE)	Slope 2 (SE)
69	0.50 (0.59)	2.29 (2.47)
70	0.77 (0.57)	1.47 (2.31)
71	0.49 (0.58)	1.12 (2.32)
72	0.55 (0.57)	1.05 (2.42)
73	0.16 (0.59)	0.38 (2.38)
74	0.19 (0.59)	1.67 (2.43)
75	0.30 (0.57)	1.04 (2.27)
76	0.48 (0.57)	0.69 (2.27)
77	0.15 (0.59)	1.16 (2.26)
78	0.69 (0.57)	1.67 (2.27)
79	0.92 (0.59)	0.70 (2.31)
80	0.89 (0.58)	-0.41 (2.23)
81	0.07 (0.57)	2.97 (2.38)
82	1.20 (0.57)	1.30 (2.22)
83	0.37 (0.59)	1.85 (2.38)
84	0.97 (0.57)	-0.74 (2.38)
85	0.90 (0.57)	-0.24 (2.33)
86	0.30 (0.57)	-0.21 (2.28)
87	0.40 (0.57)	1.41 (2.26)
88	0.71 (0.57)	0.53 (2.43)
89	0.29 (0.57)	0.68 (2.26)
90	0.80 (0.59)	0.37 (2.31)
91	0.52 (0.59)	1.28 (2.36)
92	0.85 (0.57)	-0.47 (2.26)
93	0.22 (0.58)	0.82 (2.39)
94	0.25 (0.58)	0.96 (2.32)
95 ^z	0.54 (0.57)	0.80 (2.26)
96 ^y	0.29 (0.57)	1.35 (2.27)
97 ^x	1.14 (0.48)	1.81 (1.87)

^zFC901/C817, susceptible sugar beet control.

^yFC709-2, resistant sugar beet control.

^xW364B, table beet inbred control.

Supplemental Table 3. The slopes predicted by spline regression for 62 *Beta vulgaris* subsp. *vulgaris* commercial cultivars and publicly available inbred accessions to inoculation with *Rhizoctonia solani* AG 2-2 isolate R1. Accession codes are in Table 2. Slope 1 was calculated for the diseased tissue percentage between 0 and 3 weeks postinoculation. Slope 2 was calculated between 3 and 5 weeks postinoculation. Slopes were then used to assign each accession to disease progression categories according to the following: slow followed by spike (Slope 1 $\leq$ 1.25, Slope 2 $>$ 1.25), quick followed by plateau (Slope 1 $>$ 1.25, Slope 2 $\leq$ 1.25), quick constant (Slope 1 $>$ 1.25, Slope 2 $>$ 1.25), and slow constant (Slope 1 $\leq$ 1.25, Slope 2 $\leq$ 1.25).

Accession code	Slope 1 (SE)	Slope 2 (SE)
1	1.11 (0.86)	4.74 (1.35)
2	1.63 (0.86)	-1.05 (3.26)
3	1.31 (0.86)	-0.72 (3.26)
4	0.82 (0.86)	2.50 (3.26)
5	1.58 (0.86)	2.33 (3.26)
6	0.96 (0.86)	2.19 (3.26)
7	0.87 (0.86)	2.17 (3.26)
8	1.30 (0.86)	0.81 (3.26)
9	1.37 (0.86)	0.36 (3.26)
10	1.18 (0.86)	-0.71 (3.26)
11	0.90 (0.86)	0.32 (3.26)
12	0.47 (0.86)	1.06 (3.26)
13	1.62 (0.86)	0.08 (3.26)
14	1.08 (0.86)	1.76 (3.26)
15	0.81 (0.86)	1.87 (3.26)
16	1.11 (0.86)	0.36 (3.26)
17	0.41 (0.86)	2.31 (3.26)
18	0.67 (0.86)	2.59 (3.26)
19	1.68 (0.86)	2.42 (3.18)
20	1.29 (0.86)	0.35 (3.26)
21	0.44 (0.86)	2.87 (3.26)
22	1.41 (0.86)	0.52 (3.26)
23	1.28 (0.86)	2.05 (3.26)
24	0.94 (0.86)	1.14 (3.18)
25	1.49 (0.87)	2.60 (3.32)
26	1.30 (0.86)	1.68 (3.26)
27	1.19 (0.86)	2.08 (3.26)
28	0.66 (0.86)	0.15 (3.26)
29	1.38 (0.86)	0.45 (3.26)
30	1.59 (0.86)	0.14 (3.26)
32	1.69 (0.86)	1.8 (3.26)
33	1.28 (0.86)	-1.26 (3.26)
34	1.34 (0.86)	-0.09 (3.26)
35	1.61 (0.87)	0.28 (3.32)
36	0.85 (0.86)	3.72 (3.26)
37	1.63 (0.86)	-1.19 (3.15)
38	1.25 (0.86)	0.75 (3.15)
39	1.08 (0.87)	0.40 (3.32)
40	1.04 (0.86)	0.75 (3.18)
41	1.29 (0.87)	2.04 (3.32)
42	1.51 (0.86)	1.42 (3.18)
43	1.45 (0.86)	-0.01 (3.18)
44	1.29 (0.86)	0.90 (3.18)
45	0.90 (0.86)	0.56 (3.15)
46	0.58 (0.86)	2.47 (3.15)
47	1.12 (0.87)	-0.30 (3.32)
48	1.44 (0.87)	0.57 (3.32)
49	1.69 (0.86)	0.04 (3.15)
50	1.43 (0.86)	0.11 (3.15)
51	1.17 (0.86)	-0.25 (3.15)
52	0.79 (0.86)	0.14 (3.15)
53	1.62 (0.86)	-1.76 (3.15)
54	2.11 (0.86)	-1.58 (3.18)
55	1.47 (0.96)	2.26 (3.39)
56	1.10 (0.86)	0.12 (3.15)
57 ^z	—	—
59	1.58 (0.86)	0.94 (3.15)
60 ^y	—	—
61	0.98 (0.86)	0.42 (3.15)
63 ^x	1.09 (0.87)	0.76 (3.32)
64 ^w	0.15 (0.86)	0.50 (3.15)
65 ^v	0.98 (0.41)	-0.16 (3.15)

^zSlopes not provided, observations only at the second evaluation period.

^ySlopes not provided, only one observation.

^xFC901/C817, susceptible sugar beet control.

^wFC709-2, resistant sugar beet control.

^vW364B, table beet inbred control.