PREVIOUS CROP INFLUENCES RHIZOCTONIA ON SUGARBEET

Carol E. Windels and Jason R. Brantner
Professor and Research Fellow of Plant Pathology, respectively
University of Minnesota, Northwest Research and Outreach Center, Crookston

Rhizoctonia solani is a common, soil-borne fungal pathogen of crops grown throughout the world. It is a complex fungus composed of genetically isolated populations called anastomosis groups or AGs (6). In the last decade, *R. solani* AG 2-2 has increased in prevalence and severity on sugarbeet in the Red River Valley (RRV). This trend is attributed to various factors that favor a gradual build up of inoculum in soil including wet summers and increased production of bean crops (also susceptible to stem and root rot caused by *R. solani* AG 2-2) in close rotation with sugarbeet. Between 1992 and 2003, production of soybean (*Glycine max* [L.] Merr.) in the RRV increased 182% from 817,700 to 2,307,200 acres; production of edible dry beans remained fairly stable with 323,600 and 306,000 acres sown in 1992 and 2003, respectively (4).

There are two specialized populations (intraspecific groups) within *R. solani* AG 2-2 known as IIIB and IV. *R. solani* AG 2-2IIIB tends to attack soybean and AG 2-2IV tends to attack sugarbeet, although both populations can attack both crops (2, 6, 9). In fact, *R. solani* AG 2-2IIIB is the main cause of Rhizoctonia root and crown rot on sugarbeet in Europe (3). In Germany (3) and in the southeast United States (10, 11), AG 2-2IIIB also causes Rhizoctonia crown and brace root rot on corn (not yet observed in the midwestern states, including Minnesota). The intraspecific groups can be separated by molecular techniques (3, 9) and also have different temperature requirements; *R. solani* AG 2-2IIIB grows at 95 ^{0}F but AG 2-2IV does not (9).

No data currently are available to document prevalence of *R. solani* AG 2-2, or it's intraspecific groups, on soybean and edible beans in Minnesota and North Dakota. Although our laboratory has identified *R. solani* AG 2-2 on sugarbeet in Minnesota and North Dakota, only limited research has been done to identify the intraspecific groups of this pathogen (2). In theory, both *R. solani* AG 2-2IIIB and AG 2-2IV occur in the RRV and southern Minnesota because bean crops and sugarbeet are grown in these geographic regions (one intraspecific group could predominate in a particular area or field). If *R. solani* AG 2-2IIIB is present, corn production could pose an increased risk for building up inoculum pathogenic to bean and sugarbeet crops. In 2003, corn was grown on about 622,000 acres in the RRV and on nearly 3 million acres in the sugarbeet-growing counties of the Southern Minnesota Beet Sugar Cooperative (4).

OBJECTIVE:

Our objective was to determine the effect of previous crops grown in soil infested with *R. solani* AG-2-2 IIIB on Rhizoctonia diseases on sugarbeet.

MATERIALS AND METHODS

Crops grown in 2003. On May 15, 2003, a trial was established at the University of Minnesota, Northwest Research and Outreach Center, Crookston. Plots were inoculated with an isolate of *R. solani* AG 2-2IIIB obtained from a field in a pinto bean – sugarbeet rotation; the isolate caused stem and root rot of bean crops (soybean, pinto bean, and navy bean) as well as root and crown rot of sugarbeet (2). Inoculum of the pathogen was grown on sterile barley grain for 3 weeks and then air-dried. Barley grain inoculum (3.8 ounces) was sprinkled over an 11 x 30 ft portion of each 22 x 30 ft plot and incorporated by raking to about a 2-inch depth. Later in the day, fungicide-treated seed of several crops rotated with sugarbeet were sown: soybean and pinto bean at 60 and 80 lb of seed/A, respectively; and sunflower and corn, each at 30,000 seed/A. Wheat was sown at 90 lb/A on May 16, 2003. There were two fallow controls: *R. solani*-inoculated and non-inoculated. Plots were arranged in a randomized block design of four replicates. Each crop was fertilized and maintained following recommended practices. The trial was cultivated twice (June 16 and July 8) and hand-weeded as needed, except in fallow plots which were treated with

herbicides. Near harvest, several plants were removed from plots and roots were visually inspected for rot and lesions typical of *R. solani*. Crops were harvested when ripe. Plots were chisel-plowed in October, 2003.

Sugarbeet grown in 2004. The trial was fertilized for maximum sugarbeet yield and quality based on analysis of soil samples collected from plots of each 2003 crop treatment. Seed of sugarbeet 'Beta 2820' was sown 1.5 inches apart (approximately 240 seed/30-ft row) in 22-inch rows on May 18, 2004. The trial was maintained following recommended practices.

Stands counts were made on the two middle rows of each plot (total of 60 ft) on June 3, 10, 16, 22, and 30 (= 16, 23, 29, 35, and 43 days after planting, respectively). Some dying seedlings were removed from each plot on June 23 and assayed in the laboratory to isolate pathogens. Thinning was done on July 8 in plots with more than the equivalent of 150 plants per 100 ft of row. The trial was harvested on October 4; data were collected on number of harvested roots per 60 ft row and for yield and quality.

RESULTS

2003 Crop observations. No aboveground symptoms of Rhizoctonia diseases (obvious stunting, yellowing or necrosis of foliage, necrosis at the soil line) were observed on crops grown in *R. solani*-inoculated plots in 2003. Examination of roots revealed no dark, well-defined lesions typical of infection by *R. solani.*

2004 Sugarbeet trial. At 16 days after planting, there were no differences in sugarbeet stand following any of the 2003 crop treatments and controls; stands averaged 235 plants/60 ft row or 50% emergence (Fig. 1). One week later, seedlings began to die in all the 2003 *R. solani*-inoculated treatments but not in the non-inoculated, fallow

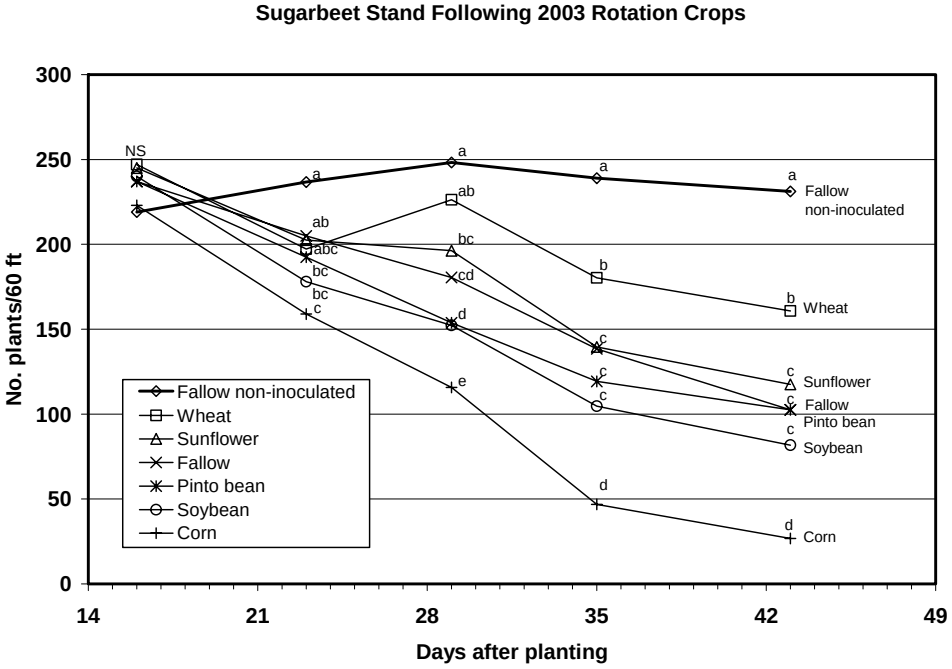

Fig. 1. Sugarbeet stand in 2004 in plots inoculated with *Rhizoctonia solani* AG 2-2IIIB in May, 2003 and then sown with several full-season crops or left fallow compared to a non-inoculated, fallow soil. Each data point is an average number of plants per 60 ft of row (about 480 seed sown per 60 ft) in 4 replicates. For each stand count date, values followed by the same letter are not significantly different ($P \leq 0.05$).

Table 1. Sugarbeet harvest data in 2004 from plots inoculated with *Rhizoctonia solani* AG 2-2IIIB in May, 2003 and then sown with several full-season crops or left fallow compared to a non-inoculated, fallow soil.

2003 Crop treatment	No roots/ 60 ft row[z]	Yield[z]			
		Tons/A	Sucrose (%)	Sucrose (lb/ton)	Rec. sucrose (lb/A)
Non-inoculated					
Fallow	81 a	22.5 a	16.6 a	310 a	6,495 a
R. solani-inoculated[y]					
Wheat	38 b	12.4 b	15.7 ab	290 a	3,639 b
Sunflower	35 bc	12.6 b	14.5 bc	257 b	3,260 bc
Pinto bean	29 bc	12.4 b	14.1 c	252 b	3,188 bc
Soybean	25 cd	9.4 bc	14.1 c	251 b	2,413 cd
Fallow	16 de	6.7 c	13.4 cd	232 bc	1,548 de
Corn	5 e	2.2 d	12.5 d	214 c	472 e
LSD ($P \leq 0.05$)[z]	11	3.7	1.3	31	1,109

[y] An 11 x 30 ft portion of each 22 x 30 ft plot was inoculated with *R. solani* AG 2-2IIIB on May 15, 2003 by sprinkling 3.8 oz of inoculum (grown on sterile barley grain) over the soil surface and raking to a 2-inch depth. Crops were planted and harvested, as appropriate. Controls included inoculated, fallow and non-inoculated, fallow plots. Plots were arranged in a randomized block design of four replicates.

[z] For each column, values followed by the same letter are not significantly different; LSD = Least Significant Difference, $P \leq 0.05$.

control (Fig.1). Sugarbeet stands in this control were statistically ($P \leq 0.05$) higher than in inoculated plots planted to pinto bean, soybean, or corn in 2003 (Fig. 1). Stands continued to decline across all inoculated plots previously sown to various crops or left fallow in 2003 compared to the non-inoculated fallow control, which had the highest stand (Fig. 1). By 43 days after planting, sugarbeet stands in *R. solani*-inoculated plots were statistically highest in plots previously in wheat and lowest in plots previously in corn. Sugarbeet stands were intermediate and equal in inoculated plots previously left fallow or sown with sunflower, pinto bean, or soybean; these treatments had statistically higher stands than where corn was previously grown and statistically lower stands than where wheat was grown in 2003. *R. solani* was recovered from all dying seedlings sampled and cultural characteristics were identical to the isolate inoculated into the field.

Stand loss continued in the *R. solani*-inoculated plots until harvest (data not shown). Numbers of sugarbeet roots harvested, yield, and quality were statistically ($P \leq 0.05$) highest in the 2003 non-inoculated, fallow control compared to all 2003 *R. solani*-inoculated plots (Table 1). For sugarbeet grown in plots inoculated in 2003, the highest number of harvested roots and yields were attained where wheat had been grown, followed in descending order by sunflower, pinto bean, soybean, fallow, and corn. Sugarbeet following corn resulted in extremely low numbers of harvestable roots and yields that generally were statistically lower than all other previous crop treatments except fallow soil.

DISCUSSION

This field trial shows previous rotation crops increase diseases caused by *R. solani* AG 2-2IIIB on sugarbeet. Initial sugarbeet stand was equal in all plots, regardless of previous season crops, because *R. solani* AG 2-2IIIB does not rot seed. Occurrence of Rhizoctonia damping-off and root rot shortly after sugarbeet emergence, however, indicated favorable environmental conditions for infection as well as high inoculum levels of the pathogen in soil. Sugarbeet seedlings are particularly susceptible to *R. solani* AG 2-2, although roots are less susceptible to infection as they

become older (1). In the 2004 field trial, sugarbeet seedlings "baited" *R. solani* from soil, similar to assaying infested soil in the greenhouse by planting sugarbeet. Thus, loss of sugarbeet stand was an indicator of inoculum levels of *R. solani* in soil. Consequently, it can be concluded that inoculum levels of *R. solani* AG 2-2IIIB were lowest in plots previously planted to wheat, followed by increasing inoculum levels for sunflower, fallow soil, pinto bean, soybean, and corn.

Severity of Rhizoctonia on sugarbeet was considerably higher than expected because plots were inoculated with only a moderate amount of inoculum and there were no obvious symptoms of root rot on rotation crops. These considerations, combined with an expected typical decline of *R. solani* inoculum during the winter (7), suggested the 2004 sugarbeet crop would not have much disease. In fields naturally infested with *R. solani* in Texas, Rush and Winter (8) also found previous crops affected severity of Rhizoctonia root and crown rot of sugarbeet, although no evidence of root rot occurred on rotation crops (the intraspecific group was not mentioned). The pathogen may increase inoculum in several ways: infecting roots of rotation crops, which remain symptomless; producing small, negligible lesions; or saprophytically colonizing crop residue after harvest. Perhaps inoculum threshold levels were too low to cause symptoms on crops grown in the 2003 field trials – but surviving inoculum levels were at a high enough threshold to cause severe damping-off of sugarbeet, a highly susceptible host. It is unclear why disease was severe on sugarbeet following inoculated, fallow soil. Weeds may have been infected by *R. solani* before herbicides were applied, thus increasing inoculum.

There was considerable sugarbeet stand loss following wheat, but losses were not as great when compared to the broad-leaf crops (soybean, pinto bean, sunflower). Inoculation of soybean and pinto bean with *R. solani* AG 2-2 IIIB (same isolate as used in the field trial) in previous greenhouse and field demonstrations, resulted in stunting, yellowing, and sometimes, plant death (*unpublished*). When sunflower, wheat, and corn were inoculated, root lesions and damping-off occasionally occurred on sunflower, but never were observed on wheat or corn (*unpublished*). In the Texas Panhandle, where *R. solani* is a common endemic pathogen, and winters are mild, Rush and Winter (8) found more Rhizoctonia root and crown rot on sugarbeet following winter wheat than when following fallow soil, sunflower, or cotton. They concluded *R. solani* survived on winter wheat by saprophytically colonizing residue. The situation in Texas is unlike the Upper Midwest sugarbeet growing areas where inoculum levels of *R. solani* typically are low and spring wheat and barley are common rotation crops.

In our trial, there was abundant corn residue on and near the soil surface when sugarbeet was sown in 2004. No attempts were made to isolate *R. solani* from this material until after sugarbeeet harvest. Then, recovery of *R. solani* was low (1 out of 48, 0.5-inch length pieces of debris, *unpublished*) but presence of the pathogen in residue may have been higher at the beginning of the 2004 growing season. Nelson et al. (5) isolated *R. solani* AG-2-2 from soybean in the RRV (presumably the IIIB population), which caused lesions on corn seedlings in greenhouse inoculations. To date, *R. solani* has not been reported as a pathogen of corn in the midwestern states. In fact, corn often is recommended as a good rotation crop to reduce inoculum levels of *R. solani.* In the southeastern United States, however, Rhizoctonia crown and brace root rot (CBRR) on corn caused by AG 2-2IIIB results in yield losses up to 30% (10). In Germany, *R. solani* AG 2-2IIIB causes severe CBRR and lodging of corn stalks (3). Weather conditions in midwestern states may be less favorable for such dramatic symptoms; inoculum levels of AG 2-2IIIB may be low; or infections could be symptomless or neglible. Perhaps *R. solani* AG 2-2IV, which is not known as a pathogen on corn, predominates in the RRV and southern Minnesota.

Crop rotation is a common practice for managing plant diseases and improving crop production. Benefits of crop rotation are complicated, vary from region to region, and are affected by a multitude of factors. Our research shows corn is a poor crop to grow in fields infested with *R. solani* AG 2-2IIIB. Unfortunately, the distribution and prevalence of AG 2-2IIIB and AG 2-2IV in the RRV and southern Minnesota are unknown. This information is critical in order to adopt crop rotation practices that will retard build-up of inoculum and to manage Rhizoctonia diseases in fields where the pathogen is established.

ACKNOWLEDGEMENTS

We thank the Sugarbeet Research and Education Board of Minnesota and North Dakota for partial funding of this research; Jeff Nielsen and Todd Cymbaluk, University of Minnesota, Northwest Research and Outreach Center, Crookston for planting, maintaining, and harvesting plots; Jeff Nielsen for statistical analysis of data; American

Crystal Sugar Co. for providing sugarbeet seed; and American Crystal Sugar Co. Quality Laboratory, East Grand Forks, MN for sugarbeet yield and quality analysis.

REFERENCES

1. Engelkes, C.A., and C.E. Windels. 1994. Relationship of plant age, cultivar, and isolate of *Rhizoctonia solani* AG-2-2 to sugar beet root and crown rot. Plant Disease 78:685-689.

2. Engelkes, C.A., and C.E. Windels, 1996. Susceptibility of sugar beet and beans to *Rhizoctonia solani* AG-2-2 IIIB and AG-2-2 IV. Plant Disease 80:1413-1417.

3. Ithurrart, M.E.F., G. Buttner, and J. Petersen. 2004. Rhizoctonia root rot in sugar beet (*Beta vulgaris* ssp. a*ltissima*) – Epidemiological aspects in relation to maize (*Zea mays*) as a host plant. J. Plant Disease Protection 111:302-312.

4. National Agricultural Statistics Service. 1992-2003. United States Department of Agriculture, Washington, D.C. (http://www.nass.usda.gov).

5. Nelson, B., T. Helms, T. Christianson, and I. Kural. 1996. Characterization and pathogenicity of *Rhizoctonia* from soybean. Plant Disease 80:74-80.

6. Ogoshi, A. 1987. Ecology and pathogenicity of anastomosis and intraspecific groups of *Rhizoctonia solani* Kuhn. Annu. Rev. Phytopathol. 25:125-143.

7. Ruppel, E.G. 1991. Survival of *Rhizoctonia solani* in fallow soil and buried sugarbeet roots at three depths. J. Sugar Beet Res. 28:141-153.

8. Rush, C.M., and S.R. Winter. 1990. Influence of previous crops on Rhizoctonia root and crown rot of sugar beet. Plant Disease 74:421-425.

9. Sneh, B., L. Burpee, and A. Ogoshi. 1991. Identification of *Rhizoctonia* species. American Phytopathological Society, APS Press, St. Paul, MN. 133 pp.

10. Sumner, D.R. 1999. Rhizoctonia crown and brace root rot. Pages 12-13 *in*: Compendium of Corn Diseases, 3rd edition. D.G. White, ed. American Phytopathological Society, APS Press, St. Paul.

11. Sumner, D.R., and D.K. Bell. 1982. Root diseases induced in corn by *Rhizoctonia solani* and *Rhizoctonia zeae*. Phytopathology 72:86-91.