Interaction of *Rhizoctonia solani* and *Rhizopus stolonifer* Causing Root Rot of Sugar Beet

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ABSTRACT

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In recent years, growers in Michigan and other sugar beet (*Beta vulgaris*) production areas of the United States have reported increasing incidence of root rot with little or no crown or foliar symptoms in sugar beet with *Rhizoctonia* crown and root rot. In addition, *Rhizoctonia*-resistant beets have been reported with higher levels of disease than expected. In examining beets with *Rhizoctonia* root rot in Michigan, over 50% of sampled roots had a second potential root rot pathogen, *Rhizopus stolonifer*. Growing conditions generally were not conducive to disease production by this pathogen alone, so we investigated the potential for interaction between these two pathogens. In greenhouse tests, four of five sugar beet varieties had more severe root rot symptoms when inoculated with both pathogens than when inoculated with either pathogen alone. This synergism occurred under conditions that were not conducive to disease production by *R. stolonifer*. Host resistance to *Rhizoctonia* crown and root rot reduced diseases severity, but was insufficient to control the disease when both pathogens were present. This raises concerns about correct disease diagnosis and management practices and indicates that a root rot complex may be important on sugar beet in Michigan.

In 2007 and 2008, beets were collected in Michigan commercial sugar beet (*Beta vulgaris* L.) fields that had severe root rot symptoms. Over the 2 years, 18 fields were sampled from five different counties, with isolations done from three to six beets per field. Two fungal genera, *Rhizopus* and *Fusarium*, were isolated from 50 to 60% of these beets, in addition to *Rhizoctonia solani* Kühn. While *R. solani* and *Fusarium* have been reported as part of a root rot complex in sugar beet (17), and the author had isolated the two pathogens together in samples from Colorado and Wyoming between 2001 and 2006 (unpublished), *Rhizopus* had not been isolated from field beets in these prior samples. The potential for interaction between *Rhizopus* and *Rhizoctonia* is not known.

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limited number of nonhost crops compatible with the sugar beet rotation often creates problems with a rotation management strategy. Commercial varieties with resistance are available, but a yield reduction usually is associated with high levels of resistance (3), and most of the beets show a tolerance that can be overcome with high inoculum densities. Fungicides have shown efficacy, but timing of application is critical for disease control (20,21), and the fungicides currently labeled are not systemic.

Rhizopus root rot, caused by *R. arrhizus* or *R. stolonifer* (13,37), has been reported in several growing areas in the United States (37,42) and in Europe (19,24,37). It causes a dark, penetrating root rot that is often soft and spongy (13). The disease most commonly is associated with conditions of excess moisture (11,13,18,37,39) or other stresses such as insect injury (13,18,24) or mechanical root damage (13). Recent reports, however, also associate the disease with drought (19,24). Regulating soil moisture and avoiding beet injury are the primary recommended disease management practices (17,18,37), but the *Rhizopus* root rot usually is a minor problem and few or no controls are necessary.

R. solani is reported as a component in a number of root rot complexes (2,7,9,25) and may be synergistic with several types of pathogens, including nematodes (1), bacteria (25,40), oomycetes (22), and fungi (9,25). The potential for interactions between *R. solani* and other pathogens of beet also have been reported (15, review by 22, 40).

This research was conducted to investigate the potential for interactions between *R. solani* and *Rhizopus stolonifer* in causing root rot of sugar beet and their effects on symptom production and host resistance, a major disease management strategy.

MATERIALS AND METHODS

Isolates of *R. solani* and *Rhizopus stolonifer* were obtained from sugar beet plants showing root rot symptoms or from culture collections of fungi from sugar beet. For *Rhizopus stolonifer*, tissue was cut from the edge of a lesion in the beet cortex, and pieces (approximately 0.5 cm² × 2 mm deep) were surface-treated for 60 s in a

This research investigates the potential for interaction between *Rhizoctonia solani* and *Rhizopus stolonifer* (Ehrenb.) Vuill.

Rhizoctonia crown and root rot (RCRR) has been the most important soilborne fungal disease of sugar beet in the United States for many years (11,13,36) and is increasingly becoming a problem in Europe (3,5). The primary causal agent is *R. solani* anastomosis group (AG) 2-2 (36,38). The crown rot stage of the disease is the most obvious form, causing sudden and permanent wilting of the leaves, with leaves having dark petioles (13,16). When the root rot stage is present, foliar symptoms may not appear, or be as obvious, until the disease is well advanced. RCRR usually causes a dry, firm, dark rot that affects primarily the root surface with little penetration deep into the root until the disease is far advanced (13,36). Two intraspecific groups (ISG) of *R. solani* can be involved, AG-2-2 IIIB and AG-2-2 IV, with AG-2-2 IIIB usually more virulent (12). We found both ISG in Michigan in recent screening of isolates (unpublished data).

Management of RCRR includes crop rotation (5,13,16), host resistance (5,16), and fungicides (16,20,21,43). However, all three have limitations. Rotation with nonhost crops for at least 3 years is recommended for RCRR management, but *R. solani* AG-2-2 isolates can infect a number of diverse crops (31,33,38,41), and the

0.5% sodium hypochlorite solution and placed on potato dextrose agar (PDA; Becton, Dickinson and Co., Sparks, MD). Plates were incubated at approximately 24°C and examined for fungal growth starting 2 days after plating and every 2 days thereafter for up to 14 days. Pure cultures were obtained by spreading spores on water agar (WA) plates, incubating 24 h at 20°C, and transferring a single germinated conidium to PDA. *Rhizopus* isolates were identified to species based on morphology on WA and malt extract agar (MEA; 35) under microscopic examination (35) and growth at 30°C but not at 40°C.

The *R. solani* isolate (R-1) was from the culture collection of E. G. Ruppel (USDA-ARS, retired, Fort Collins, CO) and originally was collected from diseased sugar beet root. We confirmed its identity as AG-2-2 IIIB by paired anastomosis testing with a known tester isolate and by temperature response (38).

Inoculum. Inoculum of *R. solani* was grown for 5 to 7 days on PDA. Plugs (6 mm diameter) of colonized medium were transferred to sterile unhulled barley grains (one plug per 500 cm³ of barley) that had been soaked in water and autoclaved. Inoculated barley was incubated in closed stainless steel pans at 26°C for 21 days. Colonized barley was air-dried and ground with a mill (Quaker City Mill F No 4, Philadelphia, PA). Ground inoculum was stored at 4°C until used.

Rhizopus stolonifer isolates were grown for 7 days on PDA at approximately 22°C in the dark. Plates were flooded with 15 ml of sterile water, and plate surfaces and fungal hyphae were scraped gently with a sterile disposable cell spreader to release spores. Spore concentration was determined with a hemacytometer, and spore concentrations were adjusted with sterile distilled water. Spore suspensions were used within 3 h of preparation.

Inoculations. Sugar beets were planted in commercial potting mix (Bacto high porosity professional planting mix, Michigan Peat Co., Houston, TX) in plastic pots (17 cm deep by 15 cm diameter). Beets were fertilized with Osmocote 14-14-14 (Scotts, Marysville, OH) and a micronutrient mix (MicroMax, Grace-Sierra, Milpitas, CA). Beets were watered when the soil surface was dry to the touch. In the first set of experiments, a RCRR-susceptible commercial cultivar, USH20 (8), and a RCRR-resistant germplasm, FC716 (30), were used to look for interactions between the pathogens. To determine whether the response was variety-specific, three commercial varieties, designated here as 27, 28, and 32, were tested separately. Pots were arranged in a completely randomized design, with four pots (one plant per pot) for each treatment. Insects were controlled by applications of a combination of potassium salts of fatty acids (M-Pede, Dow Agro-Sciences, Indianapolis, IN) and *Beauveria*

bassiana (Botanigard, Laverlam International Corp., Butte, MT). The greenhouse was maintained at 21 to 25°C.

Eight weeks after planting, beets were inoculated with *R. solani* by digging a small depression in the potting mix immediately adjacent to the beet crown and placing approximately 0.6 ml of the barley preparation into this depression with a measuring spoon. Potting mix was placed over the barley and plants were watered lightly. For *Rhizopus stolonifer* inoculations, a 10-ml spore suspension (1 × 10⁴ spores per ml) was placed on the beet crown with a sterile pipette. Control plants had sterile ground barley and 10 ml of sterile water applied, respectively. Treatments included control, *Rhizopus stolonifer* alone (with sterile barley added), *Rhizoctonia solani* alone (with sterile water applied to the crown), and a combination of both. Plants were maintained as described above. After 4 weeks, beets were removed from the potting mix and roots were washed under running tap water to remove residual potting mix. Beets were examined for root lesions, and the percent surface area showing symptoms was recorded. In addition, each root was rated for disease severity based on a 0 to 7 scale (27,32), where 0 = no disease, 1 = a small amount of surface scarring, 2 to 6 = increasing percentages of the root tissue showing rot, and 7 = root completely rotted and foliage dead. Each root was rated and an average disease severity rating calculated for each treatment. In addition, each root was cut longitudinally to examine for internal symptoms. Tissue was taken from the edge of lesions (where present) or from internal root tissue where side initials were present when no symptoms were observed. Three roots for each treatment were used from each experiment for isolation attempts, with three or four tissue pieces taken from the edge of lesions per root. Isolation was done as described above for *Rhizopus* to confirm presence of the pathogen(s). Resulting fungi were compared morphologically to those used in initial inoculations. All experiments were repeated at least once.

Statistical methods. Statistical analyses were performed using SAS (SAS Institute, Cary, NC). The Brown and Forsythe test was used to test for homogeneity of variances. Because homogeneity of variances was not found for the experiments ($P < 0.01$), analyses of variance (ANOVA) were performed for disease ratings for each experiment separately. As all ANOVA tests were significant ($P < 0.05$), mean separations were conducted for each variety using the macro PDMIX800.sas (34) with Tukey adjustments.

RESULTS

From field samples, rots from which *Rhizopus* was isolated in addition to *Rhizoctonia* were generally penetrating,

brown lesions. On the surface, lesions were dark brown to almost black, as is typical for RCRR (36). Internally, most lesions were light to dark brown, which can occur with either root rot (36,37). One sample had an acetic acid-like odor as has been described for severe *Rhizopus* root rot (37).

Rhizopus isolates were identified as *Rhizopus stolonifer* by the presence of coenocytic hyphae with sporangiophores (2 to 4 mm long) formed on stolons opposite complex, well-developed rhizoids. Isolates grew at 30°C but showed no growth at 40°C.

In the first greenhouse experiment, using USH20 and FC716, no disease symptoms were observed on plants of either variety with the control treatments (Fig. 1A). No clear rot symptoms were observed on plants treated with the *Rhizopus stolonifer* spore suspension alone (Fig. 1B), although some surface scarring was observed on the crowns. Lesions typical for RCRR were observed on the susceptible variety when treated with *R. solani* alone (Fig. 1C). More extensive root discoloration was observed on beets exposed to the combination of the two pathogens (Fig. 1D), for both the susceptible and the resistant material. Little in the way of foliar damage was observed for any treatment. In the first time through the experiment, small spots and surface scarring were observed on the resistant germplasm (FC716) exposed to *R. solani* alone and visible root rot when exposed to the combined pathogens. In the second time through the experiment, no symptoms were seen on the resistant germplasm when exposed to *R. solani* alone, but rot was present when both *R. solani* and *Rhizopus stolonifer* were combined. Analysis is shown for the first time through the experiment in which symptoms were observed on the resistant germplasm (Table 1). Results were similar for the second time through the experiment. When root ratings were compared (Table 1), disease severity ratings were increased significantly ($P < 0.05$) for the combined pathogens compared to the *R. solani* treatment alone. A significant increase in disease severity for the combined pathogen treatment was observed for all tests ($P < 0.05$). The susceptible variety had a higher disease severity rating than the resistant in the combined pathogen treatment (Table 1), but both showed a higher disease rating than with either pathogen alone.

Similar results were observed for the commercial cultivars, with no rot symptoms, just some surface scarring, on the control plants or those treated with *Rhizopus stolonifer* alone. All three varieties had significantly higher ratings following treatment with *Rhizoctonia solani* compared with the control (Table 2). Significantly greater disease severity was observed on two of the three commercial

sugar beet varieties examined following combined exposure to the two fungi. The exception was variety 28, which had no significant difference in disease severity between *R. solani* alone or in combination with *R. stolonifer* (Table 2). This variety had the highest disease rating with *R. solani* alone of any of the beet varieties or germplasm tested.

When infected roots were cut open to reveal interior symptoms, control plants and those treated with only *Rhizopus* showed little or no internal discoloration as seen with susceptible variety USH20 (Fig. 2A). Susceptible plants treated with *R. solani* had shallow, dark lesions that penetrated less than 1 cm into the interior of the root (Fig. 2B), while several plants treated with the combined pathogens had extensive internal discoloration (Fig. 2C), which usually was somewhat soft, and often lighter colored than that caused by *R. solani* alone.

All plants showing symptoms of root rot yielded *R. solani* from the tissue at the edge of lesions. No *Rhizoctonia* or *Rhizopus* was cultured from noninoculated

plants. No *Rhizoctonia* was isolated from plants inoculated with *Rhizopus* alone. *Rhizopus stolonifer* was isolated only from plants inoculated with this organism, but was not isolated from all inoculated plants. *R. stolonifer* was isolated from two *R. stolonifer*-inoculated plants in the commercial variety experiments that showed no root rot symptoms but had surface scar-

ring. Isolates were obtained from internal root tissue where side root initials were present. *R. stolonifer* also was isolated from at least one lesion from roots of each variety from the combined inoculation plants in all of the experiments. It was isolated from lesions of the deeper, more penetrating rot and softer rot areas, but it was not isolated from all lesions sampled,

Table 1. Response of a *Rhizoctonia* crown and root rot-susceptible sugar beet commercial cultivar (USH20) and a *Rhizoctonia* crown and root rot-resistant germplasm (FC716) to inoculation with *Rhizoctonia solani* AG-2-2 IIBB with and without *Rhizopus stolonifer*^w

Sugar beet type ^y	Fungal treatment ^x			
	Control	<i>Rhizopus</i>	<i>Rhizoctonia</i>	Combined fungi
USH20	0 c (0%) ^z	0.5 c (<5%)	3.5 b (20-30%)	5.5 a (60-80%)
FC716	0 b (0%)	0.5 b (<5%)	1 b (≤5%)	3 a (15-25%)

^w Plants were grown for 4 weeks after inoculation and rated for root rot severity. Results shown are from the first of two times this experiment was conducted.

^x Treatments included a spore suspension of 1×10^4 spores per ml of *Rhizopus*, 0.6 ml of a dried ground barley inoculum of *R. solani*, or a mix of the two. Control plants were treated with sterile ground barley and sterile water. Each value is the average of four plants.

^y Sugar beets tested were commercial variety USH20 (*Rhizoctonia* susceptible) or USDA released *Rhizoctonia* root rot-resistant germplasm FC716.

^z Average disease severity ratings and (range of percent surface area affected). Numbers for each variety followed by the same letter are not significantly different by Tukeys ($P = 0.05$).

Fig. 1. Response of sugar beets to inoculation with fungi. Plants shown are sugar beet variety USH20, a *Rhizoctonia*-susceptible variety. Plants were harvested 4 weeks after inoculation. **A**, Control plants treated with sterile ground barley and sterile water. **B**, Plants treated with an aqueous spore suspension of *Rhizopus* sp. and sterile ground barley. **C**, Plants treated with a barley inoculum of *Rhizoctonia solani* AG-2-2 and sterile water. **D**, Plants treated with a combination of a spore suspension of *Rhizopus* and a barley inoculum of *Rhizoctonia solani*.

particularly the dark, surface-rot areas. *R. solani* was isolated from all lesions sampled in the combined pathogen treatments.

DISCUSSION

More severe disease was observed when three of four sugar beet varieties and one germplasm were exposed to a combination of *Rhizoctonia solani* AG-2-2 IIIB and

Rhizopus stolonifer than when plants were exposed to either pathogen alone. This increased disease severity was greater than that obtained by adding the severities of the two pathogens together, and thus appeared to be synergistic. The increased disease severities occurred under conditions not reported as conducive to disease production by *Rhizopus* sp. Roots were not

exposed to excess moisture, drought, or insect damage. Despite this, in the combined inoculations, beets showed disease symptoms that appeared to be a combination of the two pathogens, as lesions penetrated well into the beet and often were soft, as would be expected with *Rhizopus* infection, rather than being the firm texture usually associated with RCRR (36,37); but

Fig. 2. Internal symptoms on a *Rhizoctonia* crown and root rot-susceptible sugar beet variety USH20 following inoculation with **A**, *Rhizopus* sp. alone, **B**, *Rhizoctonia solani* AG-2-2 IIIB alone, or **C**, a combination of both. Black arrows indicate dark, firm, shallow lesions typical of *Rhizoctonia* crown and root rot. Gray arrows indicate penetrating lesions.

the surface or parts of the lesions were firm and darker (Fig. 2C), as is typical for RCRR. In some cases, portions of the lesions were light brown, appearing more similar to symptoms of *Rhizopus* root rot (37) than the very dark brown to black lesions normally produced by RCRR (13,16,36), although both can produce light brown internal lesions (36,37).

The one variety that did not show this response may have been too susceptible to RCRR to show increased disease severity with the combined pathogens, as it had a higher disease severity rating in the presence of *R. solani* alone than did the other varieties. It might be that differences would be seen under lower *Rhizoctonia*-inoculum pressure with this variety. Alternatively, this variety might have some resistance to *Rhizopus*. No resistance to *Rhizopus* has been reported in beets and no significant difference was found among these three varieties when inoculated with *Rhizopus* as a postharvest rot (unpublished data). It is possible, however, that different responses may occur while the beet is growing compared to postharvest conditions.

Conditions in the fields where the *Rhizopus stolonifer* isolates were collected were not those generally reported for *Rhizopus* root rot. Soils where most of the samples were collected were not waterlogged. For example, in 2007, two samples that yielded *R. stolonifer* in July were from an area where the soil moisture had a maximum of 0.421 cm³/cm³ and an average of 0.186 cm³/cm³ for the 30 days prior to samples being taken, according to data from the Michigan Automated Weather Network (<http://www.agweather.geo.msu.edu/mawn/>). The maximum soil temperature was 27°C, and the minimum was 17°C for the same time period.

One concern raised by these results relates to screening for RCRR resistance in sugar beet. Resistance does occur (14, review in 29) and is in commercial use for disease management. These results (Table 1) indicate that when both pathogens were present, as well as when only *R. solani* was

present, the *Rhizoctonia*-resistant variety had a lower disease severity than the *Rhizoctonia*-susceptible variety. The increased disease severity observed with the combined pathogens in the resistant germplasm FC716 was similar to that of the susceptible variety USH20 when exposed to *R. solani* alone (Table 1). If a resistant variety were planted in a field with both pathogens without a susceptible variety for comparison, there would be a potential perceived loss of resistance, as disease levels could be similar to those observed in a susceptible variety exposed to *R. solani*. If a susceptible variety were present, the relative disease levels could indicate that the resistant variety still has less disease than a susceptible. Growers in some areas have expressed concern about poor performance of host resistance. It is not known if a mixed infection could be involved in some of these situations.

While disease severity increased with the combined pathogens, resistance to RCRR showed some promise in controlling the disease even when both pathogens were present. The germplasm FC716, with a high level of resistance to RCRR, had lower disease severity in the presence of the combined pathogens than did a susceptible variety (USH20). However, disease severity in this germplasm exposed to the combined pathogens for 4 weeks was above the level considered harvestable (27,28), while it would have been at levels acceptable for harvest if exposed to *R. solani* alone (Table 1). Thus, if the two pathogens are interacting in a field, other control measures than host resistance may be necessary.

The production of symptoms typical of *Rhizopus* from only plants treated with a combination of the two pathogens indicates that, under these growing conditions, which are not those typically reported to lead to *Rhizopus* root rot (37), *Rhizopus stolonifer* was not able to cause disease by itself. It apparently required the activity of *Rhizoctonia solani* to infect the plants. This does not appear to be simply by creat-

ing a wound, since a subset of plants of USH20 that were wounded by making a hole with a #4 cork borer at the soil surface prior to inoculation did not show any *Rhizopus* root rot symptoms (data not shown). The isolation of *Rhizopus* only from some of the tissues in the plants treated with the combined pathogens may indicate limited spread of the pathogen in the host, poor competition with *R. solani* in the host or on the isolation media, or a preferential colonization of certain tissues. This could be of concern for identifying the presence of this pathogen in the field. Also, while the crown was inoculated with *Rhizopus stolonifer*, most of the isolations of *R. stolonifer* occurred from root tissue below the soil surface, particularly in the root tissue at or below the area where the lateral roots were visible.

It may be that the isolates of *R. stolonifer* used in this study have low virulence. None of the isolates in this study had been collected as the sole pathogen in beet. All had been isolated from beets from which *R. solani* also had been isolated. This is not unexpected, as *Rhizopus* species are generally classified as weak parasites that cause disease when beets are "not developing normally" (37). The ability of these isolates to cause disease under conditions conducive to *Rhizopus* root rot, such as excessive soil moisture, 14 to 16°C temperature, and injuries to the beet is being investigated.

As noted in Figure 1, plants showed very few foliar symptoms for any of the treatments tested, in contrast to what has been reported for RCRR, where foliar symptoms of chlorosis of the foliage and black necrosis of the petioles are commonly reported (36). A lack of foliar damage (Fig. 1D) in the field can make determination of whether a field should be harvested difficult, and might result in more severely rotted beets being taken for processing than is desirable.

As well as having the potential to cause root rot in the field, *Rhizopus* spp. also can be postharvest pathogens of sugar beet (4,23, cited in 24). The presence of this pathogen in beets in the field might contribute to inoculum for postharvest rot. The contribution of field infection with both of these root rot pathogens to postharvest deterioration needs to be determined.

This study shows an interaction between the two soilborne fungi, *Rhizoctonia solani* AG-2-2 IIIB and *Rhizopus stolonifer*, that increases root rot severity in infected beets. Root rot complexes involving *R. solani* and other soilborne fungi have been reported on several crops (6,9,10,25,26) and may be an important factor for beet. Further investigations on the potential for interactions between the other major form of the RCRR pathogen of sugar beet, *R. solani* AG-2-2 IV, and *Rhizopus stolonifer* and into the conditions under which this disease interaction occurs is warranted.

Table 2. Response of three commercial sugar beet cultivars, 27, 28, and 32, to inoculation with *Rhizoctonia solani* AG-2-2 IIIB with and without *Rhizopus stolonifer*^v

Fungal treatment ^x	Average disease severity rating ^w		
	27 ^y	28	32
Control	0.5 c ^z	0.0 b	0.0 c
<i>Rhizopus</i>	0.5 c	0.3 b	0.5 bc
<i>Rhizoctonia</i>	2.8 b	4.3 a	2.3 b
<i>Rhizopus</i> + <i>Rhizoc.</i>	5.3 a	5.8 a	5.0 a

^v Plants were inoculated with a spore suspension of *Rhizopus* or sterile water and a ground barley inoculum of *R. solani* or sterile ground barley. Control plants were treated with sterile ground barley and sterile water. Plants were grown for 4 weeks after inoculation, harvested, and rated for root rot severity. Results shown are from the first of two times this experiment was conducted.

^w Disease severity rating on a 0 to 7 scale where 0 = no disease and 7 = root completely rotted. All numbers are the average of four plants.

^x Fungal treatments included a spore suspension of 1 × 10⁴ spores per ml of *Rhizopus*, 0.6 ml of a dried ground barley inoculum of *R. solani* or a mix of the two. Control plants were treated with sterile ground barley and sterile water.

^y Sugar beet commercial varieties are designated 27, 28, and 32.

^z Numbers for each variety followed by the same letter are not significantly different by Tukeys (*P* = 0.05).

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