Rhizoctonia-Bacterial Root Rot Complex in Sugarbeet

by Carl Strausbaugh

Rhizoctonia root rot is a concern worldwide in sugarbeet and other crops and seems to be on the increase in a number of production areas both in the United States and Europe. Damage caused by Rhizoctonia root rot is caused by a fungal pathogen, Rhizoctonia solani. This fungus will typically lead to a dry black rot on the exterior of the root which at times induces large cracks in the root (Fig. 1).

However, based on a series of studies in southern Idaho and southeastern Oregon, the majority of the root rot in the Amalgamated production area appears to be caused by a Rhizoctonia-bacterial root rot complex. When only fungi were isolated from the dry black rotted tissue, R. solani was the most damaging fungus isolated although other fungi (Fusarium spp., Geotrichum spp., Mucor spp., and Rhizopus spp.) could also frequently be found. This dry black rot associated with fungi could be widespread on the surface of the root but usually did not penetrate into the root very far.

Thus, on average only about 6 percent of the root mass was lost to fungal rot. When a wet type rot was evident in the root (Fig. 2), bacteria either alone or in combination with other organisms were isolated and on average 68 to 71 percent of the root mass was rotted. The bacterium that initiated the wet type rot was Leuconostoc mesenteroides subsp. dextranicum, although other bacteria such as Lactobacillus spp. and Gluconobacter spp. along with yeast were also frequently present. However, most of the organisms isolated (other than Gluconobacter) must compete for substrate or produce inhibitors of some kind since they

Figure 1. Dry black rot and cracking typical of Rhizoctonia root rot on sugarbeet.

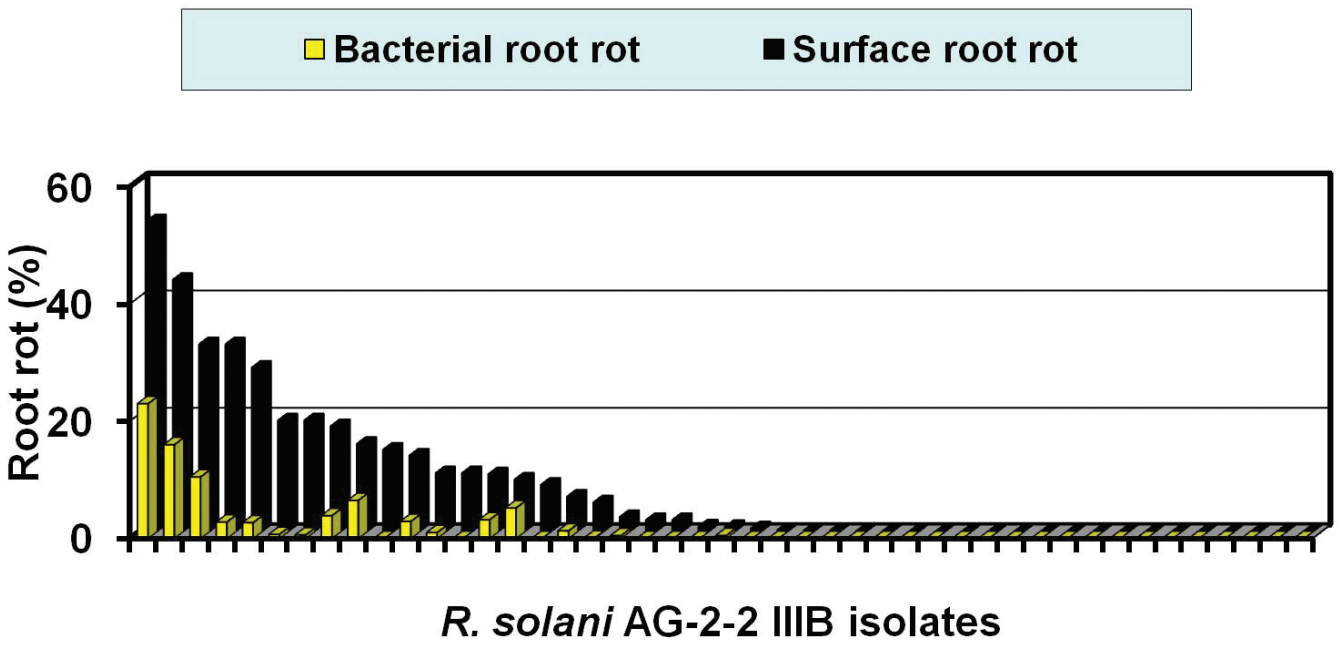
Rhizoctonia-Bacterial
Root Rot Complex in Sugarbeet

would reduce the amount of rot compared to when Leuconostoc was inoculated alone.

Leuconostoc may initiate some rot on its own, but that seems to be a rare event. Typically both in surveys and directed field studies, it appears that fungi initiate the rot on the root surface and then bacteria take over and lead to considerably more rot than if fungi were present alone. This statement certainly applies to the Rhizoctonia-bacteria root rot complex in Idaho, but observations in eastern Oregon suggest that it may also apply to Aphanomyces root rot but to a lesser extent.

The higher elevation (approx. 4,600 ft) production areas in eastern Idaho have very little root rot while western lower elevation (approx. 2,300 ft) production areas have considerable problems with the Rhizoctonia-bacteria root rot complex. When these production areas were surveyed two genetic subgroups of R. solani, AG-2-2 IIIB and AG-4, were found associated with Rhizoctonia root rot. On mature roots the AG-2-2 IIIB isolates are more aggressive than AG-4 isolates, which cause just superficial lesions. Although AG-4 causes limited rot on mature roots, it can cause damping off of sugarbeet seedlings and cause severe stem and stolon cankers on potato. Most (87 percent) of the AG-2-2 IIIB isolates were associated with the western portion of the Amalgamated production area, while 71 percent of the AG-4 isolates came from the eastern portion.

Figure 2. Wet discolored tissue and cavities typical of early (left) and advanced (right) stages of the bacterial phase of the Rhizoctonia-bacterial root rot complex.



Characteristics of fields prone to problems with the Rhizoctonia-bacteria root rot complex include longer growing season (planted in late February to early March and harvested in early November), short crop rotations (particularly with corn), furrow irrigation and over-watering. The bacterial phase of the rot tends to occur later in the season on full-sized roots accumulating sugar, so the longer grower season (more late-season heat units) seems to aid in bacterial development.

Field studies suggest that if Rhizoctonia infection is reduced then problems with the bacterial phase of the rot are limited as well. Until we develop a better understanding of this rot complex, control efforts should be targeted at controlling Rhizoctonia since Leuconostoc is naturally widespread in the environment.

Initial infection for Rhizoctonia root rot results from propagules in the soil such as sclerotia or mycelia (often associated with plant debris). The sclerotia can survive for a number of years in the soil, so using at least three-year crop rotation with non-host crops would advisable. However, R. solani has a wide host range, so finding acceptable non-host crops can be a problem. A rotation with wheat or barley should help control R. solani.

Rotation with corn also used to be encouraged but recently some isolates of AG-2-2 IIIB have been identified that can cause root lesions on field corn in Idaho and thus will likely not limit inoculum buildup. AG-4 isolates (only a few were tested) did not infect corn roots. Other crops attacked by AG-2-2 IIIB or that might maintain inoculum levels include alfalfa, carrot, dry bean, mustard, flax, snap bean, soybean and sunflower.

The application of fungicides such as azoxystrobin can delay early infection and enhance establishment of vigorous stands but does not completely prevent infections leading to crown and root rot. Resistant cultivars can also greatly help with control but need to be more widely available. However, utilizing resistance may be complicated by isolate-resistance source interactions. Field studies in Idaho suggest there is a range of virulence among AG-2-2 IIIB isolates in Idaho (Fig. 3).

Preliminary evidence from Idaho suggests that the FC709-2 source of resistance is superior to other sources tested at handling this range of virulence. ■

Editor's note: Strausbaugh is Research Plant Pathologist; NWIRSL, 3793 N. 3600 E., Kimberly, ID 83341
Email: carl.strausbaugh@ars.usda.gov