Infection by BNYVV Predisposes Sugar Beets to Rhizoctonia Root and Crown Rot

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Rhizomania was first identified in the Southern Minn production area in the mid 1990's and reported in the American Crystal area a few years later. The disease can severely reduce root yield and quality and also disrupts plant water relations. Regionally adapted cultivars possessing *Rz1* resistance were rapidly deployed but within a few years, resistant-breaking strains of BNYVV began to appear in fields planted to cultivars with *Rz1* resistance. In response, seed companies released rhizomania-resistant cultivars with other sources of resistance, or cultivars with multiple genes for resistance, but in some fields these also developed symptoms of rhizomania. It quickly became obvious that cultivars with the same dominant resistance gene, such as *Rz1*, could differ considerably in their response to resistance-breaking (RB) strains of BNYVV and that the minor genes in these cultivars significantly impacted susceptibility to rhizomania. Currently, all approved cultivars grown in MN and ND have single dominant gene resistance to BNYVV, and most also possess varying degrees to tolerance/resistance to other prevalent soilborne pathogens such as *Rhizoctonia solani*, and *Aphanomyces cochlioides*.

Rhizoctonia root and crown rot (RRCR) is caused by *Rhizoctonia solani* Ag2-2, and in recent years, incidence of this disease has increased in MN/ND production areas. Aphanomyces root rot has been a significant constraint to sugar beet production for years and like RRCR is favored by high soil moisture. When sugar beets are grown in the presence of a pathogen complex, such as BNYVV, *Aphanomyces*, *Fusarium* and *R. solani*, the strong resistance that specific cultivars display against a single pathogen is often compromised. This is especially true with a disease such as rhizomania, that greatly disrupts plant water relations, resulting in high moisture content in the soil rhizosphere. This elevated soil moisture in the rhizosphere could predispose BNYVV-infected roots to infection by other "moisture-loving" soilborne pathogens such as *R.solani* and *A.cochlioides.* It is clearly possible that the different levels of infection by BNYVV that occur in BNYVV-resistant cultivars may impact their susceptibility to infection by *R. solani.* To test this idea, a two year study was initiated to determine whether there was any correlation between BNYVV titer in rhizomania resistant cultivars and susceptibility to RRCR.

Methods

Research plots were established in 2012 and 2013 at two locations each year; Hector and Roseland in 2012, and Hector and Pennock in 2013. Research sites were selected based on past history of **farms that had losses to both RRCR and rhizomania. Nine cultivars that varied in resistance to RRCR** (based on results in previous field trials) and also possessed resistance to rhizomania were used (**TABLE 1).**

Table 1. Sugarbeet varieties with their Rhicotonia root and crown rot ratings included in the Southern Minnesota field study.

Variety	Rhizoc. Ratings	BNYVV Gene/s
Crystal 018RR	**4.25**	**Rz1+Rz2**
Crystal 265RR	**4.47**	**Rz1**
Crystal 850RR	**5.07**	**Rz1+Rz2**
Hilleshog 4017RR	**4.05**	**Rz1**
Hilleshog 4063RR	**2.99**	**Rz1**
Hilleshog 9093RR	**2.37**	**Rz1**
SV 36091RR	**5.4**	**Rz1**
SV 36835RR	**4.39**	**Rz1**
SV 36938RR	**4.39**	**Rz1**

In 2012, Hector and Roseland were planted May 14th, inoculated with *R. solani* June 18 and harvested September 12th. In 2013, plots at both sites were planted June 7, inoculated July 12 and harvested October 2. Inoculum for *R. solani* consisted of barley kernels infested with *R. solani* that was originally isolated from an infected sugar beet root from a field in southern Minnesota. At harvest, plants were topped and dug, incidence of RRCR in each plot was determined and disease severity was determined using a 0-4 scale. Root samples were taken for BNYVV detection and quantification in the lab, and realtime qPCR was used to quantify virus titer in roots of each cultivar. Data was analyzed using ANOVA and regression analysis.

Results and Discussion

Of the four field trials planted during the two years of this study, only the Hector study in 2012 yielded acceptable results. The 2012 Roseland site was not harvested because of excessive seedling disease pressure, primarily Rhizoctonia and Aphanomyces that killed over 90% of the plants even before inoculation. The two sites in 2013 had good stand establishment but neither developed adequate disease, either RRCR or rhizomania, to allow any conclusions. The Pennock site had essentially no disease of any type, and the Hector site had on average less than 3% RRCR and only 12 plants of over 100 evaluated for BNYVV tested positive. The difference in the incidence and severity of disease during the two years was likely due to differences in rainfall. In 2012, over 12 inches of rain fell between the time of planting and inoculation and approximately half that much fell in 2013.

Adequate disease developed at the 2012 Hector site to analyze the data and evaluate the impact of BNYVV titer on incidence of RRCR **(Fig. 1 & 2).**

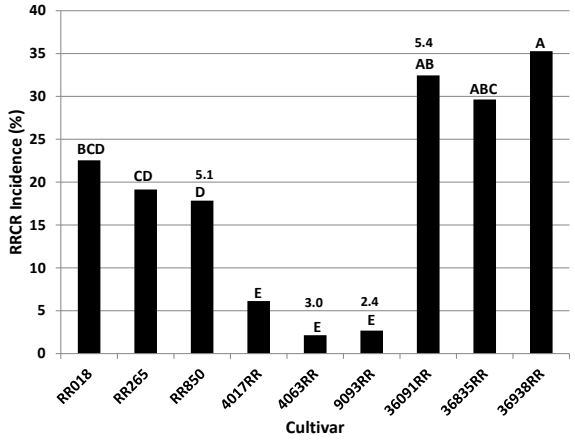

Fig. 1. Rhizoctonia root and crown root incidence in different sugar beet cultivars.

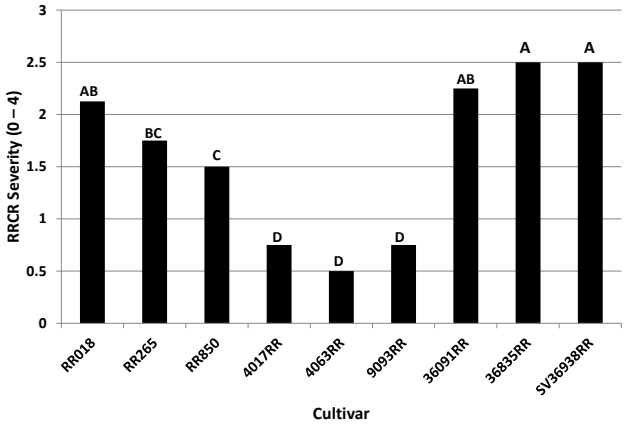

Fig. 2. Rhizoctonia root and crown root severity in different sugar beet cultivars.

RRCR developed in all cultivars but disease incidence and severity was not perfectly correlated with disease ratings from early field trials. For instance, 4017, with a RRCR rating of 4.05 was not significantly different from 4063 with a rating of 3.0 or 9093 with a rating of 2.1, but it was different from all other cultivars that had similar ratings of 4.0 or greater. As a group, the cultivars from Hilleshog had the lowest incidence and severity of RRCR.

Cultivar-specific minor genes also seemed to have a greater impact on BNYVV titer than the dominant resistant gene. In general cultivars with only *Rz1* did as well as those with *Rz1+Rz*2. For instance, 4017 with *Rz1* was not significantly different than 850 with *Rz1+Rz2*, but both were significantly different than 3609 and 3693, which have *Rz1* **(Fig. 3).**

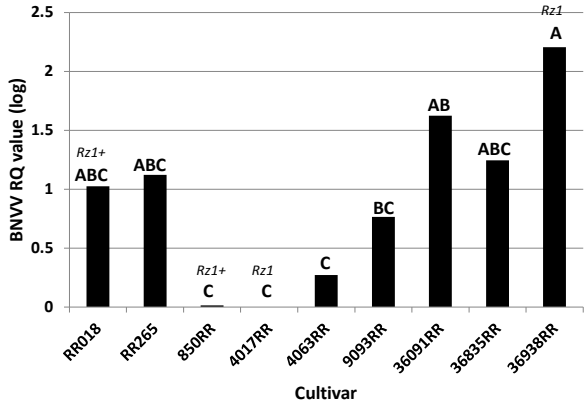

Fig. 3. BNYVV titer level in different cultivars.

As typical, there was considerable variation in BNYVV titer among samples within a single cultivar, and this explained the general lack of significant differences among cultivars.

Although there were minimal statistical differences in BNYVV titer among cultivars **(Fig. 3)**, when regression analysis was used to evaluate the relation between BNYVV titer and RRCR incidence and severity, a strong significant, positive relationship was revealed **(Fig. 4 & 5).**

In past studies, we have shown that there is a strong positive correlation between BNYVV titer a severity of rhizomania. Additionally, as severity of rhizomania increases, the less efficient the root becomes in extracting soil moisture and the rhizosphere soil becomes wetter and therefore more conducive to infection by *R. solani*. The fact that

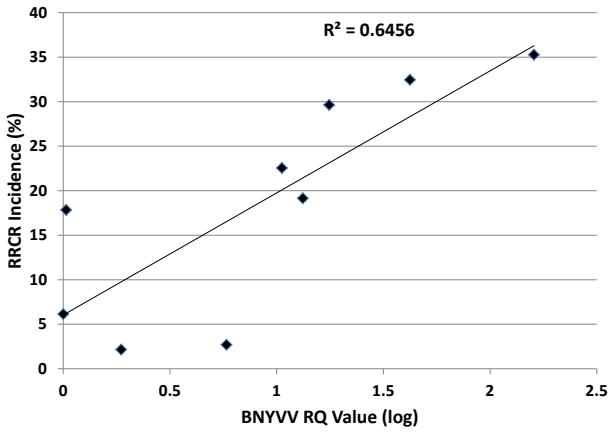

Fig. 4. Relationship between BNYVV titer levels and Rhizoctonia root and crown rot incidence.

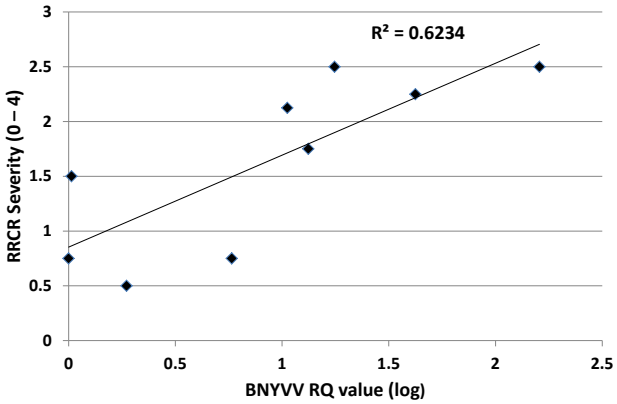

Fig. 5. Relationship between BNYVV titer levels and Rhizoctonia root and crown rot severity.

increased BNYVV titer and rhizomania severity increased incidence of RRCR was not surprising, but the susceptibility of most rhizomania resistant cultivars used in this study to severe rhizomania was somewhat surprising. At harvest, most cultivars exhibited significant rhizomania, and in greenhouse studies, using the same field soil, most cultivars exhibited extremely severe rhizomania symptoms. It is likely that RB strains of BNYVV were present in this field soil and thus the dominant single gene resistance in most of these cultivars was overcome. These results indicate the need for better diagnostic tools for detecting RB strains of BNYVV in grower's fields, and the continuing need for cultivars with new/stronger sources of resistance to RB strains of BNYVV.