INVESTIGATING POTENTIAL RELATIONSHIP BETWEEN GENETIC RESISTANCE TO BNYVV AND INCREASED SUSCEPTIBILITY TO RHIZOCTONIA ROOT AND CROWN ROT

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Rhizoctonia root and crown rot (RRCR), caused by *Rhizoctonia solani,* is wide spread in most sugar beet production areas in the USA and in recent years, incidence of this disease has increased in MN/ND production areas. It has been verified that *R. solani* AG 2-2 and 4 are the predominant pathogens of sugar beet and that two intraspecific groups of AG 2-2 exist, i.e. IIIB and IV. AG 2-2 IV is the more prevalent in the RRV while 2-2 IIIB is most common in the Southern Minn production area.

Rhizomania was first identified in the Southern Minn production area in the mid 1990's and was reported in the American Crystal area a few years later. Regionally adapted cultivars possessing *Rz1* resistance were rapidly deployed and soon all approved cultivars possessed resistance to rhizomania. Within a few years, resistant-breaking strains of BNYVV began to appear, first as blinkers and later in large diseased spots, in fields planted to cultivars with *Rz1* resistance. Seed companies began to release rhizomania-resistant cultivars with other sources of resistance, or cultivars with multiple genes for resistance. However, with experimental and commercially available rhizomania-resistant germplasm alike, susceptibility to other pathogens, such as *R. solani*, has not been adequately evaluated. The impact of low level infection of rhizomania resistant cultivars by BNYVV on susceptibility to *R. solani* has not been examined. Likewise, it is not known whether infection of Rhizoctonia-resistant cultivars by BNYVV affects resistance to *R. solani.*

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 may be compromised. These pathogens are now all present in various fields throughout Minnesota and North Dakota and in some fields multiple pathogens are present, and this pathogen complex can clearly compromise the best genetic resistance against a particular pathogen. However, whether this phenomenon can explain the increased incidence and severity of RRCR in Minnesota/North Dakota sugar beet production areas is uncertain. However, 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.* Likewise, cultivars specifically developed for resistance to RRCR may lose this resistance in the presence of BNYVV. To test this idea, studies were initiated with the following objectives: 1) Determine whether strength of resistance to BNYVV, in rhizomania-resistant cultivars growing in BNYVV-infested soil, impacts susceptibility to *R. solani* and 2) Determine if cultivars with resistance to RRCR are less resistant when grown in the presence of BNYVV.

Methods

Soil from a field in Southern Minnesota, with past history of severe Rhizomania, was used as BNYVV inoculum in a greenhouse study conducted at Texas A&M AgriLife, Bushland. Five cultivars that varied in their reported resistance to RRCR and had different dominant resistance genes against BNYVV were used in this study (Table 1). There were also four soil treatments, i.e.,1) *R. solani* + BNYVV, 2) BNYVV only, 3) *R. solani* only, and 4) a noninfested soil that was included as a healthy control. Five gallon pots were filled with potting soil and 300 g of BNYVV infested field soil were added preplant to treatment 1&2 pots that included BNYVV. Seed of the various cultivars then were planted in pots with each soil treatment and there were five replications of each cultivar x soil treatment combination arranged in a randomized complete block design. Inoculum for the *R. solani* treatments consisted of barley kernels infested with *R. solani* that was originally isolated from an infected sugar beet root from a field in southern Minnesota. Twelve weeks after emergence, three kernels were placed adjacent to the tap root on each plant in pots with soil treatments 1 & 3. Plants were watered as needed, approximately every other day and were maintained at ambient greenhouse temperatures, which ranged between 50 and 85 F.

Approximately two weeks after inoculating beets in soil treatments 1 & 3, with *R. solani,* all pots were harvested. At harvest, plants were washed from the soil, topped, root and top weights recorded, roots rated for disease severity for both RRCR and rhizomania using a 0-4 scale, and samples taken for pathogen detection and quantification in the lab. Quantitative qPCR was used to quantify virus titer in roots from treatments 1 & 2.

Results

Inoculation of five sugar beet cultivars with BNYVV and *R. solani* (BNYVV + R.s, Table 1) provided mixed results. Cultivars containing R_Z1 + *Rz2*, and R_Z2 resistance genes had significantly less rhizomania root scores than the susceptible cultivar Crystal 725. However, of the two *Rz1*cultivars used in the study, one (A) had

significantly less rhizomania root score than the susceptible cultivar while there was no significant difference between the second cultivar (C) and Crystal 725. Apparently, although these two cultivars had the same R_Z1 genes for resistance to BNYVV, they differ in minor genes which may have been reflected here. The virus quantity in the roots correlated with the visual scores (r = 0.81) indicating that visual scores corresponded well with the virus quantity in the roots. However, the five cultivars did not differ in Rhizoctonia root rot severity scores (Table 1). The cultivars had average severity scores of 2 to 3 on 0 - 4 scale.

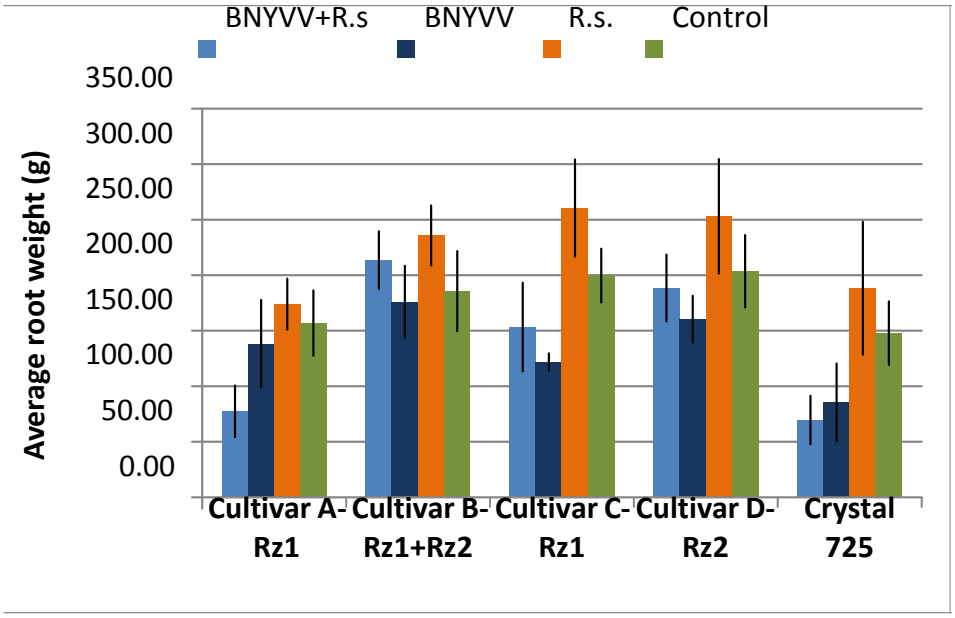
The susceptible cultivar Crystal 725 had significantly less root weight than most of the cultivars in treatments that received combinations of BNYVV + *R. solani* (Fig. 1) except in one of the R_Z1 cultivars. In all cultivars, there were no significant differences between treatments that received BNYVV+R. solani and BNYVV alone. However, there were trends in two of the cultivars that those inoculated with BNYVV + R. solani show less root weight than those inoculated with BNYVV alone, which supports our initial hypothesis. However, further studies are required to ascertain this. The uninoculated controls appeared to have less root weights than those inoculated with *R. solani* (although not significant) because the plants were inoculated close to harvest but the quality deteriorated quickly.

Table 1. Results of 2012 greenhouse study evaluating effect of BNYVV infection on RRCC.

Cultivar and source of resistance	RQ (avg)	Rhizomania rating	RRCR rating
Cultivar B-Rz1+Rz2	34.56	1.5 **a**	2
Cultivar D-Rx2	185.36	2.0 **a**	3
Cultivar A-*Rz*1	7.38	2.5 **a**	3
Cultivar C-Rz1	2133.26	3.0 **b**	3
Crystal 725	34597.3	4.0 **b**	3

Means followed by the same letter are not significantly different ($P < 0.05$; Duncan). Root severity scales: 0-4.

Figure 1. Root weights of cultivars inoculated with BYYVV +*R. solani*, BNYVV, and *R. solani*, and uninoculated control.



Discussion

Breeders and seed companies have worked hard to develop cultivars with strong resistance against soil borne pathogens of sugar beet but it is exceptionally difficult to develop cultivars with strong resistance to multiple pathogens. Even when a cultivar has strong resistance to a particular pathogen, the presence of another pathogen can adversely affect resistance to the first pathogens. For instance, Harveson and Rush, 1993 demonstrated that when BNYVV and Aphanomyces were both present, root disease was much more severe than with either pathogen

alone. In a second study, Harveson and Rush, 2002, showed that reducing soil moisture content by reducing irrigation frequency could reduce incidence and severity of sugar beet root rot in soils infested with a pathogen complex, including *Fusarium solani, Rhizoctonia solani, Aphanomyces cochlioides* and BNYVV. That study was a follow up of research conducted by Rush and Vaughn, 1993 that demonstrated that Aphanomyces root rot (which is a zoosporic pathogen similar to *Polymyxa betae*, the vector of BNYVV) could be reduced by reducing soil matric potential, i.e. reducing irrigation. An additional report on this topic was published by Piccinni and Rush, 2000 where they demonstrated that sugar beet root weight and water use efficiency, of BNYVV susceptible cultivars, were reduced when infected by BNYVV and that reduced soil water potentials, i.e., drier soils, resulted in reduced disease severity. Most recently, Price, et. al., 2010 and Workneh, et. al., 2010 reported that infection of wheat by *Wheat streak mosaic virus* (WSMV) not only reduced grain yield and quality but also reduced root function and overall plant water use efficiency. Therefore, based on previous studies with BNYVV, soil borne fungal root pathogens of sugar beet and other host/pathogen combinations, it is clear that 1) multiple pathogens greatly complicate disease management and infection by one pathogen can impact resistance to another, 2) root infection by BNYVV can negatively impact plant/water relations, and 3) increased soil moisture can result in increased losses to disease caused by soilborne fungal root pathogens.

In this present study, the severity of rhizomania on some of the cultivars was surprising, since all cultivars except the susceptible control Crystal 725 contained *Rz1, Rz2* or a combination of the two. Furthermore, cultivar A& C both have *Rz1* resistance but cultivar C had significantly more severe symptoms of rhizomania than all other cultivars, except for the susceptible control, indicating the importance of minor genes in resistance to BNYVV. Although there were significant differences in disease severity and virus titer among the different cultivars, there was not a significant correlation between rhizomania severity and severity of Rhizoctonia. This was likely due to the possibility that addition of too much inoculum of R. *solani* overwhelmed the system and all treatments developed severe RRCR. However, when looking at final root weight, it was clear that presence of both pathogens resulted in significantly greater loss than when beets were exposed to only one pathogen.

***Literature Cited*:**

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