Zebra Chip: What is the risk of disease transmission through potato tubers?

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Zebra chip disease and its impact on the Pacific Northwestern United States

Zebra chip (ZC) disease is an economically devastating disease of potatoes in the United States, Mexico, Central America, and New Zealand. The disease is attributed to a bacterium, '*Candidatus* Liberibacter solanacearum' (Lso), that is transmitted to solanaceous crops by the potato psyllid (*Bactericera cockerelli*) (Figure 1A). Potato plants that are infected with Lso show chlorosis, purple-top like discoloration, and leaf scorch in the aboveground foliage, develop aerial tubers, and produce tubers with severe internal darkening and necrosis (Figure 1B-D) (Munyaneza 2012). Lso-infection in potato fields causes a reduction in the quantity and quality of tubers harvested, leading to significant economic losses for potato growers and processors (Guenthner et al. 2012).

Figure 1. A) Potato psyllid (*Bactericera cockerelli*). B) Purple discoloration in potato plant foliage caused by Lso infection. C) Fresh ZC symptomatic tuber showing internal darkening D) Chips generated from ZC symptomatic tubers showing exacerbation of internal darkening.

To date, five haplotypes of Lso have been reported, including haplotypes A and B that affect solanaceous plants (Nelson et al. 2013). Both Lso A and B have been identified in the United States, and Mexico, but only Lso A has been reported in New Zealand (Nelson et al. 2011, 2013). Four haplotypes of the potato psyllid have also been reported, including the Central, Western, Northwestern, and Southwestern haplotypes (Swisher et al. 2012, 2014). All four haplotypes are found in the United States, while only the Western psyllid haplotype has been reported in New Zealand (Swisher et al. 2014, Thomas et al. 2011).

ZC was first identified in the United States in Texas in the early 2000s. Over the course of a decade, the disease spread across the central and western United States, until it was reported in the Pacific Northwestern states of Idaho, Oregon, and Washington in 2011 (Crosslin et al. 2012a, b). The finding of ZC in the Pacific Northwest has had negative implications on the trade of fresh and seed potatoes grown in this region of the United States to some international markets. Fear of the spread of Lso to non-infected areas has led to trade restrictions, largely in part to conflicting data concerning the importance of tuber-born ZC in the spread of the disease.

Observations made by some scientists in the United States have suggested that ZC-symptomatic tubers typically do not emerge when planted, and if they do, often die

quickly, likely before becoming a source of Lso inoculum, and thereby rendering them epidemiologically unimportant (Munyaneza, personal observation). Contrary to this, scientists in New Zealand have reported that ZC-symptomatic tubers are capable of emerging in relatively high numbers, and when they do so, generate healthy-looking foliage that often test positive for Lso based on molecular detection methods in the laboratory (Pitman et al. 2011). These conflicting observations raise significant concerns for growers and processors in the Pacific Northwestern United States as it directly affects trade of their product. Three different factors could be contributing to the conflicting reports, including Lso haplotype, potato psyllid haplotype, and potato variety used in the studies.

Assessing the risk of disease transmission through potato tubers

To assess the role of Lso haplotype, potato psyllid haplotype, and potato variety in disease transmission through seed potato, a large, multi-season field trial was begun during the 2014-growing season at the USDA-ARS farm near Yakima, Washington. In the first year, eight varieties of potatoes (Alturas, Russet Burbank, Russet Norkotah, Atlantic, Ranger Russet, Umatilla, FL 1867, and Pike) were planted in large field cages (Munyaneza et al. 2008 and Buchman et al. 2011a,b). Ten different psyllid treatments were used including (1) Central psyllid with Lso A, (2) Central psyllid with Lso B, (3) Central psyllids with Lso A and B, (4) Western psyllid with Lso A, (5) Western psyllid with Lso B, (6) Western psyllids with Lso A and B, (7) Northwestern psyllid with Lso A, (8) Northwestern psyllid with Lso B, (9) Northwestern psyllids with Lso A and B, and (10) no psyllids; each replicated three times per variety. All psyllids were released at the flowering stage, allowing ample time for disease progression during the remainder of the growing season.

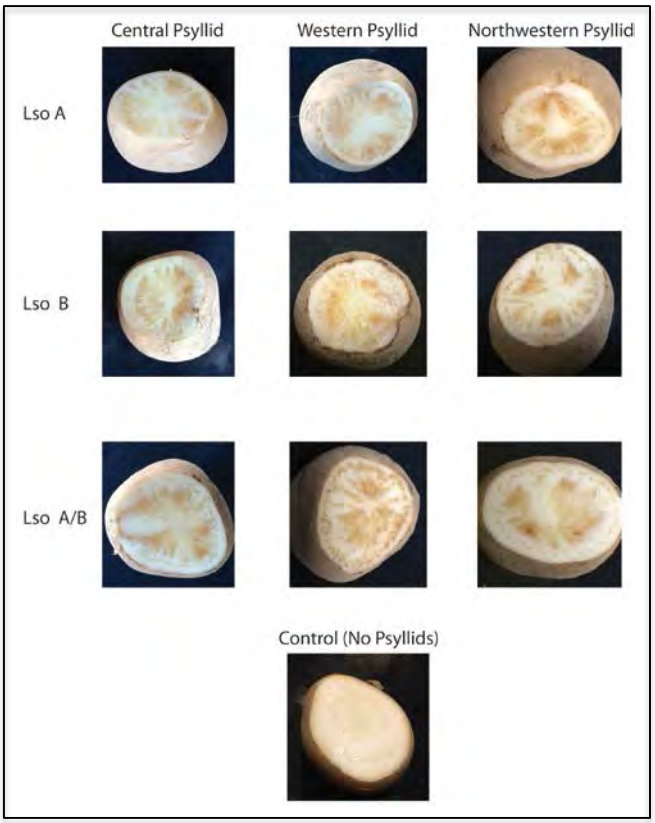

Figure 2. Severe ZC symptoms were seen in all psyllid/Lso treatments (1-9) for the Alturas potato variety. No ZC symptoms were seen in the no psyllid control.

Upon harvest, tubers from all treatments and varieties were scored for ZC disease severity. For all eight potato varieties, ZC disease severity was similar between treatments with Lso A, Lso B, and Lso A/B, regardless of psyllid haplotype used to vector the pathogen (see Figure 2 for an example of this data). For all varieties, control tubers with no psyllid/Lso pressure (treatment 10) were asymptomatic. These results

suggest that ZC disease severity caused by Lso haplotype is not the reason for the discrepancies in data between the United States and New Zealand, as has been suggested by some researchers. Current analyses are underway to determine if the rate of ZC incidence is different between Lso haplotypes. To continue this study, symptomatic tubers from the psyllid/Lso treatments (1-9), as well as asymptomatic (healthy) tubers from the no psyllid/Lso treatment (10) will be planted in the 2015 growing season for each variety. Plant emergence dates, foliar symptoms, and ZC severity in daughter tubers will be recorded during the field season.

During the 2014 field season, a second field trial was also conducted to obtain preliminary data concerning the emergence rates and quality of daughter tubers produced from ZC-symptomatic seed. In this trial, ZC-symptomatic and healthy (ZC-asymptomatic) tubers from five varieties (Alturas, Atlantic, Ranger Russet, Russet Burbank, and Umatilla Russet) were planted in large cages (Munyaneza et al. 2008 and Buchman et al. 2011a,b). Throughout the duration of the growing season, plant emergence dates and visual symptoms of the emerged plant tissue was recorded. Plants emerged from healthy tubers within 3 – 4 weeks after planting. Plants emerged at a slower rate from ZC-symptomatic tubers, with emergence dates spanning the duration of the growing season (see Figure 3 for an example of this data). In fact, plants were still emerging from ZC symptomatic tubers, and many did not even emerge, by the end of the field season (September and October), when most commercial fields in the region have been harvested. Only 2% of the plants that emerged from ZC-symptomatic seed showed ZC-like symptoms in the foliage, and these plants died after a few weeks. Upon harvest, daughter tubers from ZC-symptomatic and healthy seed were analyzed for typical tuber darkening caused by Lso, and no tubers showed visual symptoms indicating that the ZC-symptomatic seed can give rise to asymptomatic tubers.

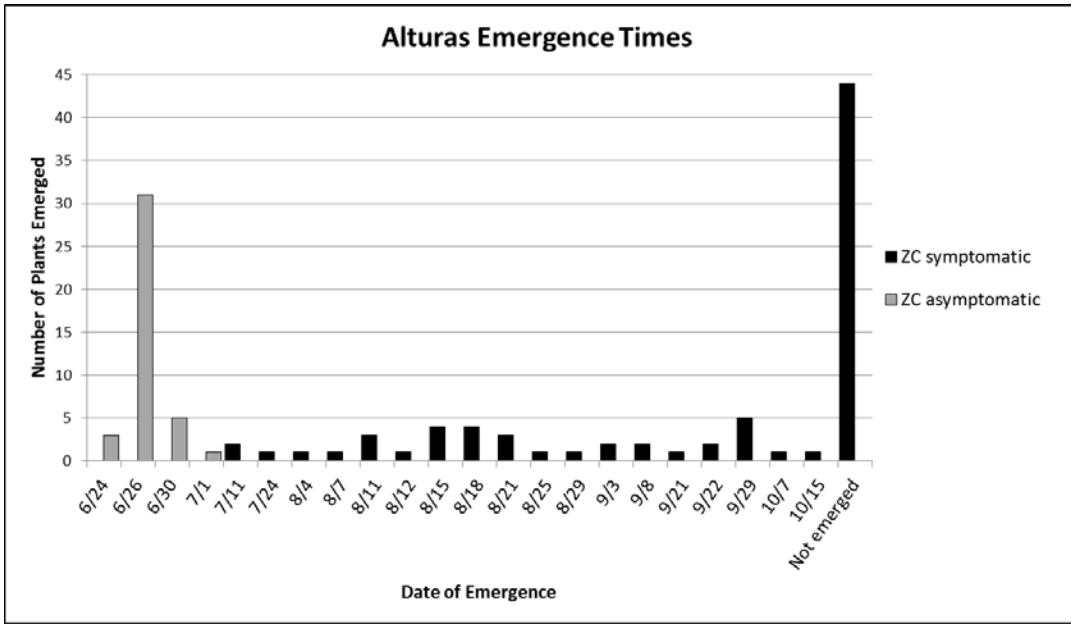

Figure 3. Emergence dates of healthy (ZC-asymptomatic) and ZC symptomatic Alturas seed during the 2014 field season.

Conclusion

In an effort to determine this risk of planting ZC tubers, thereby determining if exported ZC tubers pose a threat to spreading Lso to new areas around the world, a large, multi-year field study is underway to determine the effects of Lso haplotype, potato psyllid haplotype, and potato variety on the transmission of ZC disease. Initial data suggests that both Lso haplotypes A and B are capable of causing severe ZC symptoms in different varieties of potato, regardless of the psyllid haplotype used to vector the bacterium. Ongoing work to determine if ZC disease incidence is different between Lso haplotypes will indicate whether or not Lso haplotype was a factor that led to the conflicting reports from the United States and New Zealand. A continuation of this study in the 2015 field season will indicate whether differences in Lso infection of haplotype A or haplotype B causes differences in plant emergence rates, foliar symptoms, and daughter tuber disease severity.

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