Introduction

## 1.1. Introduction

Potatoes (*Solanum tuberosum* L.) are an important part of Idaho agriculture. Idaho is the largest producer of potatoes in the USA and for almost a third of total US production. In 2017, Idaho produced more than 131 million cwt of potatoes, estimated to be worth over a billion dollars (USDA 2017).

The potato/tomato psyllid, *Bactericera cockerelli* (Šulc), (Hemiptera: Triozidae) was described by Dr. Karel Šulc in 1909, from a specimen given to him by T. D. A. Cockerell (Šulc 1909). *B. cockerelli* is a small sternorrhynchan pest of solanaceous crops (Martin 2008, Knowlton and Thomas 1934, Wallis 1955). *B. cockerelli* was first discovered in Colorado (Šulc 1909). Potato psyllids have a history closely tied to potato growing regions and plant diseases (Richards 1973). *B. cockerelli’s* geographical distribution ranges from southern Canada to Central America and throughout the western United States (Butler and Trumble 2012, Munyanzea 2007, Rehman 2010). There has also been a recent introduction to New Zealand (Liefting et al. 2008, 2009, Martin 2008, Teulon 2009). Šulc’s original description noted the ‘large numbers’ of *B. cockerelli* on the pepper (*Capsicum annuum*) which it was collected from (Šulc 1909), foreshadowing the pest’s impact in the following century. Publications regarding the psyllid began to emerge from 1926-1928, due a disease known as ‘psyllid yellows’ (Richards 1928). Psyllid yellows infected solanaceous plants such as potatoes and tomatoes. The disease caused swollen plant nodes, aerial tubers, chlorosis, leaf purpling, dwarfing and leaf curling. (Eyer and Crawford 1933, Richards 1928, 1973) This disease was particularly damaging and could destroy entire fields if left unchecked (Binkley 1929, Richards 1973). Psyllid Yellows was controlled with chemical applications at the time (Binkley 1929, Eyer and Crawford 1933), including lime-sulfur (Daniels 1934). Chemical control was noted as a challenge due to the tendency of *B. cockerelli* to live on the undersides of leaves, which protected them from traditional spraying techniques (Binkley 1929, Daniels 1934).

Potato psyllids have become an even greater threat to agriculture since discovering their association with the phytoplasma “*Candidatus* Liberibacter solanacearum” (Lso) (Goolsby 2007a, Liefting et al. 2009, Munyaneza 2007). Lso is an unculturable gram-negative α-proteobacterium (Liefting et al 2009), which infects solanaceous plants, including potatoes (*Solanum tuberosum*). while feeding Symptoms in potato include stunting, swollen axillary buds, aerial tubers, leaf purpling, chlorosis, and reduced yield (Munyaneza et al, 2007, 2008). Infection also alters tuber sugars and phenolics, resulting in blackened stripes when tubers are fried creating a condition known as Zebra Chip (ZC) (Crosslin et al 2010, Hansen et al. 2008, Liefting 2009, Lin et al. 2009).

Multiple haplotypes of Lso have been discovered (Wen et al. 2009) with slightly different genomes (Wang et al. 2017, Wen et al. 2013, Wen et al 2009) and genetic differences which affect symptom severity (Gilkes et al. 2018, Mendoza-Herrera et al 2018, Wen et al. 2013). Additionally, Lso haplotype C has been shown to infect carrots (*Daucus carota* L.) (Alfaro-Fernandez et al. 2012, Munyaneza et al. 2010, Nelson et al. 2010, 2012, Tahzima et al. 2014) with other psyllid species acting as the vector, suggesting differences in effects between haplotypes (Nelson et al. 2010). Therefore, it may be that the historical psyllid yellows and zebra chip are the same disease (Brown et al. 2010, Mendoza-Herrera et al. 2018, Munyaneza et al. 2009, Teulon et al 2009, Wang et al. 2017), but different haplotypes which could explain the contrasting results of Venkatesan et al. (2009).

ZC was described in 1994 in Mexico (Secor and Rivera-Varas 2004, Munyanzea et al. 2009) and was detected in the United States in 2009 (Abad et. al 2009), ZC arrived in the Pacific Northwest states of Idaho, Washington, and Oregon in 2011 (Murphy et al. 2012, Crosslin et al. 2012). ZC infected tubers are unmarketable and infection caused large economic losses (Munyaneza 2007a, Rosson 2006), with yield losses ranging from 43% to 93% (Munyanzea et al. 2008, 2011). No commercial potato variety has been found with resistance to Lso (Munyaneza et al. 2011).

Various pest management practices have been investigated for management of zebra chip. Management has relied heavily on insecticides such as abamectin, imidacloprid, spiromesifen, thiamethoxam and dinotefuran (Goolsby 2007b, Guenthner et al 2012).

Biological controls have been investigated, (Walker et al. 2011) The small hoverfly *Melanostoma fasciatum* (Macquart) (Diptera: Syrphidae), the Tasmanian lacewing *Micromus tasmaniae* (Walker) (Neuroptera : Hemerobiidae) as well as other natural predators were shown to have an impact on psyllid population levels in New Zealand (Walker et al 2001), but weren’t able to suppress psyllid populations in mid or late summer.

Host plant resistance (Butler and Trumble 2012). Because multiple psyllid populations have developed resistance to common insecticides (Hernandez-Bautista et al, 2013, Liu and Trumble 2007, Prager et al, 2013, Vega-Gutierrez et al, 2008), alternative management strategies are needed.

Host plant resistance can be a valuable and desirable part of Integrated Pest Management (IPM) (Kogan 1988, Munyanzea 2012). Resistance may be based on interactions with either the vector or the pathogen itself (Kaloshian 2004). Plant resistance is traditionally divided into three categories: antibiosis, antixenosis and tolerance (Kogan and Ortman 1978, Painter 1951). Antibiosis is when feeding on the plant causes biological harm to the insect in some manner. Antixenosis is when a plant is rejected by an insect for a different, preferable host. Tolerance is when a plant resists or compensates for herbivory, reducing damage to itself without harming the insect (Mitchell et al. 2016).

Wild potatoes, such as *Solanum chacoense* L., have genes that confer natural resistance to many pests and diseases, which may be introduced to cultivated potatoes via breeding (Solomon-Blackburn and Barker 2001).

We examined psyllid settling behavior and reproductive output on three breeding clones (A07781-3LB, A07781-4LB and A07781-10LB) with high tolerance and low susceptibility to Lso (Rashidi et al. 2017), comparing responses with ‘Russet Burbank’ as a susceptible control. Results will help elucidate the mechanisms of resistance found in these germplasms and will help towards developing potato cultivars with resistance to zebra chip disease.