Plant Pathology Seminar Series

" Zebra Chip Disease and Physiological Changes of Potato in Response to '*Candidatus* Liberibacter solanacearum' Infection"

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Zebra-chip disease, caused by the vector-borne (*Bactericera cockerelli*) fastidious bacterium *Candidatus* Liberibacter solanacearum, is of recent and growing concern to potato production in North America and New Zealand (Munyaneza et al. 2007). Zebra-chip is an emerging disease that negatively affects yield, propagative potential, and both fresh and manufacturing qualities of potato tubers. Zebra-chip symptomatic plants produce tubers with distinctive brown discoloration of vascular tissue accompanied by elevated levels of free amino acids and reducing sugars which contribute to the symptoms produced by the Maillard reaction (Kumar et al. 2015). Nevertheless, little is understood about Zebra-chip disease etiology and the changes that occur within infected tubers that result in its symptoms.

Pathogen infections trigger plant biochemical defense mechanisms that may limit titer of the pathogen by means of producing hostile host tissue for microorganism reproduction and growth (Hammerschmidt 1999). Understanding the underlying mechanisms of host– pathogen interactions is of importance for developing host resistance and for developing management strategies (Kolmer 1996). However, symptoms of Zebra-chip are likely not due to the presence of the pathogen '*Ca*. L. solanacearum' itself but instead the result of pathogen-induced host biochemical responses, for instance greater levels of amino acids, carbohydrates, phenolics, and defense-associated proteins, such as peroxidases and polyphenol oxidases, compared to noninfected tubers (Navarre et al. 2009; Wallis et al. 2012).

A recent study has demonstrated that zebra-chip disease selectively induces protein catabolism in tubers through regulation of protease inhibitor levels (Kumar et al. 2015). Soluble protein content of tubers from *Ca*. L. solanacearum-infected plants was 33 % lower than from non-infected plants and substantial reductions in major tuber proteins were also observed. In contrast to non-infected tubers, the protease activity in *Ca.* L. solanacearum-infected tubers was high and the ability of infected tuber extracts to inhibit trypsin and papain proteases was greatly reduced. The selective catabolism of proteins in zebra-chip afflicted tubers undeniably affects downstream biochemical aspects of carbohydrate and amino acid metabolism, reflected by the accumulation of reducing sugars, available amino acids and reduced yield capacity. The resulting physiological changes contribute to the symptomology of zebra-chip disease in tubers and symptom severity was associated with levels of amino acid and of defense-associated proteins (Rashed et al. 2013; Kumar et al. 2015).

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