#111

Factors affecting expression of crown canker (*Leptosphaeria maculans*) in canola: implications for breeding and management

***Susan Sprague*[1]**

Angela Van de Wouw[2]
Steve Marcroft[3]
Luke Barrett[1]

[1] CSIRO Agriculture and Food, Canberra, Australia

[2] University of Melbourne, Parkville, Australia

[3] Marcroft Grains Pathology, Horsham, Australia

Background:

Blackleg disease is a serious economic constraint to canola production globally. Host resistance to yield-limiting crown canker symptoms is either qualitative or quantitative. Qualitative resistance can be rapidly overcome due to the high mutability of Leptosphaeria maculans and therefore, quantitative resistance is considered a more durable form of resistance. Phenotyping is difficult as the development of crown canker following infection of leaves by L. maculans is highly complex, involving necrotrophic and biotrophic phases of fungal growth, as well as interactions between the host, a genetically diverse pathogen population and the environment.

Objective:

Phenotyping for quantitative resistance to crown canker is critical to improve host genetic resistance. The aim was to identify canola growth stages at which quantitative resistance is expressed and factors influencing blackleg expression to improve phenotypic characterisation of canola for application to breeding.

Methods:

Multiple experiments were conducted whereby cultivars varying for quantitative resistance were inoculated using different strategies. Firstly, lines were inoculated with a genetically uniform single isolate in a controlled environment. Secondly, these same lines were inoculated with a genetically diverse blackleg population under field-like conditions. Lastly, a large experiment was carried out to determine the effect of environmental on blackleg disease, whereby a set of host lines were inoculated with the same set of three blackleg populations were grown in three different environments. Blackleg severity was determined by visual assessment of leaf lesion severity during the seedling stage, internal crown canker severity at maturity or by quantification of blackleg biomass using digital droplet PCR.

Results:

Differences in blackleg disease severity were detectable between cultivars varying in level of quantitative resistance at the seedling stage, stem elongation, flowering, and maturity. There was no relationship between the severity of lesions at the seedling stage and crown canker, but disease severity (or quantification of fungal biomass prior to the onset of visual disease symptoms) in the crown was correlated at all crop growth stages. A significant isolate x host genotype interaction was consistently measured in multiple experiments inoculated with either single isolates or diverse blackleg populations. There was a significant effect of environment on crown canker severity when host and pathogen were held constant.

Conclusions:

Our findings highlight the complex interplay between host, pathogen and environment in relation to the expression of blackleg crown canker in canola with implications for both characterisation of quantitative host resistance and disease management. In a breeding context, screening for QR must (i) focus on blackleg in the stem which can occur at any time prior to plant maturity, and (ii) consider the inoculum used (ascospore vs pycnidiospore). Screening with single isolates may be useful to detect individual QTL associated with QR but these may not contribute to QR when screened against a population. Strong QR is likely a combination of genes with additive effects requiring further breeding and characterisation to identify optimal combinations. The effect of environment should be a focus for future research with significant implications for phenotyping for breeding and identifying scenarios which lead to severe disease and yield reductions.