P-102

Soybean Stem Rot: is *Sclerotinia sclerotiorum* a necrotroph or hemibiotroph and implications for control

Laureen Blahut-Beatty, Agriculture and Agri-Food Canada, Ottawa, Canada
Yunfang Zhang, Agriculture and Agri-Food Canada, Ottawa, Canada
Asuka Itaya, Agriculture and Agri-Food Canada, Ottawa, Canada
Shea Miller, Agriculture and Agri-Food Canada, Ottawa, Canada
Michael Mahdavi, Agriculture and Agri-Food Canada, Ottawa, Canada
Suqin Zheng, Agriculture and Agri-Food Canada, Ottawa, Canada
*Daina Simmonds**, Agriculture and Agri-Food Canada, Ottawa, Canada

The success of the broad host range fungal pathogen, *Sclerotinia sclerotiorum*, is largely dependent on secretion of oxalic acid (OA). Virulence is lost in *S. sclerotiorum* mutants that do not secrete OA while transgenic plants expressing OA degrading enzymes, e.g. oxalate oxidase (OxO) attain greater resistance. For decades it has been accepted that *S. sclerotiorum* was a necrotroph and that OA was the major and possibly the only virulence factor.

To gain a better understanding of OA function, we conducted temporal and spatial studies of OA concentration [OA], pH changes and mycelial penetration of excised leaf tissue of susceptible WT soybean and its resistant isogenic OxO-transgenic line. Microscopy revealed that the fungus was capable of penetrating both germplasms to form a primary lesion but lesion expansion (host colonization) differed. To investigate the dynamics of the lesion front, [OA], pH and mycelial quantity were determined in live plant tissue, adjacent to the lesion. One day post inoculation, mycelia were detected on both germplasms, pH did not change and [OA] was low. This pH and [OA] status was maintained in the OxO germplasm over the next two days and lesion expansion was blocked; conversely, the lesion expanded rapidly in WT, concomitantly with a dramatic rise in OA, fall in pH and increase in mycelia. This underscores the role of OA for host colonization that requires high levels of OA but questions the role of OA for primary lesion formation where OA may not be needed or it may have a different, concentration-dependent role. This information suggests to us that 1. virulence factors other than OA may be contributing to primary lesion formation, 2. the fungus may be interacting with its host and may be a hemibiotroph which led us to investigate 3. alternative approaches of fungal control, e.g. HIGS (Host Induced Gene Silencing), dsRNA fungicides.