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Complexity of Leptosphaeria-Brassica interaction revealed by a novel class of disease resistance genes against blackleg disease

Nicholas Larkan
Hossein Borhan, Lisong Ma, Parham Haddadi, Isobel Parkin

AAFC, Sasktoon, Canada

Deployment of race specific resistance (R) genes is the most practical and efficient approach to control blackleg disease of canola/rapeseed (Brassica napus), caused by the fungus Leptosphaeria maculans (Lm). Lm is an apoplastic pathogen, indicating that host intercellular environment plays an important role in the initial Brassica-Leptosphaeria recognition. Not surprisingly, LepR3 and Rlm2, the two B. napus R genes against blackleg cloned so far, are membrane-localised receptor-like proteins (RLPs). The only example of molecular recognition in the Lm-B.napus pathosystem is LepR3 recognition of Lm effector (virulence) gene AvrLm1. AvrLm1's virulence function is through its interaction with the B. napus MPK9, leading to phosphorylation of MPK9 and the induction of cell death which is required for the necrotropic phase of Lm growth. We did not detect direct interaction between LepR3 and AvrLm1 by Y2H and Co-IP. AvrLm1 effector alone is sufficient convey avirulence for an Lm isolate on a B. napus line carrying LepR3 . However there are examples of epistatic interactions amongst Lm effectors, affecting the virulence of pathogen. One example we reported recently is the recognition of Lm effector, AvrLm5-9 by its corresponding R gene Rlm9 that is abolished in the presence of AvrLm4-7, another Lm effector that is genetically distinct from AvrLm9. We have recently cloned B. napus Rlm9, the AvrLm5-9 corresponding R gene, and identified the corresponding candidate alleles for Rlm3, Rlm4 and Rlm7. All these genes are from a new class of R genes distinct from the RLPs reported previously from B. napus.