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Investigation of the recognition and host target of *Phytophthora infestans* effector PiAVR2. Plant Biology Seminar Series

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Slatyer Seminar room R N Robertson Building, Research School of Biology, ANU



An important research goal in the fight against potato late blight, caused by the oomycete *Phytophthora infestans*, is to identify and characterise the host targets of key pathogen avirulence effector proteins that are likely to be delivered into host cells and to assess their contribution to the mechanism of disease resistance.

The RxLR-dEER effector gene PiAVR2 has been identified from the sequenced isolate t30-4, the product of which is recognised inside host cells by the potato R2 protein. Cloning PiAVR2 from virulent isolates revealed an additional, variant form, PiAVR2-like, which evades recognition by R2-like genes. PiAVR2 and PiAVR2-like encode proteins that differ in 13 amino acids; one or more of these specifies recognition by R2. In addition, both presence/absence polymorphism and transcriptional differences explain virulence of *P. infestans* isolates on R2 plants.

Yeast-2-hybrid analysis was used to identify a family of host proteins as candidate interactors of PiAVR2. These interactors are Ser/Thr Phosphatases from the brassinosteroid hormone signal transduction pathway. The importance of components of this pathway in plant defence against *P. infestans* was investigated using Virus-Induced Gene Silencing (VIGS).

One of the BSL family members, BSU1-like 1 (BSL1) was cloned and the interaction with PiAVR2 further investigated using Bimolecular fluorescence complementation (BiFC). In addition, PiAVR2-like was also found to interact with BSL1, suggesting that both forms share a similar function. In the model plant *Arabidopsis thaliana* BSL1 is an activator of the BR signal transduction pathway. The components upstream and downstream of BSL1 in this pathway have been found within the *S. tuberosum* cv phurjea genome suggesting that the BR pathway is intact within potato. Evidence has emerged from VIGS and BiFC experiments that BSL1 mediates indirect recognition of PiAVR2 by R2.



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