



PhD exit seminar: Signal activation of a plant defense receptor-like protein at the molecular level

Wednesday 17 July 2013 1 – 2pm

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Slatyer seminar room R.N. Robertson Building (Bldg. 46), Linnaeus Way, ANU



Plants carry resistance (R) genes that play an important role in defense to counteract attacks by invading pathogens. These R genes encode receptor proteins that enable plants to detect specific pathogen-derived molecules or so called the avirulence (Avr) factors, followed by activation of a defense response culminating in programmed cell death to inhibit pathogen growth, thereby achieving disease resistance.

My PhD research has aimed to understand plant defense activation mediated by R protein at the molecular level, in particular the leucine-rich repeat (LRR) receptor-like protein (RLP) by studying the model tomato Cf-9 RLP that confers resistance to isolates of the fungal leaf mould pathogen, *Cladosporium fulvum*, that secrete the corresponding avirulence

protein, Avr9. The RLP is a unique class of R proteins containing an extracellular LRR domain and a short cytosolic tail with no obvious signalling function, residing at the plasma membrane of plant cells. How the RLP mediates defence activation upon recognition of the avirulence factor remains elusive.

Previously, we have isolated an autoactive Cf-9 mutant, M205 that encodes a chimeric Cf-9 protein containing Cf-9A (a Cf-9 paralogue) sequence at the N terminus of the protein generated as a result of a transposon-induced recombination event. By using a domain swapping approach, we have identified a potential signalling repression domain located in the central region of Cf-9. We proposed that M205 autoactivity is a result of perturbation of signalling repression that exists at this particular region. More interestingly, this domain overlaps the region where Cf-9 detects the fungal protein Avr9, suggesting both signalling repression and recognition of Avr9 take place in the same region of Cf-9. Indeed, site-directed mutagenesis revealed a role for the six solvent-exposed Cf-9 specificity-determining residues (including one identified in this study) in signalling repression. Several models of signal activation by Cf-9 have been proposed.

During my PhD research, I also developed a transgenic *PR-5: gusA* reporter tobacco system to provide an independent and quantitative measurement of defense activation upon *Agrobacterium*-mediated transient expression of domain swaps and site-directed mutants as an alternative to qualitative assessment of necrosis. The effectiveness and sensitivity of the system for quantification of defence activation will be discussed briefly.

Presented by

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