



PhD exit seminar: mapping and functional characterization of the tomato *I-7* gene for *Fusarium* wilt resistance

Wednesday 27 November 2013 1 – 2pm

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



The tomato *I-3* and *I-7* genes, which confer resistance to *Fusarium oxysporum* f. sp. *lycopersici* (Fol) race 3, were introgressed from *Solanum pennellii* accessions LA716 (Scott and Jones, 1989) and PI414773 (McGrath et al., 1987), respectively. Lim et al. (2006) showed that *I-7* is distinct from *I-3* and does not map to the same chromosomal location as *I-3*. Since the chromosomal location of *I-7* was unknown, no markers were available for the assisted breeding of *I-7* genotypes. Therefore, the primary aim of this project was to identify the chromosomal location of the *I-7* gene and develop reliable PCR-based markers to screen for the presence of *I-7* in marker-assisted breeding.

After an intensive but unproductive marker-based search for the *S. pennellii* introgression carrying *I-7*, an RNAseq experiment was conducted to identify SNPs in root transcripts derived from genes in the introgressed region.

Transcriptome sequencing of mock-inoculated root tissue of Tristar and M82 tomato plants allowed the detection of a large number of SNPs. A plot of SNP frequency against gene position revealed a higher frequency of SNPs in 18 transcripts encoded by a cluster of genes on the long arm of chromosome 8. SNPs in four of these genes were used to design CAPS markers that confirmed the polymorphisms and showed strong linkage with *I-7*. An orthologue of Solyc08g077740, encoding an extracellular LRR receptor-like protein (RLP), was identified as the likely candidate for *I-7*.

Alleles of the *I-7* candidate gene were cloned and sequenced from *I-7* resistant (Tristar) and susceptible (M82) cultivars of tomato. Transgenic plants expressing these alleles have been generated from susceptible tomato lines (cv. MoneyMaker or cv. M82) to test for ability of the Tristar transgene but not the M82 transgene to confer resistance to *Fol* race 3. Pathogenicity tests are underway.

Experiments have also been undertaken to begin characterising the *I-7* gene. These include experiments with a mutant of the downstream signalling gene *EDS1* showing that *I-7* resistance is dependent on *EDS1* and pathogenicity tests with *Fol* race 3 carrying a knockout of *Avr3* showing that *I-7* confers resistance to *Fol* race 3 through recognition of an effector protein other than *Avr3*.

Presented by

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