

Screening natural variation in *Arabidopsis* to find genes involved in resistance, susceptibility and tolerance to clubroot disease.

William Truman

Plasmodiophora brassicae is the causal agent of clubroot disease in oilseed rape and other brassicas, and is responsible for significant crop losses. *P. brassicae* induces the formation of large galls on infected plants establishing a favourable environment for sequestration of host resources and spore multiplication. To better understand the genetic basis underpinning resistance and susceptibility to clubroot disease we carried out a screen of *Arabidopsis* accessions, quantifying pathogen levels following infection with an aggressive *P. brassicae* pathotype (P1b) prevalent in Poland. We inoculated 142 accessions and collected the hypocotyl and upper root 19 days after infection. The symptoms of disease development and gall size were scored for each accession. Quantitative PCR was performed on DNA extracted from these galls using pathogen and host primers to determine relative infection levels. 12 accessions were determined to be resistant based on pathogen quantification and absence of gall formation. Genome wide association (GWA) analysis highlighted SNPs in *ATIG32030*, a gene adjacent to the previously identified *RPBI* resistance loci, as well as *RAC1* a TIR-NBS-LRR gene and an intergenic region on chromosome 4. Confirmation of the role of *RPBI* homologs in mediating resistance to *P. brassicae* and their potential interaction with the TIR-NBS-LRR candidate is being pursued with complementation lines and a CRISPR/Cas9 approach. Higher pathogen titers largely correlated with symptoms of water deficit stress in the aboveground parts of plants, however some accessions appeared more tolerant of infection while others were hypersusceptible. Quantitative phenotyping of these symptoms in the aerial tissue of accessions is being carried out with automated collection of RGB, fluorescence and hyperspectral imaging.