

Molecular basis of the *Arabidopsis-Botrytis cinerea* interaction

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The signal transduction pathways involved in establishing plant disease resistance are currently being dissected using *Arabidopsis thaliana* as a model organism. Two major signalling pathways exist mediated by salicylic acid and jasmonic acid/ethylene respectively. Many other components of defence pathways have been identified although the role of these components in defending against different pathogens is not clear. We are investigating the molecular changes that occur during infection of a plant by *Botrytis cinerea* and how these affect the virulence of the pathogen.

One approach is the use of *Arabidopsis* mutants with altered disease resistance. A transgenic line expressing luciferase under the control of a defence-related promoter (*PR-1*) was mutagenised and a dominant mutant, *cir3*, isolated. This mutant displayed luciferase activity in the absence of pathogen infection. Additional defence genes (*PR-1*, *PR-2*, *PR-5* and *PDF1.2*) are also constitutively expressed in *cir3* and the mutant shows enhanced resistance to *B. cinerea*. The lesion size and fungal content of infected leaves is reduced in *cir3* compared to the parental line. Resistance to *B. cinerea* in *cir3* is dependent on both salicylic acid and the EIN2 protein (part of the ethylene signalling pathway).

We are also investigating how the plant-pathogen interaction is influenced by the pathogen genotype. Camalexin (the main phytoalexin in *Arabidopsis*) has been shown to impact on the severity of *B. cinerea* infection and camalexin-deficient *Arabidopsis* mutants develop much larger lesions than wild-type. We have screened *B. cinerea* isolates for camalexin-insensitivity and so far have identified one such isolate. These studies will help us tease apart how the pathogen and host are interacting and possible points at which disease progression can be influenced.