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Onset and maintenance of plant immune priming

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Plants can increase the responsiveness of their immune system after perception of specific environmental signals. This priming of immunity provides broad-spectrum resistance against pests and diseases. Our previous discovery of the IBI1 receptor of the chemical priming agent beta-aminobutyric acid (BABA) has allowed us to study the molecular mechanisms controlling the onset of immune priming in Arabidopsis. Using this system, we found that VOZ transcription factors physically interact with the IBI1 receptor after pathogen attack, mediating SA-independent priming of cell wall through interaction with ABA-dependent signalling pathways. This work also led to the discovery of a structural BABA analogue, R-beta-homoserine (RBH), which primes distinct defence pathways against biotrophic and necrotrophic pathogens, but is less toxic to plant metabolism than BABA. In parallel, we are studying the epigenetic mechanisms controlling long-lasting immune priming, which stems from our previous discovery that diseased Arabidopsis produces epigenetically primed progeny. Characterization of Arabidopsis mutants that are altered in genome-wide DNA (de)methylation revealed that nearly half of the defence-related transcriptome is directly or indirectly controlled by DNA (de)methylation. Further investigation of the transgenerational effects of disease on DNA methylation indicated global impacts on gene body DNA methylation. Finally, we are mapping and characterizing epigenetic quantitative trait loci (epiQTLs), using a fully characterized mapping population of Arabidopsis epigenetic recombinant inbred lines (epiRILs). Transcriptome analysis of the immune response in a selection of resistant epiRILs points towards a trans-regulatory role of stress-responsive transposable elements in transgenerational immune priming.