



S. Bradbrook/NPG

WRKY transcription factors that bind PopP2 and AvrRps4 and that are acetylated by PopP2, which inhibited their interaction with nuclear DNA. Catalytically active PopP2 promoted *R. solanacearum*-induced disease in *Arabidopsis*, and PopP2 acetyltransferase activity dampened the upregulation of host PTI genes in *Nicotiana benthamiana* (Le Roux *et al.*) and interfered with resistance to AvrRps4-carrying bacterial strains in *Arabidopsis* (Sarris *et al.*). Thus, PopP2 is a virulence factor that dampens host immunity by altering the DNA binding of host transcription factors.

In uninfected tissues, RRS1-R is a negative regulator of RPS4 activation. This is consistent with the autoimmune phenotype of the *Arabidopsis slh1* mutant, which has a single amino acid insertion in the WRKY domain of RRS1-R that disrupts functional DNA binding. The authors propose that the targeting of the WRKY domain of RRS1-R by PopP2 and AvrRps4 is a trigger for activation of the RRS1-R–RPS4 pair, which results in ETI. Le Roux *et al.* showed that mimicry of RRS1-R acetylation by the Lys1221Gln substitution activates RPS4-dependent immunity in *Arabidopsis*.

Both groups conclude that PopP2 and AvrRps4 have evolved to block the activity of host WRKY transcription factors and that the WRKY domain of RRS1-R is an integrated effector target for the detection of such pathogen virulence activity. As many other NLR pairs contain additional domains of unknown function, it is possible that these could also be mimics that might lead to the discovery of novel host virulence targets.

Kirsty Minton

Two studies published in *Cell* describe the presence of a DNA-binding domain in a plant immune receptor that mimics the host transcription factors targeted by pathogen virulence proteins. This binding domain enables the host to detect attempted manipulation of its transcriptional response to infection.

The plant immune response to pathogens triggered by plasma membrane pattern recognition receptors (known as pattern-triggered immunity (PTI)) can be suppressed by pathogen virulence factors (effectors). In turn, plant hosts detect these pathogen effectors or their effects on host proteins through intracellular receptors that mount a second line of defence known as effector-triggered immunity (ETI). ETI is mediated by nucleotide-binding leucine-rich repeat (NB-LRR) receptors that resemble mammalian NOD-like receptors (NLRs). Plant NLRs often function in pairs, of which one or both members are fused to other protein domains of unknown relevance. For example, detection of the bacterial effectors PopP2 and AvrRps4 requires the *Arabidopsis* NLR pair RPS4 (Resistance to *Pseudomonas syringae* 4) and RRS1-R (Resistance

to *Ralstonia solanacearum* 1). RRS1-R, unlike most NLRs, contains at its carboxyl terminus a ‘WRKY’ DNA-binding domain that is conserved in plant WRKY transcription factors that orchestrate biotic stress responses. Le Roux *et al.* and Sarris *et al.* now show that the integrated WRKY domain of RRS1-R functions as a decoy to detect pathogen interference with transcription factors involved in host defence.

Both groups showed that catalytically active PopP2 acetylates lysine residues in the invariant WRKY domain of RRS1-R, in particular Lys1217 and Lys1221. Sarris *et al.* also showed that the WRKY domain of RRS1-R is necessary for recognition of AvrRps4. In both studies, acetylation of RRS1-R by PopP2 reduced its affinity for W-box DNA sequences, such as those that are found in the target promoters of WRKY transcription factors. Le Roux *et al.* used homology modelling to predict that Lys1221 acetylation of RRS1-R would markedly decrease the electrostatic potential at the interface with DNA.

This effect of PopP2 on DNA binding of RRS1-R mimics its effect on host transcription factors. The two groups identified several *Arabidopsis*

“ the integrated WRKY domain of RRS1-R functions as a decoy to detect pathogen interference ”

