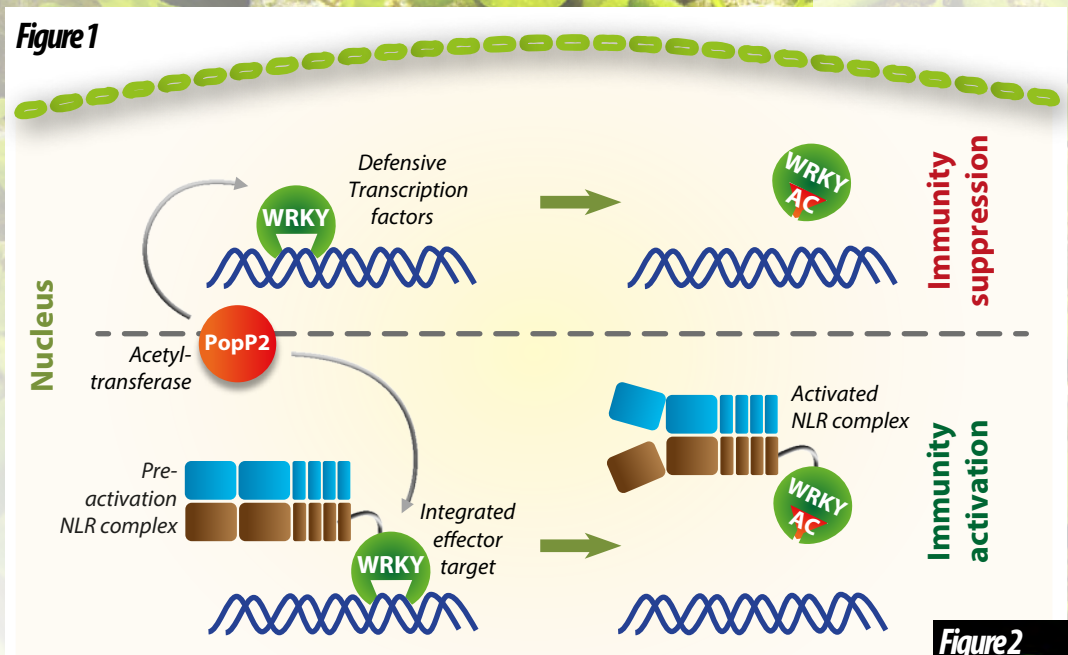
An immune receptor pair with an integrated decoy converts pathogen disabling of defensive transcription factors into resistance

Microbial pathogens infect host cells by delivering virulence factors (effectors) that interfere with defenses. In plants, intracellular nucleotide-binding/oligomerization domain (NOD)-like receptors (NLRs) detect specific effector interference and trigger immunity by an unknown mechanism. The Arabidopsis interacting NLR pair, RRS1-R with RPS4, confers resistance to different pathogens including *Ralstonia solanacearum* bacteria expressing the acetyltransferase effector, PopP2. We show that PopP2 directly acetylates a key lysine within an additional C-terminal WRKY transcription factor DNA-binding domain of RRS1-R. This disrupts RRS1-R DNA association and activates RPS4-dependent immunity (see figure 1).

Figure 1



PopP2 employs the same lysine acetylation strategy to target multiple defense-promoting WRKY transcription factors, causing loss of WRKY-DNA binding and transactivating functions needed for defense gene expression and disease resistance. Thus, RRS1-R integrates an effector decoy with an NLR complex at the DNA to switch a potent bacterial virulence activity into defense gene activation.

Figure 2: *Response of* A. thaliana *plants root-inoculated with* Ralstonia solanacearum. *RRS1-R/RPS4-dependent immunity is activated by PopP2 type III effector activity (left). In plants lacking the RRS1-R receptor, the bacterial pathogen is not recognized, allowing rapid spread of the pathogen inside tissues (right).*

Figure 2

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