URLs

PLANT PATHOGENS

Coordinated defence

An important link between the two defence systems that plants use to detect and combat microbial pathogens has been reported in *Cell*.

Innate immune systems in plants and animals detect pathogen-associated molecular patterns (PAMPs), which are pathogen-specific molecules such as bacterial lipopolysaccharide. In animals, Toll-like receptors (TLRs) detect PAMPs, whereas in plants, the innate immune response (basal defence) is mediated by PAMP receptors. Following PAMP detection, receptors activate signalling pathways that effect defence responses.

A second system in plants recognizes bacterial virulence factors (type III effectors) that are injected into plant cells by the type III secretion system. When a resistance (R) protein that recognizes a specific type III effector is present, a defence response that includes cell death, named the hypersensitive response (HR), occurs and the type III effector is known as an avirulence protein (Avr). If the R protein is absent, there is no HR and the type III effector mediates its virulence function. Some R proteins recognize interactions between type III effectors and cellular proteins instead of interacting with the type III effector. Such R proteins 'guard' the cellular protein that is targeted by the type III effector — the basis of the guard hypothesis.

Here, Kim *et al.* investigated how pathogen Avr proteins affect plant defences using a model system of *Pseudomonas syringae* infection of *Arabidopsis thaliana*. The *P. syringae* type III effector AvrRpm1 phosphorylates *A. thaliana* RIN4, and the R-protein guard RPM1 detects RIN4~P. A second *P. syringae* type III effector, AvrRpt2, degrades RIN4, which activates the R-protein guard, RPS2. Using molecular and cellular approaches, Kim *et al.* showed that both AvrRpm1 and AvrRpt2 inhibit PAMP-induced defences. Plus, overexpression of RIN4, the target of these bacterial proteins, also inhibited the PAMP response. As plants lacking RIN4 had increased PAMP responses, RIN4 is a *bona fide* negative regulator of the PAMP response.

However, the plant is not easily outsmarted by its pathogen and uses the R-protein guards RPM1 and RPS2 to oversee RIN4. If RIN4 functions are perturbed, a second line of defence is activated by the R-protein guards. This is the first study to show a link (RIN4) between the induction of PAMP responses and the R-protein-mediated defence system, which indicates that plants coordinate their defences — the plant uses RPM1 and RPS2 to guard against pathogens that perturb the PAMP-signalling system.

The authors speculate that their findings might have parallels in animal immunity. A *Salmonella enterica* sv. Typhimurium protein AvrA inhibits innate immune responses in animals and can also induce apoptosis, akin to the induction of the HR in plants by bacterial virulence proteins — so perhaps animals also use guard proteins.

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References and links

ORIGINAL RESEARCH PAPER Kim, M.G. *et al.* Two *Pseudomonas syringae* type III effectors inhibit RIN4-regulated basal defense in *Arabidopsis*. *Cell* **121**, 749–759 (2005)