PLANT DISEASE RESISTANCE

Triggering resistance

In plants, as in animals, invading microorganisms are recognized by the detection of conserved pathogen proteins, known as pathogen-associated molecular patterns (PAMPs). However, how the detection of PAMPs contributes to disease resistance has so far been unclear. A study in *Nature* by Thomas Boller and colleagues now describes an important step in understanding innate immunity in plants, showing that recognition of a bacterial PAMP, flagellin, has a central role in triggering resistance.

Flagellin is a key component of the bacterial flagellum, which is important for motility in many pathogenic species, and recognition of this protein triggers a number of defence responses in plants. Boller and colleagues investigated whether the plant flagellin-perception system also has a role in resistance to bacterial infections. To do this, they made use of the flg22 peptide, which represents a highly conserved region of flagellin and is sufficient to trigger defence responses. The authors pre-treated *Arabidopsis thaliana* leaves with flg22 and studied the host response to a subsequent challenge with a pathogenic strain of *Pseudomonas syringae*. Plants that had received the pre-treatment showed an approximately 100-fold lower level of bacterial replication than those that had not been treated. This flg22-induced resistance was shown to depend on *A. thaliana* FLS2 — a Toll-like receptor that recognizes flagellin; in plants that expressed a non-functional form of FLS2, flg22 pre-treatment was no longer able to induce resistance.

Boller and colleagues then tested whether FLS2 has a role in resistance under natural infection conditions, in which plants are exposed to the flg22 sequence in the context of pathogenic bacteria rather than by treatment with a flg22 peptide. When plants were infected with *P. syringae* by infiltration directly into the intercellular leaf space, no significant difference in bacterial replication or disease symptoms was seen between wild-type plants and those lacking functional FLS2. But when the bacteria were sprayed onto the leaf surface, disease symptoms developed far more rapidly in *fls2* mutants. So, resistance stimulated by FLS2-mediated flagellin detection seems to be involved in preventing invading bacteria from penetrating the leaf surface, rather than in later stages of infection.

This study also showed that PAMPs other than flagellin must also be able to trigger resistance in plants. Pre-treatment of *A. thaliana* with a crude extract from another pathogen, *Agrobacterium tumefaciens*, provided protection from *P. syringae*, despite the fact that *A. tumefaciens* lacks a flg22 sequence. In addition, resistance was also stimulated in *fls2* mutants, indicating that PAMP recognition must be mediated through other receptors, as well as by FLS2. The identification of other PAMPs that stimulate disease resistance in plants, and their host-encoded receptors, will be key aims for future studies.

Louisa Flintoft

References and links

ORIGINAL RESEARCH PAPER Zipfel, C. *et al.* Bacterial disease resistance in *Arabidopsis* through flagellin perception. *Nature* **428**, 764–767 (2004)