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IMMUNITY

One receptor, many pathogens

Most plant pattern recognition receptors induce immune responses by detecting molecular patterns typical to one group of microbes. A newly identified complex, on the other hand, monitors effector proteins widely distributed among bacteria, fungi and oomycetes, casting a new light on the evolution of pattern recognition in plants.

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Monitoring potentially pathogenic microbes through the perception of conserved microbe-associated molecular patterns (MAMPs) is a common principle of innate immunity in both plants and animals[1]. A focus in the field of plant immunity has been the discovery of new pattern recognition receptors (PRRs) and their cognate MAMPs[2]. This is due in part to their potential for conferring broad-range disease resistance in crops important for food security, which are constantly under the menace of new pathogenic microorganisms. PRRs identified so far recognize molecular patterns specific to one group of microbes; for example, the PRRs FLS2 and EFR recognize the bacterial flagellin and EF-Tu proteins, respectively, and CEBiP/CERK1 recognizes chitin in the fungal cell wall[2]. Contrastingly, Albert *et al.*[3] report in this issue of *Nature Plants* that a novel PRR complex in *Arabidopsis* recognizes a common epitope that induces immune responses, from a group of proteins called Nep1-like proteins (NLPs), which are widely distributed among bacteria, fungi and oomycetes[4]. These findings not only extend our understanding of PRRs but also open possible applications in molecular breeding, given the wide distribution of their ligands. Further, involvement of NLPs in microbial pathogenicity[4] also raises an interesting question regarding the evolution of pattern recognition in plants.

NLPs are widespread proteins among a taxonomically diverse group of microbes, especially those associated with plants, and many of them induce cytotoxicity in dicotylenous plants[4]. Recently it was shown that the 20- or 24-amino-acid long peptides (nlp20 or nlp24) conserved in NLPs were sufficient to induce immune responses similar to other MAMPs in *Arabidopsis*, indicating that NLPs are a MAMP with cross-kingdom distribution, and these peptides represent their recognition motif[5,6]. Searching for the receptor of the conserved motif in NLPs, Albert *et al.* screened mutant collections of leucine-rich repeat (LRR) type receptor-like kinases (LRR-RLKs) and receptor-like proteins (LRR-RLPs) for nlp20-induced ethylene production, and found that the mutants for RLP23 did not respond to nlp20. *Arabidopsis* accessions mutated for RLP23 were also blind to the peptide. Expression of RLP23 restored the responsiveness to nlp20. Furthermore, pre-treatment with nlp20 increased disease resistance against bacterial and oomycete infection in Col-0 but not in an *rlp23* mutant. The LRR domain of RLP23 was shown to specifically bind the nlp20 peptide, leading the authors to conclude that RLP23 is a receptor for nlp20.

How does RLP23 activate immune signalling? As it does not carry any

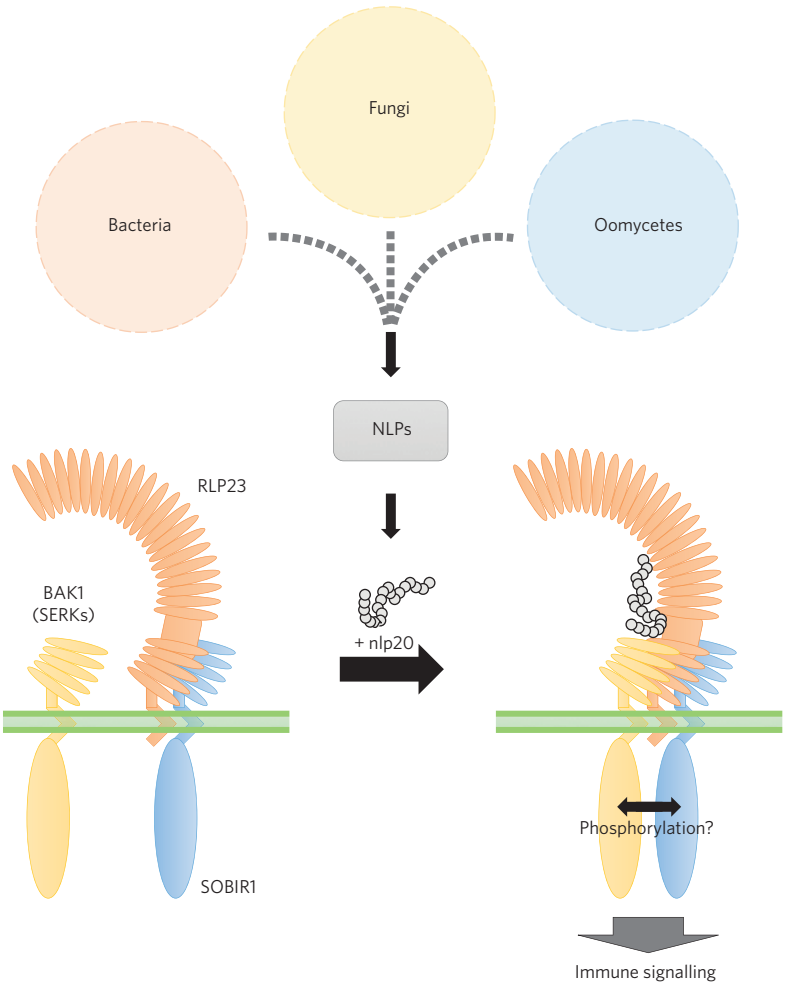

Figure 1 | Activation model for the RLP23–SOBIR1–BAK1 complex. NLPs are secreted proteins produced by divergent microorganisms including bacteria, fungi and oomycetes. The conserved peptide motif nlp20 in NLPs induces defence responses in a subset of plant species. RLP23 and SOBIR1 form a complex in the absence of the nlp20 ligand. After nlp20 binds to RLP23, BAK1 (a member of the SERK family) is recruited into the receptor complex, which then activates immune signalling.

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intracellular signalling domain, the authors tested the idea that RLP23 could form a receptor complex with RLKs carrying a signalling kinase domain. As expected, immunoprecipitation of RLP23 followed by MS/MS analysis of bound partners identified SOBIR1 and BAK1, two RLKs already known for their involvement in RLP signalling[7,8]. RLP23–SOBIR1 interaction was ligand-independent but RLP23–BAK1 interaction was nlp20-dependent. A requirement for SOBIR1 and BAK1 in immune signalling was also confirmed with corresponding mutants. Combined, these results strongly suggest that the ligand-dependent recruitment of BAK1 to the preformed RLP23–SOBIR1 complex forms a tripartite signalling complex that activates downstream responses (Fig. 1).

As NLPs are present in a wide range of pathogenic microbes across different kingdoms, the receptor for a common NLP epitope is expected to recognize those pathogens and confer disease resistance in the plants lacking the corresponding receptor. The authors showed that the transfer of *RLP23* gene into potato, which does not respond to nlp20 (ref. 6), increased resistance against devastating oomycete (*Phytophthora infestans*) and fungal (*Sclerotinia sclerotiorum*) pathogens, supporting this notion. The rather limited distribution of the nlp20 sensing system among plant species[6], also suggests the usefulness of RLP23 for such an application. Although the present results are encouraging, it should further be clarified in future studies to what extent this strategy is effective by using different sets of crops and pathogens.

Plant immunity is described as a multi-layered system, consisting of pattern-triggered immunity (PTI) that covers a wide range of potential pathogens and effector-triggered immunity (ETI) mediated by a much more specific recognition of pathogen effectors by corresponding resistance proteins in host plants, which were obtained in the course of co-evolution between plants and pathogens[9,10]. The unique features of the nlp20–RLP23 system raise questions about such a clear-cut discrimination between PTI and ETI. The suggested function of NLPs as microbial effectors that enhance host susceptibility to pathogens[4] indicates that the system could be considered as belonging to ETI. On the other hand, widespread distribution of NLPs among taxonomically diverse microbes and perception of the conserved motif, nlp20, by a typical LRR-RLP and LRR-RLK plasma membrane complex seems to be a hallmark of pattern recognition in PTI. As such, this pathogen detection system appears a good prompt to rethink the evolutionary relationships between PTI and ETI. In relation to this, the authors suggested an intriguing possibility that the acquisition of an NLP effector in pathogenic microbes might have driven the emergence of corresponding PRRs in host plants[6]. As Thomma *et al.* argued a few years ago, "there is a continuum between PTI and ETI"[11]. Support for this idea is certainly increasing today. ❐

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