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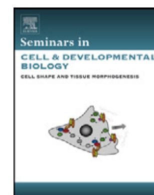

Review

In the trenches of plant pathogen recognition: Role of NB-LRR proteins

Maryam Rafiqi [a,1,2], Maud Bernoux [b,1,3], Jeffrey G. Ellis [b,4], Peter N. Dodds [b,*]

[a] Plant Cell Biology Group, Research School of Biological Sciences, Australian National University, Canberra, ACT 2601, Australia
[b] CSIRO Plant Industry, GPO Box 1600, Canberra, ACT 2601, Australia

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ABSTRACT

As in nearly every discipline of plant biology, new insights are constantly changing our understanding of plant immunity. It is now clear that plant immunity is controlled by two layers of inducible responses: basal responses triggered by conserved microbial features and specific responses triggered by gene-for-gene recognition of pathogen effector proteins by host resistance (R) proteins. The nucleotide-binding domain leucine-rich repeat (NB-LRR) class of R proteins plays a major role in the combat against a wide range of plant pathogens. The variation that has been generated and is maintained within these conserved proteins has diversified their specificity, subcellular localisations, activation and recognition mechanisms, allowing them to specifically adapt to different plant–pathogen interaction systems. This review addresses recent advances in the molecular role of NB-LRR proteins in pathogen recognition and activation of plant defence responses.

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Contents

1. Introduction 1017
2. Pathogen recognition 1018
 2.1. Plant R proteins encode conserved molecular sensors 1018
 2.2. Indirect and direct recognition: diverse pathways to defence response activation 1018
 2.3. Diverse and dynamic intracellular localisation patterns of NB-LRR proteins 1020
3. NB-LRR protein activation: from resting to action 1020
 3.1. Resting NB-LRR proteins are kept in a competent state by molecular chaperones 1021
 3.2. Intramolecular interactions are crucial for regulation of R protein activation 1021
 3.3. The NB domain acts as a molecular switch and regulator of protein activity 1021
4. From activation to signalling: what's hidden behind? 1022
5. Future perspectives 1022
 References 1023

1. Introduction

Like animals, plants are engaged in a constant battle against the wide range of microbial pathogens that seek access to host nutrients. However, plants lack the circulating adaptive immune system of animals and have responded to this pathogen challenge by developing a cell-autonomous innate immunity system to combat the diversity of pathogen populations [1]. Plant disease resistance is based mainly on two interconnected forms of innate immunity [2,3]. In the front line of the plant immune response, cell surface-located transmembrane receptors, referred to as pattern recognition receptors (PRRs), recognize pathogen-associated molecular patterns (PAMPs) which are conserved molecular signatures present in many classes of microbes, including non-pathogens. Examples of PAMPs include peptide motifs in bacterial flagella, lipopolysaccharides of Gram-negative bacteria, or the conserved epitope elf18 from the bacterial translation factor EF-Tu [4,5]. Perception of the PAMPs derived from microorganisms activates MAP kinase signalling cascades that result in the induction of primary or basal defence responses that inhibit microbial invasion. This system, referred to as PAMP-triggered immunity, or PTI, suc-

* Corresponding author. Tel.: +61 2 6246 5039.
E-mail addresses: maryam.rafiqi@anu.edu.au (M. Rafiqi), maud.bernoux@csiro.au (M. Bernoux), jeff.ellis@csiro.au (J.G. Ellis), peter.dodds@csiro.au (P.N. Dodds).
[1] These authors contributed equally to this work.
[2] Tel.: +61 2 6125 4539.
[3] Tel.: +61 2 6246 5377.
[4] Tel.: +61 2 6246 5421.

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cessfully prevents the growth of non-pathogenic microbes in plant tissues [2,4]. PTI mechanisms probably also underlie many examples of non-host resistance, that is the resistance of, for example, rice to a pathogen that has evolved to infect tobacco.

Of course many microbes have evolved to cause disease on certain host plants, which necessitated the development of strategies to evade PTI. This is apparently largely achieved through the action of virulence effector proteins that are produced by the pathogen to directly suppress PTI-based host surveillance mechanisms. In fact, many plant pathogens have been shown to secrete and deliver effector proteins inside host cells, and a primary function of many of these is to suppress plant basal defence [2,3,6–9]. For instance AvrPto of *Pseudomonas syringae* directly inhibits the intracellular kinase signalling domains of several PRRs in *Arabidopsis* [10]. Other fungal and oomycete effectors have been shown to suppress plant defence responses [11–14]. In the co-evolution of host–pathogen interactions, plants in turn have acquired highly specific cognate resistance (R) proteins that either directly or indirectly (see Section 2.1) recognize pathogen effector proteins. This recognition activates a second molecular defence layer known as effector-triggered immunity (ETI). ETI triggers strong disease resistance by boosting basal defence reactions and activating programmed cell death at pathogen infection sites, known as the hypersensitive reaction (HR) [15]. The function of R genes was recognised long before the advent of molecular biology, in the classical gene-for-gene model of plant disease resistance [16]. In this model resistance was ascribed to the genetic interaction between dominant plant R genes, now known to encode immune receptors, and dominant pathogen avirulence (Avr) genes, which we now know to encode the effector molecules recognised by the R protein receptors.

Although engaging different molecular receptors and activating different signalling pathways, PTI and ETI networks are believed to interconnect to stop pathogen infection [3,17]. Here we review recent advances in understanding plant resistance to infection by host-adapted pathogens, focussing on the role of the specificity determinants of ETI, the R proteins.

2. Pathogen recognition

2.1. Plant R proteins encode conserved molecular sensors

Many plant R genes have now been cloned, conferring resistance to numerous classes of pathogens such as fungi, oomycetes, bacteria, viruses, nematodes and sucking insects. Most belong to one of two main classes which present a recognition domain to either the extra- or intracellular environment [18,19]. One class encodes membrane bound proteins with an extracellular leucine-rich repeat (LRR) domain, either with or without an intracellular kinase domain. The corresponding Avr proteins are secreted into the apoplastic space during infection, where they may be detected. However, the majority of known *R* genes encode intracellular proteins with an LRR domain and a nucleotide-binding (NB) domain. These are among the largest proteins found in plants and form the largest group of plant R proteins with about 150 NB-LRR genes found in *Arabidopsis* and about 600 genes in rice [20]. These domains are also present in mammalian nucleotide-binding oligomerisation domain (NOD)-like immune receptors which play a role in PAMP recognition and subsequent induction of innate immunity responses in animals [1,21,22]. Some plant NB-LRR proteins also contain an N-terminal TIR domain related to the intracellular signalling domain of the *Drosophila* Toll protein and mammalian interleukin-1 receptor proteins. These animal proteins are part of the Toll-like receptor (TLR) family involved in triggering innate immunity in response to extracellular PAMPs [23]. The second common class of NB-LRR proteins, also called non-TIR-NB-LRR, contains either a coiled-coil (CC) domain (CC-NB-LRR) instead of the N-terminus TIR domain [24], or an N-terminal domains of as yet unknown function (X-NB-LRR) [25].

The LRR domain appears to be the major determinant of recognition specificity. Most amino acid variation occurs in this domain as a result of strong positive selection [26] and domain swaps between related R proteins with different recognition specificity show that this region indeed controls recognition specificity [27–30]. LRR domains are characterised by a 25–30 amino acid repeat motif containing Leu or other aliphatic residues at conserved positions and occur in a diverse range of proteins where they are generally implicated in protein–protein interactions [31]. The basic repeat motif corresponds to structural units containing a β-strand and α-helix, with the β-strands forming a parallel β-sheet, in which adjacent strands are more closely packed than the opposed helices, resulting in a characteristic curved structure [32]. Most variation is clustered in the β-sheet region which appears to form a protein interaction surface. Further confirmation of the role of the LRR regions of NB-LRR R proteins in Avr recognition comes from the work of Jia et al. [33] who showed that the LRR domain of the rice Pi-ta resistance protein interacts in a yeast two-hybrid assay with the corresponding Avr-Pita protein from *Magnaporthe grisea*. However there is also evidence that the TIR domain can influence recognition specificity [34] and that intramolecular interactions between the TIR-NB and LRR regions are important for the function of these proteins (discussed in Section 3).

2.2. Indirect and direct recognition: diverse pathways to defence response activation

The principal function of NB-LRR proteins is as the recognition component of the plant ETI system. NB-LRR proteins can recognise pathogen effectors either indirectly (Guard Model), by sensing biochemical modifications on plant proteins targeted by the pathogen Avr protein, or directly through physical association with the Avr protein (Receptor-Ligand Model) (Fig. 1A) [35,36].

The Guard Model was first suggested to explain the mechanism by which *P. syringae* AvrPto is detected by both tomato proteins Pto (a serine threonine kinase) and Prf (an NB-LRR). Prf protein was found to associate with the Pto protein and their association is thought to keep Prf in an inactive state (see Section 3.1). Prf is activated upon Pto-AvrPto interaction which disrupts the inhibitory action of Pto and allows Prf to induce plant defence responses [36,37]. In this recognition model, R proteins are postulated to act as 'guards' that monitor the state of other host proteins (guardees) that may be targeted by pathogen effectors. The recognition mechanism proposed by the Guard Model supports therefore the ability of a limited number of NB-LRR R proteins to recognise a multitude of pathogen effectors, by focussing on the more limited number of potential host protein targets [18]. Another classical example that conforms to the Guard Hypothesis is the *Arabidopsis* RIN4, RPM1 and RPS2 proteins. The guardee RIN4 (a negative regulator of plant defence) is targeted by three *P. syringae* Avr proteins, AvrRpm1, AvrB and AvrRpt2, and guarded by two CC-NB-LRR proteins, RPM1 and RPS2. RIN4 is either phosphorylated or cleaved upon interaction with AvrRpm1, AvrB or AvrRpt2, and this modification of RIN4 results in disruption of its inhibitory action on RPM1 or RPS2 [38,39]. Another example of the Guard Hypothesis is illustrated by the *Arabidopsis* protein PBS1 which is degraded by the *P. syringae* effector protein HopAR1 (previously AvrPphB). The CC-NB-LRR protein RPS5 is able to detect this degradation to activate resistance mechanisms [40,41]. Interestingly, RIN4 and PBS1 (the guardees) associate with the CC domain of their respective "guards", RPM1 and RPS5 [42,43], and Pto interacts with the N-terminal domain of Prf [37]. The corresponding NB-LRR proteins could therefore sense the presence of the pathogen indirectly through the mod-

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