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Review

Plant systems for recognition of pathogen-associated molecular patterns

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ABSTRACT

Research of the last decade has revealed that plant immunity consists of different layers of defense that have evolved by the co-evolutional battle of plants with its pathogens. Particular light has been shed on PAMP- (pathogen-associated molecular pattern) triggered immunity (PTI) mediated by pattern recognition receptors. Striking similarities exist between the plant and animal innate immune system that point for a common optimized mechanism that has evolved independently in both kingdoms. Pattern recognition receptors (PRRs) from both kingdoms consist of leucine-rich repeat receptor complexes that allow recognition of invading pathogens at the cell surface. In plants, PRRs like FLS2 and EFR are controlled by a co-receptor SERK3/BAK1, also a leucine-rich repeat receptor that dimerizes with the PRRs to support their function. Pathogens can inject effector proteins into the plant cells to suppress the immune responses initiated after perception of PAMPs by PRRs via inhibition or degradation of the receptors. Plants have acquired the ability to recognize the presence of some of these effector proteins which leads to a quick and hypersensitive response to arrest and terminate pathogen growth.

Contents

1. Introduction

Recognition of non-self and subsequent activation of defense against the attacking pathogen is known from all multicellular organisms. These hosts express pattern recognition receptors (PRRs) that specifically recognize the so-called microbe or pathogen-associated molecular patterns (M/PAMPs) [1,2]. Such patterns are invariant (surface) structures that are indispensable to the microorganism, do not exist in the host and thereby allow the host to recognize them as non-self to fend off invading pathogens. As a consequence of the observation of striking similarities between plant and animal innate immune systems the plant immunity community adopted the nomenclature from animal innate immunity that was proposed by Medzhitov and Janeway in the nineties of the last century [3]. The formerly named basal or non-cultivar-specific resistance now designated PAMP-triggered immunity (PTI) is fully in agreement with the definition of animal innate immunity. It is an ancient conserved first layer of defense, it is based on the perception of conserved microbial structures by PRRs and it is effective against a broad spectrum of invading microorganisms. That PTI is indeed

Abbreviations: P/M/DAMP, pathogen/microbe/danger-associated molecular pattern; PTI, PAMP-triggered immunity; ETS, effector-triggered susceptibility; ETI, effector-triggered immunity; LRR-RLK, leucine-rich repeat receptor kinase; PRR, pattern recognition receptor; TLR, TOLL-like receptor; LysM, lysine motif.

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sufficient to restrict pathogen growth was shown by mutants in PTI components that are more susceptible to the invaders [4]. To successfully grow and proliferate on their host, virulent pathogens have to override the first line of defense. Therefore, these pathogens inject effector proteins into the plant cell that can suppress PTI. Together with additional effectors, that make use of the host's nutrients, the pathogens can survive and complete their life cycle [5,6]. This phenomenon is called effector-triggered susceptibility (ETS). Notwithstanding the fact that plants do not possess an adaptive immune system, plants have evolved a plant specific second line of defense. Specific detection molecules, the so-called resistance (R) proteins guard effector-mediated dysfunction of host components [7]. By this, virulence factors are turned into avirulence factors that allow the plant to specifically detect formerly successful pathogens. The perception of the presence of these avirulence factors leads to a drastic and fast hypersensitive response that restricts the growth of the aggressor. While evolutionary older PTI restricts diseases on most plants against most pathogens, effector-triggered immunity (ETI) has evolved by the evolutionary battle of successful pathogen races with specific plant cultivars that have acquired the ability to recognize the interaction of effector proteins with their targets in the cell [8]. For further information on ETI, please refer to the respective chapter in this issue.

2. Principles of innate immunity in plants and animals

Striking similarities between the animal and plant innate immune systems became obvious when innate immune receptors from both kingdoms were identified [9]. The first PRR identified in animals (the TOLL receptor from *Drosophila*) shows a modular structure consisting of an extracellular leucine-rich repeat domain, one transmembrane domain and a cytoplasmic TIR (TOLL-interleukin receptor) domain that interacts via adaptor proteins with the cytoplasmic kinase IRAK [10]. This structure is similar to the first identified PRR from plants, the flagellin receptor FLS2, that also contains the LRR- and transmembrane domain but already includes the kinase domain in the same polypeptide [11]. In vertebrates, PRRs were identified as perception molecules for PAMPs [1]. Bacterial LPS (lipopolysaccharide) for example is recognized by the best-studied PRR, the TOLL-like receptor 4 (TLR4). Upon ligand perception the activation of an inflammatory response is initiated to restrict microbial growth in the host [12]. Other bacterial PAMPs recognized in animals are peptidoglycan, flagellin and unmethylated DNA fragments. In addition, fungus-derived PAMPs (e.g. glucans, zymosan and mannans), as well as virus-derived single and double-stranded RNA, proteins or CpG-DNA fragments are sensed as non-self by animal PRRs [13].

Similarities in the molecular strategy of animal and plant innate immune systems expand to the receptors' ligands as well. PAMPs such as LPS, flagellin, and peptidoglycans are also recognized in plants [14–17]. These molecules are often also referred to as MAMPs (microbe associated molecular patterns) since their origin is not restricted to pathogenic microbes [18]. Besides patterns of microbial origin, endogenous host molecules can trigger defense reactions in animals and plants. These so-called danger-associated molecular patterns (DAMPs) are released upon damage of host tissue caused, e.g. by infection and alert the host that its integrity is threatened [19].

In addition, principles of the molecular architecture of immune signaling pathways are conserved across kingdom borders. Immune responses triggered by these PAMPs comprise the activation of MAP kinase cascades that lead to the induction of an (inflammatory) defense response and the production of antimicrobial peptides as well as the activation of defense-related genes in plants and animals [9]. These immune responses comprise the first layer of defense in both kingdoms. In animals, an adaptive immune system is superimposed on the ancient innate immune system that confers specific and efficient immunity based on non-heritable recombination-derived receptors that are specifically adapted to the invading pathogens. Differentiation of the respective lymphocytes is triggered by the perception of PAMPs by PRRs underlining the importance of the innate immune system for the whole defense potential of the organism. Crosstalk between the two immunity layers also exists in plants. Even though plants lack an adaptive immune system, a plant specific second layer of defense, the effector-triggered or resistance/avirulence gene-specific immunity, interacts with PTI via derepression and amplification of PAMP-induced defense responses [20].

3. Signals activating plant innate immunity

PAMPs are defined as follows: indispensable to the microorganisms, structurally conserved and unique to microbes and thus not present in the hosts. The first molecule that was characterized to fit the definition as a PAMP perceived by plants was PEP13, a 13 amino acid peptide motif of a *Phytophthora sojae*-derived cell-wall transglutaminase [21]. The *in planta* recognized motif is coincidentally the most conserved sequence shared by a number of *Phytophthora* species. Another proteinaceous PAMP is the elongation factor Tu from *Pseudomonas* [22]. Although this protein is not surface exposed, an 18 amino acid acetylated minimal motif (elf18) is recognized by plants and triggers ion fluxes, calcium influx, and MAP kinase activation in plants [23]. The best studied bacterial PAMP peptide is flg22 derived from bacterial flagella that induces very similar defense reactions as elf18 in Arabidopsis and with differing epitope specificity also in other plants [16,24]. The structural and functional conservation of flg22 within bacterial species and its importance for both activation of defense and flagellum function was recently shown by Naito et al. [25]. Other proteinaceous PAMPs are the cold shock protein [26], elicitins [27], HrpZ1 and NEP1-like proteins (NLP). HrpZ1 is an effector protein that is secreted into the plants apoplast by the bacterial type-III secretion system but is not injected into the plant cell as described for other effector proteins [28,29]. HrpZ1 together with the NLPs form a special class of PAMPs as they induce cell death in plants [30,31]. Both are able to form pores in the host membranes and induction of typical PAMP-induced defense responses might be due to toxic action on the plant. PAMPs also comprise non-proteinaceous patterns such as lipids (cerebrosides, ergosterol, cutin monomers), carbohydrates (glucans, chitins, uronides, cellodextrin) and combinations of the former such as peptidoglycan or lipopolysaccharides [9].

The perception of pathogens must be expanded to the surveillance of the integrity of the plant itself. The so-called danger-associated molecular patterns (DAMPs) are signals that are encoded by the host and that are released upon plant damage. One representative is Arabidopsis AtPep1, a peptide released from a propeptide that is induced after infection. Upon perception of the peptide by its cognate receptor PEPR1 the plants get alerted and activate defense responses [58]. For an overview about P/M/DAMPs perceived by plants see Table 1).

4. Pattern recognition receptors

In animals as well as in plants the perception of the invading pathogens is based on perception of PAMPs by pattern recognition receptor complexes. PRRs are predominantly located on the plasma membrane but can also localize to endosomal compartments or can even be cytoplasmic [9].