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Review

The role of chitin detection in plant–pathogen interactions

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Abstract

Despite the deployment of antifungal defence strategies, fungal diseases occur in all types of multicellular organisms. In plants, the role of fungal chitin as pathogen-associated molecular pattern that activates host defence is well established. Interestingly, plants employ homologs of the chitin immune receptors to initiate microbial symbiosis. Accumulating evidence shows that fungal pathogens developed secreted effectors to disarm chitin-triggered host immunity.

Keywords: LysM; Immune receptor; Fungal disease; Resistance; Pathogenicity; Symbiosis

1. Introduction

Together with cellulose, a D-glucose homopolymer that constitutes the primary structural component of plant cell walls, chitin is the most abundant carbohydrate found in nature. Chitin is an *N*-acetyl-D-glucosamine (GlcNAc) homopolymer that is found as primary structural component in the cell walls of fungi, the exoskeleton of arthropods and egg-shells of nematodes.

Fungal cell walls are largely composed of carbohydrate polymers that include β-glucans, chitin and mannans in addition to glycoproteins. While the three carbohydrate components are interspersed throughout the cell wall, chitin presumably localizes near the plasma membrane, whereas the mannans are thought to line the outer cell wall [1,2]. Importantly, extensive differences in cell wall composition occur not only between fungal species, but even between strains of the same species and between morphological structures of the same strain [3,4].

Fungi are the most important plant pathogens that cause significant yield losses worldwide [5]. Similar to animals, also plants possess an innate immune system to sense the presence of microbial invaders by means of receptors that detect microbial-derived molecules, or by receptors that detect plant-manipulating activities of pathogens [6]. As plants do not contain chitin, this molecule is recognized as non-self component and activates host immune responses [7,8].

2. Plant defence against microbial infections

The first obstacles in plants against pathogen attack are formed by physical, enzymatic and chemical barriers that are constitutively present and include the cuticle, a waxy layer that is deposited on the plant surface, the plant cell wall and anti-microbial compounds that include enzymes, peptides and secondary metabolites [9,10]. In addition to these preformed defences, inducible defence mechanisms can be activated upon recognition of pathogen attack [11,12]. These include structural fortifications at the site of attempted pathogen ingress and the production and release of anti-microbial molecules at the site of infection as well as in tissues away from the initial infection site. Furthermore, a localized apoptosis-like hypersensitive cell death response may occur at the infection site. Eventually this all can result in systemic acquired resistance (SAR) a long-lasting (weeks to months) state of induced immunity against a broad range of pathogens [13].

Although over-simplified [14], the current view of the evolution of inducible defence responses in plant pathogen interactions is nicely captured in the so-called zig-zag model

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[15]. In this model, the first inducible defences are activated by pattern recognition receptors (PRRs), which are cell surface receptors that recognize microbial-associated molecular patterns (MAMPs; also referred to as pathogen-associated molecular patterns or PAMPs) as non-self components; so-called MAMP-triggered immunity (MTI; also referred to as PAMP-triggered immunity or PTI; Fig. 1). This defence response includes local cell wall fortifications, production of reactive oxygen species (ROS), and the production and release of anti-microbial compounds, which collectively will stop most microbial invaders. The key element of the zig-zag model is the notification that successful pathogens are able to overcome MTI by the use of secreted effectors that perturb host defences in a pro-active manner, thus establishing effector-triggered susceptibility (ETS; Fig. 1) [15–17]. Many of these effectors appear to have molecular targets inside host cells. While pathogenic Gram negative bacteria typically inject such effectors directly into the host cytoplasm by means of their type-III secretion machinery, fungal effectors carry host cell uptake motifs that mediate autonomous translocation into the host cytoplasm [16,17]. It has been shown that some effectors directly target and destabilize MAMP receptor complexes [18,19]. So far, however, the molecular targets of most fungal effectors remain enigmatic [17].

During evolution, plants have evolved to intercept the activity of particular pathogen effectors through novel receptors that are typically called resistance proteins. Although several resistance proteins have been characterized as cell surface receptors, the majority of these receptors are cytoplasmic proteins of the nucleotide binding leucine-rich repeat (NB-LRR) type that again activate inducible host defences, generally referred to as effector-triggered immunity (ETI; Fig. 1). It was initially proposed that defence responses associated with ETI occur more quickly, are more prolonged and stronger than MTI responses, and generally include the hypersensitive cell death, an apoptosis-like programmed cell death response at the site of attempted penetration [15]. However, it rather appears that ETI and MTI both can be robust or weak, depending on the specific interaction between effector and receptor, and possibly also on environmental conditions [14]. In any case, ETI halts pathogen ingress and poses a selection pressure on the pathogen to either lose or mutate the effector that is recognized to avoid the activation of host immunity [20], or to acquire novel effectors to suppress the ETI response [21]. This arms race will continue because new plant receptors will evolve that recognize either the mutated or the newly acquired effectors to again activate ETI [15].

According to the zig-zag model, MTI is generally triggered by a wide range of microbes because MAMPs are conserved throughout classes of microbes, whereas the propensity to trigger ETI is typically pathogen strain-specific [15]. However, it is increasingly being appreciated that the distinction between MAMPs and effectors, and, therefore, between MTI and ETI, cannot strictly be maintained, and that there is a continuum between MTI and ETI [14].

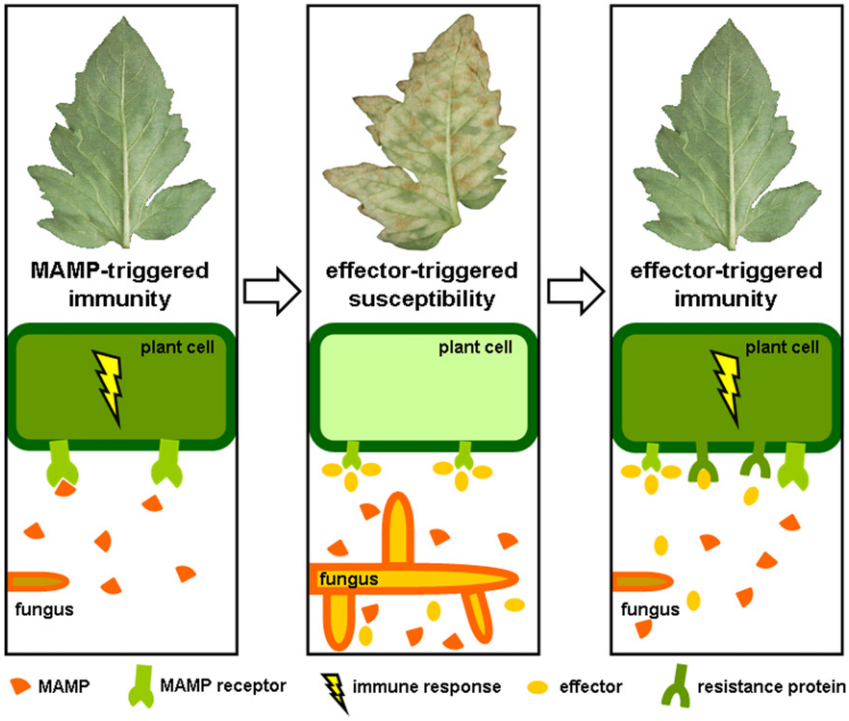

Fig. 1. The co-evolutionary arms race between pathogen and plant. Initially, perception of microbe-associated molecular patterns (MAMPs) by plant receptors on the cell surface triggers defence responses leading to MAMP-triggered immunity (left panel). Successful pathogens will evolve to secrete effector proteins that perturb MAMP-triggered immunity. The employment of these effectors results in susceptibility of the host, which is known as effector-triggered susceptibility (middle panel). In turn, evolution drives plants to evolve receptors, so-called resistance proteins, which recognize these effectors and resurrect defence responses. The renewed state of immunity is referred to as effector-triggered immunity (right panel). This arms race continues when pathogens alter effectors to avoid recognition or develop novel effectors to perturb host immunity, which triggers plants to develop novel recognition specificities to reactivate defence.