I. Introduction

The plant immune system can be activated by two different types of signals, by microbial signatures and by features signifying malfunctioning of plant processes. In other words, plants respond to signals indicating ‘non-self’ or to signals specifying ‘disturbed self’. Perception of these signals is mediated by two different types of receptors: a class of membrane-resident receptors that identify extracellular pathogen-derived molecules and a class of mainly intracellular receptors that recognize the presence or the activity of pathogen-derived effector molecules inside the host cell. Extracellular ligands can be evolutionarily conserved, broadly occurring molecules of functional importance for the microbe although without being specifically intended for the interaction with a host and, hence, cannot easily be modified without loss of functionality. These molecules that are absent from the potential host have been termed microbe-associated molecular patterns (MAMPs; Mackey and McFall 2006) or pathogen-associated molecular patterns (PAMPs; Medzhitov and Janeway 1997; Nürnberger et al. 2004). In the following, the former more general term is used, because plant-recognized PAMPs can also be found in non-pathogenic microbes. MAMPs are recognized on the surface of plant cells by specific pattern recognition receptors (PRRs; Nürnberger and Kemmerling 2006).

The current view of the plant immune system and its evolution was outlined in a recent review article as a four-phased model (Jones and Dangl...
Most plants are resistant to most invading pathogens due to a basic resistance strategy, in which conserved MAMPs are recognized by PRRs and pathogen development is prevented by MAMP-triggered immunity (MTI; in Jones and Dangl (2006) termed PAMP-triggered immunity; PTI). To get access to the plant food market and to allow microbe accommodation, pathogens need to avoid recognition or suppress its consequences. For this purpose, they secrete effectors that interfere with MTI, thus causing effector-triggered susceptibility (ETS), formerly called basic susceptibility (see Chap. 9). Once the plant evolves a receptor (resistance protein) to specifically recognize one of these effectors directly or through its activity, the consequence is effector-triggered immunity (ETI), formerly called cultivar-specific resistance. Meanwhile, hundreds of plant genes encoding putative resistance proteins of the NB-LRR type have been identified in plant genomes (Meyers et al. 2003). In the next phase, the pathogen 'learns' to avoid or to suppress ETI, but selection can also produce new resistance gene specificities, resulting in re-established ETI. This chapter focuses on the plant perception of MAMPs from fungal and Oomycete pathogens and on signaling molecules that are involved in the intracellular signal transduction leading to plant immunity (MTI). Further details on ETI (with an emphasis on bacteria-plant interactions) have been recently reviewed (Abramovitch et al. 2006; Chisholm et al. 2006; DeYoung and Innes 2006; Jones and Dangl 2006).

II. MAMP Perception

A. MAMPs of Oomycete or Fungal Origin

Most of our knowledge on MAMP perception originates from studying bacterial MAMPs. For instance, highly conserved parts of the protein building block of bacterial flagellin are recognized by the innate immune system of many plant species and animals (Zipfel and Felix 2005). Plants and animals also have perception systems for lipopolysaccharides, the major structural components of the outer membrane of Gram-negative bacteria (Zipfel and Felix 2005). Some MAMPs are less widely recognized. For instance, the most conserved motif of bacterial cold-shock proteins, the RNA-binding motif, serves as a MAMP in members of the Solanaceae (Felix and Boller 2003). In contrast, the Brassicaceae are able to perceive the N-terminus of elongation factor Tu (EF-TU), the most abundant and highly conserved protein in the bacterial cytoplasm (Kunze et al. 2004).

Fungi and Oomycetes are also characterized by the presence of surface-localized or secreted MAMPs. Typical cell wall components, such as Oomycete β-glucans and fungal chitin, have long been recognized as inducers (‘general elicitors’) of plant defense (Ayers et al. 1976; Hadwiger and Beckman 1980). Two additional cell wall proteins were characterized as Oomycete MAMPs: a Phytophthora transglutaminase with its conserved Pep-13 epitope (Brunner et al. 2002) and a cellulose-binding elicitor lectin protein (CBEL; Gaulin et al. 2006). Also secreted proteins, such as Oomycete lipid transfer proteins (elicinits), necrosis and ethylene-inducing protein 1 (Nep1) from Fusarium oxysporum (Bailley 1995) and its structural homologues in various Oomycetes, fungi and bacteria (Nep1-like proteins, NLPs; Pemberton and Salmond 2004; Qutob et al. 2006), as well as a fungal endopolygalacturonase (Poinssot et al. 2003) and ethylene-inducing xylanase (EIX; Bailley et al. 1990) were described as ligands in MAMP perception. Finally, the typical fungal sterol, ergosterol (Granado et al. 1995), as well as fungus-specific sphingolipids, cerebroside A and C (Koga et al. 1998), need to be mentioned in this context as well. All these components are not found in higher eukaryotes and, hence, represent molecular signatures that characterize putative microbial plant invaders. Although a variety of different fungal and Oomycete MAMPs was shown to trigger defense reactions in plants, knowledge on the corresponding receptors and the biochemical mechanisms linking receptor activation and intra-cellular signaling has remained sparse with only very few exceptions. Three PRRs involved in the perception of different fungal or Oomycete cell wall components and a secreted fungal protein, respectively, are treated in the following to exemplify concepts for signal perception at the plant plasma membrane and its conversion into an intracellular response.

B. Pattern Recognition Receptors

1. β-Glucan Receptor – Enzymatic Ligand Amplification and Optimization

Binding sites for β-glucans were described 20 years ago (Schmidt and Ebel 1987), but isolation, cloning