Plant defence strategies against phytopathogenic bacteria

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Abstract

Plants have developed a highly effective innate immunity which uses trans-membrane/surface-localised pattern recognition reporters (PRRs) to respond to slowly evolving microbial- or pathogen-associated molecular patterns (MAMPS or PAMPs). Plants also use the polymorphic nucleotide-binding domain and leucine-rich repeat (NB-LRR)-type immune receptors to respond to the pathogen virulence factors. The NB-LRR protein products are encoded by resistance (R) genes. Plant pathogenic bacteria use a type III secretion system (T3SS) to inject proteins called type III effectors (T3Es) into the plant cell. The T3Es can induce defence if recognised by specific host resistance proteins. In susceptible plants, T3Es promote virulence and enhance susceptibility by interfering with host innate immunity. This paper reviews strategies used by plants to resist attack by plant pathogenic
bacteria.

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Introduction

Plants have sophisticated mechanisms to resist attack by the majority of potential pathogens (Chisholm *et al.,* 2006, Caplain *et al.,* 2008; Zhou and Chai, 2008). In contrast, plant pathogens use diverse life strategies to attack their hosts (Büttner and Bonas, 2003; Alfano and Collmer, 2004; Nomura *et al.,* 2006; Grant *et al.,* 2006; Jones and Dangl, 2006). This current review gives insights into infection by phytopathogenic bacteria and mechanisms that host plants use to recognize and respond to attack by bacteria.

Strategies used by phytopathogenic bacteria to attack the host

Many Gram-negative plant pathogenic bacteria, including Pseudomonas, Xanthomonas, Ralstonia, Erwinia, Pectobacterium, Pantoea and Brenneria enter the plant through stomata, hydathodes or via wounds and proliferate in intercellular spaces (the apoplast). They inject a set of type III effector proteins (T3Es) into the host cells using a type III secretion system (T3SS) to suppress the host's innate immunity (Büttner and Bonas, 2002 and 2003; Alfano and Collmer, 2004; Nomura *et al.,* 2006; Grant *et al.,* 2006; Jones and Dangl, 2006; Cunha et al., 2006 and 2007; Block *et al.,* 2008; Zhou and Chai, 2008). These T3Es can induce defence if recognised by specific corresponding host resistance (R) proteins (Fig.1). In susceptible plants, T3Es can alter host protein turnover either by direct cleavage or by modulating ubiquitination and targeting the 26S proteasome. The T3Es that alter host protein turnover have been intensively studied in animal and plant bacterial pathogens and examples of such effectors include AvrPtoB (HopAB2), HopM1, AvrPphB and AvrRpt2 from Pseudomonas syringae (Büttner and Bonas, 2003; Debroy *et al.,* 2004; Abramovitch *et al.,* 2006a and b; Cunha *et al.,* 2007 and Block *et al.,* 2008), XopD from Xanthomonas spp. (Hotson *et al.,* 2003; Chosed *et al.,* 2007) and YopJ from a human pathogen Yersinia spp. The family members of YopJ in Xanthomonas spp. include AvrXv4, AvrBsT, AvrRxv and XopJ (Mukherjee *et al.,* 2006). The second strategy that plant pathogenic bacteria use to attack and suppress plant innate immunity is alteration of transcriptional regulation through for example transcriptional activation or ADP-ribosylation of RNA-binding proteins (Block *et al.,* 2008; Cunha *et al.,* 2008). The T3Es that target host transcription include AvrBs3, PthXo6, PthXo7 and TALEs from Xanthomonas spp., and HsvG and HsvB from Pantoea agglomerans (Kay *et al.,* 2007; Romer *et al.,* 2007; Bogdanove *et al.,* 2010). In P. syringae HopU1 (HopPto2) targets RNA metabolism and suppresses pathogen-associated molecular pattern-triggered immunity (PTI) and effector-triggered immunity (ETI) (Fu *et al.,* 2007). Plant pathogenic bacteria can also inhibit the kinases involved in plant defence signalling, either by addition or removal of phosphates or by direct inhibition (Block *et al.,* 2008). Two T3Es (HopAI1 and HopAO1) from P. syringae are known to employ this

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(phosphorylation or dephosphorylation of host protein) strategy (Li *et al.,* 2007). In addition to secreted pathogen effector proteins, other small molecules such as toxins, plant hormones, autoinducers (quorum sensing regulation) and exo-polysaccharides (EPS) are also used as virulence factors to promote disease.

Bacterial toxins such as coronatine, syringomycin, syringopeptin, tabtoxin and phaseolotoxin (Bender *et al.,* 1999) have diverse mechanisms of action, including mimicking plant hormones, forming pores in host membranes, or inhibiting host metabolic enzymes to cause necrosis or chlorosis (Abramovitch *et al.,* 2006). For example, P. syringae strains produce the jasmonic acid mimic that suppresses salicylic acid-mediated defence against biotrophs. Plant pathogenic bacteria such as P. syringae have also evolved virulence factors such as coronatine to cause stomatal re-opening as a pathogenesis strategy (Melotto *et al.,* 2006; Hofius *et al.,* 2007).

For the pathogen to be successful in infecting a host it has to gain entry into the tissue, acquire nutrients, multiply and spread to uninfected tissues or neighbouring plants (Jackson, 2009). However, plants on the other hand, have a sophisticated mechanism to recognise and resist the attack by the majority of potential pathogenic bacteria. This molecular struggle between plant pathogenic bacteria and the host plants is complex.

Plant defense network

Plants have developed a sophisticated defence network that resists attack by potential pathogens. First, the epidermis with its hydrophobic cuticle and gated entry sites (stomata) prohibits direct access to more exposed plant cells and nutrient flow. The plant cell wall provides an additional barrier that blocks direct contact between pathogen and host cell. Second, plants have developed several active layers of defence to prevent microbial access to nutrients (Jackson, 2009).

Plants response to infection using two-branched innate immune system. The first branch uses transmembrane/surface-localised pattern recognition receptors (PRRs) that respond to microbial- or pathogen-associated molecular patterns (MAMPS or PAMPs). The microbial molecules that can be recognised include bacterial flagellin, lipo-polysaccharide (LPS), peptidoglycan or elongation factor Tu (EF-Tu) and proteins. The identified MAMP perception in plants are the perception of flagellin, elongation factor Tu (EF-Tu) and type I-secreted sulphated protein activator of XA21-mediated immunity (Ax21) by FLS2, EFR and XA21, respectively (Boller and Felix, 2009; Lee *et al.,* 2009; Zipfel, 2009). The perception of the other MAMPS and transduction in plants are not clearly understood. Recognition of some classes of MAMPs such as flagellin is relatively conserved but others such as EF-Tu are not widely recognised across the species. Since non-pathogens also synthesize these molecules, a more appropriate term is 'microbe-associated molecular pattern' 'MAMPs' (Ausbel, 2005). The MAMPs/PAMPs are essential for survival, fitness or virulence of the pathogens and they are evolutionary conserved molecules (He *et al.,* (2007).

However, based on Zipfel et al. (2004) and Sun et al. (2006) some MAMPs may exist as variable but not conserved products. For example, the variation of defence-eliciting ability among flagellin purified from different strains of Xanthomonas campestris pv. campestris (Xcc). It was found that, a single amino acid polymorphism in Xcc flagellin was critical for the FLS2-mediated defence eliciting ability. However, the mutation of FLS2 did not affect the growth of virulent Xcc strains and symptom development in Arabidopsis using different infection assays (Sun *et al.,* 2006). In contrast, FLS2 was found to be important in the defence against the virulent Pseudomonas syringae pv. tomato DC3000 shown in Arabidopsis (Zipfel *et al.,* 2004). The MAMPs recognition can be variable in different plants. For example, the recognition of flg22 in Arabidopsis by FLS2 was found to display characteristics which were distinct from those observed in tomato and rice, suggesting the specificity in ligand–receptor interactions (Felix *et al.,* 1999; Chinchilla *et al.,* 2006).

Plant immunity to pathogens can occur if the PRRs perceive the PAMPS (Chisholm *et al.,* 2006). This type of immunity is referred to as PAMP-triggered immunity (PTI). The PTI can be inhibited by T3Es delivered into the host cell to enhance virulence by many bacterial pathogens. The second branch acts on T3Es largely inside the cell, using the polymorphic nucleotide-binding domain and leucine-rich repeat (NB-LRR)-type immune receptor protein products encoded by most R genes (Dangl and Jones, 2001; Zipfel and Felix, 2005; Chisholm *et al.,* 2006, Caplain *et al.,* 2008; Zhou and Chai, 2008). Recognition of some of the T3Es inside the cell by the corresponding resistance proteins activates the effector-triggered immunity (ETI).

Jones and Dangl, (2006) described a four phased 'zigzag' model showing how PAMPS are recognised in plants. In phase 1, PAMPs/MAMPs are recognised by PRRs resulting in PAMP-triggered immunity (PTI). In phase 2, successful pathogens release effectors that contribute to pathogen virulence and can interfere with PTI resulting in effector-triggered susceptibility (ETS). If the effectors are recognized by one of the NB-LRR proteins, it results into effector-triggered immunity (ETI) (Phase 3). Recognition can either be indirect, or through direct NB-LRR recognition of an effector. The ETI is an accelerated and amplified PTI response, resulting in disease resistance and, usually, but not always a hypersensitive response (HR) or programmed cell death at the infection site. In phase 4, pathogens can avoid ETI either by shedding or diversifying the recognized effector gene, or by acquiring additional effectors that suppress ETI (Ausubel, 2005; Jones and Dangl 2006; Chinchilla *et al.,* 2006).

Pathogen recognition by NB-IRR

Pathogen recognition by NB-LRR immune receptors has been suggested to involve three models (Caplan *et al.,* 2008). Direct recognition occurs by direct physical association of the pathogen effectors with the R immune receptor, which is similar to a ligand binding to its receptor (Fig. 1). Three effectors: AVR-Pita from the fungus Magnaporthe grisea, PopP2 from the bacterial wilt and AvrL567 from the flax rust fungus seemed to have a direct interaction with: the LRR domain of Pita (Bryan *et al.,* 2000), Arabidopsis RRS1-R immune receptor and flax multigenic loci (Dodds *et al.,* 2004 and 2006), respectively resulting in resistance. The indirect recognition of pathogen T3Es by NB-LRR immune receptors involve detecting products of action on host targets. This is consistent with the guard hypothesis (Van der Biezen and Jones 1998; Dangl and Jones, 2001; Jones and Dangl, 2006). The key principles of this hypothesis are (i) an effector acting as a virulence factor has a target(s) in the host, (ii) by manipulating or altering this target(s) the effector contributes to pathogen success in susceptible host genotypes and (iii) effector perturbation of a host target generates a 'pathogen-induced modified self' molecular pattern, which activates the corresponding NB-LRR protein, leading to ETI. The R immune receptor can be constitutively bound to its guardee host factor or alternatively R immune receptor may bind to its guardee only after the guardee is bound to the pathogen effector. The pathogen effectors can mimic a transcription factor and directly induce the expression of a non-NB-LRR resistance protein. It is known that the majority of NB-LRRs use indirect recognition of pathogens and that, immune receptors employ components originating from the plasma membrane, cytoplasm, chloroplasts and nucleus during recognition (Caplan *et al.,* 2008).

Members of NB-LRR class of resistance genes can be subdivided by the N termini: the TIR class, containing resistance genes with homology to the Toll protein of Drosophila and the Interleukin1 receptor protein of mammals, and the non-TIR class, which is further subdivided into proteins containing putative coiled-coli (CC) regions and those with no recognisable protein structure (NX) at their N termini (Hammond-Kosack and Jones, 1997). The NX class require some specific intra-molecular domain interaction for its function. For instance, work with Rx protein, a closely related NB-LRR protein from potato (Moffett *et al.,* 2002) and interaction of Bs2 and SGT1in co-immuno-precipitation studies (Leister *et al.,* 2005). The formation of Bs2-SGT1-containing protein complex is required for the expression of bacterial disease resistance. This Bs2/AvrBs2-mediated resistance was found to require two genes namely NbSGT1 and NbNPK1 (Leisser *et al.,* 2005).

The outcome of interaction between plants and pathogenic bacteria can be classified as compatible, incompatible and non-host interactions. Compatible interactions are thought to lack ETI, whereas incompatible interactions are cultivar-specific and determined by ETI. Non-host interactions refer to those between a plant species and non-adapted pathogen (Thordal-Christensen, 2003). Non-adapted pathogens refer to those that have no ability to circumvent the PTI. The PTI and basal resistance have been used interchangeably (Boller and Felix, 2009). However, Zhang *et al.,* (2010) showed that ETI can also contribute to weak basal resistance. It has been established that PTI primarily acts on pre-penetration resistance to compatible P. syringae but largely ineffective once the bacteria has gained access to the apoplast (Zhang *et al.,* 2010). In non-host interaction, PTI acts on both pre-and post-penetration resistance (Li et al., 2005). Zhang et al. (2010) demonstrated how the PTI and ETI differentially contribute to basal resistance during compatible and non-host interactions. ETI and PTI act synergistically during non-host resistance, for instance, ETI contributes to basal resistance during both compatible and non-host Arabidopsis-P. syringae interactions, while, PTI appears to play a greater role in non-host resistance than basal resistance during compatible interactions. Basal resistance (minimum resistance) has been defined as PTI plus weak ETI minus ETS (Jones and Dangl 2006).

The innate immune response is also controlled by a family of peptide signalling molecules (AtPeps) and their plasma membrane receptor AtPepR1. The AtPeps can act as danger-associated molecular patterns (DAMPs) by giving signals during its interaction with AtPepR1 leading to expression of pathogen-defence genes in a Ca2+-dependent manner (Qi *et al.,* 2010).

Activation of immune responses

The activation of innate immune responses is controlled by plant innate immunity using mitogen-activated protein kinase (MAPK) cascades and hormones in the salicylic acid (SA) defence pathway (Loake and Grant, 2007). These defences include the deposition of lignin and callose in the cell wall, transcription of pathogenesis-related (PR) proteins, production of antimicrobial compounds and reactive oxygen species (ROS). The MAPK cascades are involved in both positive and negative regulation of the PTI pathway. It is also known that several MAP kinases including MPK3, MPK4, and MPK6 are activated in Arabidopsis within minutes after stimulation by PAMPs/MAMPs (Bittel and Robatzek, 2007).

The SA plant defence pathway is important in the establishment of local and systemic-acquired resistance (SAR) and response to challenge by a diverse range of biotrophic phytopathogens (Loake and Grant, 2007). The plant defence in nectrotrophic pathogens is controlled by the SA-antagonistic pathways referred to as jasmonic acid (JA) and/ or ethylene (ET)-signalling pathways (Glazebrook, 2005). Plant cells are capable of producing phosphatidic acid (PA) to interact with infection of the pathogen. However, some pathogens e.g. P. syringae pv. tomato is capable of manipulating host enzymes to prevent accumulation of PA. The loss of function of gene encoding α/β hydrolase called

SOBER1 (SUPPRESSOR OF AVRBST-ELICITED RESISTANCE 1) led to susceptibility of A. thaliana due to low accumulation of PA which is important for induction of HR to P. syringae pv. tomato AvrTSB in the host (Hok *et al.,* 2010).

Postulates describing how plants resist infection by pathogens

Plants resistance to infection (non-host resistance) by most pathogens is based on two postulates. 1) A pathogen's effectors could be ineffective on a potential new, but evolutionarily divergent, host, resulting in little or no suppression of PTI and a failure of pathogen growth. 2) One or more of the effectors complement of the would-be pathogen could be recognised by the NB-LRR repertoire of plants other than its co-adapted host, resulting in ETI (Jones and Dangl, 2006). The most characteristic feature of the defence is the development of the hypersensitive response (HR) that results in a layer of dead cells to restrict pathogen growth (Fig. 1). The HR is possibly a by-product of defence. It is a rapid and highly localized reaction near the site of recognition, which results in death of both the plant cell and the attacking pathogen, thus limiting pathogen spread (Hofius *et al.,* 2007). This dramatic response is generally induced by the gene-for-gene recognition of a pathogenic determinant by a host encoded resistance (R) protein (Cunha *et al.,* 2006). As previously described, the recognition of pathogen associated molecular patterns (PAMPs) also contributes to resistance activation (Cunha *et al.,* 2006).

Conclusions

There are a lot of reports on how plant pathogenic bacteria and plant innate immunity interplay (Ausubel, 2005; Jones and Dangl, 2006; Cunha *et al.,* 2006; Hofius *et al.,* 2007; Boller and Felix, 2009; Zhang et al. 2010). Such reports indicate that bacteria use T3SS to secrete and deliver T3Es into the host cells. These T3Es together with hormones and toxins are used by plant pathogenic bacteria to attack the host. Although there has been much progress in understanding how these (T3Es, hormones and toxins) molecules function individually, their collective function remains unknown. Therefore future studies should be targeted into understanding how these molecules collectively function to the advantage of the pathogen. Progress has also been made in understanding how plants resist pathogen attack by using a two branched innate immune system. We now know that both PTI and ETI contribute to basal resistance (minimum resistance). The relationship between PTI and ETS has been summarised by both Jones and Dangl (2006) and Zhang et al. (2010) who defined basal defence as PTI plus weak ETI minus ETS. This area requires further studies in order to understand better the interaction between plants and phyto-pathogenic bacteria.

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Figure 1. A model illustrates how bacterium infects its host cell. In this scheme, a bacterium employs a type III secretion system (T3SS) to inject type three effector proteins (T3Es, red circles) into the plant cell in stage A. The T3Es can activate R genes of the resistant host plant (Path II-B). The activated R genes recognise the bacterium leading to plant defense response (blue arrows). In susceptible plants (Path I A), recognition of T3Es is not possible (red arrows) thus the bacterium acquires nutrient, multiplies and causes disease.