Plant Innate Immunity

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The ability to discriminate between self- and nonself-molecules is characteristic of all living organisms. This feature forms the basis for the activation of innate immune responses upon microbial attack. If microbes bypass the external physical barrier, plants have evolved two classes of immune receptors to detect nonself-molecules to prevent further pathogen progress. One class consists of membrane-resident pattern-recognition receptors that sense molecules from microbes, the so-called microbe-associated molecular patterns (MAMPs). The second class consists of plant resistance (R) proteins that have capacity to detect directly or indirectly isolate-specific pathogen effectors encoded by avirulence genes. These receptors are mainly intracellular. An alternative route is effector molecules that act as transcription factors. Signal transduction cascades link recognition and defence responses through second messengers, transcription factors and crosstalk between plant hormones to fine-tune the overall response.

Advanced article

Article Contents

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Introduction

Innate immunity is an ancient trait where various recognition systems distinguish between 'self' and 'nonself'. This distinction provides the fundamental basis for altruistic behaviour in 'social' microbes and ultimately how multicellular organisms could arise. Like all living systems, plants need to protect themselves from parasites. Large, multicellular organisms with a comparably slow generation time must race against small and rapidly evolving parasites. Despite this, plants and other multicellular organisms are not fighting a losing battle and infectious disease is more an exception than a rule. In fact, the immune response itself is a double-edged sword and must be tightly controlled in order not to cause disease itself or otherwise be detrimental for the organism. **See also**: Eukaryotes and Multicells: Origin; Innate Immune Mechanisms: Nonself Recognition

It is also important for the plant, just like for any other organism, to distinguish between parasites and other microorganisms that are commensalistic. Sometimes the discrimination between a pathogen and a commensalistic microorganism can be very indistinct and both often acquire nutrients from the plant in a very similar manner (Holub, 2006; Soto *et al*., 2006). To make this distinction, specific recognition events must occur which determines the host response. Furthermore, different pathogens are acquiring their nutrients from the host in different manners and a defence strategy that may be efficient against one pathogen could actually benefit another (Spoel *et al*., 2007), which implies that the plant also needs to monitor the type of attack and determine its defence response accordingly.

Plants, unlike mammals, lack mobile defender cells and a somatic adaptive immune system. Instead, they rely on innate (nonadaptive) immunity of each cell and on systemic signals emanating from infection sites. The first barrier of defence against pathogens is by preventing access to the host through various physical barriers on the outer surfaces, mainly the plant cell wall. This is however not enough and specific detection systems have evolved to cope with rapidly evolving pathogens, which will be the main focus of this article. One solution of the problem is to evolve recognition of general 'patterns', which are indispensable for various types of pathogens. The challenge of pathogens is probably one reason why sexual reproduction has been maintained throughout evolution despite its fitness costs (Kover and Cacedo, 2001).

Plant Host–Pathogen Models

The majority of our current knowledge about plant innate immune receptors and signalling components originate from genetic analyses from the model organism *Arabidopsis thaliana*. In *Arabidopsis*, some of the most important model pathogens where most of the genetics and understanding of plant innate immunity has been delineated are *Pseudomonas syringae* (a bacterium), *Hyaloperonospera parasitica* (an oomycete) and *Alternaria brassicicola* (a fungus). Importantly, many of the models presented

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here are valid in *Arabidopsis* but there are observations that some signalling may occur differently in other plant species. Systems where additional important information are generated are the interactions between the leaf mould fungus *Cladosporium fulvum* on tomato, the rust fungus *Melampsora lini* on flax, the powdery mildew causing fungus *Blumeria graminis* f. sp. *hordei* on barley, the rice blast fungus *Magnaporthe grisea* and the potato late blight oomycete *Phytophthora infestans*. **See also**: *Arabidopsis thaliana* as an Experimental Organism

Entering Plant Cells

To invade a plant, phytopathogenic bacteria, fungi and oomycetes have evolved strategies to subvert host immunity and actively penetrate plant tissue. Wounded plants are sensitive since wounds constitute an easy entrance for many types of pathogens. Many bacteria such as *Ps. syringae* swim towards openings like stomata and hydathodes and enter the apoplastic space of plant tissue. From there, bacterial effectors can be injected into the cytoplasm via various secretion systems, like the type III secretion system in *Pseudomonas* and the type IV secretion system found in *Agrobacterium*. Several fungi have spores that upon germination on a host form an appressorium. This is a fungal structure that exerts high pressure on the plant cell walls allowing the fungal hyphae to enter and invaginate cell membranes. At this stage pathogens need either to bypass or suppress pathogen-associated molecular patterns (PAMP) triggered immune responses to proliferate and colonize the host tissue.

The plant endomembrane system exhibits a much higher degree of complexity than that of mammals or yeast (Jurgens, 2004). This is accompanied by an increased complexity of genes coding for proteins regulating vesicle trafficking. In plants *N*-ethylmaleimide-sensitive factor adaptor protein receptors or SNARE (soluble N-ethylmaleimide-sensitive factor attachment protein receptor)-mediated vesicle trafficking are of outmost importance in basal plant defence. Much of our current understanding of these events derives from studies of *Bl. graminis* f. sp. *hordei* interactions in barley and *Arabidopsis*. Recently, it was found that vesicle-associated membrane proteins bind to PEN1 (penetration 1) syntaxin but they can also operate redundantly in a default secretory pathway suggesting dual function (Kwon *et al*., 2008).

How Plants Identify Different Parasites through General Patterns

General elicitors, or microbe-associated molecular patterns (MAMPs), are conserved structures typical of classes of microbes that are sensed by a broad spectrum of host species. MAMPs are recognized by cognate pattern-recognition receptors (PRRs) that trigger immediate defence responses leading to basal or nonhost resistance (**Figure 1a**), or more commonly denoted PAMP-triggered immunity (PTI). To date, all known PRRs in plants are plasma membrane-resident proteins, allowing the perception of MAMPs to occur at the cell surface (He *et al*., 2007). MAMPs represent a broad category of compounds but typically all are essential for microbial life. These include chitin, ergosterol and xylanse from fungi and β-glucans from oomycetes. Similarly, lipopolysaccharides flagellin, cold shock protein and elongation factor Tu (EF-Tu) all from bacteria act as MAMPs in plants (Zipfel, 2008). Interestingly, MAMPs such as glucans, chitin, lipopolysaccharides and flagellin can act in both plants and animals, but the individual epitopes that are recognized differ. For example, the *Arabidopsis* flagellin insensitive 2 (FLS2) protein recognize a 22-amino acid motif in flagellin, whereas mammalian toll-like receptor 5 recognize an epitope formed by the *N*- and *C*-termini of the flagellin peptide chain (Zipfel, 2008). The FLS2, EFR (EF-Tu receptor) and LeEix1/2 receptors display extracellular leucine-rich repeat (LRR) domains for either direct or indirect recognition of the MAMPs. The chitin receptor LysM RLK1/CERK1, however, has a different extracellular domain (Miya *et al*., 2007; Wan *et al*., 2008). The *Arabidopsis* LysM chitin receptor also has an intracellular kinase domain analogously to that of EFR and FLS2 and signalling appear to converge partly with components downstream of those two receptors of bacterial MAMPs. This is in contrast to the rice LysM chitin receptor CEBiP that lack intracellular domains.

In addition, knowledge on gene silencing and thereby resistance mechanisms in plants derives from studies on degradation of viral ribonucleic acid (RNA) (Hamilton and Baulcombe, 1999) which contributed to the discovery of short RNA species. The research on small noncoding RNA has expanded enormously the last 10 years and we have learnt much of their important roles in gene expression not least in plant immunity including PAMP and PRR recognition affecting PTI, and *R* gene recognition that modify effector-triggered immunity (ETI) (Jin, 2008). Recently, evidence was also presented where bacteria-like viruses were shown to have evolved mechanisms to suppress transcriptional activation of some PAMP-responsive microRNA (miRNAs) to cause plant disease (Navarro *et al*., 2008).

Effector-Triggered Susceptibility and Effector-Triggered Immunity

Specialized pathogens are able to overcome basal (MAMP-triggered) host immunity by either circumventing the detection of PAMPs or interfering with PTI by delaying, suppressing or reprogramming host responses. The secretion of effector molecules promotes pathogen virulence and leads to susceptibility. Most effector molecules have been identified as products of an avirulence (*Avr*) gene

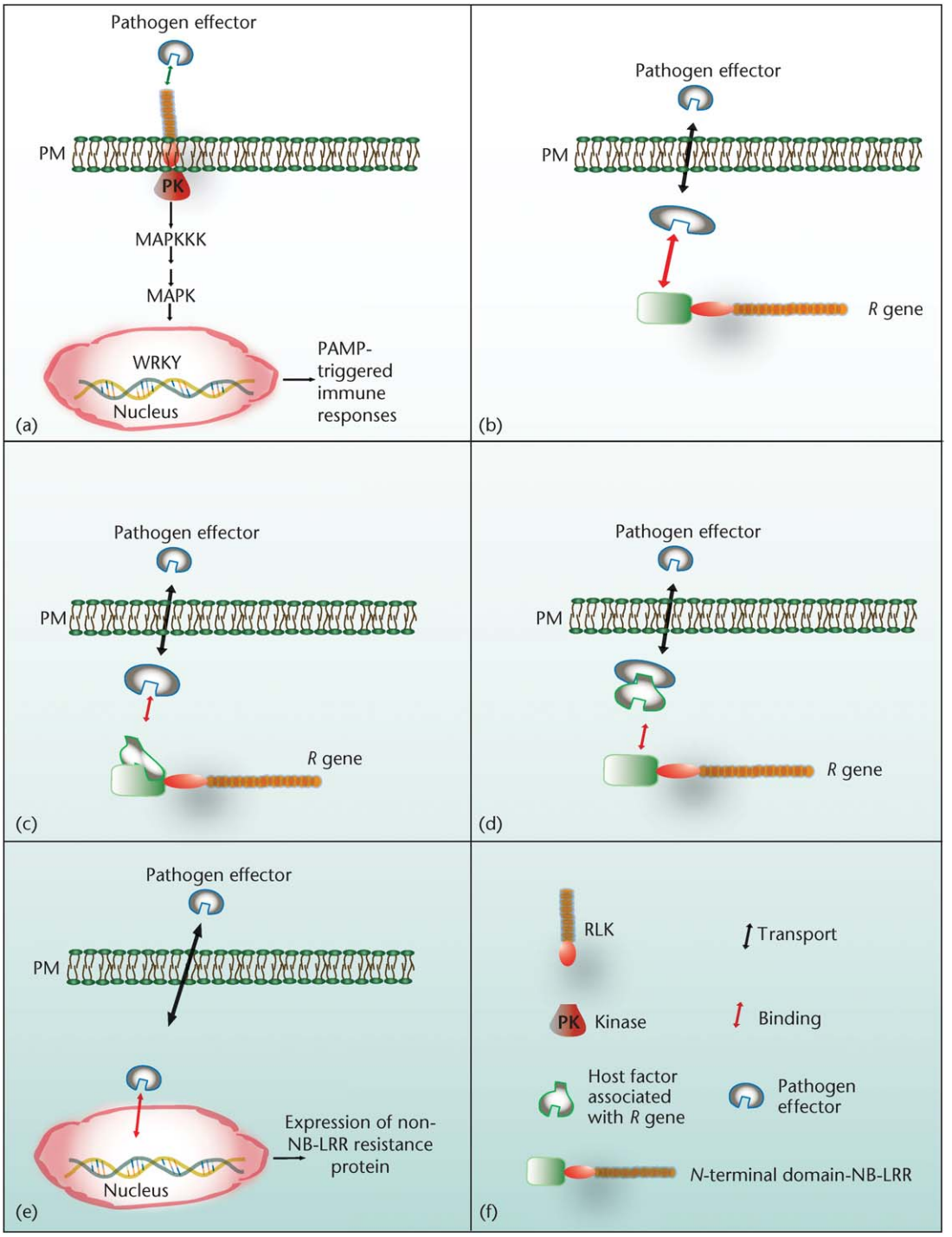

Figure 1 Different modes of pathogen recognition. (a) Recognition of microbe-associated molecular patterns (MAMPs) by extracellular receptor-like kinases (RLKs) triggers basal immunity mediated via MAP kinase signalling and activation of WRKY transcription factors. (b) Direct recognition can occur between pathogen effectors and an NB-LRR encoded *R* gene. (c) NB-LRRs can indirectly recognize pathogens through the *N*-terminal domain via a host protein (guardee), alternatively (d) the guardee component can first be associated with the pathogen effector and subsequently become recognized by the *N*-terminal of a NB-LRR encoding gene. (e) The most recently found interaction is based on recognition of a pathogen effector that mimics a transcription factor and binds directly to a non-NB-LRR R protein. (f) Explanation of the symbols used.

specific to a certain microbial strain that are recognized by a matching *R* gene. This gene-for-gene-mediated resistance is now referred to as ETI. The plant resistance in these interactions is phenotypically recognized as a local host cell death reaction, the hypersensitive response (HR). The genetic relationship between an *R* and *Avr* genes was determined already during the 1940s in the flax – *Me. lini* system (reviewed by Flor, 1971). The work on flax and flax rust formed the basis of the gene-for-gene hypothesis and mimics the ligand–receptor systems in animals (**Figure 1b**). Although, the gene-for-gene-type response often is very potent, it can also be broken and the durability of a single resistance trait is dependent on the population structure and reproduction strategy of the pathogen (McDonald and Linde, 2002). In essence, the problem of breaking *R* genes is an effect of the monoculture whereas the pluralism in a natural population is regarded as much more durable (Jones and Dangl, 2006). Based on accumulated data on resistance genes and pathogen effectors, an extended model termed the 'guard hypothesis' was suggested by van der Biezen and Jones, which described the indirect recognition between pathogen effectors and R proteins via the so-called guardee proteins (**Figure 1c** and **d**), where the guardee is thought to represent a protein targeted by the pathogen effector/Avr protein to suppress PTI (van der Biezen and Jones, 1998). Both hypotheses are now proved to be correct in different plant–pathogen systems showing that various strategies have been taken by plants to defend itself. **See also**: Resistance Genes (*R* Genes) in Plants

In an attempt to illustrate the co-evolution between plant defence responses and virulence factors by pathogens, where both partners evolve mechanisms to overcome each others defence or attack strategies over time, the zig-zag model was put forward (Jones and Dangl, 2006). In essence, the theory is based on how a plant first recognize PAMPs and PTI is triggered. Over time the pathogen adjusts and develops effectors, breaking the defence and causing susceptibility (effector-triggered susceptibility, ETS). Recognition of intruding effectors is the plant response in the next phase, resulting in ETI. New effectors and *R* gene alleles will evolve in a more or less constant battle and the outcome is a variation of the magnitude of the defence, an overall process resembling a zig-zag pattern.

New data on indirectly recognized effectors have however emerged that are inconsistent with the direct R–Avr protein binding and the guard model. For example, the AvrBs3 effector protein from *Xanthomonas campestris* is directly localized to the nucleus and binds to the promoter of the *Bs3* resistance gene (**Figure 1e**), which leads to *Bs3* transcript accumulation followed by HR induction (Römer *et al*., 2007). Based on this and additional findings, the decoy model has been proposed (van der Hoorn and Kamoun, 2008). This model takes into account the evolutionary aspects of opposing selection forces on guarded effector targets. Experimental evidence to distinguish between variants of the guard model and the new proposed decoy model are to be expected in near future.

Receptors Recognizing Pathogens, Blurring the Borderline between PTI and ETI

Receptors detecting microbial patterns can be divided into surface and intracellular receptors. A thorough description of PTI and ETI in different pathosystems can be found in Chisholm *et al*. (2006) and Jones and Dangl (2006). Since 1992, approximately 40 *R* genes have been cloned. These genes confer resistance to several classes of pathogens, including viruses, bacteria, fungi, oomycetes, insects and nematodes (Hammond-Kosack and Parker, 2003). Surprisingly, the protein products of these *R* genes are structurally similar to each other and contain a few, conserved domains. The LRR domain is the most common domain among R proteins, and it is also found in animal innate immunity molecules, including Toll from *Drosophila*, and TLRs and nucleotide-binding oligomerization domain proteins (NODs) from mammals (Nürnberger *et al*., 2004; Staal and Dixelius, 2007). Members of the largest class of R proteins comprise, in addition to the LRR, a central nucleotide-binding (NB) site domain that is similar to the NB of the NODs and the animal cell death effector proteins Apaf1 and CED4 (cell death 4), denoted NB-ARC (Apa1, R protein and Ced4 domain homology). The NB-LRR class of R proteins is further subdivided according to the *N*-terminal domain of these proteins. Some proteins contain a Toll–interleukin 1 receptor (TIR) homology region domain, whereas others possess a coiled-coil domain. Like the LRR and NB domains, the TIR domain is found in animal innate immunity proteins, specifically Toll, the TLRs and their adaptor proteins TRIF (Toll/IL-1 receptor (TIR)-domain-containing adaptor protein-inducing IFN-beta) and MyD88 (Pålsson-McDermott and O'Neill, 2007). The surface receptors mainly detect PAMPs and include receptor-like kinases (RLK), receptor-like proteins (RLP) and extracellular binding proteins. In fact the conceptually clear distinction between PAMPs and effectors can be indistinct. For example, the quorum-sensing signal protein AvrXa21, detected by the rice RLK Xa21, is an indispensable structure and at the same time a race-specific elicitor (Lee *et al*., 2006). RLKs reside in plasma membranes and are composed of a putative extracellular ligand-binding domain, a single transmembrane domain and an intracellular serine/threonine kinase domain. Thus, RLKs show to have structural similarities to animal receptor tyrosine kinases. In contrast, RLPs are composed of an extracellular domain and a membrane-spanning domain. Since RLPs lack intracellular activation domains, they require interaction with adaptor molecules for signal transduction. In the *Arabidopsis* genome, 610 RLKs and 57 RLPs are present, but only a limited number have been functionally characterized. A recent global phenotyping of mutants in all *Arabidopsis* RLPs revealed a role in development, hormone sensing and nonhost resistance towards a bacterial pathogen (Wang *et al*., 2008).

Stabilization, Signalling and Degradation of R Protein Complexes

RAR1 (required for Mla12 resistance) is an important component of *R* gene-mediated disease resistance, a protein which contains two zinc-binding finger motifs termed CHORD (cysteine- and histidine-rich domains)-I and CHORD-II (Shirasu *et al*., 1999). In some plant defence systems, it has been shown that RAR1 interacts directly with SGT1 (suppressor of the G2 allele of *skp1*) and the heat shock protein HSP90 (Takahashi *et al*., 2003). HSP90 is an abundant, highly conserved, adenosine triphosphate (ATP)-dependent molecular chaperone that is essential for eukaryotic cell viability. SGT1 is required for disease resistance mediated by diverse R proteins (Azevedo *et al*., 2002) and is also required for the function of an SCF (for Skp1/Cullin/F-box) protein complex. RAR1, SGT1 and HSP90 were shown to play an important role in regulating the stability of R proteins that contain the nucleotide binding site (NBS)-LRR domains (Azevedo *et al*., 2006). It has furthermore been suggested that SGT1 could be involved in R protein-mediated signalling by targeting negative regulators for degradation via the SCF complex.

The current understanding on R protein structures and function suggest that the LRR domain is under diversifying selection, and specifically the surface-exposed residues in the β-sheet are involved in ligand or guardee protein recognition (van Ooijen *et al*., 2007). In addition to a role in recognition specificity, the *N*-terminal (CC or TIR domains) is involved in downstream signalling. The central NB-ARC, however, is thought to act as a molecular switch that controls the activation state of the protein. It has been shown that the *N*-terminal part of LRR domains can physically interact with HSP90 whereas the *C*-terminal part can bind to protein phosphatase 5. However, despite having a conserved interaction pattern with NB-LRR proteins (together with HSP90, SGT1 and RAR1/CHP1 – CHORD protein 1), in both animals and plants, the function of protein phosphatase 5 is still elusive. Recent data suggest that additional components in these protein complexes display important roles in innate immunity. For example, it has been shown that RAR1, HSP90, HSP70 and Rac/Rop GTPase (guanosine triphosphatase) can form protein complexes of importance for immunity in rice (Thao *et al*., 2007).

Hypersensitive Response and Cell Death Signalling

To incite local cell death is a quick suicide response where the plant sacrifices a few cells to prevent further tissue colonization of an invading pathogen. This is a very efficient defence to biotrophic pathogens. In the onset of the cell death process several other events take place or are initiated. One of the most rapid responses in this context is the oxidative burst, which leads to the transient production of large amounts of reactive oxygen species, including superoxide, hydrogen peroxide and hydroxyl radicals (Ma and Berkowitz, 2007). Reactive oxygen species accumulate preceding cell death in HR. Neither salicylic acid (SA) nor ethylene (ET) alone can trigger HR, but both hormones are regarded as positive regulators of HR in contrast to jasmonic acid (JA) that display a negative role in plant cell death regulation. In addition, nitric oxide another potent molecule is required for signalling downstream of Ca^{2+} influx to result in HR (Zeidler *et al*., 2004). However, far from all HR components and their role in signalling cascades are elucidated today.

Defence Signal Transduction

Stimulation of immune receptors by their cognate ligands results in a chain of signal transduction events. This includes activation of a mitogen-activated protein kinase cascade, function of WRKY transcription factors, and an array of gene expression changes. Besides local immune responses, PTI and ETI activate long-distance defence reaction, such as systemic acquired resistance (SAR). In *Arabidopsis* and other higher plants, local and systemic defence responses are controlled by balanced action of distinct, but partially interconnected pathways involving SA, JA and ET. However, additional hormones such as abscisic acid, auxin, gibberellic acid can also play active roles in defence signalling (Robert-Seilaniantz *et al*., 2007).

Based on *Arabidopsis* host–pathogen models, two major response pathways have been identified. One depends on SA and is mainly effective against biotrophs. Some major characteristics of this response are the HR and upregulation of various pathogenesis-related (PR) proteins (van Loon *et al*., 2006; Sels *et al*., 2008). The second response is dependent on the JA and ET. This reaction is mainly effective against necrotrophic pathogens and the requirement of JA and ET can differ depending on the type of pathogen studied (Glazebrook, 2005). However exceptions from these two categories exist.

Genetic evidence for JA antagonism of SA signalling pathways is well documented, but emerging data suggest a more complex signalling network evoking both positive and negative regulatory interactions (**Figure 2**). The two responses are mutually antagonistic via the cytoplasmic nuclear protein, nonexpressor of pathogenesis-related 1 (NPR1) and various WRKY-family transcription factors (Li *et al*., 2006; Ndamukong *et al*., 2007), but can also show some synergism at low concentrations, possibly by upregulation of NPR1 by SA (Mur *et al*., 2006). Interestingly, recent dual infection models have shown that the defence response against a biotroph (*Ps. syringae*) only repressed the defence response against a necrotroph (*Al. brassicicola*) when the interaction was compatible, indicating an advanced risk-benefit evaluation by the plant immune system (Spoel *et al*., 2007). JA is mainly involved in wound, insect and necrotrophic defence responses besides a number of developmental processes. Recently, it was

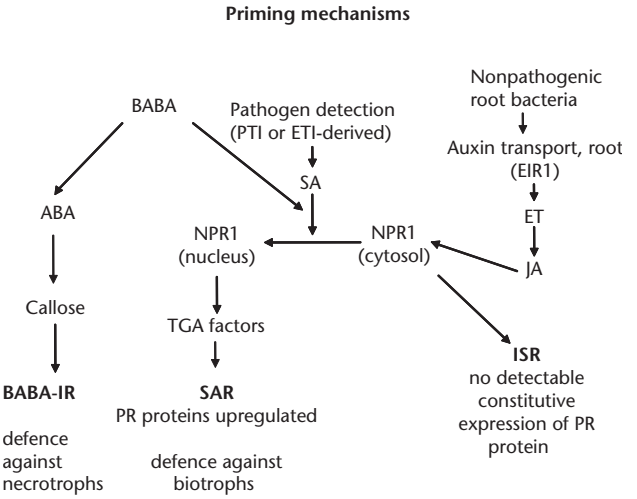

Figure 2 Priming is a nonacute plant pathogen response, which renders the plant more resistant to future attack. In ISR priming models, a role for NPR1 in JA responses has been established, whereas this role is not apparent during infection with necrotrophs – where the detrimental role of NPR1-mediated antagonism by SA is more important. The chemical agent β-aminobutyric acid (BABA) induces priming of callose deposition via interference of ABA signalling, resulting in BABA-induced resistance (BABA-IR). BABA-IR can act independently of SAR and ISR against some pathogens and via the SAR pathway against others.

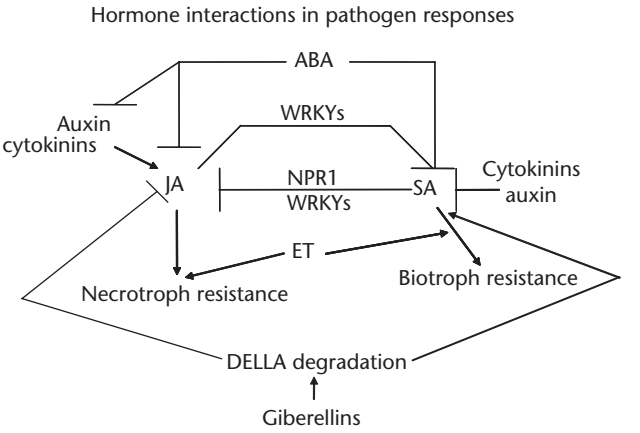

Figure 3 Through observations of disease outcome in various mutants, the mutual antagonism between defences against necrotrophs and biotrophs was observed. Most classical models have focused on the two main pathways with SA-dependent resistance against biotrophs and JA and/or ET-dependent responses versus necrotrophs. As more mutants in other hormone pathways have been studied, a complex web of interactions has become more and more apparent. Some pathogens where the three main plant biotic defence hormones SA, JA and ET play no or a minor role in resistance, mutants in other hormone pathways have rendered susceptibility – indicating that there are yet undiscovered pathogen response pathways that need to be delineated and characterized in relation to the more well-established models. Each interaction is also more complex than indicated by simplified model drawings, since the combination of signals can cause some subsets of the responses to be antagonistic whereas others are synergistic or unaffected. The models are thus not representative for every response gene, but rather for the final disease outcome in those plant–pathogen systems hitherto studied.

found that JAsmonate Zim-domain (JAZ) proteins act to repress transcription of jasmonate-responsive genes (Chini *et al*., 2007; Thines *et al*., 2007). Jasmonate treatment causes jasmonate zim protein degradation and this degradation is dependent on activities of the SCF^{COI1} ubiquitin ligase and the 26S proteosome. Furthermore, the jasmonoyl–isoleucine conjugate, but not other jasmonate-derivates promotes physical interaction between coronatine-insensitive protein 1 (COI1) and JAZ proteins in the absence of other plant proteins. One current hypothesis that remains to be shown is that SA antagonism of JA signalling could be achieved by blocking JA-mediated degradation of JAZ proteins. JA defence responses are generally monitored via expression of the storage protein VSP2 (vegetative storage protein 2) or the defensin PDF1.2 (plant defensin). The latter marker gene is shared with ET-induced responses and ET biosynthesis is activated in many plants challenged by pathogens. *PR* genes such as *PR-3*, *PR-4* and *PR-12* (*PDF1.2*) are induced synergistically via ET and JA signalling pathways.

Remembering Previous Attacks

A plant can 'remember' a former infection attempt and signal information to distal parts of the plant. This systemic response implies that a plant can stay in an activated defence state for a relatively long time. This memory, the SAR, is an SA-dependent resistance response. SAR is incited by avirulent pathogens that usually attacks leaves or stems of plants and is induced simultaneously with local primary and secondary immune responses inciting accumulation and the induction of a subset of *PR* genes. SA itself is not the mobile signal but methyl salicylate together with other candidate molecules seems to interact in signal perception and amplification (Vlot *et al*., 2008). The SAR signalling networks appear to share significant overlap with MAMP-induced basal defence. NPR1 is a central positive regulator of SAR signalling (Pieterse and van Loon, 2004). SA accumulation induces a change in cellular redox potential triggering the reduction of NPR1 from cytosolic, disulfide-bound oligomers to active monomers that translocate to the nucleus and interact with TGA transcription factors. These interactions stimulate the binding of TGA factors to SA-responsive elements in the promoters of *PR* genes, and the subsequent transcriptional reprogramming contributes to the establishment of SAR (Dong, 2004).

Nonpathogenic root microbes or rhizobacteria can induce another type of long-lasting activated state denoted induced systemic resistance (ISR), which depends on NPR1 and JA. This primed state can promote defence to a variety of fungal and bacterial pathogens, and has been proposed to be a more energy-efficient way to cope with the threat of infections rather than keeping the defence response fully active (van Hulten *et al*., 2006).

Lately, it has become apparent that many other plant hormones also influence the defence responses of pathogens (**Figure 3**). Alternatively those pathogens have learnt to

trigger selected plant hormones to facilitate infection. Abscisic acid, a hormone primarily associated to abiotic stress such as salt, cold and drought, has been shown to negatively influence resistance towards *Botrytis cinerea* in tomato and interfere with JA/ET signalling (Ghassemian *et al*., 2000; Mohr and Cahill, 2007). Abscisic acid can also be a crucial resistance component in some plant–pathogen interactions (Adie *et al*., 2007; Kaliff *et al*., 2007) or be linked to infection (de Torres-Zabala *et al*., 2007). Auxin physiology was changed in the absence of *R* genes in the *Ps. syringae* interaction, and upregulation of auxin signalling and the SA signalling pathway rendered the plants more susceptible to the *Ps. syringae* DC3000 strain (Zhang *et al*., 2007). Importantly in this context, the fact is that many pathogens can produce various hormones themselves. It has been proposed that endogenous pathogen-produced hormones are one strategy to disturb the hormone balance in their hosts, leading to suppression of defence responses.

Plant immune responses are also associated with the concerted modulation of a large number of WRKY transcripts and proteins. For example, upon triggering of SA-dependent defences, at least 49 AtWRKY genes exhibited differential regulation representing separate waves of transcript accumulation or repression (Dong *et al*., 2003). Several WRKY factors act as negative regulators of plant defence whereas others positively modulate this response implying their association with distinct regulatory complexes (Wang *et al*., 2006).

The understanding of the diversity and complexity of recognition mechanisms in plants has become rather extensive over the last years and additional new mechanisms and functions can be expected since a limited number of pathosystems have been explored to date. Superimposed, factors like plant growth hormones and modulation of developmental processes, not earlier recognized being of importance in a defence context, make the overall understanding of plant immunity exceedingly complex.

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