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Plant Immunity

Sonhita Chakraborty, Wolfgang Moeder, and Keiko Yoshioka, University of Toronto, Toronto, ON, Canada

Glossary

Biotrophic pathogen Plant pathogenic microorganisms that obtain nutrients from living host cells.

Elicitor Any molecules that are recognized by plant cell receptors and trigger a defence response.

Endophytes Microorganisms that live within their living host plant without causing apparent disease.

Epiphytes Microorganisms that grow on the surface of its living plant host.

Hemi-biotrophic pathogen Plant pathogenic microorganisms that switch from a biotrophic mode of infection to a necrotrophic mode.

Lesion mimic mutant Mutant plants that exhibit constitutive activation of immune responses, often including constitutive programmed cell death, also known as autoimmune mutants.

Necrotrophic pathogen Plant pathogenic microorganisms that kill host cells and extract nutrients from its host for sustenance.

1 Introduction

The ability of organisms to respond to the constantly changing environment in a timely manner is essential for their survival on earth. Over the course of millions of years, organisms have developed sophisticated defence mechanisms and the ability to distinguish non-self invaders and the self. In animals, this is accomplished through the innate immune system, and with specialized cells such as T-lymphocytes and B-lymphocytes produced by the acquired immune system (Janeway *et al.*, 2001). Furthermore, most animals can physically move out of harm's way. In contrast, plants are sessile organisms that have to be able to withstand both biotic and abiotic stresses without being able to move. Plants did not develop the corresponding mobile immune cells found in their animal counterparts. Instead, almost every plant cell is capable of defending itself without the aid of specialized cells (Nürnberger *et al.*, 2004). For this, they have developed different, but equally and uniquely sophisticated strategies to protect themselves from harm (Nürnberger *et al.*, 2004).

Like animals, a wide range of phylogenetically diverse organisms such as bacteria, viruses, fungi, oomycetes, and nematodes can infect plants. However, only a very small percentage of such organisms can cause disease (Agrios, 2005). This is mainly due to the elaborate defence system that plants have evolved over the course of time as part of the evolutionary arms race with pathogenic organisms. This article summarizes the various forms of plant immunity and the mechanisms to activate effective defence systems.

2 Pre-Existing (Also Called "Constitutive" or "Passive") Defences

Parallel to animals, plants possess two types of defence systems; pre-existing or passive defence and induced (or active) defence systems. Former includes structural barriers such as a rigid cell wall, a waxy layer at the cuticle, and trichomes (microscopic hair-like structure on leaf surfaces) similar to the animal skin layer. In addition, plants also possess a wide variety of preformed chemical compounds with anti-microbial properties (Agrios, 2005; Bednarek and Osbourn, 2009). Examples of such chemicals are phenolics, sulphur compounds, alkaloids, and saponins. Saponins are amphipathic glycosides and many saponins possess antifungal activity. The oat saponin, avenacin A-1, is produced in the roots of oats and accumulates in a circle around the periphery as an active form (**Fig. 1**). Avenacin A-1 confers resistance to the root-infecting fungus *Gaeumannomyces graminis* var. *tritici*, which causes the "take-all" disease of wheat and barley but is unable to infect oats. The oat-attacking variety of *G. graminis* var. *avanae* produces the saponin-detoxifying enzyme, avenacinase, indicating the important antifungal function of avenacin A-1 (Bednarek and Osbourn, 2009).

Some preformed antimicrobial secondary metabolites occur as inactive precursors as well. They will be converted to their active forms in response to pathogen infection; likely because they are also toxic for plant cells themselves. For instance, hydrogen cyanide, a toxic product from the breakdown of cyanogenic glycosides, is known to deter herbivores and fungal pathogens (Gleadow and Møller, 2014). Wounding can also induce the activation of various enzymes such as myrosinases, which hydrolyze endogenous glucosinolates to antimicrobial compounds by releasing glucose (Bones and Rossiter, 1996).

3 Inducible (Active) Defence Systems

The vast majority of microorganisms are unable to overcome the aforementioned pre-existing defence systems. However, a handful microorganisms have succeeded to acquire the ability to overcome such defence systems and became pathogenic to particular host plant species. To fight against such pathogenic organisms, plants rely on its robust inducible (active) defence systems. Unlike the pre-existing (therefore constitutive) defence systems, the inducible defence system is only activated upon recognition of invading pathogens, since induced defence is often harmful for themselves and an energy consuming process.

3.1 Examples of Induced Defence Response

3.1.1 Cell wall fortification

As mentioned, the plant cell wall is a first line of pre-existing defence. However, it is also actively reinforced through the deposition of papillae at sites of infection upon the recognition of invaders. Pathogens such as fungi produce penetration pegs in attempts to breech the plant cell wall. The formation of papillae structures just below the cell wall at the point of foreign penetration can prevent the ingress of the fungi (Heath, 2000). The deposition of proteins and other phenolic compounds at the cell wall accompany papillae formation and cell wall fortification. The deposition of callose, a plant polysaccharide, at the cell wall during the formation of papillae, is often accompanied by production of reactive oxygen species (ROS) such as hydrogen peroxide,

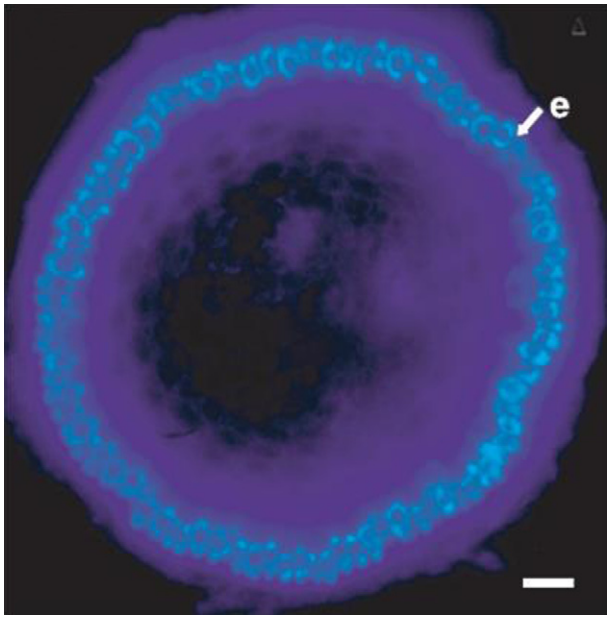

Fig. 1 Avenacin A-1 in oat roots Cross-section of an oat root showing auto-fluorescence of Avenacin A-1 under UV illumination.

superoxide and peroxides, which also contribute to cell wall strengthening (Heath, 1998). Such massive production of ROS upon pathogen infection is called oxidative burst (OB, described later in detail).

3.1.2 Phytoalexins

A pathogen attack can also induce the *de novo* synthesis of small antimicrobial compounds called phytoalexins and they accumulate rapidly at the infection site (Smith, 1996). They are chemically diverse and are host specific, not pathogen specific. Phytoalexins include alkaloids, glycosteroids, and terpenoids (Ahuja *et al.*, 2012). For example, camalexin, a simple indole alkaloid produced by the model plant, *Arabidopsis thaliana*, is induced by both biotic and abiotic stress (Ahuja *et al.*, 2012). Many crop species produce a wide variety of phytoalexins after elicitor treatment (see Glossary) or exposure to pathogens, such as resveratrol, pisatin, medicarpin, capsidiol, glyceollin (Agrios, 2005).

Interestingly, the consumption of many plant-derived phytoalexins provides beneficial effects to humans. Phytoalexins derived from commercially important crops like soybean (*Glycine max*), grapes (*Vitis vinifera*), sorghum (*Sorghum bicolor*) have been shown to have anti-cancer properties (Ng *et al.*, 2011; Smoliga *et al.*, 2011; Yang *et al.*, 2009).

3.1.3 Pathogenesis-related proteins

Pathogenesis-Related (PR) proteins are a category of proteins that accumulate at high levels in plants following a pathogen attack. Their initial discovery in tobacco (*Nicotiana tabacum*) following infection by tobacco mosaic virus (TMV) has prompted years of research and characterization of these proteins (van Loon and Pieterse, 2006). Various PR proteins have been shown to have antimicrobial properties. For instance, some PR proteins have been shown to have chitinase and glucanase activity (Kauffmann *et al.*, 1987; Legrand *et al.*, 1987). Chitins and glucans are well-known components of fugal and oomycete cell walls, respectively. The accumulation of PR proteins is also closely associated with the induction of the hypersensitive response (HR), accumulation of the plant hormone, salicylic acid (SA), and systemic acquired resistance (SAR), a form of long-lasting, broad-spectrum resistance (See below for HR, SA and SAR, Vlot *et al.*, 2009).

3.1.4 Hypersensitive response

Hypersensitive response (HR) is a form of programmed cell death (PCD) at the site of pathogen infection, which is thought to quarantine biotrophic pathogens (**Fig. 2**, see Glossary) at the site of pathogen entry and thus prevent spreading outward towards healthy tissue (Heath, 1998). However, the necrosis associated with HR is not effective to contain necrotrophic pathogens, thus this type of resistance is activated only against biotrophic or hemi-biotrophic pathogens (see Glossary).

HR shares many similarities with the apoptotic cell death (apoptosis) observed in animal cells (Lam *et al.*, 2001). DNA laddering is one of these common feature between HR and animal apoptosis and mitochondria are known to be an important regulator of PCD in both animals and plants (Lam *et al.*, 2001). The possible involvement of Cysteine-aspartic proteases (Caspase) type enzymes has been reported to be an important step in initiating HR similar to apoptosis (Lam and del Pozo, 2000). Caspases are protease enzymes that play crucial roles to induce PCD including apoptosis in animals. However, plant genomes do not possess *bona fide* caspase genes, instead they have metacaspase (MCPs) that likely play a similar role in plant PCD (Watanabe and

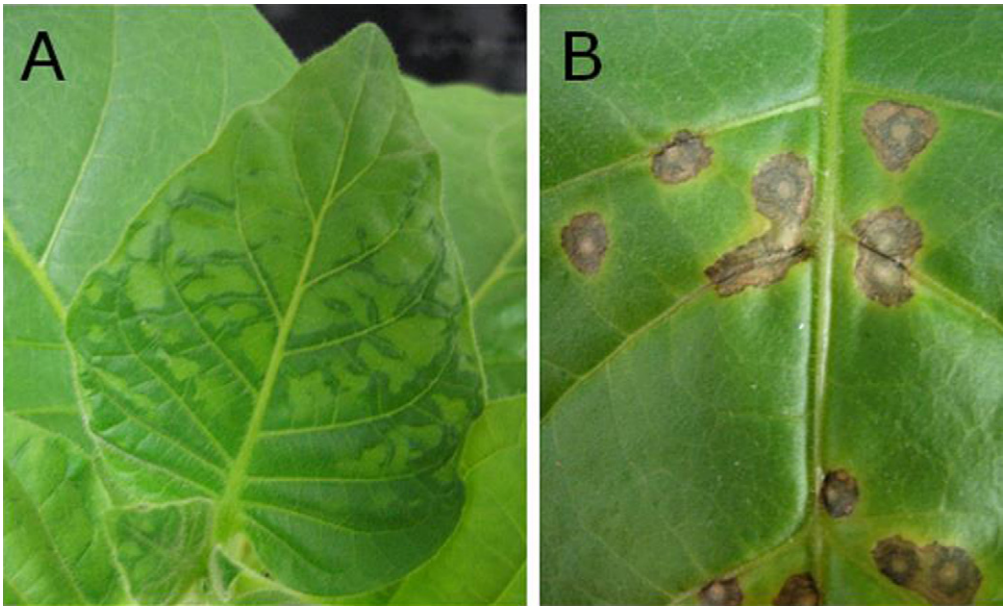

Fig. 2 Hypersensitive response (HR) in tobacco (A) Mosaic symptoms caused by tobacco mosaic virus on a susceptible tobacco leaf. (B) Hypersensitive cell death by tobacco mosaic virus on a resistant tobacco leaf. Photo K.B.G. Scholthof.

Lam, 2011; Lam and Zhang, 2012). Other physiological processes associated with HR include accumulation of pathogenesis-related (PR) proteins and cell wall modifications (Heath, 1998).

3.1.5 Oxidative burst

The oxidative burst (OB) or respiratory burst was first reported as the rapid release of reactive oxygen species (ROS) such as hydrogen peroxide and superoxide from animal immune cells, for example, neutrophils and monocytes, to degrade internalized pathogens. A similar burst of ROS is also observed after pathogen recognition in plant cells (Lamb and Dixon, 2003; Nürnberger *et al.*, 2004). The biological functions of the OB in plants are threefold: 1. direct antimicrobial effect, 2. aforementioned promotion of cell wall fortification and 3. downstream signaling molecules (Lamb and Dixon, 2003). OB in plants is believed to be mostly mediated by a family of plasma membrane localized NADPH oxidases, called respiratory burst oxidase homologs (RboH) (Torres *et al.*, 1998, 2002). RboH proteins have similar domain organization to mammalian NADPH oxidases (i.e., NOXs) that are involved in animal OB, indicating their parallel evolution. Knockout *A. thaliana* mutants of some RboH isoforms have been reported to exhibit altered ROS accumulation and subsequent PCD upon pathogen infection, providing evidence of involvement of OB in proper immune activation in plants (Torres *et al.*, 2002).

3.2 Local and Systemic Acquired Resistance (LAR and SAR) – Memory of Attack

In animal systems, adaptive immunity generates an immunological "memory" after an initial defence response. This response is very specific and leads to enhanced immune responses to subsequent invasion by the same pathogen. This phenomenon is the basis of modern-day vaccination. On the contrary, plants do not possess the same system, but they have evolved a conceptually similar immunological memory, called Local Acquired Resistance (LAR) and Systemic Acquired Resistance (SAR). In both cases, plants can activate induced resistance more quickly and strongly than naïve plants, since the memory of attack can "prime" plants to respond more quickly to a subsequent pathogen attack (Vlot *et al.*, 2009). Such priming can be observed not only in the local leaves (LAR) that have encountered the inducing microorganism prior, but also in systemic leaves that have never encountered to pathogens (SAR), indicating the existence of systemic signaling molecules that can induce priming in naïve leaves. This enhancement of immunity is manifested by a faster and stronger induction of defence responses (Fu and Dong, 2013).

Besides pathogens, various chemicals that do not possess antimicrobial properties can mimic pathogen infection and induce LAR and SAR (van Loon *et al.*, 1998; Kessmann *et al.*, 1994). Such chemicals are called SAR activators and were proposed to be used as environmentally safer alternatives of agrochemicals to control plant diseases (Vlot *et al.*, 2009). In fact some chemicals are commercially available as disease control agents (e.g., Actigard, Oryzaemate). Although LAR and SAR resemble the adaptive immunity of animals they, in fact, provide broad-spectrum resistance against taxonomically diverse pathogens. This suggests that acquired resistance in plants is only conceptually similar to vaccination in animals; the molecular mechanisms behind these phenomena are completely different.

3.3 Transgenerational Memory

Recently it has been suggested that plants may have transgenerational memory of pathogen attack (Alvarez, *et al.*, 2010; Luna *et al.*, 2012). This may be due to epigenetic changes related to the methylation and acetylation status of DNA and histones (Alvarez, *et al.*, 2010; Jaskiewicz *et al.*, 2011). Increased somatic homologous recombination events have been associated with the activation of immune responses. In addition, in the naïve progeny of plants that were exposed to Microbial-Associated Molecular Patterns/Pathogen-Associated Molecular Patterns (see below 5.1) increased homologous recombination events in the absence of pathogens were observed Molinier *et al.* (2006). Genome-wide high-resolution mapping of DNA methylation will shed further light on this phenomenon.

3.4 Induced Systemic Resistance (ISR)

Non-pathogenic bacteria that are closely associated to the root system can also prime plants against subsequent pathogen infection in systemic naïve tissues. This type of resistance is called Induced Systemic Resistance (ISR) (Pieterse and Van Loon, 1999). The rhizosphere, the narrow region of soil surrounding plant roots, contains an astonishing number and variety of microorganisms. Plant root secretions (called exudates) influence the composition of these microorganisms and some cases, they recruit beneficial microorganisms (endophytes or epiphytes) that significantly affects plant growth, development, nutrient and water acquisition, and tolerance to abiotic and biotic stresses such as drought, temperature, and pathogens (Pieterse *et al.*, 2014). Due to the diverse and dynamic roles of microbes, the rhizosphere microbiome is frequently compared to the animal gut microbiome, which can influence many physiological aspects in animals including immunity. In Arabidopsis ISR has been reported to be induced by the non-pathogenic bacteria *Pseudomonas florescences WSC417r* (Pieterse *et al.*, 1996). This form of priming has a different spectrum of effectiveness than SAR and is predominantly effective against necrotrophic pathogens (Ton *et al.*, 2002).

4 Hormonal Change

Not only proper growth, development, but also responses to environmental stressors rely on the induction of a plethora of plant hormones. The accumulation of the plant hormone salicylic acid (SA) is a hallmark of activation of pathogen resistance against certain types of pathogens (i.e., biotrophic pathogens) in plants (Vlot *et al.*, 2009).

SA is one of many phenolic compounds that are synthesized by plants. It regulates many aspects of plant physiology, such as growth, thermogenesis and disease resistance. Its role in disease resistance has been studied extensively after the discovery of SA being an endogenous signaling molecule to control disease resistance (Malamy *et al.*, 1990; Métraux *et al.*, 1990). It is of interest that SA is also used as medication for humans. Its acetylated derivative, known as aspirin, is one of the most widely used drugs for pain, fever, inflammation and heart attack (Klessig *et al.*, 2016). In plants, exogenous treatment with SA as well as aspirin can induce PR proteins and pathogen resistance as well as SAR (Dempsey and Klessig, 2017). As SA is predominantly required for resistance against biotrophic pathogens, the induction of SAR primarily provides resistance against biotrophic pathogens (Pieterse *et al.*, 2009). In *nahG* transgenic plants that cannot accumulate endogenous SA due to the transgenically express bacterial NahG gene, a SA hydrolyzing enzyme, no induction of PR gene expression and a deficiency in SAR is observed (Gaffney *et al.*, 1993).

While SA has been known to accumulate in distal tissues and as such, is essential for the induction of SAR in distal tissues, (Malamy *et al.* 1990; Gaffney *et al.*, 1993), SA is not the systemic signal for SAR (Vlot *et al.*, 2009). Instead, a number of other mobile compounds such as methyl salicylate, azelaic acid, glycerol-3-phosphate, and pipecolic acid, have been implicated to move through the phloem to distal tissues of the plant to prime them against a subsequent pathogen attack (Dempsey and Klessig, 2012; Shah and Zeier, 2013).

In addition, other plant hormones, especially jasmonic acid (JA) and ethylene (ET), are also essential for resistance against necrotrophic pathogens (Bari and Jones, 2009). The signal cascades of various phytohormones are now known to occur synergistically or antagonistically in accordance with the nature of the pathogen. As such, the signaling pathways for SA and JA-ET act antagonistically to fine tune immune responses to particular types of pathogens (Koornneef and Pieterse, 2008).

5 Recognition of Attack – Non-Self and Infected-Self

Since induced resistance often involves transcriptional reprogramming, it is an energy consuming activity for plants (Tian *et al.*, 2003). This can be seen as serious growth restrictions in autoimmunity mutants, such as lesion mimic mutants (LMMs, see section Glossary) that show HR cell death and elevated immunity-related gene expression constitutively without pathogen attack (Moeder and Yoshioka, 2008). Thus, generally speaking, the activation of resistance is strictly controlled and activated only when plants cells recognize an attack by a pathogen. Induced resistances are often categorized into two types of resistance, 1. Basal Immunity or PAMP (Pathogen Associated Molecular Patterns)-Triggered Immunity (PTI) and 2. *R*-gene mediated resistance or Effector-Triggered Immunity (ETI) (Jones and Dangl, 2006).

5.1 Basal Immunity or Pattern Triggered Immunity (PTI)

When a pathogen is not limited by passive or constitutive barriers in its host, the successful recognition of microbial elicitors in the form of evolutionarily conserved molecular patterns, called Microbial-Associated Molecular Patterns (MAMPs) or Pathogen-Associated Molecular Patterns (PAMPs) such as the bacterial flagella, lipopolysaccharide (LPS), and fungal chitin, will lead to the activation of defence. Such recognition is achieved by Pattern Recognition Receptors (PRRs) at the plant plasma membrane, which subsequently trigger the downstream signal transduction to activate the first line of defence, PAMP Triggered Immunity (PTI) (Jones and Dangl, 2006; Dodds and Rathjen, 2010). Many plant PRRs are receptor like kinase (RLK) proteins similar to animal receptor kinases that can activate immunity in animals (Shiu and Bleecker, 2001). One well-studied PRR is *FLAGELLIN INSENSITIVE 2* (FLS2). The FLS2 is a Leucin-rich-repeat (LRR) serine/threonine kinase that can recognize a conserved 22 amino acid sequence of the bacterial flagellin protein (Felix *et al.*, 1999; Gómez-Gómez *et al.*, 2001). Recognition of bacterial flagellin by FLS2 initiates immune signaling by association with BAK1, another RLK protein that interacts with FLS2 (Chinchilla *et al.*, 2007). Recently it has been revealed that upon recognition of flagelin, FLS2, BAK1 and BIK1, a receptor-like cytoplasmic kinase, are phosphorylated and BIK then phosphorylates the aforementioned NADPH oxidase, Rboh which initiates the OB (**Fig. 3** Macho and Zipfel, 2014).

Interestingly, similar to animal cells, infected, therefore, damaged-self is also recognized as a sign of pathogen invasion. Damage-Associated Molecular Patterns (DAMPs) such as cleaved or degraded endogenous plant cellular components released from dying cells such as oligogalacturonides from the cell wall, ATP, or High Mobility Group Box (HMGB, which is also an animal DAMP) can activate PTI or amplify the resistance responses (Dodds and Rathjen, 2010; Choi and Klessig, 2016). This form of basal resistance conferred by the recognition of PAMPs/MAMPs is also termed "non-host resistance" as it provides resistance against pathogens that have not adapted to its host plant (Dodds and Rathjen, 2010). Above mentioned cell wall fortification, OB, production of phytoalexins, induction of PR proteins can be seen upon recognition of pathogens by PRRs. However, various plant pathogens have developed "effector" proteins that can suppress PTI signal transduction leading to effector triggered susceptibility (ETS) (Bari and Jones, 2009). Therefore, these effector proteins are also known as "virulence factors".

5.2 Effector-Triggered Immunity (ETI)

As a part of the on-going evolutionary arms race between plants and disease causing pathogens, plants in turn have developed strategies to combat ETS. Plant *Resistance* (*R*) genes can recognize specific effector proteins released by pathogens to trigger a second line of defence called Effector-Triggered Immunity (ETI) (Jones and Dangl, 2006). This form of immunity in plants was first noted by

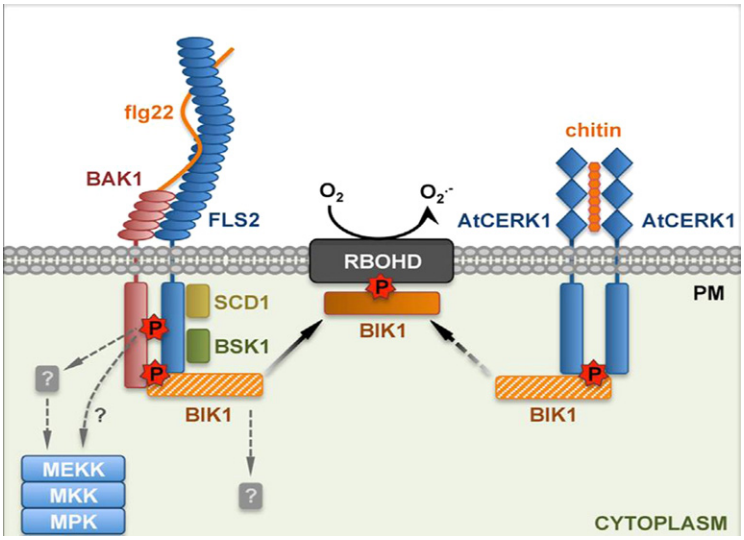

Fig. 3 Examples of Pattern Recognition Receptor (PRR) Complexes in Arabidopsis: Perception of the bacterial PAMP flg22 triggers the phosphorylation of the cytoplasmic domains of FLS2 and BAK1, as well as the RLCK, BIK1. Activated BIK1 gets released from the receptor complex, leading to phosphorylation and activation of the NADPH oxidase AtRBOHD. How PRR activation leads to the activation of MAPKs and other downstream substrates is currently unclear (Macho and Zipfel, 2014).

Flor (1942), who then coined the term "gene-for-gene" resistance and the researcher hypothesized the existence of genes that can work as receptors. Currently, many *R*-genes have been cloned and we now know that the majority of *R*-genes are categorised as nucleotide binding leucine rich repeat (NB-LRR) genes. ETI in plants is similar to how animals perceive the self from non-self. Humans and Drosophila also possess NB- and LRR-containing proteins that recognize PAMPs (Nürnberger *et al.*, 2004; Ausubel, 2005). Cytoplasmic receptor NB-LRR R-proteins often contain a Toll/interleukin receptor (TIR)-like domain or a coiled coil (CC) domain at the N-terminal region (Jones *et al.*, 2016). The TIR domain is a well-known signaling domain found in the interleukin (IL)-1 receptor, Toll receptor and MyD88 of animals that plays an important role in the mammalian immune system (Gazzinelli and Campos, 2004). Successful ETI (*R*-gene mediated resistance) is thought to arise from direct binding of R-proteins (receptors) with pathogen-derived effectors (ligand). However, it can also be achieved by the indirect interaction between R-proteins and plant proteins that are targeted by effector proteins. This concept has described as the "guard hypothesis" (Jones and Dangl, 2006).

5.3 Common Pathogen-Activated Signaling Events in PTI and ETI

Although elicited by different pathogen-associated signature molecules, PTI and ETI share many downstream signaling events, such as OB and PR protein accumulation that only vary in duration and intensity (Tsuda and Katagiri, 2010; Dodds and Rathjen, 2010). Though HR has been considered as a hallmark of ETI, recent evidence also suggests that HR may also occur upon PAMP-induced PTI (Mishina and Zeier, 2007; Naito *et al.*, 2007). Hence, it has been suggested that ETI might essentially be a more robust form of PTI (Thomma *et al.*, 2011). Both forms of immunity activate ion flux changes, changes in redox state including oxidative burst (OB), and activation of mitogen-activated protein kinase (MAPKs), and ultimately, changes in defence related gene expression (Dodds and Rathjen, 2010).

6 The Master Switch of Plant Immunity, NPR1

As mentioned, SA is an important signaling molecule to activate plant immunity including SAR. Several genetic screens for SA insensitive mutants independently identified the same gene, *NPR1* (*Nonexpresser of PR genes 1*, also called *NIM1*, *SAI1*), as a master regulator of the SA signaling pathway (Yan and Dong, 2014). Over the last two decades, NPR1 has been studied extensively and we now know that upon pathogen infection SA induces redox changes in the cell, which will convert NPR1 from its oligomeric form to monomers. The monomeric form then can translocate from the cytosol into the nucleus, where it acts as transcriptional co-activator for the TGA transcription factors to induce the expression of defence genes such as *PR1* (Yan and Dong, 2014).

NPR1 has been reported to be a key factor for ISR as well (Pieterse *et al.*, 2014). Interestingly, during ISR, an SA-independent but JA and ET-dependent process occurs that relies on NPR1 through mechanisms that have yet to be identified (Pieterse, 1998; Pieterse *et al.*, 2009).

Although SAR and ISR work through different signaling pathways, both forms confer resistance in un-inoculated systemic tissues following initial resistance induction at the site of inoculation/colonization. Furthermore, the concurrent activation of both forms of resistance can provide plants with even stronger defence activation (van Wees *et al.*, 2000).

7 Concluding Remarks

The goal of this article was to provide a brief overview of plant immunity. Both plants and animals possess immune systems that allow them to protect themselves from harm posed by invaders and pathogens. To reiterate, plants lack a circulatory system and specialized immune cells. Instead, every plant cell can defend itself. In contrast, a pathogen attack in animals leads to a series of events, including the mobilization of immune cells to unaffected tissues (Nürnberger *et al.*, 2004).

However, despite their differences, plant immunity shares many similarities with the animal immune system (Ausubel, 2005). For instance, both plants and animals have pre-formed structures (e.g.,rigid cell wall in plants, epithelial layer in animals) that act as the first barrier to potentially malicious pathogens. Additionally, the conserved nature of pattern-recognition receptor kinases (PRKs) and TIR receptors across both kingdoms suggests either evolutionarily conserved relationships or convergent evolution (Nürnberger *et al.*, 2004). Animals and plants also share downstream signaling events, including production of ROS, activation of MAPK cascades and ion flux changes to induce immunity (Nürnberger *et al.*, 2004).

Plants have been an essential source of food, medicine and commercially important goods for humans since the dawn of mankind. However, history is also full of cases of destructive famines and disease caused by pathogens that affect these plants. The devastating effects of Irish potato blight caused by *Phytophthora infestans* led to the loss of many lives in Ireland and the subsequent displacement of an entire diaspora of people to other parts of the world. Today, plant pathogens pose serious threats to global food security, as they can cause massive losses to crop yields (Strange and Scott, 2005). Increasing temperatures associated with climate change can negatively influence plant architecture and response to the biotic stresses as well (Patel and Franklin, 2009; Bebber *et al.*, 2013). The increasing resistance of pests to insecticides and pesticides also means that the heavy reliance on such potentially toxic chemical agents is not sustainable (Feyereisen, 1995). Understanding how plants defend themselves against pathogens is important to breed disease-resistant crops that pose a more sustainable alternative to the use of harmful agrochemicals.

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Further Reading

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