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REVIEW

The Genetic and Molecular Basis of Plant Resistance to Pathogens

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ABSTRACT

Plant pathogens have evolved numerous strategies to obtain nutritive materials from their host, and plants in turn have evolved the preformed physical and chemical barriers as well as sophisticated two-tiered immune system to combat pathogen attacks. Genetically, plant resistance to pathogens can be divided into qualitative and quantitative disease resistance, conditioned by major gene(s) and multiple genes with minor effects, respectively. Qualitative disease resistance has been mostly detected in plant defense against biotrophic pathogens, whereas quantitative disease resistance is involved in defense response to all plant pathogens, from biotrophs, hemibiotrophs to necrotrophs. Plant resistance is achieved through interception of pathogen-derived effectors and elicitation of defense response. In recent years, great progress has been made related to the molecular basis underlying host−pathogen interactions. In this review, we would like to provide an update on genetic and molecular aspects of plant resistance to pathogens.

KEYWORDS: Resistance to pathogen; Innate immune system; *R* genes

INTRODUCTION

Plants are in a continuous evolutionary battle against a multitude of microbial and other pathogens. Pathogens usually access the plant interior either by penetrating the leaf and root surfaces directly or by entering through wounds and natural openings such as leaf stomata. During the invasion process, plant pathogens degrade the cell wall by synthesizing and liberating cell wall-degrading enzymes, then deliver pathogen effectors by specialized infection structures, and eventually interfere with the normal activities of the host (Pajerowska-Mukhtar and Dong, 2009; Tilsner and Oparka, 2010; Horbach et al., 2011). Plants in turn have evolved sophisticated defense mechanisms to combat pathogen invasion by blocking pathogen entrance and activating a range of defense responses. The first barriers that pathogens face are the waxy cuticular layers and cell wall as well as preformed antimicrobial compounds. Once pathogens penetrate the cell wall, the plant two-tiered innate immune system is activated to counter-attack pathogen invasion. The first tier of plant immune system is PAMP-triggered immunity (PTI), which is based on the sensitive perception of pathogen- or microbe-associated molecular patterns (PAMPs or MAMPs), and the second tier is effector-triggered immunity (ETI), which perceives effectors produced by pathogens that have evaded PTI (Jones and Dangl, 2006).

In this review, the genetic and molecular aspects of plant resistance will be discussed, including the genetic basis for plant resistance, signaling pathways, perception of pathogens and defense mechanisms of plants.

GENETIC BASIS OF PLANT DISEASE RESISTANCE

Qualitative and quantitative disease resistance

Plant disease resistance is generally divided into qualitative and quantitative resistance. The former is controlled by major

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R gene(s), and the latter is conditioned by multiple genes with minor effects (Poland et al., 2009).

R gene usually confers complete resistance to a specific pathogen or pathogen race and is easy to manipulate for basic research and crop improvement. Many *R* genes have been cloned, and the downstream responses triggered by *R* genes are becoming increasingly well understood. As the resistance mediated by major *R* genes can be rapidly overcome by new virulent pathogens, *R* genes represent a frustrating battle in disease control for plant breeders and farmers.

Quantitative disease resistance (QDR) is controlled by multiple genes, each contributing to partial resistance. QDR leads to lower selection pressure against pathogen variants, and those that overcome an individual quantitative resistance locus (QRL) have little advantage. Thus, quantitative disease resistance tends to be more durable than *R* gene-mediated resistance (Parlevliet, 2002). Many studies on quantitative disease resistance have indicated its importance in crop disease improvement, and variation in quantitative disease resistance can be exploited by traditional or marker-aided plant breeding methods, leading to a commercially acceptable level of disease control.

Major genes for disease resistance

In the last two decades, numerous *R* genes for qualitative resistance have been cloned from many plant species, and they can be generally divided into seven classes based on their amino acid motif organization and membrane-spanning domains (Gururani et al., 2012). The largest class consists of NBS-LRR genes, which contain a nucleotide-binding site (NBS) and a leucine-rich repeat (LRR) domain (Young, 2000). NBS-LRR genes in plants are typically divided into two groups according to their N-terminal domains: one is the TIR group genes that are composed of an N-terminal domain having homology to the intracellular domain of the *Drosophila* Toll and mammalian interleukin-1 receptors, and the other group of genes code for the CC (coiled-coil) domain (Gururani et al., 2012). There are about 200 genes that encode proteins with similarity to the nucleotide-binding site and other domains characteristic of plant resistance proteins in *Arabidopsis*, in which 149 are NBS-LRR proteins (Meyers et al., 2003). The second class is characterized by extra-cytoplasmic LRRs and a C-terminal membrane anchor, represented by the tomato *Cf* genes (Jones, 2001). The rice *Xa21* gene, which confers resistance to a bacterial pathogen, is characteristic for the third class of *R* genes, as it features both extracellular LRRs and a transmembrane protein kinase (Song et al., 1995). The *Arabidopsis* RPW8 protein is an example of the fourth class of resistance gene encoding proteins, which contains a membrane protein domain and a putative coiled-coil domain (CC) (Wang et al., 2009; Gururani et al., 2012). The fifth class of resistance gene encoding protein contains putative extracellular LRRs, a PEST (Pro-Glu-Ser-Thr) domain and short motifs that might target the protein for receptor-mediated endocytosis. This class is represented by the *Ve1* and *Ve2* genes in tomato (Kawchuk et al., 2001). The sixth class is represented by the *RRS1-R* gene in *Arabidopsis*, which has a C-terminal extension with a putative nuclear localization signal (NLS) and a WRKY domain (Deslandes et al., 2002). The seventh class encodes enzymatic proteins which contain neither a LRR nor NBS domain. For example, the maize *Hm1* gene that provides protection against corn leaf blight caused by the fungal pathogen *Cochliobolus carbonum* encodes an enzyme, HC toxin reductase, which detoxifies a specific cyclic tetrapeptide toxin produced by the fungus (Johal and Briggs, 1992).

In addition to the above mentioned classification for mostly dominant resistance genes, several recessive resistance genes have been identified in plants. For example, two recessive genes, *bs5* and *bs6*, which control resistance to all known races of *Xanthomonas euvesicatoria* in peppers, have been characterized (Vallejos et al., 2010). In rice, a novel recessive resistance gene, *xa34*, was mapped to a 204-kb interval and confers resistance against *Xanthomonas oryzae* pv. *oryzae* (Zhu et al., 2011). A question for recessive resistance genes is whether they are really resistance genes or are simply mutant forms of susceptibility alleles? If susceptibility is an active process in which a host gene is targeted by a pathogen protein to induce susceptibility, then the recessive resistance may be a passive process due to lack of susceptibility instead of activation of defense responses. Though investigations of recessive resistance genes are still in the early stages, the current available evidence favor the hypothesis that the recessive resistance is a passive process. For instance, the resistance mediated by a recessive rice *xa13* gene can be overcome by the disease susceptibility gene *Os11-N3* (Yang et al., 2010). Several evidence suggested that *xa13* has atypical *R* gene responses, which indicates that *Xa13* may act as susceptibility allele and *xa13* is the mutant form (Iyer-Pascuzzi and McCouch, 2007). The *pvr2* locus in pepper corresponds to an eukaryotic initiation factor 4E (*eIF4E*) gene, conferring recessive resistance against strains of potato virus Y (PVY) (Ruffel et al., 2002). The novel function for eIF4E supports virus (Potyvirus, pea seed-borne mosaic virus) movement from cell-to-cell, in addition to its probable support for viral RNA translation (Gao et al., 2004). The interaction between the Potyvirus genome-linked protein (VPg) and eIF4E is important for virus infectivity; and the recessive resistance results from incompatibility between the VPg and eIF4E in the resistant genotype (Ruffel et al., 2002).

Quantitative loci for disease resistance

Quantitative resistance loci (QRL) may be involved in a wide range of biological activities in plants, including 1) regulating morphological phenotypes and developmental process, 2) promoting basal defense, 3) encoding enzymes to detoxify pathogen-produced phytotoxins, 4) assisting with defense signal transduction, 5) circadian clock-associated genes, and 6) weaker alleles of *R* genes or a unique set of previously unidentified genes.

Flowering time is closely correlated with resistance to many pathogens, as susceptibility is enhanced after

flowering, indicating that QRL may be genes that regulate flowering time (Collins et al., 1999). PAMP-triggered immunity, a form of basal defense, confers resistance to broad-spectrum pathogens in plants. In this case, QRL represent mutants or different alleles of genes involved in basal defense (Dunning et al., 2007). The level of camalexin is correlated with quantitative disease resistance based on a biochemical study of the *Arabidopsis*−*Botrytis* pathosystem, and camalexin sensitivity of different pathogen isolates contributes to isolate specificity (Denby et al., 2004; Kliebenstein et al., 2005; Schlaeppi et al., 2010), indicating that QRL may be related to the production of anti-pathogen chemicals. Activation of the salicylic acid (SA)-dependent signaling pathway can lead to expression of certain pathogenesis-related proteins that contribute resistance to biotrophs. Likewise, activation of the jasmonic acid (JA)- and ethylene-dependent signaling pathways strengthens plant defense responses to necrotrophs (Thomma et al., 1998; Pieterse et al., 2009). Moreover, SA, JA, and ethylene signaling pathways interact extensively with one another (Koornneef and Pieterse, 2008; Robert-Seilaniantz et al., 2011). There are many examples of signaling components that regulate variable levels of susceptibility when mutated (Koornneef and Pieterse, 2008). Therefore, different alleles of the genes involved in the regulation of these signaling pathways are speculated to be QRL (Poland et al., 2009). Recently, some novel genes involved in *R* gene-mediated resistance against downy mildew were identified in *Arabidopsis* and they were controlled by the circadian regulator, CIRCADIAN CLOCK-ASSOCIATED 1 (CCA1). In addition, an interconnection between *R* gene-mediated programmed cell death (PCD) and basal resistance was also established, as an alteration in the former was also affecting the latter (Wang et al., 2011b). There are reports of quantitative disease resistance that is controlled by non-classical resistance genes. For instance, *pi34*, which shows partial resistance to rice blast, is located in a 65.3-kb interval, spanning 10 open reading frames and none of the candidate genes have sequence similarity to any previously reported defense genes (Zenbayashi-Sawata et al., 2007).

To date, only a few QRL conferring QDR have been cloned and characterized. In rice, some QRL represent genes that have not been previously reported to function in disease resistance, such as the *pi21* gene for rice blast, which encodes a mutated proline-rich protein and has no similarity to currently known defense-related genes (Fukuoka et al., 2009). In wheat, *Yr36* confers resistance to a broad spectrum of stripe rust races under high temperatures (25°C−35°C) in adult plants, and encodes both kinase and putative START (steroidogenic acute regulatory protein-related lipid transfer) lipid binding domains, which were essential to confer resistance (Fu et al., 2009). *Lr34* confers resistance to slow rust, leaf rust, and powdery mildew in wheat and has been used for over 50 years in breeding. The protein encoded by *Lr34* is a putative adenosine triphosphate-binding cassette (ABC) transporter of the pleiotropic drug resistance subfamily (Krattinger et al., 2009).

MECHANISMS UNDERLYING PLANT RESISTANCE TO PATHOGENS

Biotrophic and necrotrophic pathogens

Plant pathogens are broadly divided into biotrophs and necrotrophs, according to their lifestyles. Biotrophic pathogens gain nutrients from living host tissue, whereas necrotrophic pathogens kill host tissue and feed on the remains. There are, however, many hemi-biotrophic pathogens that behave as both biotrophs and necrotrophs, depending on the conditions or the stages of their life cycles. Many fungi that are commonly considered necrotrophs but have a biotrophic stage early in their infection process, and may thus be better described as hemibiotrophs (Pieterse et al., 2009). The molecular mechanisms underlying activation of plant defense responses are quite different between biotrophs and necrotrophs.

Plant resistance to biotrophs

Previous studies suggest that plant defense against biotrophic pathogens is largely due to gene-to-gene resistance (Glazebrook, 2005) (Fig. 1). *R* gene-mediated resistance usually results in hypersensitive response (HR), which is thought to be very important for plants to combat biotrophic pathogens, such as *Peronospora parasitica*, *Pseudomonas syringae*, and *Erysiphe* spp., by restricting their access to water and nutrients (Glazebrook et al., 1997; Aarts et al., 1998; Feys et al., 2001). *R* gene-mediated resistance also activates SA-dependent signaling, leading to an activation of a string of presumed defense effector genes. This activation of SA signaling occurs throughout the plant to develop systemic acquired resistance (SAR) against subsequent pathogen infections (Glazebrook, 2005). During SAR, deposition of callose and lignin occurs in the plant cell walls, and plants acquire the ability to mount a rapid HR. Analysis of *Arabidopsis* mutants with defects in various defense-related signaling pathways provides support for the idea that SA signaling can result in resistance to biotrophic pathogens. For instance, both *EDS1* and *PAD4* play important roles in SA signaling, and mutations in these two loci weaken resistance to some *P. parasitica* isolates. The *NPR1* (*Nonexpressor of pathogenesis-related genes 1*) gene is a master regulator for SA signaling. The *npr1* mutant is more susceptible to a variety of pathogens (Cao et al., 1994; Bi et al., 2011). Mutants with defective SA synthesis, including *eds5* (Nawrath and Metraux, 1999) and *sid2* (Nawrath and Metraux, 1999) show enhanced susceptibility to *P. syringae* as well. Two plant-specific DNA-binding proteins, SAR Deficient 1 (SARD1) and CBP60g, are key regulators for SA synthesis. Knocking out *SARD1* compromises basal resistance and SAR. In the *sard1-1/cbp60g-1* double mutant, pathogen-induced SA synthesis are blocked in both local and systemic leaves, resulting in compromised basal resistance and loss of SAR (Zhang et al., 2010c). In addition, the expression of CBP60g/SARD1-dependent resistance-related genes are modulated by

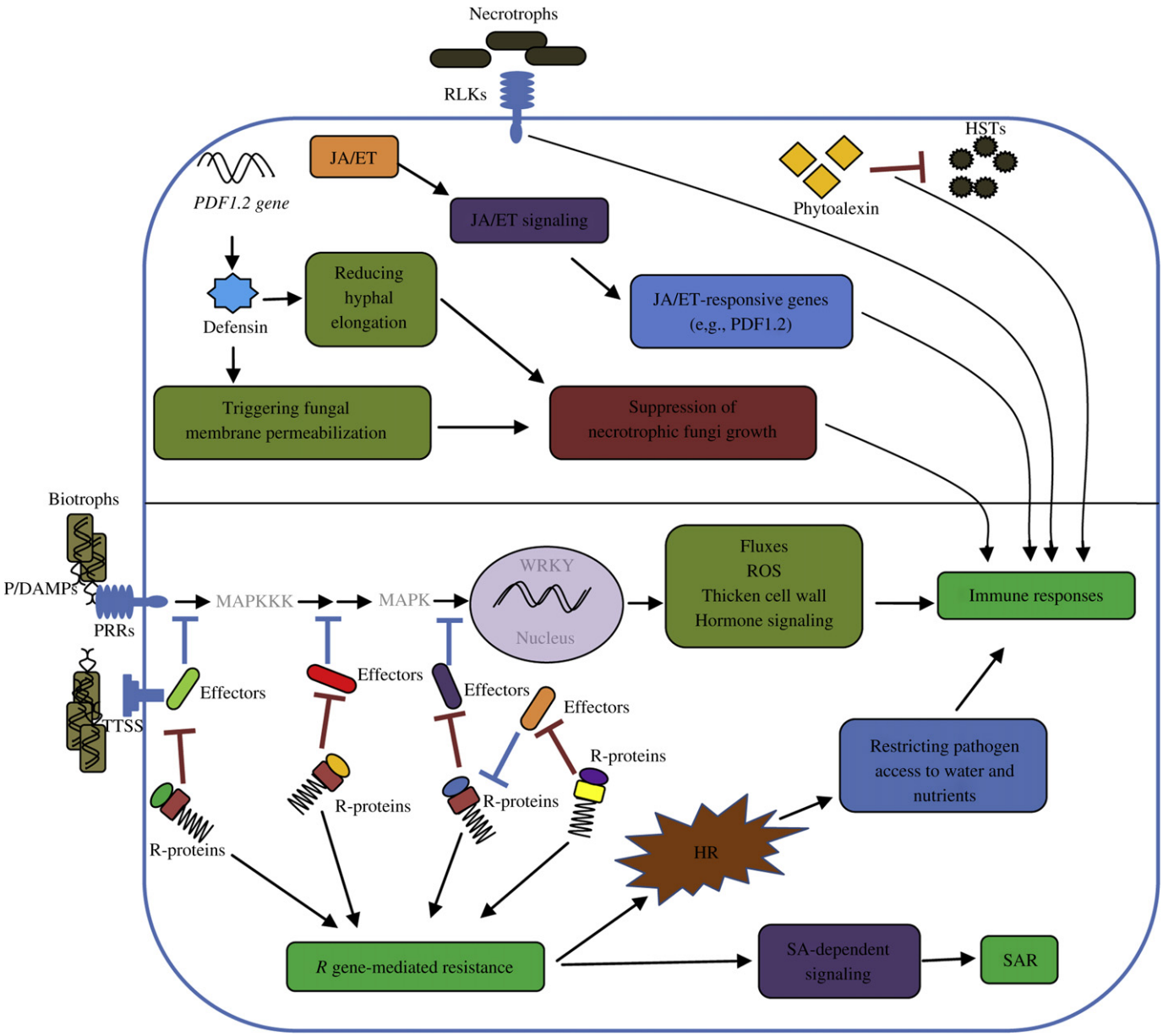

Fig. 1. **Molecular basis of plant resistance to pathogens.**
The upper part of the diagram is the defense response to necrotrophic pathogens, conferred by RLKs, defensin, phytoalexin, and JA/ET signaling. The lower part of the diagram is the two-tiered immune system of plant resistance to biotrophic pathogens. The first tier of defense (PTI) is triggered on perception of P/DAMPs by membrane-anchored PRRs, followed by activation of MAPK cascade and downstream transcription factors, leading to immune responses. The second tier of defense is elicited by pathogen effector *via* an interaction with R protein (ETI), in which the interaction between R protein and pathogen effector oscillates between compatible and incompatible reactions over time. The *R* gene-mediated resistance to biotrophic pathogens usually results in hypersensitive response (HR), and meanwhile activates SA-dependent signaling, leading to systemic acquired resistance (SAR). Abbreviations: HSTs, host-selective toxins; PRRs, pattern recognition receptors; TTSS, type III secretion system; ROS, reactive oxygen species; HR, hypersensitive response; P/DAMPs, pathogen/damage-associated molecular patterns.

CBP60g/SARD1, suggesting that CBP60g and SARD1 affect defense responses in addition to SA production (Wang et al., 2011a).

Plant resistance to necrotrophs

Generally it was assumed that no gene-for-gene resistance functions in resistance to necrotrophic pathogens, but it was indicated in a number of reports that several plant pattern recognition receptors (PRRs) are involved in the perception of necrotrophic fungi, such as receptor-like protein kinases (RLKs) (Llorente et al., 2005; Berrocal-Lobo and Molina, 2008) (Fig. 1). One of the putative RLKs is encoded by *BIK1* (*Botrytis-induced kinase 1*) gene, which is predicted to be involved in early stages of plant defense against *B. cinerea* and *A. brassicicola* (Veronese et al., 2006). Host-selective toxins (HSTs) are considered as efficient weaponry of necrotrophic fungi and the diseases caused by necrotrophs are

manifested by the appearance of necrotic lesions (Ciuffetti and Tuori, 1999; van Kan, 2006). For instance, tan necrosis caused by the fungus *Pyrenophora tritici-repentis* results from the action of the HST toxin, Ptr ToxA (Ciuffetti and Tuori, 1999). Exogenous application of the phytotoxin botrydial causes severe chlorosis and collapse in plant tissue to facilitate penetration and colonization of fungal *Botrytis cinerea* (Colmenares et al., 2002). Phytoalexins are low-molecular-weight antimicrobial compounds produced in response to pathogen infection or treatments with various abiotic elicitors. One of them, phytoalexin camalexin, is an indole derivative produced by *Arabidopsis* in response to infection by the bacterial pathogen *P. syringae* (Fig. 1). The *pad3* mutation, which inhibits camalexin production, has no negative effect on resistance to the biotrophic pathogen *P. syringae*, but does lead to decreased resistance to the necrotrophic fungal pathogen *A. brassicicola* (Thomma et al., 1999a; Zhou et al., 1999). This clearly demonstrates that camalexin plays an important role in *Arabidopsis* resistance to necrotrophs (Chassot et al., 2008). Camalexin was later shown to enhance the resistance against two other necrotrophic pathogens, *B. cinerea* and *L. maculans* (Bohman et al., 2004). In plants, a group of small cysteine-rich proteins, known as defensins, displays antimicrobial activities against micrographic fungi, which is considered an active participant in the plant innate immunity by triggering fungal membrane permeabilization and reducing hyphal elongation (Aerts et al., 2007) (Fig. 1). A pathogenesis-related gene *PDF1.2*, encoding defensin in *Arabidopsis*, was regulated by JA. It was reported that plants decreased in JA signaling exhibit a low level of *PDF1.2* expression during fungal infection and show enhanced susceptibility (Mengiste et al., 2003; Veronese et al., 2004).

JA- and ethylene-mediated defense responses are expected to play key roles in resistance to necrotrophic pathogens (Xie et al., 1998; Thomma et al., 1998; Thomma et al., 1999a) (Fig. 1). As known, JA activity in *Arabidopsis* requires the function of *COI1* (Xie et al., 1998; Pre et al., 2008). Plants with mutations in *coi1* show enhanced susceptibility to infection by the fungal pathogen *B. cinerea*, indicating that JA signaling is required for resistance to necrotrophs (Thomma et al., 1998). In *Arabidopsis*, the ethylene-insensitive *ein2-1* mutants are more susceptible than wild-type plants to infection by two different strains of *B. cinerea* (Thomma et al., 1999a). Inoculation of wild-type *Arabidopsis* plants with the fungus *Alternaria brassicicola* results in an activation of three resistance genes, and these genes fail to function in the *ein2-1* mutant (Thomma et al., 1999a). Furthermore, the *A. brassicicola* and *B. cinerea* necrotrophs are restricted by JA- or ethylene-dependent defense responses (Thomma et al., 1999b; Ferrari et al., 2003; Bohman et al., 2004). These findings support the idea that JA- and ethylene-controlled responses play vital roles in *Arabidopsis* resistance to necrotrophic pathogens. In addition, treatment of *pad3* plants with exogenous JA reduces the susceptibility to infection by *Botrytis cinerea* (Thomma et al., 1998), further supporting the idea that JA signaling is required for resistance.

HR is associated with increased resistance against biotrophs but decreased resistance to necrotrophs. HR does not protect *Arabidopsis* against infection by *B. cinerea*, which is the necrotrophic pathogen that attacks more than 200 plant species including many crops (Kliebenstein and Rowe, 2008). The high level of HR activated in biotroph-plant pathosystems may also provide an entry for necrotrophs in the local environment.

RNA silencing in plant resistance

A primary means by which plant defend against viral infection is RNA silencing (Dinesh-Kumar, 2009), which is triggered by double-stranded RNA. The RNA sequence that is homologous to the dsRNA is degraded and the gene that encodes the RNA is effectively silenced (Meister and Tuschl, 2004). It was reported that most plant viruses are RNA viruses. The RNA silencing process is composed of the dsRNA trigger, the processor Dicer or a Dicer-like (DCL) protein, small RNAs (siRNAs or miRNAs) of 21−24 nt in length and the effector complex RISC in which the Argonaute (AGO) protein is the key player. Viruses encode RNA-dependent RNA polymerases (RdRPs) and produce the opposite-sense of the viral genome in the first step of replication, thus generating many long dsRNA species that trigger RNA silencing (Waterhouse et al., 1998; Dalmay et al., 2000;). It was also suggested that viral RNA secondary structures might be the trigger of RNA silencing (Vance and Vaucheret, 2001). In plants, there are several homologues of the DICER endonuclease, and these DCL enzymes generate siRNA (short interfering RNA) in an antiviral response (Xie et al., 2004). Virus-induced gene silencing (VIGS) functions as a natural antiviral defense mechanism, in which host RNA silencing machinery targets and processes the virus derived dsRNA into vsiRNAs (virus-derived siRNAs) that are then recruited to host RISC complexes, which target and inhibit gene expression and protein translation in the viral genome. Viral effectors could suppress the host RNA silencing responses. Such suppressors have been identified in many plant viruses (Dinesh-Kumar, 2009). These studies indicate that the different suppressors interfere with the host-silencing machinery, and many viruses independently developed means to suppress RNA silencing.

A TWO-TIERED INNATE IMMUNE SYSTEM IN PLANTS

Plants lack mobile defender cells and a somatic adaptive immune system. Instead, they have evolved an innate immune system that efficiently detects potentially dangerous microbes and then counter-attacks their invasion. There are two tiers in the plant immune system. The first tier is based on the sensitive perception of PAMPs (or MAMPs) through PRRs on the cell surface of the plant. Immune responses to PAMPs are categorized as PTI. Successful pathogens have evolved to produce effectors to inhibit PTI, and plants can perceive such effectors through additional receptors (R proteins) to form the second tier of defense, ETI. There is a dynamic co-evolution

between plants and pathogens, and this dynamic process continues, as some pathogens have acquired effectors that interfere with ETI (Jones and Dangl, 2006).

PTI

As the first tier of the plant resistance system, PTI is triggered by PAMPs. Although numerous PAMPs have been described, only a few pattern recognition receptors (PRRs) have been identified so far (Zipfel, 2009) (Fig. 1). A typical example is the leucine-rich repeat-receptor-like protein kinase (LRR-RLK), named FLS2, which is located in the plasma membrane to bind a bacterial flagellin that contains a 22-amino acid epitope (flg22) (Gomez-Gomez and Boller, 2000). FLS2 initiates immune signaling by association with another leucine-rich repeat-receptor-like kinase, BAK1. A receptor-like cytoplasmic kinase BIK1 is rapidly phosphorylated upon flagellin perception by both FLS2 and BAK1. Phosphorylated BIK1 then transphosphorylates FLS2/BAK1 to propagate flagellin signaling (Lu et al., 2010). The mitogen activated protein kinases (MAPKs) are involved in various processes in plants, including plant immunity (Asai et al., 2002). A complete MAPK cascade and downstream transcription factors are then activated after flg22 detection, which activates the defense response (Nicaise et al., 2009). FLS2 was reported to activate two MAPK cascades. One consists of an MEKK-MKK4/5-MPK3/6 complex and acts positively on PTI, while the other consists of MEKK1-MKK1/2-MPK4 and acts negatively on PTI (Nicaise et al., 2009; Pandey and Somssich, 2009). During PTI, activation of the MAPK cascade leads to the activation of WRKY-type transcription factors and key regulators of plant immunity (Pandey and Somssich, 2009). Elongation factor Tu (EF-Tu) is one of the most abundant bacterial proteins and is recognized as a PAMP in *Arabidopsis* and other Brassicaceae (Kunze et al., 2004; Zipfel et al., 2006). The EF-Tu-derived peptide elf18, a highly conserved N-acetylated 18-amino acid peptide, is sufficient to trigger immune response. The plant PRR for EF-Tu is the LRR-RLK EF-Tu receptor (EFR), which belongs to the same subfamily (LRRXII) as FLS2 (Zipfel, 2009). FLS2 and EFR may oligomerize with BAK1, a general regulator of LRR-RLKs, and other SERK proteins in a ligand-dependent manner (Zipfel, 2009). The two receptor-like proteins LeEIX1 and LeEIX2, which contain a Leucine zipper, an extracellular Leu-rich repeat domain with glycosylation signals, a transmembrane domain, and a C-terminal domain with a mammalian docytosis signal, have been identified in tomato for perception of the ethylene-inducing xylanase, (Ron and Avni, 2004). Chitin is a major constituent of the cell wall of most fungi, and products degraded from chitin, *N*-acetylglucosamine and *N*-acetylchito-oligosaccharides, are potent PAMPs in several plant species (Kaku et al., 2006). The rice chitin-binding protein CEBiP is a transmembrane protein, and silencing of *CEBiP* in rice reduces chitin binding, suggesting that it constitutes the chitin PRR (Zipfel, 2009). In legumes, a soluble β glucan-binding protein can potentially release and then bind two ligands, 1,6-β-linked and 1,3-β-branched heptaglucoside, that are present in the cell wall of the oomycete *Phytophthora sojae* during contact (Fliegmann et al., 2004; Zipfel, 2009). However, the signaling pathway after perception is unclear. A semidominant *Arabidopsis* mutant, *snc2-1D*, constitutively activates defense responses. A suppressor screen of *snc2-1D* revealed that mutations in *WRKY70* suppress the constitutive defense responses in *snc2-1D*. Since WRKY70 may have a role in the regulation of conversion of SA to salicylic acid glucoside (SAG), this suggests that WRKY70 functions in an SA-independent pathway downstream of *snc2-1D* (Zhang et al., 2010d).

Other receptor-like proteins were identified in tomato and *Arabidopsis*, but their detailed interactions and signaling mechanisms are unknown (Ferrari et al., 2007; Schwessinger and Zipfel, 2008). In addition to PAMPs, plant cells can recognize molecules from damaged host cells upon microbial attack, which are referred to as damage-associated molecular patterns (DAMPs) (Lotze et al., 2007; Qutob et al., 2006a). For example, plants can recognize oligo-α-galacturonides released from damaged cell walls by fungal hydrolytic enzymes and Nep1-like (necrosis and ethylene-inducing peptide1-like) proteins secreted by many pathogens (Nurnberger et al., 2004).

Recognition of PAMPs is associated with a series of responses to prevent microbial growth (Nicaise et al., 2009). The first physiological response to PAMP recognition in plant cells is alkalinization of the growth medium (Garcia-Brugger et al., 2006). There are fluxes of H^+, K^+, Cl^-, and Ca^{2+} after PAMP treatment (Jabs et al., 1997). In addition, elevation of cytoplasmic Ca^{2+}, which is mediated by an increase in Ca^{2+} influx, is a critical step in plant innate immunity (Nicaise et al., 2009). Plant recognition of PAMPs induces rapid and transient production of reactive oxygen species (ROS) in an oxidative burst (Zhang et al., 2007), and the production of RbohD-dependent ROS appears to be downstream or independent of MAPK activation (Nicaise et al., 2009). The accumulation of callose, which is synthesized between the cell wall and the plasma membrane, as well as stomatal closure, are classic markers of PTI (Spoel and Dong, 2008; Bari and Jones, 2009) (Fig. 1). In addition, the SA, JA, and ethylene defense hormones are induced in PTI (Jones and Dangl, 2006).

ETI

During the development of ETI, plants evolved *R* genes to detect pathogen effectors and trigger defense responses (Fig. 1), and pathogens, in turn, have evolved new effectors to evade ETI. Such interactions between R proteins and effectors oscillate between compatible and incompatible reactions over time (Qutob et al., 2006b). Genes encoding pathogen effectors that induce *R* gene-mediated resistance are defined as *Avr* genes, which qualitatively reduce virulence but only when the host has the cognate *R* gene (Martin et al., 1993; Oh and Martin, 2011). Several plant bacterial pathogens contain members of the type III secretion system (TTSS) protein family, which has the ability to deliver bacterial virulence effectors directly into the host cells (He et al., 2004). Pto was

the first disease resistance gene cloned from plants, which encodes an intracellular Ser/Thr protein kinase that activates ETI in tomato (Oh and Martin, 2011). In concert with Prf, a NBS-LRR protein, Pto triggers a resistance response by interacting with either the AvrPto or AvrPtoB effector proteins delivered into the plant cell by *Pseudomonas syringae* pv. *tomato* (Oh and Martin, 2011). During the past 15 years, about 25 genes that play a role in Pto-mediated ETI have been identified by loss-of-function studies (Oh and Martin, 2011).

As mentioned above, plants can overcome pathogen suppression of PTI and re-establish ETI, but it is uncertain how they do this. During infection of *Arabidopsis* by *Pseudomonas syringae* pv *tomato* DC3000, the pathogen effector HopM1 destabilizes a host ADP ribosylation factor guanine nucleotide exchange factor, AtMIN7, through the host 26S proteasome (Nomura et al., 2006; Nomura et al., 2011). AtMIN7 is required not only for PTI but also for ETI, and the posttranscriptional AtMIN7 level increases in response to activation of PTI (Nomura et al., 2011). Blocking pathogen degradation of AtMIN7 is a critical part of the ETI mechanism to counter bacterial suppression of PTI. AvrPphB is a cysteine protease that cleaves the *Arabidopsis* receptor-like cytoplasmic kinase PBS1 to trigger cytoplasmic immune receptor RPS5-specified ETI. It was shown that AvrPphB can inhibit PTI by cleaving PBS1-like (PBL) kinases in plants lacking *RPS5*. AvrPphB-mediated degradation of PBS1 is monitored by RPS5, to initiate ETI, and AvrPphB targets other PBL kinases for PTI inhibition (Zhang et al., 2010a). SUMM2 is a nucleotide-binding leucine-rich repeat (NB-LRR) R protein, and SUMM2-mediated immunity is negatively regulated by the MEKK1-MKK1/MKK2-MPK4 cascade. Inhibition of MPK4 kinase activity by the *Pseudomonas syringae* pathogenic effector HopAI1 resulted in the activation of SUMM2-mediated defense responses (Zhang et al., 2012)

As an indicator of the evolutionary battle between plants and pathogens, ETI itself can be suppressed by other effectors. HopZ1a is a *P. syringae* pv. *syringae* type III effector, a member of the HopZ effector family of Cys-proteases that triggers immunity in *Arabidopsis*. HopZ1a-triggered immunity is independent of salicylic acid (SA), EDS1, jasmonic acid (JA), and ethylene-dependent pathways. Moreover, HopZ1a suppresses the induction of PR-1 and PR-5 associated with Pto-triggered ETI-like defenses, AvrRpt2-triggered immunity, and Pto activation of SAR, and that suppression requires HopZ1a Cys protease activity (Macho et al., 2010).

As a PRR, FLS2 not only recognizes a part of bacterial flagellin but also is physically associated with three R proteins, RPM1, RPS2, and RPS5 (Qi et al., 2011), indicating a possible association of PTI and ETI receptors in *Arabidopsis*. Moreover, PTI and ETI differentially contribute to basal resistance (Tsuda and Katagiri, 2010; Zhang et al., 2010b).

Comparing signaling pathways engaged in PTI and ETI

PTI and ETI are two separate tiers of the plant immune system, but, at least in some cases, PTI and ETI extensively share downstream signaling machinery. Transcriptional reprogramming, programmed cell death, and hormonal changes are triggered during both PTI and ETI as common plant immune responses. However, there are differences in how plants use these common signaling networks in PTI and ETI.

Analysis of the *Arabidopsis* transcriptome using a whole-genome DNA microarray revealed that exposure to a specific MAMP treatment induced a large transcriptional response (Navarro et al., 2004; Gust et al., 2007). In addition, the transcriptome responses triggered by various MAMPs are very similar in the early stages (Navarro et al., 2004), indicating that these PTI responses involve a common downstream signaling mechanism. Interestingly, the genes induced by flg22 and by effector recognition overlap significantly (Tsuda and Katagiri, 2010), suggesting that ETI may have adapted a part of its immune machinery from the pre-existing PTI, in addition to developing a new set of recognition molecules. HR is a form of rapid plant programmed cell death that may restrict pathogen growth, which is often associated with ETI. HR triggered by AvrRps4, which is derived from the bacterial pathogen *P. syringae*, is dependent on autophagy components, whereas AvrRpt2-triggered HR is not (Hofius et al., 2009), indicating that mechanisms leading to HR are different among different ETI triggers. Surprisingly, flagellin derived from *P. syringae* pv. *tabaci6605* induces cell death. Hence, plant cell death can be mediated by different signaling mechanisms and occur both in PTI and ETI (Tsuda and Katagiri, 2010). ROS may function as signaling molecules following pathogen recognition, and their generation is one of the earliest cellular responses (Torres et al., 2006; Tsuda and Katagiri, 2010). MAMP recognition triggers rapid ROS production, which is dependent on the NADPH oxidase AtRbohD (Torres et al., 2006), and recognition of a pathogen effector by an R protein also elicits ROS accumulation (Tsuda and Katagiri, 2010). Flg22 perception triggers the activation of the MAPK cascade, and MPK3 and MPK6 are also activated by *P. syringae* infection, and this latter activation is much more prolonged than MAMP treatment when *P. syringae* carries the effector AvrRpt2 (Tsuda and Katagiri, 2010). Hence, different durations of MAPK activity may be the marker to the differentiation of downstream responses between PTI and ETI (Katagiri, 2004; Liu and Zhang, 2004). In addition, SA, JA, and ethylene signaling can all be activated in PTI and ETI, but they lead to different outcomes (Tsuda et al., 2008; Grant and Jones, 2009; Halim et al., 2009; Pieterse et al., 2009; Robert-Seilaniantz et al., 2011). In other plants, hormones such as abscisic acid, gibberellins, and auxin also play roles in plant immunity (Robert-Seilaniantz et al., 2011).

CONTRASTING MODELS OF PATHOGEN RECOGNITION BY PLANTS

In the 1930s, Flor defined the basic elements of gene-for-gene complementarity in plant−pathogen interactions, in which single plant resistance genes and single complementary avirulence genes account for plant recognition of pathogens, which results in HR (reviewed by Flor, 1971). Functional

alleles are generally inherited as dominant characters. If either partner lacks a functional allele, recognition and resistance do not occur and the plant becomes infected (Keen, 1990).

The elicitor−suppressor and elicitor−receptor models

There are two main models proposed to explain the molecular basis of recognition and specificity in gene-for-gene systems. In the elicitor−suppressor model, pathogens are thought to provide general elicitors that initiate defense reactions in plants until a specific suppressor is produced by a particular pathogen race (Fig. 2A) (Bushnell and Rowell, 1981; Keen, 1990). The model assumes that many pathogen species have substances that elicit defense responses in plants, and the responses elicited are mostly the production of phytoalexins and the induction of hypersensitive cell death. Such defenses are assumed to be elicited nonspecifically by binding of the elicitor to a receptor in the non-host plant. The model further assumes that pathogens produce specific suppressors, which prevent the nonspecific elicitors from acting, and the plant becomes infected (Gabriel and Rolfe, 1990). In the elicitor−receptor model, either proteins from primary avirulence genes or metabolites resulting from elicitor-mediated catalytic activities are predicted to be recognized by specific plant receptors encoded by disease resistance genes, and these then trigger the resistance response (Fig. 2B) (Dangl and McDowell, 2006). Both models indicate the specific recognition of pathogen Avr proteins by plant R proteins, in which the former are the ligands for the latter (Mcdowell and Simon, 2006). Although these models provide a simple parallel to the immune system, they are not strongly supported by molecular evidence for direct interactions between R proteins and their cognate Avr proteins. To date, only three pairs of direct R-Avr interactions have been demonstrated (Jia et al., 2000; Deslandes et al., 2003; Dodds et al., 2006).

The guard model

There is increasing evidence for indirect interactions between pathogen effectors and R proteins, resulting in the proposal of the 'guard hypothesis' model (Fig. 3A). In this model, R proteins guard a limited number of host proteins that are targets of pathogen effectors during pathogenesis (van der Hoorn and Kamoun, 2008). The guard model suggests that multiple effectors can be perceived by a single R protein and that a relatively few *R* genes can target the broad spectrum of pathogens that attack plants. This model highlights that the guarded effector target (also called the guardee) is indispensable for the virulence function of the effector protein in the absence of the cognate R protein (Jones and Dangl, 2006). The guard model is supported by the findings of RIN4 and PBS1 in *Arabidopsis* and RCR3 and Pto in tomato (Jones and Dangl, 2006). In *Arabidopsis*, AvrRpm1 and AvrRpt2, two distinct effectors from *Pseudomonas* species, target the host protein RIN4, the status of which is closely monitored by the R proteins RPM and RPS2 (van der Hoorn and Kamoun, 2008). EDS1 (ENHANCED DISEASE SUSCEPTIBILITY1) behaves as an effector target and activated TIR-NB-LRR signal transducer for defenses across cell compartments (Heidrich et al., 2011).

The decoy model

Recently, many pathogen effectors were found to have multiple targets in the host and the guardee proteins are often dispensable for the virulence of effectors in the plants lacking the R protein (Zipfel and Rathjen, 2008). New data on additional targets of AvrPto and AvrBs3 promoted the concept that some host targets of effectors act as decoys to detect pathogen effectors *via* R proteins (van der Hoorn and Kamoun, 2008). van der Hoorn and Kamoun (2008) proposed the decoy model (Fig. 3B) to explain the recent knowledge of evolution in plant−pathogen interactions. This model is based on an

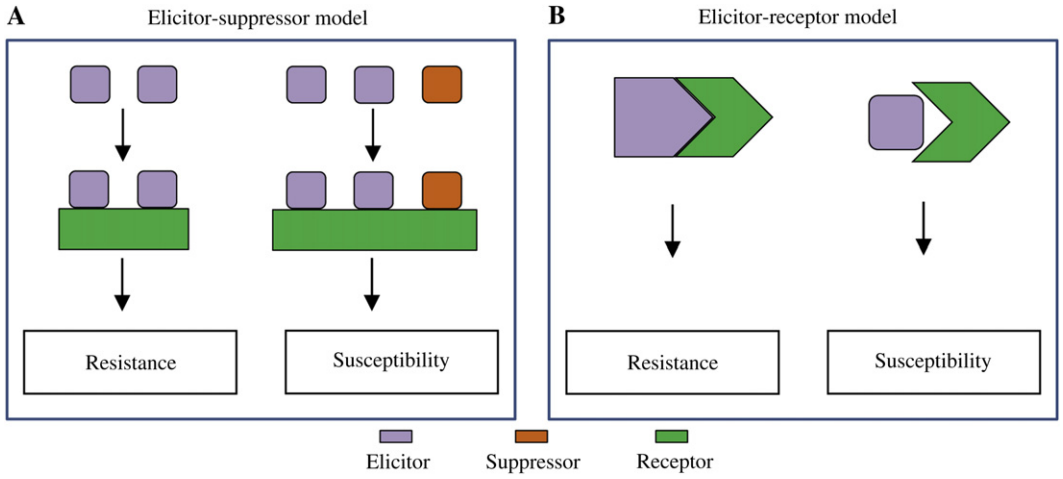

Fig. 2. **Comparison of the elicitor−suppressor model and the elicitor−receptor model.**
A: the elicitor−suppressor model. Elicitor initiates plant defense reaction (resistance) until appearance of a specific suppressor in a particular pathogen race, which leads to failure of defense reaction (susceptibility). **B:** the elicitor−receptor model. Protein encoded by avirulence gene is recognized by a specific plant receptors, which then triggers the resistance response. If the receptor does not fit the avirulence protein, this would inevitably lead to susceptibility.

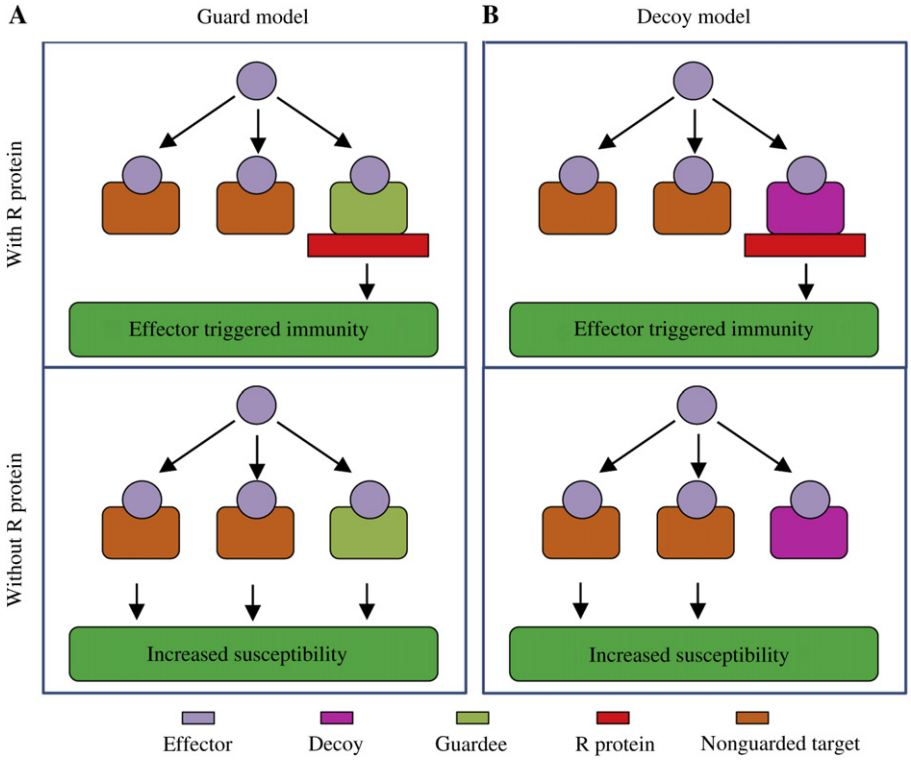

Fig. 3. **Comparison of the guard model and the decoy model.**
A: the guard model. Multiple effectors could be perceived by a single R protein, and a relatively small number of *R* genes could target the broad spectrum of pathogens that attack plants. **B:** the decoy model. The guardee is in an evolutionarily unstable situation named as 'decoy'. In the presence of functional *R* genes, natural selection is expected to favor guardees with improved interaction with an effector to enhance pathogen detection. In the absence of *R* genes, natural selection is expected to drive the guardee to decrease its binding affinity with the effector and evade detection and modification by the effector.

evolutionary point of view, whereby the guardee is in an evolutionarily unstable situation, as it is subjected to two opposing natural selection forces in plant populations where polymorphic *R* genes are either present or absent. In the presence of functional *R* genes, natural selection is expected to favor guardees with improved interaction with an effector to enhance pathogen perception. In the absence of *R* genes, natural selection is expected to drive the guardee to decrease its binding affinity with the effector, and evading detection and modification by the effector then results in a host 'decoy' protein to be relaxed selective constraint during evolution. This decoy specializes in effector perception by the R protein. The *P. syringae* effector AvrPto binds FLS2 to block plant immune responses in the plant cell and the ability to target FLS2 is required for the virulence function of AvrPto in plants. Pto competes with FLS2 for AvrPto binding, which in association with Prf, recognizes the bacterium and triggers strong resistance (Xiang et al., 2008). The decoy model is distinct from the guard model, which indicates that the manipulation of the guarded effector target benefits pathogen fitness in the absence of the R protein.

Although different models have been proposed, each one might apply to specific pathosystems to explain the complicated interactions between plants and pathogens. More experimental evidence is, however, needed to differentiate between these models, which may lead to novel approaches to manipulate plant innate immunity and improve pathogen resistance.

APPLICATION OF MAJOR *R* GENES AND QRLs

Utilization of major *R* genes

Although *R* genes have been extensively used in crop improvement, there is a high risk because of their potentially transient effectiveness and availability, as the cognate pathogen has a high potential for evolving new race specificity. One typical example is the outbreak of powdery mildew in wheat caused by *Blumeria graminis* (DC.) E.O. Speerf. sp. *tritici* (Bgt), which overcame several major resistance genes used in the Chinese wheat breeding program (Tao et al., 2000). Although more durable resistance may also be obtained by wide deployment of multiple *R* genes, approaches that yield long-term effectiveness and long-lasting specificity are needed in plant resistance breeding (St Clair, 2010).

Utilization of QRLs

Several QRLs have been discovered in numerous crop plants, but only a few have been used in breeding programs (Pumphrey et al., 2007). However, the practical use of QRLs indicates that quantitative disease resistance is an exciting field with the prospect of valuable applications in crop improvement as compared with qualitative resistance, because of its broad-spectrum and long-lasting resistance. Resistance genes with minor-to-intermediate additive effects can result in long-

lasting resistance to yellow (stripe) and leaf (brown) rusts caused by *Puccinia striiformis* and *Puccinia triticina*, respectively (Singh, 2005). *Fhb1* is a major QRL for resistance to *Fusarium* head blight (FHB) in wheat. A total of 19 QTL-NIL pairs were developed by using microsatellite markers flanking the QTL region, and each NIL pair was tested under point-inoculation in a greenhouse (Pumphrey et al., 2007). On average, NILs with *Fhb1* significantly ($P < 0.001$) reduced the disease severity rating by 23% and the percentage of infected kernels by 27% in harvested grain. Six QRLs (*Rphq1*, *Rphq2*, *Rphq3*, *Rphq4*, *Rphq5*, and *Rphq6*) resistance to leaf rust (causal agent *Puccinia hordei*) in barley were detected by 103 recombinant inbred lines (RILs) by single-seed descent from a cross between the susceptible parent L94 and the partially resistant parent Vada (Qi et al., 1998), and three of them (*Rphq2*, *Rphq3*, and *Rphq4*) were confirmed by using NILs created by marker-assisted backcrossing (MAB) (van Berloo et al., 2001). The QRL *Rphq2* was introgressed by MAB into the susceptible L94 background to obtain NIL L94-*Rphq2*. By contrast, NIL Vada-*rphq2* contained a susceptibility allele in the resistant Vada background. The latency period was prolonged by 28 h for L94-*Rphq2* and shortened by 23 h for Vada-*rphq2* (Marcel et al., 2007), and this delay is sufficient to help keep the damage below economic thresholds for pesticide treatment. In bean, common bacterial blight is caused by *Xanthomonas axonopodis* pv. *phaseoli*. Mutlu and colleagues developed the advanced backcross bean lines NE-01-8, NE-01-15, and NE-01-17, which possess the QTL on linkage group B8 derived from the highly resistant line XAN 159 as well as the QTL on linkage group B10 from the moderately resistant line 'chase' (Mutlu et al., 2005). A major resistance QTL, *qHSR1*, on bin 2.09 confers resistance to head smut, one of the most disastrous diseases in maize (Chen et al., 2008). The *qHSR1* was introduced *via* marker-assisted backcrossing into 10 maize inbred lines that have high yield potential but are susceptible to head smut. The resulting 10 converted inbred lines all showed enhanced resistance to head smut, but remained unchanged for other agronomic traits (Zhao et al., 2012).

PERSPECTIVES

Understanding the fundamental mechanisms underlying plant disease resistance is of vital importance to sustainable agriculture and human health. The past decades have witnessed surprising advances in the field of plant−microbe interactions, including experimental demonstrations of the functional role of PRRs in plant disease resistance and the discovery that many pathogen virulence factors are involved in suppressing PRR signaling and PTI-associated immune responses. However, the research on plant resistance is still limited because of reliance on information derived from a limited number of pathosystems, mainly on the interactions between *Arabidopsis* and bacteria. The incredibly diverse interactions between plants and microbes indicate the existence of many other novel mechanisms. For studying plant pathosystems, progress in genome sequencing and *R* gene isolation will be beneficial for the research on molecular plant−microbe interactions.

Over the past several years, detailed models for plant−pathogen interactions have emerged involving recognition, evasion, and defense. It does, however, appear likely that the molecular basis of plant resistance will draw upon an even broader mechanistic base. Aspects such as components of the signal transduction system, antimicrobial compounds such as phytoalexins, and other unknown factors are also likely to be important components of plant resistance responses that remain to be characterized. Cloning additional resistance genes and QTLs that underlie plant resistance will reveal how they contribute to plant defenses. This knowledge will enable more efficient and effective utilization of these genes in crop improvement and protection.

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