

Nonhost resistance: how much do we know?

Kirankumar S. Mysore and Choong-Min Ryu

Plant Biology Division, Samuel Roberts Noble Foundation, Ardmore, OK 73401, USA

Nonhost disease resistance is the most common form of disease resistance exhibited by plants against the majority of potentially pathogenic microorganisms. Recently, several components of nonhost disease resistance have been identified. Nonhost resistance exhibited against bacteria, fungi and oomycetes can be of two types. Type I nonhost resistance does not produce any visible symptoms whereas type II nonhost resistance results in a rapid hypersensitive response with cell death. Strong similarities exist between nonhost and gene-for-gene resistance responses but it is still not clear if the same mechanism is involved in producing these resistance responses.

Plants are continually exposed to a vast number of potential pathogens and, as a result, they have evolved intricate defense mechanisms to recognize and defend themselves against a wide array of these disease-causing agents by inducing a set of defense responses that can defeat the invading pathogens. These responses include a hypersensitive response (HR; rapid localized cell death at the site of infection), increased expression of defense-related genes [e.g. pathogenesis-related (*PR*) genes], and the oxidative burst [1–3]. Often, the plant disease resistance described is cultivar or accession specific and is referred to as host resistance. A second resistance, operating under less-understood mechanisms, provides resistance against pathogens throughout all members of a plant species. This type of resistance is referred to as nonhost resistance [1,4]. A pathogen that cannot cause disease on a nonhost plant is referred to as a nonhost pathogen.

Nonhost resistance, shown by an entire plant species to a specific parasite or pathogen, is the most common and durable form of plant resistance to disease-causing organisms [5]. In spite of tremendous progress in plant science, nonhost resistance is still poorly understood in contrast with host resistance. Host resistance is often governed by single resistance (*R*) genes, the products of which directly or indirectly interact with the specific elicitors produced by the avirulence (*avr*) genes of pathogens [6,7]. Considerable progress has been made in our understanding of gene-for-gene resistance (*R-avr* interactions [8]) and because of this, we now better understand how plants defend themselves against certain pathogens. In spite of this progress, it is still not clear why

a pathogen fully virulent on one plant species is nonpathogenic on others. Studying nonhost resistance is key to deciphering the complex plant defense mechanisms against pathogens. Recent developments in genomic technologies and sequencing of plant genomes have given us hope of dissecting the complex mechanisms of nonhost resistance. To better understand the complex phenomenon of nonhost resistance, it is important to answer the following questions. What are the components of nonhost resistance? Are there different types of nonhost resistance? Are there any similarities between nonhost and gene-for-gene resistance? This review answers these questions and summarizes the recent progress in the study of nonhost resistance.

Components of nonhost resistance known to date

Little progress has been made during the past several years to identify key components of nonhost disease resistance. This section describes some of the known plant cellular components that can contribute to nonhost disease resistance. Disruption of any of these components leads to loss of nonhost resistance against certain pathogens.

Preformed or passive defense mechanisms

Preformed defense is the first obstacle a pathogen faces before invading the plant. For example, the plant cytoskeleton provides a physical barrier against most invading plant pathogens. Plant actin microfilaments have been implicated in playing a role in defense against fungal penetration [9], and their disruption leads to the loss of nonhost resistance against several nonhost fungi. Treatment of several nonhost plants (barley, wheat, cucumber and tobacco) with cytochalasins, specific inhibitors of actin polymerization, allows several nonhost fungi (*Erisiphe pisi*, *Erisiphe graminis* f. sp. *hordei*, *Erisiphe graminis* f. sp. *tritici*, *Sphaerotheca fuliginea*, *Colletotrichum graminicola*, *Colletotrichum lagenarium*, *Mycosphaella pinodes*, *Alternaria kikuchiana* and *Corynespora melonis*) to penetrate the cells of these plants [10]. Furthermore, a combination of loss of actin cytoskeletal function and EDS1 activity severely compromises non-host resistance in *Arabidopsis* against wheat powdery mildew (*Blumeria graminis* f. sp. *tritici*) [11]. These data provide evidence that the plant cytoskeleton plays a significant role during nonhost disease resistance.

Plants constitutively produce a plethora of secondary metabolites, many of which can act as antimicrobial

Corresponding author: Kirankumar S. Mysore (ksmysore@noble.org).

compounds during defense against microorganisms. Saponins are glycosylated triterpenoid, steroid, or steroidal alkaloid molecules with antifungal activity [12]. Saponins are constitutively produced in many plants and can also be induced as a result of a pathogen infection. The root-infecting fungus *Gaeumannomyces graminis* var. *tritici* is a wheat pathogen and is unable to infect oats; it produces the root-specific avenacins, a class of triterpene saponin. Saponin-deficient (*sad*) mutants of a diploid oat species, *Avena strigosa*, lack or have only trace amounts of avenacin and are compromised for disease resistance against the nonhost fungal pathogens *G. graminis* var. *tritici* and *Fusarium culmorum* [13]. *sad* mutants do not show any alteration of leaf saponins and hence do not compromise nonhost resistance against the leaf-infecting fungus *Stagonospora nodurum*, a non-pathogen of oat [13]. Saponins are widely distributed in the plant kingdom; however, it is not clear how broad a role they play in nonhost resistance.

Inducible plant defense mechanisms

The second obstacle an invading pathogen has to face is the inducible plant defense mechanisms. Phytoalexins are low molecular weight antimicrobial compounds that are synthesized *de novo* in response to pathogen attacks. In spite of the large literature on the properties and induction mechanisms of phytoalexins, there are few examples of genetic proof for a role of phytoalexins in host or nonhost resistance. Several phytoalexin-deficient (*pad*) mutants have been identified in *Arabidopsis* [14]. Wild-type *Arabidopsis* plants are resistant to *Alternaria brassicicola* and produce a typical HR upon inoculation with the spores of this fungus [15]. One of the *Arabidopsis pad* mutants, *pad3-1*, is compromised for nonhost resistance against *Alternaria brassicicola* [16]. *PAD3* is required for the biosynthesis of camalexin (an indole phytoalexin) in *Arabidopsis* and encodes a putative cytochrome P450 monooxygenase [17]. It is not known whether *pad3* is also compromised in nonhost resistance against other non-pathogens of *Arabidopsis*.

Plant defense signaling

Several plant signaling components are involved during the induction of plant defense. An invading pathogen has to bypass many of these signaling components to cause disease successfully in plants. For example, the plant hormone ethylene is an important signaling component during plant–pathogen interactions. Ethylene perception is often required for basal resistance against pathogens and it can also induce disease resistance in plants. An ethylene-insensitive tobacco has been shown to lack nonhost resistance against several soil-borne fungi [18]. Transgenic tobacco plants expressing the *Arabidopsis etr1-1* gene (which causes loss of ethylene perception) were unable to support induction of basic *PR* genes upon tobacco mosaic virus (TMV) infection, and developed spontaneous stem necrosis during growth in soil. The stem necrosis was attributed to fungal infections by *Pythium sylvaticum*, *Pythium splendens*, other uncharacterized *Pythium* spp., *Rhizopus* spp. and *Chalara elegans* [18]. None of these soil-borne fungi infects

wild-type tobacco plants, indicating that tobacco is a nonhost for these fungi and that ethylene signaling might play a role in nonhost disease resistance. A recent report suggests that the *Arabidopsis* ethylene-insensitive mutants *etr1-1* and *ein2-1* exhibit increased susceptibility to several *Arabidopsis* pathogens [19]. However, the *Arabidopsis etr1-1* mutant has not been reported to lack nonhost pathogen resistance. Thus, the requirement of ethylene perception for nonhost resistance could be plant species specific.

Salicylic acid is one of the key signaling molecules that activate plant defense responses against invading pathogens [20]. Recently, salicylic acid has been implicated in playing a role in nonhost resistance. *Arabidopsis* is a nonhost for cowpea rust fungus (*Uromyces vignae*) and hence restricts the growth of this fungus. The *Arabidopsis* mutant *sid2*, defective in an enzyme that synthesizes salicylic acid, and *Arabidopsis NahG* plants (which express salicylate hydroxylase that can degrade salicylic acid) support growth of *U. vignae* indicating that the salicylic acid pathway is required for nonhost resistance against the rust fungus in *Arabidopsis* [21]. *Arabidopsis NahG* plants are also susceptible to *Pseudomonas syringae* pv. *phaseolicola*, a pathogen normally unable to infect *Arabidopsis* [22]. The loss of nonhost resistance of *Arabidopsis NahG* to *P. syringae* pv. *phaseolicola* has recently been shown to be the result of the presence of catechol, a degradation product of salicylic acid, and not to the lack of salicylic acid itself [23]. However, the susceptibility of the *sid2* mutant to *U. vignae* suggests that salicylic acid synthesis is still required and might play a key role during nonhost disease resistance.

Wound-induced protein kinase (WIPK) and salicylic acid-induced protein kinase (SIPK) have been previously implicated as signaling components of plant defense reactions [24]. A recent report shows that silencing of WIPK and SIPK in *Nicotiana benthamiana* compromises nonhost resistance against *Pseudomonas cichorii* by allowing multiplication and growth of this nonhost pathogen [25]. It is interesting to note that the silencing of either WIPK or SIPK did not alter the HR mediated by the incompatible pathogen *P. cichorii* on the nonhost *N. benthamiana*. It is intriguing that the avirulent pathogen could multiply up to 20-fold more in the WIPK- and SIPK-silenced plants, compared to the control, in the presence of an HR. It is possible that there was a slight delay or reduction of the HR in the WIPK- and SIPK-silenced plants, but this was not detectable by the naked eye. Silencing of WIPK and SIPK also did not affect the HR mediated by INF1 (an elicitor from *Phytophthora infestans* that produces HR when inoculated on wild-type *N. benthamiana*, a nonhost for *P. infestans*) on *N. benthamiana* [25].

Heat-shock proteins (Hsps) are highly conserved proteins that are induced during various forms of environmental stress. Kanzaki *et al.* recently showed that *N. benthamiana* Hsp90, a cytosolic protein, interacts with SIPK in a yeast two-hybrid system [26]. They also showed that silencing of Hsp70 and Hsp90 in *N. benthamiana* individually compromised nonhost resistance by allowing the multiplication and growth of the

nonhost pathogen *P. cichorii* when compared to the wild-type *N. benthamiana* plants [26]. Silenced plants did not produce nonhost HR after inoculation with *P. cichorii* or *P. infestans* INF1 elicitor. It is not known whether silencing of Hsp70 and Hsp90 will also compromise nonhost resistance against other potential pathogens of *N. benthamiana*. The role of Hsps in nonhost resistance warrants further study.

SGT1 is a highly conserved component of the SCF (Skp1–Cullin–F-box protein) ubiquitin ligase complex that targets regulatory proteins for degradation. SGT1 has previously been shown to interact with the resistance signaling gene product RAR1 and is required for several *R* gene-mediated disease resistances [27–29]. Silencing of *SGT1* in *N. benthamiana* affects not only several *R* gene-mediated disease resistances, but also allows the growth of nonhost pathogens *P. syringae* pv. *maculicola* and *Xanthomonas axonopodis* pv. *vesicatoria* [30]. However, silencing *SGT1* did not affect nonhost resistance against *X. campestris* pv. *campestris* and cauliflower mosaic virus [30]. The exact role of *SGT1* in some, but not all examples of nonhost disease resistance has yet to be determined.

Broad-spectrum disease resistance genes

Several nonhost disease resistance genes have now been identified and they are required for nonhost resistance against certain nonhost pathogens. An *Arabidopsis* nonhost resistance gene, *NHO1*, was recently identified and subsequently cloned [22,31]. *NHO1* encodes a glycerol kinase and is required for resistance against *Botrytis cinerea* and *Pseudomonas syringae* isolates from bean or tobacco for which *Arabidopsis* is a nonhost [31]. The *nho1* mutation does not compromise resistance to several other nonhost pathogens including *Alternaria brassicicola*, *Peronospora trifoliorum* or *Xanthomonas oryzae* pv. *oryzae*, suggesting that *NHO1* is required for resistance only to certain pathogens [31]. Interestingly, the expression of *NHO1* was suppressed by infection with *P. syringae* pv. tomato DC3000, a virulent pathogen of *Arabidopsis* [31]. These results suggest that *NHO1* plays a key role in nonhost resistance against some pathogens in *Arabidopsis* and is targeted by the pathogen for parasitism. The exact role of this glycerol kinase in conferring nonhost resistance against pathogens remains unclear.

The *Arabidopsis EDS1* (enhanced disease susceptibility 1) gene has previously been shown, by mutational analysis, to encode an essential component of race-specific disease resistance [32–35]. *EDS1* encodes a novel protein with homology to eukaryotic lipases [36]. Interestingly, the *Arabidopsis eds1* mutant is partially susceptible to several isolates of *Peronospora parasitica* and *Albugo candida* for which *Arabidopsis* is a nonhost [32]. Both *P. parasitica* and *Albugo candida* are pathogens of *Brassica oleracea*. The *eds1* mutant did not lose resistance against several other nonhost pathogens. Partial loss of nonhost resistance in *eds1* suggests that, in addition to *EDS1*, other plant factors are required for nonhost resistance against *P. parasitica* and *A. candida*.

In many cases, nonhost resistance against fungal pathogens is associated with the penetration process. *Arabidopsis pen* (penetration) mutants were identified by

screening for mutants that showed increased penetration of the nonhost fungal pathogen *Blumeria graminis* f. sp. *hordei*, which causes powdery mildew in barley [37]. Mutations in the *PEN1* and *PEN2* genes reduced the ability of the plants to arrest conidia of *B. graminis* f. sp. *hordei* to ~20% of that of wild-type plants [38]. Map-based cloning of *PEN1* revealed that it encodes syntaxin and that it might play a crucial role in papilla-related vesicle trafficking in the plasma membrane [37]. Syntaxins are members of the SNARE super family of proteins that mediate membrane-fusion events. The *pen2* mutant shows alteration of cell-wall-related structure suggesting that the cell wall structures play an important role as physical barriers against fungal infections [38]. Similar screening with the *B. graminis* f. sp. *hordei* pathogen on the host plant barley isolated two mutants, *ror1* and *ror2* (required for MLO-specified resistance), which enhance penetration of *B. graminis* f. sp. *hordei* [39]. Interestingly, *ROR2* gene is a functional homolog of *PEN1* gene [37]. These results provide a mechanistic link between non-host and basal penetration resistance.

Types of nonhost resistance

Plants have evolved to defend themselves against most phytopathogens by recognizing them and triggering an array of defense responses. An incompatible interaction of a pathogen and a nonhost plant often induces several different defense signaling cascades, including generation of active oxygen species, programmed cell death or HR in infected cells, and induction of *PR* genes. HR is commonly used as a visual marker for incompatible plant–pathogen interactions. In many instances, inoculation of a pathogen on one plant species into another nonhost plant species elicits the HR associated with nonhost resistance (Table 1). Interestingly, in some instances, nonhost disease resistance is not associated with induction of a HR (Table 1). Based on these observations, we propose that nonhost resistance against bacteria, fungi and oomycetes be classified into two types: type I and type II. The type I nonhost resistance does not produce any visible symptoms (necrosis) and the type II nonhost resistance is always associated with rapid localized necrosis (HR). The type of nonhost resistance triggered in a nonhost plant is dependent on both the plant species and the pathogen species, such that a nonhost plant species can show type I nonhost resistance against one pathogen species and type II resistance against another pathogen species. For example, *N. benthamiana* exhibits type I nonhost resistance against *Xanthomonas campestris* pv. *campestris* and type II nonhost resistance against *P. syringae* pv. tomato [30]. A single pathogen species can trigger both type I and type II nonhost resistances on different plant species. For example, *P. syringae* pv. *phaseolicola* triggers type I nonhost resistance in *Arabidopsis* and type II nonhost resistance in tobacco [22,40]. It is still not clear if nonhost resistance against viruses can be classified in the same manner because this is mainly caused by a passive mechanism when essential host components are missing [41]. However, there are reports of HR-like responses exhibited by plants during nonhost resistance against

Table 1. Examples of type I and type II nonhost resistance

Pathogen	Strain or isolate	Nonhost plant(s)	Visible symptoms	Refs
Type I nonhost resistance				
<i>Pseudomonas syringae</i> pv. phaseolicola	NPS3121	<i>Arabidopsis</i>	None	[22]
<i>P. s.</i> pv. phaseolicola (at 30 °C)	S2	<i>Nicotiana tabacum</i>	None	[54]
<i>P. s.</i> pv. syringae	B76	<i>Arabidopsis</i>	None	[55]
<i>P. s.</i> pv. savastanoi	213-3 (IAA ⁻)	<i>Arabidopsis</i>	None	[55]
<i>P. s.</i> pv. delphinii	PDDCC529	<i>Arabidopsis</i>	None	[55]
<i>P. s.</i> pv. morsprunorum	B60-1	<i>Arabidopsis</i>	None	[55]
<i>P. s.</i> pv. atrofaciens	B143	<i>Arabidopsis</i>	None	[55]
<i>P. s.</i> pv. coronafaciens	B142	<i>Arabidopsis</i>	None	[55]
<i>Xanthomonas campestris</i> pv. campestris	8004	<i>Nicotiana benthamiana</i>	None	[30]
<i>Gaeumannomyces graminis</i> var. <i>tritici</i>	T5	<i>Avena strigosa</i>	None	[13]
<i>Puccinia recondita</i> f. sp. <i>tritici</i>	WBR I	Oat	None	[56]
<i>Puccinia graminis</i> f. sp. <i>tritici</i>	ANZ	Oat	None	[56]
<i>Phytophthora infestans</i>	88069	<i>N. alata</i> cv. lime green	None	[57]
<i>P. infestans</i>	88069	<i>N. clelandii</i>	None	[57]
<i>P. infestans</i>	88069	<i>N. tabacum</i> cv. <i>xanthi</i>	None	[57]
Type II nonhost resistance				
<i>Pseudomonas syringae</i> pv. <i>maculicola</i>	m2	<i>Nicotiana benthamiana</i>	HR	[30]
<i>P. s.</i> pv. tomato	DC3000	<i>N. tabacum</i>	HR	[58]
<i>P. s.</i> pv. phaseolicola	NPS3121	<i>N. tabacum</i>	HR	[40]
<i>P. s.</i> pv. glycinea	PG4180	<i>N. tabacum</i>	HR	[59]
<i>P. s.</i> pv. pisi	ATCC # 11055	<i>N. tabacum</i>	HR	[54]
<i>P. s.</i> pv. syringae	61	<i>N. tabacum</i>	HR	[54]
<i>P. cichorii</i>	83-1	<i>Arabidopsis</i>	HR	[55]
<i>Xanthomonas axinopodis</i> pv. <i>vesicatoria</i>	82-8	<i>N. benthamiana</i>	HR	[30]
<i>X. campestris</i> pv. <i>glycinea</i>	8ra	Pepper, tomato	HR	[60]
<i>X. citri</i>	3213	Cotton, bean	HR	[61]
<i>Erwinia rubrifaciens</i>		<i>N. tabacum</i>	HR	[62]
<i>Alternaria brassicicola</i>	MUCL20297	<i>Arabidopsis</i>	HR	[16]
<i>Blumeria graminis</i> f. sp. <i>tritici</i>	bgtA95	Barley	HR	[63]
<i>Phytophthora infestans</i>		<i>Arabidopsis</i>	HR	[64]
<i>P. infestans</i>	88069	<i>N. benthamiana</i> , <i>N. rustica</i> , parsley	HR	[57]
<i>P. sojae</i>		<i>Arabidopsis</i>	HR	[64]
<i>Fusarium solani</i> f. sp. <i>phaseoli</i>	W-8	Pea	HR	[65]

certain viruses [42]. RNA silencing in plants against viruses can also be a mechanism of nonhost immunity [43].

Type I nonhost resistance

Even though the symptom-less (no HR) type I nonhost resistance is probably the most common type of nonhost resistance, it has received little attention in the past years. Only a few cases of non-HR-mediated gene-for-gene resistance have been reported. For example, the *Arabidopsis dnd1* (defense no death) mutant confers resistance against avirulent bacteria in a gene-for-gene manner without a HR [44]. This suggests that HR is not always required for disease resistance. We have proposed a model for the mechanism of non-HR-mediated type I nonhost resistance (Figure 1). After a pathogen lands on a nonhost plant, it tries to enter the host tissue in search of nutrition. The first obstacle that the pathogen will face is preformed plant barriers (passive defense mechanisms), such as cell walls, antimicrobial compounds and other secondary metabolites [38,45]. The second obstacle the pathogen will face is the inducible plant defense responses (active defense mechanisms). Plants recognize general elicitors from pathogens in a nonspecific manner to activate defense responses. For example flagellin, a general elicitor protein from bacterial flagella, activates defense responses through a MAP kinase cascade in *Arabidopsis* [46]. Plants can also recognize pathogen surface molecules, also referred to as pathogen-associated molecular patterns (PAMPs), to induce innate immunity [47,48].

PAMPs are shared among members of a pathogen group and are known to induce innate immunity in both plants and animals. Some of the plant defense responses that are induced because of general elicitors and PAMPs include cell wall thickening, cell wall lignification, accumulation of phenolics, production of saponins, production of phytoalexins, papilla formation and induction of *PR* genes [49–52].

During type I nonhost resistance, the pathogen will not be able to get past the first or the second obstacle, and the multiplication and penetration into the plant cell will be completely arrested. Even though the plant looks normal (without any visible symptoms) during the type I nonhost resistance, several molecular changes might be happening. For example, *Arabidopsis* is a nonhost for *P. syringae* pv. phaseolicola and when infected with *P. syringae* pv. phaseolicola, *Arabidopsis* activates *PR* gene expression without any visible symptoms [22]. Recently, Yi Tao *et al.* showed that in addition to *PR* gene expression, a wide array of plant defense genes, similar to those induced during a gene-for-gene resistance response, was activated in the *Arabidopsis*–*P. syringae* pv. phaseolicola nonhost interaction [53]. Several instances of type I nonhost resistance have been reported against bacterial, fungal and oomycete pathogens, and are listed in Table 1.

Type II nonhost resistance

The most commonly discussed phenomenon of nonhost resistance is the type II that produces a nonhost HR.

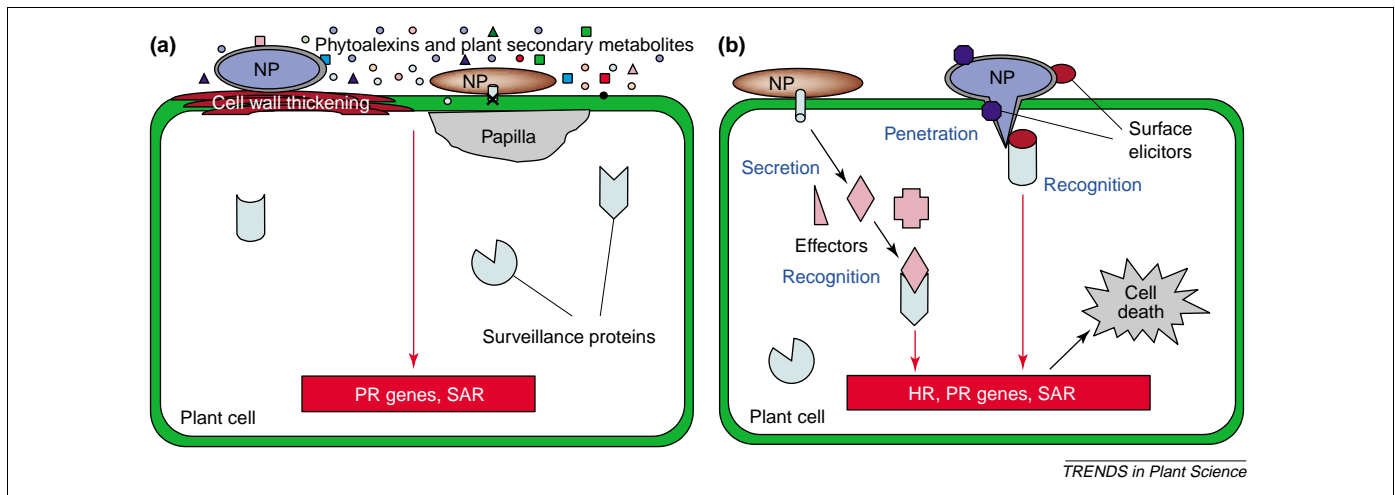


Figure 1. A model for type I and type II nonhost resistance. Blue-colored NPs (nonhost pathogens) represent fungi or oomycetes and brown-colored NPs represent bacteria. (a) During type I nonhost resistance the nonhost pathogen is not able to overcome the preformed and general elicitor-induced plant defense responses such as cell wall thickening, phytoalexin accumulation, other plant secondary metabolites and papilla formation. Pathogenesis-related (PR) gene expression as a component of systemic-acquired resistance (SAR) can be induced by general elicitors of the nonhost pathogen. (b) During type II nonhost resistance the nonhost pathogen is able to overcome preformed and general elicitor-induced plant defense responses, probably by producing detoxifying enzymes. Specific pathogen elicitors are then recognized by the plant surveillance system and this triggers plant defense leading to a hypersensitive response (HR). PR gene expression and SAR are also induced during type II nonhost resistance.

Type II nonhost resistance is phenotypically more similar to an incompatible gene-for-gene interaction and is a more sophisticated plant defense mechanism than type I nonhost resistance. Some pathogens can conquer early obstacles by producing detoxifying enzymes to overcome the toxic effect of preformed antimicrobial plant secondary metabolites [45]. The next obstacle the pathogen will face is the plant cellular defense surveillance mechanism. Plants have evolved to recognize certain pathogen elicitors, either in the plant cytoplasm or at the plant cell membrane, which trigger a defense mechanism that will often lead to HR. Such pathogen elicitors that can be recognized by plants to activate defense responses are called avirulence (Avr) proteins. Avr proteins when not recognized by plants can enhance the virulence of pathogens [66].

Once a pathogen can overcome preformed and general elicitor induced barriers, fungal and oomycete pathogens can directly penetrate a plant cell whereas most plant bacterial pathogens inject virulence and avirulence proteins into the plant cell through a *hrp* gene-encoded type III secretion system (TTSS) [67] (Fig. 1). For fungal and oomycete pathogens, the extracellular proteins on the hyphae or secreted proteins serve as elicitors whereas the injected avirulence proteins serve as elicitors for bacterial pathogens. An elicitor(s) will be recognized by the plant surveillance system and a defense reaction leading to HR will be activated. This will prevent the further spread of the pathogen from the infected cell. For example, several isolates of the oomycete pathogen *P. infestans* produce the extracellular protein INF1, which is required for producing an HR on nonhost plants such as *N. benthamiana* [57]. A functional *hrp*-encoded TTSS is required for bacteria to cause HR on nonhost plants [68]. Thus, *P. syringae* pv. *phaseolicola* caused nonhost HR on tobacco [40], but three Tn5-induced *hrp* mutant strains of *P. syringae* pv. *phaseolicola* did not [69]. These mutants were unable to grow in nonhost leaf tissue even in the

absence of a HR [69]. This is probably because, in addition to the avirulence factors, the virulence factors required for bacterial growth were also not secreted into the plant cell owing to a nonfunctional TTSS.

Similarities between nonhost and gene-for-gene resistance

Several plant defense responses are induced during the nonhost resistance response. Many of these defense responses are similar to those induced during gene-for-gene or host resistance. For example, an HR is associated with both host and nonhost resistance to *P. infestans* [70]. Reactive oxygen species (ROS) are also produced during both gene-for-gene and nonhost resistance, even though the timing and amount might slightly vary between the two [63,71]. Lack of penetration by fungi into nonhost tissue can be associated with the deposition of lignin in the upper and lateral host epidermal cell walls [72,73]. Lignification is a typical response during an incompatible gene-for-gene plant-pathogen interaction [74,75]. Even though both the host and nonhost resistance responses have similarities, it is still questionable if they involve the same signal transduction pathways.

Evidence of similar mechanisms for host and nonhost resistance comes from a recent finding showing that ubiquitin ligase-associated protein SGT1 is required for both host and nonhost resistance in plants [30]. Silencing of *SGT1* affected diverse types of disease resistance in *N. benthamiana*, supporting the idea that R protein-mediated and nonhost resistance might involve similar mechanisms [30]. However, not all examples of nonhost resistance were suppressed in SGT1-silenced *N. benthamiana* plants. These data indicate that the signal transduction pathway of nonhost resistance, against some pathogens, might converge with the host resistance pathway. Further evidence for similar mechanisms of host and nonhost resistance comes from the characteristics of the *Arabidopsis* *nho1* and *eds1* mutants.

The *nho1* mutation not only compromises nonhost pathogen resistance against *Pseudomonas syringae* pv. phaseolicola and *B. cinerea* but also compromises gene-for-gene resistance mediated by *RPS2*, *RPS4*, *RPS5* and *RPM1* [22]. The *eds1* mutant is compromised for *R*-gene-mediated disease resistance against several pathogens and also for nonhost resistance against isolates of *Peronospora parasitica* and *A. candida* [32,33]. These data again suggest that both host and nonhost resistances can share a common pathway.

Gene expression profiling has been used by several researchers to identify genes that are differentially expressed during nonhost disease resistance [76–78]. Recently, using *Arabidopsis* GeneChip, expression of ~8000 *Arabidopsis* genes were monitored after infection with the nonhost pathogen *Pseudomonas syringae* pv. phaseolicola and it was found that the gene expression profile during this nonhost resistance was similar to gene-for-gene resistance mediated by *RPS2* and *avrRpt2* [53]. However, differences were also observed. It will be worthwhile to further characterize these genes to identify gene expression markers specifically for nonhost disease resistance, if such exist.

Even though significant similarities exist between nonhost and gene-for-gene resistance, there are also differences that exist between the two. For example the *PEN1/ROR2*-mediated nonhost resistance or penetration resistance against *B. graminis* f.sp. *hordei* is dispensable for immunity mediated by race-specific *R* genes [37]. Transgenic tobacco plants expressing the *Arabidopsis etr1-1* gene lost nonhost resistance against several nonhost fungal pathogens but the *N* gene-mediated gene-for-gene resistance against TMV was not compromised [18]. It is possible that both nonhost and gene-for-gene resistances have separate signal transduction pathways with significant amount of cross-talk between them and the two pathways might converge at a later time.

Conclusions and future prospects

Extensive studies on gene-for gene resistance have made it possible to clone >40 *R* genes from plants [79]. Such single dominant *R* genes can be readily transferred within closely related species to protect the more agronomically useful crop plants. Resistance conferred by single dominant *R* genes is specific to a particular pathogen race that can express the corresponding avirulence gene(s). Pathogen avirulence genes can be easily mutated or eliminated and hence protection conferred by *R* genes is not durable. By contrast, nonhost resistance can be more durable. Durable disease resistance is a long-sought goal of plant breeders and plant pathologists. Preformed defenses and general elicitor-induced defense responses are the major components of type I nonhost resistance. Because type I nonhost resistance does not involve a HR, it is ideal to exploit this type of resistance for durable disease control in crop plants. It is still not clear whether the durability is because of general or multiple elicitors that can be recognized by the plant surveillance system or because of the presence of several *R* genes that function simultaneously by recognizing their corresponding *avr* genes from a pathogen.

To date, little progress has been made in our understanding of nonhost resistance even though it has been a topic of interest to plant pathologists for many years. One of the reasons for this could be the belief that nonhost resistance is a multigenic trait and thus difficult for genetic manipulation [80]. However, several components of nonhost resistance have now been identified. Mutations in any one of these components have been shown to compromise nonhost resistance against some, although not all, nonhost pathogens. Hence, genetic studies can be used to dissect nonhost resistance pathways. Even though many of the nonhost resistance components are known, we are still far away from understanding the complex phenomenon of nonhost resistance. Recent progress in functional genomic technologies has made available tools such as gene expression profiling and virus-induced gene silencing that can be used to dissect the complex phenomenon of nonhost resistance. In depth study of the mechanism(s) of nonhost resistance will bring us a much better view of the dynamics of plant disease resistance in general and the potential to genetically engineer plants for resistance against a broad range of pathogens.

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