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THE INTRICACIES OF NON-HOST RESISTANCE MECHANISM IN PLANTS: A COMPREHENSIVE REVIEW

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ABSTRACT

Non-host resistance (NHR) is a vital defense mechanism in plants that provides complete immunity to a plant species against all strains of a pathogenic microorganism that typically affects other plants, and it functions independently of canonical resistance (R) genes. When potentially harmful microorganisms, incapable of infecting any variant of a particular plant species, are termed non-host pathogens whereas, plants showcasing resistance against all variants of a specific pathogenic species are categorized as non-host plants. NHR reduces a potential pathogen's ability to penetrate or thrive within a non-host plant. Unlike R-gene mediated resistance, which often relies on a single genetic element, NHR is orchestrated by a multitude of genes, governing diverse protective mechanisms. These encompass both inherent barriers and inducible responses, forming a multi-layered defense. These defenses include natural barriers, pre-existing or triggered under specific conditions, such as signal-mediated pathogen deterrence, physical defenses like waxy coatings and robust cell walls, and pre-formed chemical deterrents like phytoanticipins. Induced defense responses, like lignin accumulation, antimicrobial compound production, hypersensitive response (HR), and pathogenesis-related (PR) protein activation, play a vital role.NHR exemplifies nature's intricate strategies for protecting plants against potential pathogenic threats.

Key words : Non-host Resistance, Basal defense, Durable resistance, Hypersensitive Reaction, Pathogenesis-related (PR) protein.

Introduction

Non-hostresistance (NHR) is a comprehensive defense mechanism in plants that grants immunity to an entire plant species against all strains of a microorganism that causes disease in other plant species. Coined by Michele Heath in the late 1970s, the term NHR signifies the intrinsic capability of organisms to fend off pathogenic organisms without the involvement of canonical resistance (R) genes. This form of resistance is exhibited when potentially harmful microorganisms, incapable of infecting any variant of a specific plant species, are labeled as heterologous pathogens or non-host pathogens. Conversely, plants that display resistance against all variations of a particular pathogenic species are categorized as non-host plants.

The outcome of NHR is a diminished ability for a potential pathogen to penetrate or propagate within a plant species that is not its usual host. In contrast to R-gene mediated resistance, often governed by a single genetic element, non-host resistance is predicted to be regulated by an assortment of protective mechanisms influenced by numerous genes. Non-host resistance tends to be multilayered, involving various barriers that a specific host deploys to prevent the establishment of a potential pathogen. These obstructions encompass a variety of natural defenses found in plants, both pre-existing and triggered under specific conditions. Examples of these barriers encompass the presence or absence of signals that prompt pathogen development, the physical structures and pre-formed chemical deterrents. Induced defense reactions also play a crucial role and encompass processes

such as the accumulation of lignin, the production of antimicrobial compounds like phytoalexins, the hypersensitive response (HR) and the activation of pathogenesis-related (PR) proteins.

Despite shared defense themes among plants, variations exist between different species and sometimes even among different genetic types within the same species. These distinctions manifest in the structure, biochemical composition, and the specific cues that trigger these defense mechanisms.

Types of Non-host Resistance

Non-host resistance against bacteria, fungi, and oomycetes can be classified into two types (Mysore and Ryu, 2004).

Type I : This type of non-host resistance does not produce any visible symptoms because of the absence of a hypersensitive response. The pathogen does not get past the first (preformed plant barriers) or the second obstacle (inducible plant defense responses) and the multiplication and penetration into the plant cell arecompletely arrested. Plants recognize general elicitors from pathogens in a nonspecific manner to activate defense responses (Table 1).

Table 1 : Type I non-host resistance.

Pathogen	Strain	Non-host plant(s)
Pseudomonas syringae pv. *phaseolicola*	NPS3121	*Arabidopsis*
Xanthomonas campestris pv. *campestris*	8004	*Nicotiana benthamiana*
Gaeumannomyces graminis var. *tritici*	T5	*Avena strigosa*
Puccinia graminis f. sp. *tritici*	ANZ	Oat
P. infestans	88069	*N. clevelandii*

Table 1 : Type II non-host resistance.

Pathogen	Strain	Non-host plant(s)
X. campestris pv. *glycines*	8ra	Pepper, tomato
Alternaria brassicicola	MUCL20297	*Arabidopsis*
Blumeria graminis f. sp. *tritici*	bgtA95	Barley
Fusarium solani f. sp. *phaseoli*	W-8	Pea
X. citri	3213	Cotton, bean

Type II : This type of non-host resistance produce necrotic symptoms as a result of Hypersensitive Response. Pathogens may conquer early obstacles by producing detoxifying enzymes to overcome the preformed constitutive barriers. Plants have evolved to recognize specific pathogen elicitors, either in the plant cytoplasm or at the plant cell membrane, which trigger a defense mechanism that will often lead to HR (Table 2).

The type of non-host resistance triggered in a non-host plant is dependent on both the plant species and the pathogen species, such that a non-host plant species can show type I non-host resistance against one pathogen species and type II resistance against another pathogen species. For example, *N. benthamiana* exhibits type I non-host resistance against *Xanthomonas campestris pv. campestris* and type II non-host resistance against *P. syringae* pv. Tomato. A single pathogen species can trigger both type I and type II non-host resistances on different plant species. For example, *P. syringae* pv. *phaseolicola* triggers type I non-host resistance in *Arabidopsis* and type II non-host resistance in tobacco.

Components of Non-host Resistance

Preformed or Passive Defense Mechanism

Pre-existing defenses constitute the initial hurdle that pathogens must surpass before infiltrating a plant. These defenses encompass both physical barriers and chemical barriers that restrict the entry of pathogens (Heath MC1997, Aires *et al*., 2009; Fan *et al*., 2011). Certain structural defenses are inherently present within the plant even before any contact with the pathogen occurs. These structural attributes encompass characteristics such as the quantity and quality of waxy coverings and cuticles on epidermal cells, the composition of epidermal cell walls, the arrangement, size, shapes of stomata and lenticels, as well as the presence of thick-walled cell tissues that obstruct pathogen advancement. A dense layer of hairs on the plant surface can also exert a comparable water-repellent effect, potentially diminishing the likelihood of infection. The plant's cytoskeleton serves as a tangible defense against a variety of invading pathogens. For instance, actin microfilaments within the plant play a vital role in safeguarding against fungal penetration. Disruption of these microfilaments results in the loss of non-host

resistance to several non-host fungi (Wang *et al*., 2022).

Plants are naturally equipped with an assortment of secondary compounds that are synthesized constitutively for defense against microorganisms. Phenolic compounds, Peptides (Broekaert, 1995) tannins and fatty acid-like molecules such as dienes are among these compounds. Phytoanticipins, a group of secondary metabolites are notably active within plants before any pathogenic intrusion. For example, fragarin, a phytoanticipin derived from the cytosolic fraction of strawberry leaf tissues, effectively impedes the growth of various bacterial pathogens (Filippone *et al*., 1999). Saponins, another class of compounds, are consistently produced in numerous plants and can also be induced in response to a pathogen attack. An illustrative instance of this is observed with the fungus *Gaeumannomyces graminis* var. *tritici*, a wheat pathogen that cannot infect oats. In response to this challenge, *Gaeumannomyces graminis* produces avenacins, a category of triterpene saponins specific to root infection (Papadopoulou *et al*., 1999). Within the Brassicaceae family, glucosinolates and their derivatives, sulfur- and nitrogen-containing secondary metabolites, play a pivotal role in boosting plant defense mechanisms against an extensive spectrum of pathogens (Chhajed *et al*., 2020; Singh, 2017). These pre-existing defenses collectively exemplify the intricate strategies that plants have evolved to resist and counteract potential pathogenic threats.

Inducible Plant Defense Mechanisms

The subsequent challenge faced by an invading pathogen pertains to the induction of plant defense mechanisms. These defense reactions impede the growth of non-host pathogens through the creation of structural barriers, the initiation of new synthesis of antimicrobial compounds and proteins and the activation of various defense pathways at the molecular level. Recognition of non-host pathogens often triggers the reinforcement of the cell wall through the deposition of callose, lignin and suberin. These compounds are widely acknowledged for enhancing the structural integrity of the cell wall as an induced defense response.

In addition to these induced physical barriers, numerous chemical compounds are newly synthesized or transformed from non-toxic forms to toxic forms, thereby assuming antimicrobial roles. Phytoalexins, for instance, represent a class of low molecular weight antimicrobial compounds that are manufactured anew in response to pathogenic assaults. In the case of lettuce, hydroxyproline-rich glycoproteins, phenolic compounds, and a phytoalexin called lettucenin A are produced as defensive measures against non-host pathogens (Senthil-Kumar, 2013). Another example is camalexin, a phytoalexin known for its ability to disrupt bacterial membranes. It accumulates in *Arabidopsis* plants when they are inoculated with the non-host pathogen *Pseudomonas syringae* pv. *syringae*, the causative agent of bacterial brown spot in beans (Rogers, 1996). Organosulfur compounds like sulforaphane have been identified in *Arabidopsis*. They are synthesized and released into the apoplast to restrain the growth of non-host pathovars of *Pseudomonas syringae* (Wang *et al*., 2020). Collectively, these inducible defense mechanisms exemplify the intricate strategies plants deploy to mount a robust response against potential pathogenic intruders.

R gene-mediated Resistance

Effector Triggered Immunity (ETI) is one of the major components of host resistance in plants (Cui *et al*., 2015). It is activated by direct or indirect interaction between one or more pathogen effectors and one or more plant R proteins, often resulting in HR. Numerous cases of effector recognition non-host plants have been reported (Table 3).

Plant Defense Signaling

A multitude of plant signaling components play a crucial role in instigating plant defense responses. In the context of non-host pathogen invasion, plants discern signals from these non-host pathogens, subsequently activating various defense pathways at the molecular level. Notably, the efficiency of signal perception and the robustness of individual pathogen recognition events characterize non-host resistance. To effectively cause disease in plants, invading pathogens must successfully navigate through many of these signaling components. Classical genes linked to defense pathways, such as phytoalexin-deficient 4 (PAD4) and pathogenesis-related 1 (PR1), exhibit significant induction in *Arabidopsis* upon non-host pathogen introduction (Zhang *et al*., 2018).

An integral player in activating plant defense responses against invading pathogens is salicylic acid, a key signaling molecule (An, 2011; Saleem *et al.*, 2021; Vidhyasekaran, 2015). For instance, *Arabidopsis* is a non-host for the cowpea rust fungus (*Uromyces vignae*), leading to the inhibition of its growth. The role of the salicylic acid pathway in non-host resistance is evident in experiments involving *Arabidopsis* mutants. Mutants like sid2, which lack the enzyme responsible for salicylic acid synthesis and NahG plants, which degrade salicylic acid due to the expression of salicylate hydroxylase, permit the growth of *U. vignae* (Mellersh, 2003). This underscores the necessity of the salicylic acid pathway

Table 3 : R-gene mediated genes involved in NHR.

Gene/protein	Function in non-host resistance (NHR)	Reference
EDS1	This gene is necessary for R-gene-mediated resistance to many pathogens in *Arabidopsis* and is also involved in the execution of NHR against isolates of *Peronospora parasitica* and *Albugo candida*.	Parker *et al.* (1996)
avrRxv	Induces HR reaction in non- host common bean against *Xanthomonas campestris* pv. *Vesicatoria* tomato race 1	Whalen *et al.* 1988
SGT1	Silencing of SGT1 in *N. benthamiana* compromises NHR against *P. syringae* pv. *maculicola* and *Xanthomonas axonopodispv. vesicatoria*	Peart *et al.* (2002)
Heat-shock proteins (Hsps)	Silencing of Hsp90 and Hsp70 in *N. benthamiana* individually compromised NHR against *P. cichorii*.	Kanzaki *et al.* (2003)
WIPK and SIPK	In *N. benthamiana* virus-induced gene silencing of NbSIPK and NbWIPK allowed multiplication of non-host bacterium *P. cichorii*.	Sharma *et al.* (2003)
PAD4/SAG101	Pad4 and sag101 single mutation have little effect on the frequency of Bgh haustoria formation in *Arabidopsis*. But along with pen mutation (pen2 sag101pad4) NHR was compromised.	Lipka *et al.* (2005) and Stein *et al.* (2006)

Table 4 : Broad-spectrum disease resistance genes

Gene/protein	Function in non-host resistance (NHR)	Reference
PAMPs involved in NHR		
Pep-13	Induces defense responses in non-host plants like potato	Nürnberger *et al.* (2005)
Harpin (Hrp Z)	Elicits HR-like cell death and defense responses in various plants	
Avirulennce (avr) genes	These genes from bacterial pathogens are recognized by previously unidentified R-genes in non-host plants.	Kobayashi *et al.* (1989) and Arnold *et al.* (2001)
Genes involved in NHR		
PEN1 (Penetration 1) /ROR2	This gene is involved in timely deposition of papillae during non-host interactions.	Collins *et al.* (2003)
PEN2	Encodes myrosinase involved in hydrolysis of indole glucosinolates to release potential antimicrobial components at the site of non-host interaction.	Lipka *et al.* (2005) and Bednarek *et al.* (2009)
PEN3/PDR8	May be involved in exporting toxic materials to the site of non-host pathogen interaction and intracellular accumulation of toxins.	Stein *et al.* (2006)
ETR1-1	Ethylene insensitive (etr1-1) tobacco plants lost resistance against many non-host pathogens; but N-gene-mediated gene-for-gene resistance against TMV was not compromised.	Knoester *et al.* (1998)
NHO1	Required for NHR of *Arabidopsis* against *Pseudomonas syringae* pv. *phaseolicola*)	Kang *et al.* (2003)

for non-host resistance against the rust fungus in *Arabidopsis*. Ethylene perception is pivotal not only for basal resistance against pathogens but also for inducing disease resistance in plants. For example, tobacco plants engineered to express the *Arabidopsis* etr1-1 gene (causing a loss of ethylene perception) failed to induce basic Pathogenesis-Related (PR) genes upon infection with tobacco mosaic virus (TMV) (Knoester, 1998). Heat-shock proteins (Hsps), which are highly conserved and induced during environmental stress, also contribute significantly. In plants like *Nicotiana benthamiana*, silencing Hsp70 and Hsp90, cytosolic proteins, compromises non-host resistance against pathogens like *Pseudomonas cichorii* (Kanzaki, 2003). This leads to the proliferation and growth of the non-host pathogen, highlighting the protective role of these proteins in non-host resistance. Wound-induced protein kinase (WIPK) and salicylic acid-induced protein kinase (SIPK) have previously been linked to plant defense responses. Recent research shows that silencing these components in

Nicotiana benthamiana compromises non-host resistance against *Pseudomonas cichorii* (Segonzac *et al.*, 2011) (Table 4).

Conclusion

The molecular basis of NHR continues to be elucidated and more examples of interspecies transfer of NHR genes that provide resistance against non-adapted pathogens also providing resistance in host species, are apparent. This type of exploitation of NHR for crop protection will undoubtedly increase with an increased understanding of the molecular mechanisms underlying this resistance. The forecasted change in plant and pathogen distributions as a consequence of climate change may unfortunately act as a catalyst to uncover other NHR pathosystems that are currently also based upon limited resistance.

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