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What is Host-Pathogen Initial Interaction Telling Us? an Essential Component of Biotic Stress Response Mechanism!

Pavitra Kotari[1], Vinod Jatav[2] and Ravishankar KundapuraV[1]*

[1]*Department of Biotechnology, ICAR-Indian Institute of Horticultural Research, India*

[2]*Department of Vegetable Crops, ICAR-Indian Institute of Horticultural Research, India*

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***Corresponding author:** KV Ravishankar, Department of Biotechnology, ICAR-Indian Institute of Horticultural Research, Hesaraghatta Lake Post, Bengaluru 560 089, India

Abstract

Plants, in their natural environment are encountered by numerous pathogens. However, plants have evolved with the innate immune system that can deal with attack of these pathogens. Nevertheless, pathogens also keep modifying themselves to counter the host immunity and thus making the plants susceptible. Over the years, several studies in various crop species on plant pathogen interactions, showed that understanding of host-pathogens is still an intriguing area of investigation. In this review, we s discuss the recent studies, which show the active role of pathogens and their effector proteins in bringing up the disease development in the host system.

Keywords: Biotic stress; Plant pathogen; Signalling; Elicitors; Effectors; Disease development; Defense mechanism

Abbreviations: CWEDs: Cell Wall Degrading Enzymes; PAMPS: Pathogen Associated Molecular Patterns; PRRs: Pattern Recognition Receptors; PTI: Pattern Triggered Immunity; TALEs: Transcription Activator-Like Effectors; EBEs: Effector-Binding Elements; PR: Pathogenesis-Related

Introduction

Presently, biotic stress has become a global challenge, thereby significantly imposing intense selection pressure on plants leading to huge economic losses. Additionally, the changing climate is further worsening this situation. These stress factors (Bacteria, Fungi, Viruses, nematodes, insects and pests) act as un favourable conditions that bring about physiological and biochemical changes in plants resulting in inhibited growth, reduced economical yield, rendering acclimatization and *adaptation* of species to changing environment. Pathogens are those mediators that cause diseases in plants by manipulating the normal functioning of plant cells. There are mainly two types of pathogens Necrotrophs (Kill their host viz., *Fusarium*; *Botrytis, Cochliobolus*), and biotrophs (rarely kill their hosts viz., *Erysiphe, Ustilago,* Phytophthora) causing vascular wilts, leaf spots, cankers, wilting, stunting, chlorosis, malformations and affecting different parts of the plants.

Plant pathogens have evolved different strategies to infect their host and thus suppress host defense mechanism. By understanding the mode/mechanism of infection brought about by the pathogens would help to unravel the disease controlling strategies. Over the years lot of studies have been reported on plant pathogen interactions from both the pathogen and the plant viewpoint. Adapted pathogens circumvent and disturb host immunity, thus making plants susceptible. Sometimes, the failure of the host's immune system also contributes towards plant disease susceptibility.

Host-pathogen interaction occurs on different levels during infection. Firstly, at the cell-wall level (First ground of battle field) and secondly at nucleus or cytoplasm level (Second ground of battle field). Pathogens, to make their entry into the host cell, make certain alterations in the plant cell walls by secreting different types of enzymes/elicitors that lead to destruction of cellulose, xylan and pectin eg. Cell Wall Degrading Enzymes (CWDEs). In turn, plants have evolved to combat the initial mode of infection by recognizing the Pathogen Associated Molecular Patterns (PAMPs) by Pattern Recognition Receptors (PRRs) leading to induce Pattern Triggered Immunity (PTI). In response to first line of defense, pathogens in turn modify them to bring about the infection at the second ground of battle field by secreting different types of virulence proteins through different secretion pathways.

Elicitors

Plants in their natural habitat exposed to several pathogens, releasing various chemical substances to exhibit a cascade of interactions between the host and its pathogen (Figure1). An elicitor is a molecule that triggers defense response in the plant

system. They are diverse molecules eliciting the similar response in plants. Elicitors are basically of two types based on its origin, exogenous elicitors (pathogen origin) endogenous elicitors (plant origin). Elicitors are further divided into two categories based on its host interaction, General elicitors (infectious to all host) and Specific elicitors (infectious to specific host).

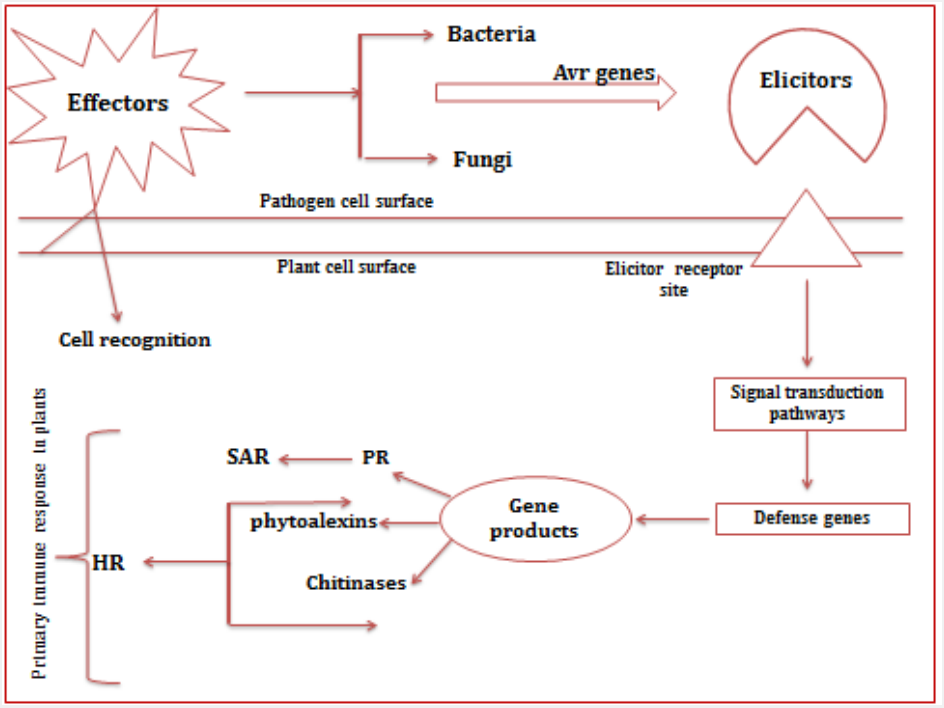

Figure 1: Illustration of host-pathogen interaction.

When pathogen encounters a host, it tries to overcome the important barrier (cell wall) that plants use to limit pathogen attack by producing enzymes that bring about the destruction of different components (Cellulose, xylan and pectin) of the cell wall resulting in establishment of pathogenesis. Recently, several studies have reported the role of elicitors upon pathogen infection. One such study reported describes about a novel protein CfPDIP1 secreted by *Colletotrichum falcatum*, exhibiting defense responses in sugarcane and triggering hypersensitive response in tobacco. Another study focused on β-glucans and chitin oligomers from Phytophthora and Pythium, Produced phytoalexins in soybean and rice plants and lignification in wheat leaves [1,2] studied the effect of oligogalacturonides from bacteria and fungi leading to synthesis of protein inhibitors and thus activating defense in *Arabidopsis* and tomato. Few studies have also been reported in viruses to elicit HR response in tobacco and tomato using viral coat protein hairpin from TMV [1]. Another report by [3], focused on *Flagellin*, flg 15 from *Ralstonia* bacteria, leading to deposition of callose and activation of defense genes in *Arabidopsis*.

Effectors

Table 1: Effector(s) and their functions of different pathosystem.

Crops	*Pathogen*	Effector (s)	Function	Reference (s)
Tomato	*Ralstonia solanacearum*	RSp0846	Injecting into host cell through Hrp type III secretion system (TTSS)	Mukaihara et al. [64]
		RipAY	Suppresses Plant Immunity	Mukaihara et al. [63]
		AWR5	Inhibitor of the TOR signalling pathway	Popa et al. [68]
		RipP2 (PopP2)	RRS1-R and RPS4 are the corresponding r genes	Deslandes et al. [51]
		RipAX2	EBWR9 is the corresponding r gene locus in brinjal	Morel et al. [62]
	Meloidogyne incognita	Misp12	Down-Regulation of Defensive Genes (NF1, PAL5)	Xie et al. [10]
		MiPFN3	tip2 (AY731066) is the corresponding tagert	Leelarasamee et al. [59]
		Msp40	Facilitate parasitism	Niu et al. [65]
		MiISE5	Suppresses programmed cell	Shi et al. [73]
		MiISE6	Facilitate parasitism	Shi et al. [74]
		8D05	Promote the parasitic interaction	Xue et al. [9]

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	Alternaria alternata f. sp. lycopersici	AAL-toxins	Asc is the corresponding r locus	Wolpert et al. [82]
	Fusarium oxysporum f.sp. lycopersici	Avr1	I-1, I-2 and I-3- are the corresponding r genes, respectively	Takken and Rep [76]
		Avr2		
		Avr3		
Potato	*Phytophthora infestans*	Avr3a	R3a is the corresponding resistance gene	Armstrong et al. [40]
		AVR1	R1 is the corresponding resistance protein	Du et al. [52]
		SFI	Suppressive to Host Defense	Yin et al. [83]
		AVRblb2,	Targeted the host papain-like cysteine protease C14	Bozkurt et al. [42]
		Pi04314	Deutralized host immunity.	Wang et al. [80]
		Pi14054	Suppressor of RNA silencing	Vetukuri et al. [79]
Watermelon	*Fusarium oxysporum f. sp. Niveum*	FonSIX6	Fusarium wilt avirulence factor	Niu et al. [65]
Onion	*Fusarium oxysporum f. sp. cepae*	SIX, CRX1 and C5	Strongly related to pathogenicity	Taylor et al. [77]
Grape	*Plasmopara viticola*	RxLR	ETI to downy mildew in grape	Brilli et al. [43]
Banana	*Fusarium Oxysporum f. sp. Cubense*	Six 1 and Six 6	Suppressing plant immunity	Covey et al. [47] and Chakrabarti et al.[44]
Arabidopsis thaliana	*P. syringae*	AvrRpm1, AvrRpt2 AvrPphB AvrRps4	RPM1, RPS2, RPS5, RPS4 are the corresponding r genes	Tamaki et al. [78] Whalen et al. [81] Jenner et al. [56] Hinsch et al. [55]
Rice	*Xanthomonas oryzae*	PthXo1, AvrXa7 and AvrXa27	xa5 is the corresponding r gene	Ma et al. [26]
		AVR-Pik	Pikp-HMA and Pikm-HMA are the corresponding r genes	Concepcion et al. [46]
	Magnaporthe oryzae	AVR-Pia	Delivered to the plant cytosol	Sornkom et al. [75]
		SPD4	Avoided recognition by the host	Sharpee et al. [72]
Wheat	*Blumeria graminis f. sp. tritici*	AVRPM	PM3A and PM3F are the corresponding immune receptor	McNally et al. [61]
	Puccinia striiformis f. sp. tritici	PstSCR1	Involved in PTI	Dagvadorj et al. [50]
	Puccinia graminis	Shr7	Suppress Multiple Plant Defense Responses	Ramachandran et al. [69]
	Zymoseptoria tritici	SSP15 and SSP18	Required for pathogenesis	Gohari et al. [54]
Maize	*Ustilago maydis*	Cce1	Suppress plant defense responses	Seitner et al. [71]
Brassica napus	*Leptosphaeria maculans*	AvrLm4-7	Rlm4 and Rlm7 are the corresponding r genes	Parlange et al. [67]
Soybean	*Phytophthora sojae*	PsCRN63	Suppress plant immunity	Li et al. [60]
		Avr1b	Rps1b is the corresponding r gene	Cui et al. [49]
		PsAvh262	Suppresses ER stress-triggered cell death	Jing et al. [57]
Cotton	*Xanthomonas citri subsp. Malvacearum*	Avrb6	ETS to bacterial blight	Cox et al. [48]
Flax	*Melampsora lini*	AvrL567	L6 is the corresponding r gene	Ellis et al. [53]

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For the establishment of compatible interaction, pathogens must overcome Pattern Triggered Immunity (PTI) by secreting effector proteins that leads to Effector Triggered Susceptibility (ETS) and thereby be recognized by the host defense system, to exhibit Effector Triggered Immunity (ETI) [4]. In this review, we discuss the role of specific effectors of various pathogens affecting different crop species (Table 1).

Effectors are the protein molecules produced by virulence genes of pathogens. In order to initiate an infection, plant pathogens secrete a wide range of virulence proteins into the plant system through different types of secretion systems. There are different types of secretion pathways. Type I and II pathways secrete proteins to the host intercellular spaces, whereas type III and IV pathways deliver proteins or nucleic acids directly into the host cell. Examples of plant pathogen effectors secreted via the type I pathway are proteases and lipases from the soft-rot pathogen *Erwinia chrysanthemi* [5]. Type II secretion system is used by *Ralstonia* to translocate CWDEs. The prokaryotic pathogens (bacterium) mainly use a type three secretion system (T3SS).

While, the nematodes use the stylet to secrete effectors into the plant cells [6,7]. Further the fungal pathogens secrete the effectors into the plant apoplast or into the host cells and thus bring about the suppression of defense responses and alterations of the host metabolism. For example, the smut pathogen of maize, *Ustilago* maydis, encodes several hundreds of effector proteins that are virulence in nature and establish susceptibility [8]. Several nematode effectors responsible for parasitism have been reported by several researchers (Misp12, Msp40, 8D05) [9-13].

In order to counteract the effector molecules, plants have inculcated an inbuilt immune system to recognize effector proteins by producing specific R proteins (NLR receptors) [14]. A study reported by Gururania et al. [15] and Ali et al. [16] describe about the effector proteins from crop-infecting fungi, out of eighty-three, 43 are encoded by Avr genes. Recent study, conducted by Bourras et al. [17] explains the effector protein (SvrPm3a1/f1) suppressing the immunity against powdery mildew pathogen in wheat.

Effectors have seen to perform diverse functions (pathogen self- defence, liberation of nutrients and manipulating signal transduction) as reported by Fatima and Senthil-Kumar [18]. Example SnTox1 effector from *Parastagonospora nodorum* is seen to have multiple roles in host manipulation. Another Zt6 effector from the wheat pathogen *Zymoseptoria tritici* is seen to have phytotoxic and antimicrobial activity that affects both host and other microbes [19,20]. Another study reported by Rodriguez-Moreno et al. [21] has described about the various infectious strategies used by the fungal pathogens to mediate host penetration, recognition escape and host defense inhibition.

Transcriptional Reprogramming

Recent transcriptomic studies demonstrated the fact that transcriptional changes in response to avirulent and virulent pathogens plays a vital role in both pathogenesis and resistance mechanism via up regulation or down regulation of the corresponding genes. Transcriptional changes in susceptible and resistant cultivars in response to pathogen, differ quantitatively bot not qualitatively [22]. Pathogen evolved to increase fitness (mutation in effector gene) in response of host by modulating host physiology in various ways. Some examples of diverse effector functions include modifying components of the immune system to evade detection and redirecting nutrients to the apoplast to support pathogen growth [23,24].

Transcriptional Reprogramming can broadly be classified into two groups. Firstly, effector proteins act as direct signal transducers, where the effector proteins are directly recognised by R- protein and thus induce the defense system of plant by up or down regulation of pathogenesis responsive genes. On the other hand, pathogens evolve to avoid recognition of effector protein by R-protein and hijack the transcriptional machinery of plants in favor of itself. For instance, *Xanthomonas oryzae pv. oryzae* injects Transcription Activator-Like Effectors (TALEs), directly into plant host cells, where they bind to specific promoter sequences in host genes (Xa5), which are designated as TAL Effector-Binding Elements (EBEs) [24,25], and promote the transcription of susceptibility (S) genes (OsSWEET11 and OsSWEET14). In response plant evolved naturally via mutating the Xa5 gene as xa5 (naturally occurring mutant form of Xa5) and these defective rice lines show reduced expression of the TALE-targeted susceptibility (S) genes, which results in increased resistance against bacterial blight in rice [26].

Secondly, effector proteins can act as indirect signal transducers; here the effector protein induces the signal transduction through other molecules. Virulent pathogens again evolved to use the plant transcription machinery according to their *adaptation*. For instance, CaCDPK15 was found to regulate pepper response to Pseudomonas solanacearum attack by indirectly activating CaWRKY40 expression, and CaWRKY40 in turn activated transcriptional expression of CaCDPK15 by binding to the CaCDPK15 [27]. *Hyaloperonospora arabidopsidis*, effector (HaRxL44), decreased SA immune processes by interfering with mediator function bringing enhanced susceptibility to downy mildew in *arabidopsis* [28]. Another previous report on Salicylic Acid (SA) required for the activation of genes encoding Pathogenesis-Related (PR) proteins such as PR1, potentiates the hypersensitive response triggered when pathogen effectors are directly or indirectly detected by R proteins [29]. Example. The biotrophic fungus *Ustilago* maydis causes smut disease in maize. Cmu1, an effector translocated into the cytoplasm of maize (Zea mays) plants by U. *maydis,* interferes with the SA pathway during disease development. Xanthomonas species have been shown to use a novel class of effectors, known as transcription activator-like effectors, which function to bind specific host DNA sequences, resulting in the expression of host genes that aid pathogen colonization and infection [30,31]. Another review reported by Bezrutczyk et al. [32] has focused on the role of some pathogens that produce transcription activators, that induce the expression

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of sugar uptake transporters involved in bringing about plant susceptibility to pathogens.

Discussion and Conclusion

The co-evolution of plants and pathogens involves complex interaction mechanisms between the virulence factors (Elicitors and Effector proteins) of pathogens and the plants immune system (PTI and ETI). Cell wall being the initial battlefield for the pathogens and plants, both adopt contrasting wall-breaking and wall –reinforcing strategies to attack or to defend the entry of the foreign molecule [33].

Modification of cell walls is not the only strategy employed by fungal pathogens to prevent plant recognition. Where, pathogens make use of the CWDEs, as their first line of gadget of virulence to dissolve the cell wall components and establish the susceptible reaction/disease incidence. In response to this, plants also have developed a first line of defense mechanism known as basal immunity by recognizing the conserved microbial molecular patterns or PAMPs through PRRs leading to induction of PTI. An extensive review on plant Pattern Recognition Receptors (PRRs) role in detection of microbe-associated molecular patterns and activation of intracellular signaling leading to PTI has been reported by Saijo et al. [34]. To overcome PTI induced defense, pathogens have further evolved with and developed different types of secretion systems to deliver and transport virulence factors (effectors) directly into the host system (cytosol) by bypassing / eluding the recognition facilitating the parasitism [35].

One such study reported, describes about the fungal pathogen that has evolved several strategies to overcome host immune responses by bringing about alterations in cell wall compositions and the secretion of effectors [36,37]. For instance, the tomato leaf mould fungus *Cladosporium fulvum* secretes the carbohydrate-binding effector protein Ecp6 that suppresses chitin-triggered host immunity [38].

In turn, to fight back, plants also have developed second line of defense (R gene mediated immunity and/or gene-gene recognition) by recognizing the effector proteins through specific, leading to induction of ETI. This is an ongoing process, in which pathogens keep evolving with new races and secreting modified effector proteins to increase their virulence. For example. Phytophthora infestans, encode RXLR-type effectors that silence the activity of AVR3a that leads to a reduction in disease incidence [39-50].

Transcriptional re-programming is another gadget, that both pathogen and host make use of to exhibit a complex interaction mechanism. Here, both the pathogen and the host try to control transcription of different genes on a competition basis; the success decides the course of transcriptional mechanism for that particular gene. If the pathogen succeeds, the virulence gene(s)/gene product(s) bring about the re-programming in the expression of plant genes either by up-regulating susceptible genes or down-regulating resistant genes. On the other side, if the plant succeeds, it elicits the defense mechanism by bringing about the transcriptional re-programing of various defense related molecules (SA, JA and ET), R-genes and down regulation of susceptible genes. Several transcription factors (WRKY, ERF, AP2, bHLH, NAC, bZIP and MYB) also play a vital role in bringing about re-programing in the transcriptional mechanism [51-65].

Deciphering the role of pathogenicity factors (effector/ elicitor proteins) in the manipulation of host system would help us in understanding the mechanisms contributing towards the disease establishment and could also potentially lead to improve disease control methods. A thorough knowledge on the molecular mechanisms complementing with the virulence factors and receptor proteins would also lead to understand the gene for gene relationship among host and its pathogen, which finally would help in formulating pathogen specific resistance breeding programmes [66-83].

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Conflict of Interest

The authors declare no conflict of interest.

Informed consent

All authors give informed consent to publish the manuscript.

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