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Modern Molecular Host-Pathogen Interaction is an Old Story of the Rivalry Between Host and Pathogen

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ABSTRACT

Pulses are important food crops cultivated globally for their high nutritional For almost 16 years, the Zig-Zag model successfully explains the compatibility and incompatibility in host-pathogen interaction. This model fits well in the case of biotrophs and hemi-biotrophic but is least applicable in the case of necrotrophs. To regulate the immunity a Crosstalk happens between PTI and ETI that intersect the downstream pathways. In this brief review, we have elaborated the concept of immunity with specific reference to the story of Troy for easy understanding the resemblance with host-pathogen interaction.

Keywords : PTI, ETI, Zig-Zag model, Immunity, R-gene, Effectors

Introduction :

There are several characters representing the classical story of the movie "Troy"andcorrelated the story with molecular interaction started with the Zig zag are model of resistance which has given by Jones and Dangle (2006). The concept of innate mmunity in vertebrate animals is old there, here like in the movie troy plants have also a different level of defensesystem present there, In the Zigzag model four phases there as depicted in Fig 1.

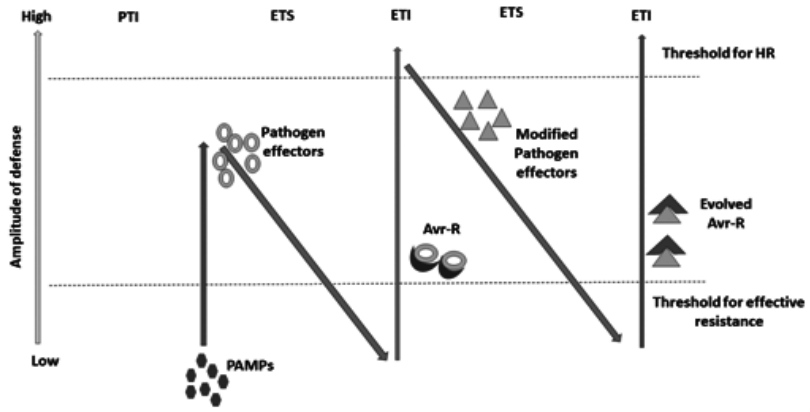

Figure 1. Zigzag Model of Plant immune system adapted from the model given by Jones and Dangal, 2006

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During Phase 1, main interacting factor PAMPs (Pathogen-associated molecular patterns) or MAMPS (Microbe-associated molecular patterns) recognized by specific Pattern recognition receptors (PRR) present on the surface of the plant or host. After successful recognition of PAMP/MAMP with their respective PRR leads to becomes weak but broader defense reaction that is termed as PAMP-triggered Immunity or **PTI**. This type of interaction is also seen in the case of nonpathogenic microbes that triggers the immunity inside plant from here story of romance started between microbes and plant like in the story of Troy. PAMP-triggered Immunity restricts the colonization of pathogens, for example, it induces a cell-wall-associated immune response against bacterial blight in pomegranate via callose deposition on cell-wall (Kumar and Mondal, 2013). After PTI, the second phase started where the pathogen uses the same strategy asthe Trojan horse that breach the strong wall of defense by entering inside the city silently without raising alarm. Here in case of bacterial pathogens uses Type3 secretion system to mask the alarm raised by MAP Kinase Pathway as reported in case of *Xanthomonasaxonopodis*pv. *punicae* (**Xop)** effectors uses the same strategy and reduce the ROS production *via* accumulation on plasma membrane (Kumar *et al.*, 2016). Hence,its resulting in susceptibility known as Effector-Triggered Susceptibility (**ETS**).

In Phase 3, over again immunity system of the plant is on alert and uses the polymorphic NB (nucleotide-binding) and Leucine-rich repeat (LRR) domain similar to the animal system, these proteins recognize their respective cognate effectors and produce strong Effector Triggered Immunity (**ETI**). This recognition sometimes may be directly or indirectly with the effectors molecules. These effectors are virulence factors of pathogen due to selection pressure or mutation modify its structural organization so that their specific R-gene (NB-LRR proteins) cannot identify or recognize their respective effectors, this result in effector triggered susceptibility during Phase 4. The same rivalry is going on sometimes pathogens win and sometimes host. Consequently, the host develops a new NB-LRR protein that again recognizes the new mutated effector molecules. Thus, this theory is well worked for Biotrophs or Hemi-biotrophs but not necrotrophsas their mode of action is different, which use toxins (Jones and Dangle, 2006).

PTI-ETI crosstalk :

PTI and ETI activate a specific type of receptors present {i.e. PRRs and NLRs, (Nucleotide oligomerization domain (NOD)-like receptors (NLRs) respectively}, however both types of immunity intersect during signalling cascades. Both types of Immunity intersect and produce several overlapping products such that mitogen-activated protein kinase (MAPK) cascades, calcium flux, reactive oxygen species burst, reprogramming of transcriptional activators and signalling of phytohormones (Tsuda and Katagiri, 2010; Thulasi and Zhang, 2018; Peng *et al.,* 2018). From experimental findings from the Arabidopsis interaction module it is evident that several plant component plays dual roles in ETI and PTI and converse both pathways; for example, two Arabidopsis receptor-like kinases, ANXUR1 (ANX1) and ANX2

interact with BAK1 and BIK1 and similarly interact with RPS2 that promotes RPS2 degradation, therefore negatively regulate both PTI and ETI (Mang *et al.*, 2017). In a similar wayin rice OsRac1 interacts with both the PRR co-receptor OsCERK1 and NLR Pit forming different complexes and PTI and ETI signals were positively transduced (Akamatsu *et al.,* 2020). In Arabidopsis, both PTI and ETI were negatively regulated via a gene silencing mechanism that post-transcriptionally modifies mRNAs encoding CNL proteins (Bocaara *et al.*, 2014; Jiang *et al.,* 2020). But the detail of how these components are cross-regulated by PRRs and NLRs are still unknown. Major key components of PTI pathway, such as BAK1 and MPK4, are guarded by NLRs suggesting cross-talk between PTI and ETI in different contexts (Wu *et al.*, 2020; Zhang *et al.*, 2012, Takagi *et al.*, 2019).

Gene for gene hypothesis (H.H. Flor, 1946) :

In case of a plant cultivar that expresses a specific resistance gene known as the R gene gives an incompatible response to a pathogenic strain after interacting with its cognatea virulence (Avr) gene from Pathogen. Where, either of these combinations missing produces a susceptible or compatible response in the particular cultivar that results in disease occurrence (Fig 2). In this context, *Pseudomonas syring aepvto mato* DC 3000 effect or AvrPtoB interact with Pto which trigger resistance via Prf resistance gene and this mechanism is Known as guard mechanism of interaction.

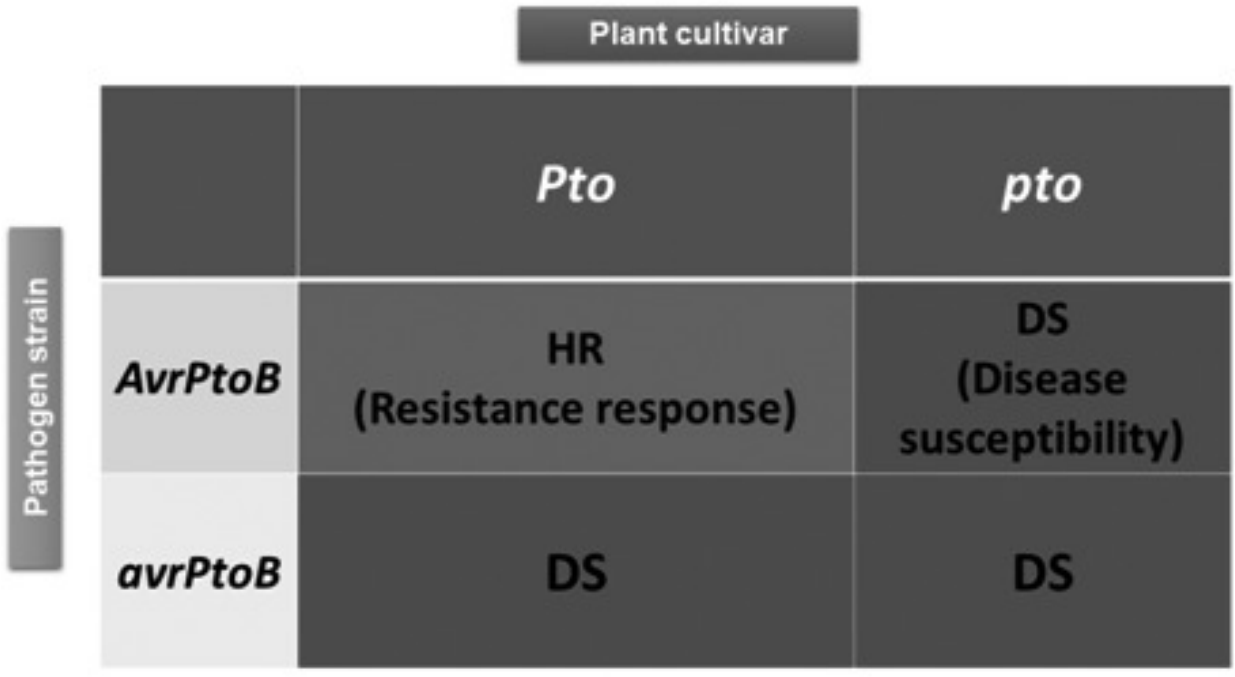

Fig 2. : Gene for Gene Hypothesis

After 16 years of the Zigzag model established, this is still the most acclaimed model for describing plant immunity with some limitations as described by Pritchard and Birch (2014) are as follows :

1. This model doesn't consider Damage-associated molecular patterns (DAMPs) molecules (Boller and Felix, 2009).
2. ETI may trigger plant cell death, which is a favourable condition for necrotrophs (Dickman, and de Figueiredo, 2013).
3. Environmental factors were ignored like drought or flooding, temperature effect, and photoperiods these factors affect the metabolism of pathogens and host. Thus, the same plant reacts differently to these factors. Overall abiotic stress and its correlation is not considered in the Zigzag model.

Conclusion :

- Zigzag model opened the avenue for molecular host-pathogen interaction that is still valid in the case of several classical plant pathogens.
- This system works well in both fungal and bacterial plant pathogens.

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