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Effector Triggered Immunity in Plants

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SUMMARY

Plants have several modes of action to defend the attack by the pathogens. With the evolution in plants, the defense response against pathogens also evolves and so do the pathogen and its infection strategies. The process of innate immunity in plants is necessary for survival. Pattern Triggered Immunity and Effector Triggered Immunity are important plant pathogen interactions which decide the resistance and susceptibility for a particular disease. In case when the first layer of the immune response that is Pattern Triggered Immunity is no longer effective as a result of pathogenic effectors, effector-triggered immunity (ETI) comes into action and often provides resistance.

INTRODUCTION

Plants, in nature are resistant to most pathogens, but some pathogenic microbes are capable of causing severe diseases. Plant cell wall and pre-produced metabolites are primary barriers against pathogenic invasion. To successfully respond to and defend against pathogenic microbes, plants developed multilayered protective and surveillance networks. The first layer of the plant immune system is pattern-triggered immunity (PTI), which is activated by pathogen associated molecular patterns (PAMPs), the conserved molecular structures of pathogens such as fungal chitin or bacterial flagellin, or damage-associated molecular patterns, which are molecules resulting from plant–pathogen interactions such as peptides and oligosaccharides. These inducers can be recognized by pattern recognition receptors (PRRs), plasma membrane-localized plant immune receptors, which are mainly found in the forms of receptor-like protein kinases and receptor-like proteins. Activation of these receptors provokes an array of plant defense responses to halt pathogen spread and colonization.

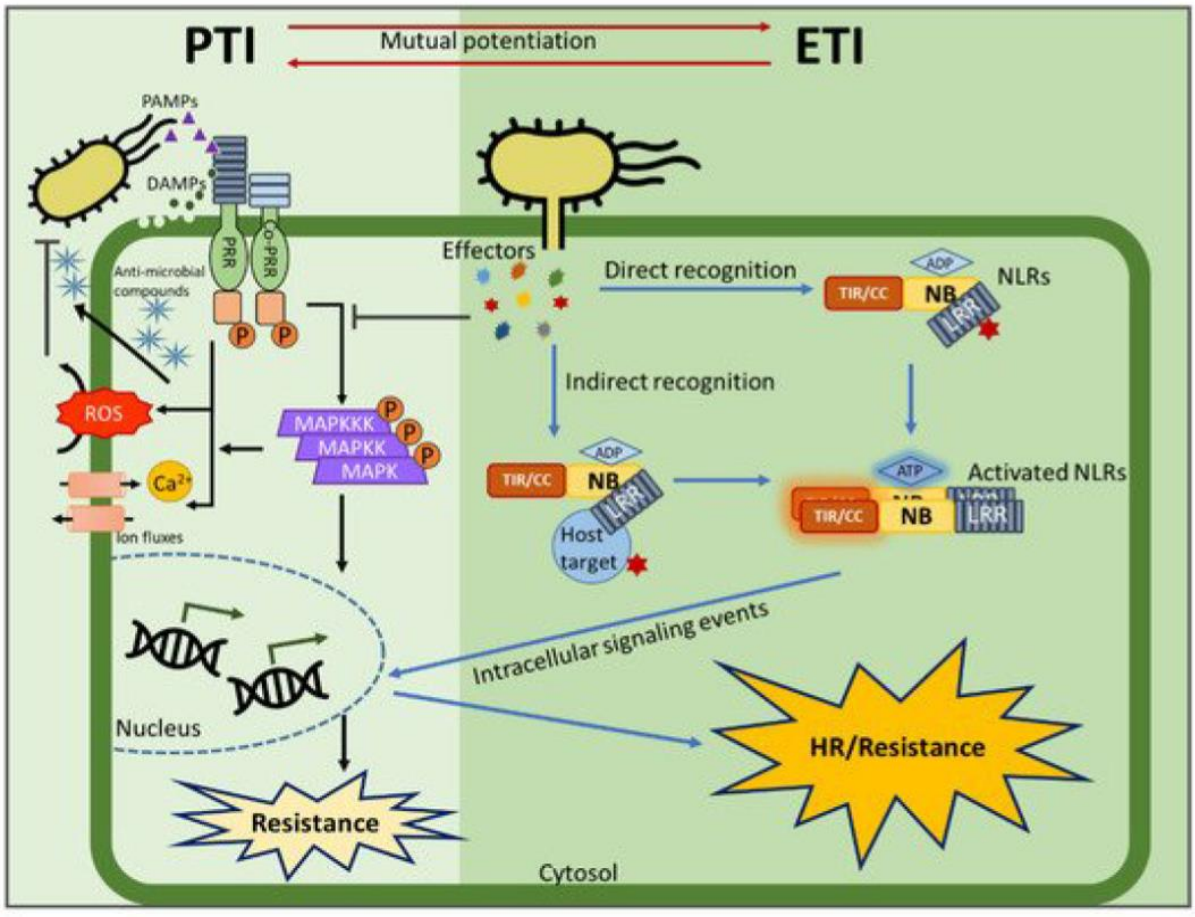

Figure: Diagrammatic representation of PTI and ETI

Effector Triggered Immunity

PTI activates multiple signalling pathways in the host cells. One of the rapid responses is an influx of extracellular Ca into the cytosol, followed by the activation of mitogen-activated protein kinases, reactive oxygen species (ROS) signaling, and other signalling molecules, such as reactive nitrogen species, lipids, callose, salicylic acid, n-hydroxypipecolic acid, jasmonic acid, ethylene, and cytokinin. The role of Effector triggered immunity comes into action when many pathogens deploy a variety of effector proteins to defeat PTI responses by plants. When these effectors are recognized by specialized receptors in the plant called resistance (R) proteins, the second layer of plant immune responses is activated, which is effector-triggered immunity (ETI). Host plants employ nucleotide-binding (NB) and leucine-rich repeat (LRR) domains, known as NLRs to detect effectors rapidly during pathogen invasion.

NLR and its function

Plants are known to bear some genes to provide protection towards one or more genotypes of a pathogen, known as resistance (R)-genes. R-genes most commonly encode NLRs, and resistance occurs when the NLR-protein detects an effector, either directly or indirectly, and subsequently triggers efficient ETI. As per the structural and functional analysis studies of R proteins, there are two conserved features in R protein. The structure of other NLR domains depends on whether a Toll-interleukin 1-like receptor (TIR) or a coiled-coil (CC) is attached at the N terminus. CNLs are found in dicot and monocot plant lineages, whereas TNLs are restricted to dicots. Host plants employ a diverse family of NLRs to detect effectors rapidly during pathogen invasion. NLRs selectively recognize the effectors, either directly or indirectly, and such recognition often leads to a hypersensitive response, a form of rapid localized programmed cell death. The immune responses elicited by PRRs and NLRs are similar, although the duration and amplitude of ETI responses are often vastly larger than those of PTI responses.

Recognition of pathogen effectors

Direct NLR-Effector Interactions

Gene-for-gene model, given by H.H. Flor's by genetic studies on flax rust disease resistance states that complementary pairs of genes in a plant host and pathogen determine disease resistance specificity. This theory was used as a base for exploring modes of receptor effectors recognition. Molecular interpretation of the gene-for-gene model, in terms of NLR-effector recognition, is that specific interaction between the receptor and its recognized (cognate) effector protein triggers resistance. Yeast two-hybrid and in vitro interaction assays of several NLR-effector combinations indeed support direct NLR-effector interaction as underlying resistance specificity.

Indirect NLR-Effector Interaction

NLR indirect sensing of the actions of diverse pathogen effectors converging on a limited set of host proteins might also increase the NLR recognition space to help keep pace with rapid pathogen evolution. One indirect recognition model, the receptor is activated by a pathogen effector modifying a host factor that is bound to and monitored (or guarded) by the NLR. Plant decoy strategy in which the monitored host factor has no measurable resistance function but serves as a bait to trap pathogen effectors that target structurally related basal defense components, thereby triggering ETI, also depicts an indirect mode of interaction.

Advantages of ETI over PTI

Both ETI and PTI plays an important role in plant defense against pathogens but since both can have some pros and cons, the pros with ETI signaling is that it is more robust against genetic mutations than PTI and is also resilient against pathogen effector perturbations Because genetically redundant defense components in a single pathway are unlikely to offer much of a barrier to effectors, ETI robustness might be achieved at the level of the resistance network, buffering against perturbations through compensation between different pathways. Although the immune pathway framework is shared in PTI and ETI, the activation kinetics for Reactive Oxygen Species production, Ca2+ spikes, and MAPK signaling are much more prolonged in ETI compared with PTI.

Recent findings suggest that quantitative differences in the strength and duration of these pathways produce qualitatively different resistance responses and network properties. For example, prolonged MAPK activation in Arabidopsis ETI regulates the expression of specific genes. During PTI, regulation of a subset of genes is dependent on SA when MAPK activation is transient. During ETI, when MAPK activation is extended, the same genes become less dependent on SA, and defects in SA signaling are compensated for by prolonged MAPK activation. The, quantitative changes in activation kinetics can lead to qualitatively different defenses. This leads to a model in which sustained activation of certain immunity pathways contributes to the strength and robustness of ETI.

CONCLUSION

Effector triggered immunity is an essential part of plant defense mechanism and studies of several novel effector proteins which are responsible for inducing disease resistance in plants can be greatly utilised to reduce the disease incidence and prevent the crop losses. Though the exact mechanism involved in ETI is still under several folds, efforts can be made in the direction of revealing the main mechanism.

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