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Original Article

Layered Defence in Plants: How Surface and Intracellular Immune Receptors Cooperate Against Microbial Pathogens

Aman Kumar Sah*, Tonmoy Ghosh

Birjhora Mahavidyalaya, Assam.

ABSTRACT

Plants cannot flee from danger, so every cell must be able to detect and answer microbial attack on its own. For two decades the field has described this ability as a two-tier system: receptors on the cell surface that detect conserved microbial molecules, triggering pattern-triggered immunity (PTI), and receptors inside the cell that detect pathogen-delivered effector proteins, triggering effector-triggered immunity (ETI). Recent work has shown that these tiers are not independent. They share signalling components, strengthen one another, and together produce a defence that neither could achieve alone. This review summarises how plants perceive pathogens, how early signals such as calcium influx and reactive oxygen species are generated, how PTI and ETI are now understood to potentiate each other, and where environmental change and crop improvement enter the picture. We close with open questions and priorities for future research.

Keywords: plant immunity, pattern-triggered immunity, effector-triggered immunity, pattern recognition receptors, NLR, reactive oxygen species, plant-pathogen interaction

INTRODUCTION

A plant in a field is exposed to bacteria, fungi, oomycetes and viruses throughout its life, yet disease is the exception rather than the rule. This is because plants carry a sophisticated innate immune system, and because most microbes are simply unable to overcome it. Lacking mobile defender cells or an adaptive antibody system, plants depend on immune capacity built into each cell, supplemented by signals that travel from infected tissue to distant parts of the plant.

The conceptual framework that organised the field was laid out by Jones and Dangl, who described plant immunity as a sequence of recognition events with an evolutionary arms race between host and pathogen at its core (Jones and Dangl, 2006). In that picture, surface receptors detect widely shared microbial signatures and give a baseline level of resistance. Successful pathogens respond by delivering effector proteins into host cells to disable this first layer. Plants, in turn, have evolved intracellular receptors that detect those effectors or their effects, mounting a stronger second layer of defence. Reviews of the receptor families involved have described how some proteins once classified purely as resistance genes were later recognised as surface sensors, which has blurred the neat boundary between the two tiers (Ortiz-Morea et al., 2022; MPMI review, 2024).

The aim of this article is to bring together current understanding of the two tiers, emphasising the evidence that they operate as one integrated network, and to consider what this means for managing plant disease.

2. Perception at the Cell Surface: Pattern-Triggered Immunity

2.1 Pattern recognition receptors and their ligands

The first layer of immunity begins at the plasma membrane. Pattern recognition receptors (PRRs) are of two broad structural kinds: receptor-like kinases, which carry an intracellular kinase domain, and receptor-like proteins, which lack one and rely on partner kinases (Yu et al., 2024). They bind microbe-associated molecular patterns, which are conserved molecules that microbes cannot easily discard, such as components of bacterial flagellin, fungal chitin, and cell-wall fragments. They also bind damage-associated patterns, which are endogenous molecules released when plant tissue is wounded or broken down during infection (Boller and Felix, 2009).

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Because the ligands are essential for microbial survival, PRR-based recognition tends to be durable and applies to many pathogen species, but it gives only a modest level of protection by itself.

2.2 Early signalling events

Binding of a ligand usually induces association between the PRR and a co-receptor kinase, followed by a burst of phosphorylation. Among the earliest outputs are an influx of calcium ions into the cytoplasm, activation of mitogen-activated protein kinase cascades, and production of reactive oxygen species at the cell surface by NADPH oxidases (Yu et al., 2024). These signals lead to transcriptional reprogramming, reinforcement of the cell wall through callose deposition, closure of stomata that would otherwise admit bacteria, and synthesis of antimicrobial compounds. Plant hormones, especially salicylic acid, jasmonic acid and ethylene, shape the balance and duration of these responses and help determine whether the plant favours defence against biotrophic or necrotrophic pathogens.

3. Pathogen Counter-Measures: Effectors

Successful pathogens evade or dismantle PTI. Bacteria deliver effector proteins directly into host cells through specialised secretion systems, while fungi and oomycetes release effectors from feeding structures that contact host cells. Effectors act on many targets: they can degrade or modify PRR complexes, block MAP kinase signalling, interfere with hormone pathways, or reprogram host gene expression. This ability to suppress the first layer explains why many pathogens can cause disease on a compatible host, and it provides the selective pressure that drove evolution of the second layer of defence.

Pathogens can also change the host's susceptibility to other microbes. An infection that suppresses immunity can open the door to secondary infection, which makes co-infection an important problem in natural and agricultural settings (Plants, 2024).

4. Intracellular Recognition: Effector-Triggered Immunity

4.1 NLR receptors

The main intracellular sensors are nucleotide-binding, leucine-rich repeat receptors, usually abbreviated NLRs. These proteins detect effectors either directly or indirectly, by monitoring host proteins that effectors modify. NLRs are typically grouped by their N-terminal domain, and many act in networks in which a "sensor" NLR detects the effector and a "helper" NLR carries out the downstream signal (Yu et al., 2024).

4.2 The resistosome

Structural studies gave a striking picture of how an activated NLR works. Biochemical and cryo-electron microscopy work on the Arabidopsis protein ZAR1 showed that, after recognising effector activity, NLR proteins assemble into a wheel-shaped oligomer, termed a resistosome (Wang et al., 2019). Later studies indicated that certain resistosomes can function as calcium-permeable channels at the plasma membrane, linking effector detection directly to the calcium signal that is also central to PTI. This finding illustrates why the two tiers cannot be treated as isolated: they converge on the same early signals.

4.3 The hypersensitive response

ETI often culminates in the hypersensitive response, a localised programmed cell death that deprives biotrophic pathogens of living tissue and limits their spread. The response is accompanied by transcriptional changes, a strong oxidative burst and often by the establishment of systemic resistance in tissues away from the infection site.

5. One Network, Not Two Branches

For many years, PTI and ETI were presented as separate pathways that differed in strength and timing. It was difficult, however, to study ETI in the absence of PTI, because pathogens usually activate both. Two independent studies overcame this problem in Arabidopsis by activating one tier while holding the other silent. Their conclusion was that the tiers strengthen each other: intracellular receptors raise the abundance of key surface-signalling proteins, and surface receptors strongly enhance the hypersensitive response that intracellular receptors control (Ngou et al., 2021; Yuan et al., 2021). Neither tier, activated alone, provided strong resistance to the bacterial pathogen *Pseudomonas syringae*, but together they did.

This discovery also helps explain how pathogens can suppress PTI but still fail: effector-triggered activation of NLRs can restore the signalling components

that effectors have depleted. It is consistent with reviews that now describe PTI and ETI as sharing signalling components and reciprocally boosting each other (Yu et al., 2024).

The practical consequence is that the old binary classification, although useful for teaching, can hide the integrated nature of defence. Future work needs to ask how the network is balanced, how autoimmunity is avoided when receptors are over-active, and how pathogen effectors target connections between tiers rather than single receptors.

6. Other Layers of Defence

PTI and ETI are not the only defences. RNA silencing is a major antiviral system in plants: viral double-stranded RNA is cut into small interfering RNAs that guide degradation of viral genomes, and many viruses encode suppressors of silencing in response. Autophagy, the controlled recycling of cell components, also contributes to defence and is itself manipulated by some pathogens (Plants, 2024). Together with systemic acquired resistance and priming, which prepare tissues away from the infection site, these processes broaden the immune toolkit available to the plant.

7. Environment and Disease

Immunity does not operate in isolation from environmental conditions. Temperature, humidity, drought and elevated carbon dioxide can alter both plant defence and pathogen growth, which means that disease outcomes depend on the interaction of host, pathogen and environment. Drought stress, for example, changes plant-pathogen interactions and disease development (Plants, 2024). For countries with variable climates and large agricultural sectors, such as India, understanding these interactions is a practical priority, since changing weather patterns may alter which pathogens threaten which crops.

8. Applications for Crop Protection

Knowledge of immune receptors has opened several approaches to resistance breeding. First, NLR genes from wild relatives can be introduced into cultivated varieties by conventional breeding or transgenic methods. Second, PRR genes from one plant family can be transferred to another so that crops gain the ability to recognise new pathogen patterns. Third, understanding of effector targets makes it possible to edit those targets so effectors can no longer act, or to engineer receptors that recognise new effectors. Because the immune network integrates signals from both tiers, combining receptors for both layers may provide stronger and more durable resistance than relying on one receptor type. A recurring concern is that single-gene resistance is often overcome as pathogen populations evolve, which argues for stacking several resistance genes and for monitoring pathogen populations.

9. Open Questions

Several important questions remain unresolved:

- How general is the mutual potentiation of PTI and ETI across plant species and pathogen types, including fungi and viruses?
- How do plants limit the cost of strong immunity to growth and yield, and how can this trade-off be reduced in crops?
- Which pathogen effectors target the connections between PTI and ETI, and can these be exploited for engineering resistance?
- How will changing climate conditions alter the balance of the immune network?

CONCLUSIONS

The plant immune system is best understood as an integrated network in which surface and intracellular receptors share signals and reinforce each other, rather than as two independent layers. This view has changed how researchers interpret resistance and offers new routes to improving crop protection. Progress will depend on combining molecular studies in model species with testing in crops under realistic field and climate conditions.

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