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# Molecular Basis of Crop Disease Resistance

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Plant diseases causes huge crop losses caused by pathogenic fungi, bacteria, nematodes, oomycetes, and viruses. Hence improvement of disease resistance in crops has great potential to increase productivity by preventing the huge losses. Plant disease resistance is typically divided into complete resistance (qualitative resistance) and partial resistance (quantitative resistance).

Complete resistance is usually controlled by resistance genes (R), which typically encode surface immune receptors (such as receptor-like kinases [RLKs]) or intracellular immune receptors (such as nucleotide-binding leucine-rich repeat proteins [NLRs]), that can detect pathogenic molecules *i.e.* effectors/avirulence (*Avr*) proteins. However, most R-mediated resistance belong to race-specific "gene-for-gene" resistance (Flor, 1971), which can easily be broken down in the field because pathogens can evolve to evade host recognition by mutating the *Avr* gene. Partial resistance is a durable and broad-spectrum resistance (BSR) against various pathogen races, controlled by quantitative resistance loci (QTL). However, it is difficult to use single QTLs in crop breeding because of their minor effects.

Plant immune system has been established along with the identification and characterization of genes that encode immune receptors, pathogen-associated molecular patterns (PAMPs), pathogen *Avr* proteins/effectors, and key signaling components of plant immune responses. Plant immune system includes two-tiered plant immune machinery: PAMP-triggered immunity (PTI) and effector-triggered immunity (ETI).

The widely recognized zigzag evolutionary model of plant innate immunity recapitulates the stepwise co-evolution of the host and microbes (Jones and Dangl, 2006). PTI is a basic defense response activated by plasma member-anchored pattern recognition receptors (PRRs), which detect conserved PAMPs, such as flagellin, elongation factor Tu (EF-Tu), and chitin. PTI tends to be effective in preventing infection by a vast majority of microbes, and most PTI-related pathways share highly similar signaling modules. Therefore, the simultaneous activation of multiple PRR pathways is likely to increase the robustness of overall plant defense against pathogen infection.

Pathogens secrete effectors into the host cell to suppress host PTI, which leads to effector-triggered susceptibility (ETS). To counter ETS, plants have evolved numerous intracellular immune receptors. These immune receptors directly or indirectly recognize microbial effectors, thereby trigger a response called ETI, which in turn typically induces the local hypersensitive response (HR) cell death to prevent pathogen growth.

Most intracellular receptors are NLRs, which are generally subdivided into TIR-NLRs (TNLs) with an N-terminal Toll-like/IL-1 receptor (TIR) domain, CC-NLRs (CNLs) with an

N-terminal coiled-coil (CC) domain, and CCRNLRs with an N-terminal RPW8-like CC domain NLR (Baggs *et al.*, 2017). Plant genomes encode hundreds of such NLR receptors, whose roles in immunity have functionally diversified during the co-evolution of plants and microbes.

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