

## THE MOLECULAR BASIS OF PLANT IMMUNITY AND SUSCEPTIBILITY

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Both plants and animals possess innate defense mechanisms to resist microbial infection. Although innate immune systems from both lineages share conceptual and mechanistic features, they are likely the result of convergent evolution. Efficient plant disease resistance is based on two evolutionarily linked forms of innate immunity. The primary plant immune response is referred to as PAMP-triggered immunity (PTI) and has evolved to recognize invariant structures of microbial surfaces termed pathogen or microbe-associated molecular patterns (PAMPs/MAMPs). Subversion of PTI by microbial effectors is believed to be one of the key strategies of successful pathogens in order to grow and multiply in host plants. In the co-evolution of host-microbe interactions individual plant cultivars have acquired resistance (R) proteins that guard microbial effector-mediated perturbations of host cell functions and thereby trigger plant immune responses. This type of plant defense is referred to as effector-triggered immunity (ETI) and is synonymous to pathogen race/host plant cultivar-specific plant disease resistance. This talk will feature our current knowledge on the molecular basis of plant immunity.

## MECHANISMS OF RHIZOBACTERIA-MEDIATED INDUCED SYSTEMIC RESISTANCE

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Non-pathogenic soilborne microorganisms can promote plant growth, as well as suppress diseases through microbial antagonism or induction of systemic resistance (ISR) in the plant, but it is not clear in how far both mechanisms are connected. Induced resistance is manifested as a reduction of the number of diseased plants or in disease severity upon subsequent infection by a pathogen. Such reduced disease susceptibility can be local or systemic, results from developmental or environmental factors and depends on multiple mechanisms. The spectrum of diseases to which PGPR-elicited ISR confers enhanced resistance overlaps partly with that of pathogen-induced systemic acquired resistance (SAR). Both ISR and SAR represent a state of enhanced basal resistance of the plant that depends on the signaling compounds jasmonic acid and salicylic acid, respectively, and pathogens are differentially sensitive to the resistances activated by each of these signaling pathways. Root-colonizing *Pseudomonas* were shown to alter plant gene expression in roots and leaves to different extents, indicative of recognition of one or more bacterial determinants by specific plant receptors. Conversely, plants can alter root exudation and secrete compounds that interfere with quorum sensing regulation in bacteria. Such two-way signaling resembles the interaction between root-nodulating Rhizobia and mycorrhizal fungi within legume species. Although ISR-eliciting rhizobacteria induce typical early defense-related responses in cell suspensions, in plants they do not necessarily activate defense-related gene expression. Instead, they appear to act through priming of effective resistance mechanisms, as reflected by earlier and stronger defense reactions once infection occurs.

