S9-3

# *Pseudomonas* Type III Effector AvrPtoB Activates Plant Immunity with Pto Kinase and Induces Plant Disease Susceptibility by Inhibition of Host Programmed Cell Death

Young Jin Kim
Graduate School of Biotechnology, Korea University

An important recent advance in the field of plant-microbe interactions has been the cloning of genes that confer resistance to specific viruses, bacteria, fungi or insects. Disease resistance (R) genes encode proteins with predicted structural motifs consistent with them having roles in signal recognition and transduction. Plant disease resistance is the result of an innate host defense mechanism, which relies on the ability of plant to recognize pathogen invasion and efficiently mount defense responses. In tomato, resistance to the pathogen *Pseudomonas syringae* pv. *tomato* is mediated by the specific recognition between the tomato serine/threonine kinase Pto and bacterial protein AvrPto or AvrPtoB. This recognition event initiates signaling events that lead to defense responses including an oxidative burst, the hypersensitive response (HR), and expression of pathogenesis-related genes.

Resistance to bacteria speck disease in tomato is mediated by the tomato Pto kinase which recognizes strains of *Pseudomonas syringae* pv. *tomato* that express the effector protein AvrPto. AvrPto likely enters the plant cell via the *Pseudomonas* type III secretion system where it physically interacts with Pto kinase and activates signaling pathways leading to variety of defense responses. We have recently identified another *Pseudomonas* effector protein, AvrPtoB, that also interacts specifically with Pto. AvrPtoB and AvrPto are similar in several discrete regions which might define their contact points with Pto. Both proteins also appear to confer virulence of *Pseudomonas* when the Pto kinase is not present in plant. The AvrPtoB type III effector protein is conserved among diverse plant pathogens suggesting it plays an important but hitherto unknown role in pathogenesis. Here we report that *Pseudomonas* AvrPtoB suppresses plant immunity and does so by inhibiting defensive programmed cell death (PCD). AvrPtoB acts inside the plant cell and inhibits PCD initiated by disease resistance proteins and the pro-apoptotic mouse protein Bax. Using deletion mutants, we identified distinct AvrPtoB domains that are necessary for host recognition and PCD inhibition. We also discovered a suppressed host resistance activity that triggers AvrPtoB-dependent immunity only in the absence of AvrPtoB PCD inhibitory activity. These findings suggest a new model, with mechanistic and structural details, of how a type III effector protein promotes disease.

**Keywords** : Pto kinase; AvrPto; AvrPtoB; disease resistance; signal transduction; recognition specificity; PR genes