Molecular basis of resistance to bacterial speck disease in tomato

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Pseudomonas syringae pv. *tomato* causes bacterial speck disease of tomato (*Solanum lycopersicum*) which can negatively impact both yield and quality of the crop. Tomato plants have two defense systems that act to prevent infection by this pathogen. The first system is activated when host 'pattern recognition receptors' (PRRs) detect certain 'pathogen-associated molecular patterns' (PAMPs) expressed by the bacterium. PAMPs are typically highly-conserved molecules that are indispensable for bacterial survival and include flagellin, lipopolysacharide, and peptodoglycan. PAMP-triggered immunity (PTI) can be effective, but successful pathogens like *P. s.* pv. *tomato* have evolved a mechanism to undermine this initial immune response. This mechanism is the type III secretion system which allows the pathogen to deliver ~30 virulence proteins ('effectors') into the plant cell. In the plant cell, the effectors disrupt a variety of host processes to compromise immunity allowing the bacteria to multiply and eventually cause symptoms of bacterial speck. Two of the best-studied effectors are AvrPto and AvrPtoB. These sequence-unrelated proteins act early in the infection process by binding to the kinase domains of PRRs preventing them from activating PTI.

The second plant defense system, effector-triggered immunity (ETI), is activated when a plant resistance (R) protein detects the presence of a specific effector protein. ETI is a strong response that is associated with localized programmed cell death (the hypersensitive response). In tomato, varieties that are resistant to speck disease express the *R* gene *Pto* which encodes a protein kinase that recognizes and binds either AvrPto or AvrPtoB, thereby activating ETI. Recently, the crystal structures of Pto in a complex with AvrPto or AvrPtoB have been solved shedding light on the underlying structural basis of this 'gene-for-gene' interaction. Another resistance protein, Fen, is closely related to Pto and also can prevent *P. s.* pv. *tomato* infection but it has been defeated by the acquisition of an enzymatic activity by the AvrPtoB protein. Both Pto and Fen act in concert with *Prf* a gene that encodes an NB-LRR protein. In the past 15 years, ~25 genes have been identified by loss-of-function studies as playing a role in Pto-mediated ETI (Oh and Martin, 2011).

We have recently identified wild relatives of tomato that recognize only AvrPto, only AvrPtoB, both effectors, or neither one. Our current work is focused on integrating knowledge of the Pto and AvrPto/AvrPtoB crystal structures with what we are learning about natural variation in *P. s.* pv. *tomato* recognition in the *Solanaceae* from characterization of wild tomato species. All plants appear to use fundamentally similar resistance mechanisms and therefore knowledge about the 'molecular arms race' between tomato and *P. s.* pv. *tomato* is probably relevant to many economically important plant species and to the control of diverse pathogens (bacteria, fungi, oomycetes, and viruses). *Supported by NIH R01GM078021, NSF-IOS 0841807, NSF-IOS 1025642, USDA-2010-65108-20503, USDA-BARD IS4159-08C and the Triad Foundation.*

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