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IN BRIEF

The Tomato Pto Kinase Uses Shared and Unique Surfaces to Recognize Divergent Avirulence Proteins

The interaction between *Solanum lycopersicum* (tomato) and *Pseudomonas syringae* pv *tomato*, which causes bacterial speck disease, is a model of gene-for-gene disease resistance in plants (reviewed in Pedley and Martin, 2003). In *Pto* resistant tomato varieties, defense responses are elicited when the tomato Pto (for *P. s. tomato*) kinase recognizes either one of two *P. syringae* type III effector proteins, AvrPto or AvrPtoB. Previous work from Jijie Chai and colleagues on the crystal structure of the AvrPto-Pto complex showed that the interaction of Pto with AvrPto likely relieves negative regulation by Pto of another protein, Prf (for Pto resistance and fenthion sensitivity) (Xing et al., 2007). Now, **Dong et al. (pages 1846–1859)** have solved the crystal structure of the AvrPtoB-Pto complex and found further support for the idea that interaction with the *P. syringae* effectors abrogates Pto inhibition of Prf-mediated defense responses.

The authors began by demonstrating that AvrPtoB and Pto directly interact in vitro and then solved the structure of a fragment of AvrPtoB that is responsible for interaction with Pto. They found that its fold is strikingly different from that of AvrPto. Intriguingly, AvrPto and AvrPtoB are unrelated in primary amino acid sequence and in structure, even though they have similar affinities for Pto and cause the same downstream effects. Dong et al. obtained the structure of the AvrPtoB-Pto complex, which shows two contact surfaces (see figure). One of these surfaces is unique, while the other is similar to one found in the AvrPto-Pto complex and may interfere with Pto's substrate binding ability. When AvrPtoB residues from those interfaces were mutagenized, interaction with Pto was disrupted and AvrPtoB avirulence activity in planta was lost.

As predicted, when Dong et al. mutated Pto residues from the shared interface, Pto complex formation with both AvrPto and AvrPtoB was disrupted, while mutations in the other interfaces affected interaction with one or the other. The authors went on to test the activity of these proteins in a transient expression assay. When Pto and AvrPtoB are coexpressed in *Nicotiana benthamiana*, they induce cell death. The authors identified several Pto mutations that did not interact with AvrPtoB and also failed to cause cell death. In addition, they found a Pto mutation in the unique AvrPtoB interface that did not interact with AvrPtoB but that did induce cell death, even in the absence of AvrPtoB. This constitutive gain of function was Prf dependent and did not occur in a *Prf*-silenced tobacco line. Thus, it seems likely that the mutation interferes with Pto's negative regulation of Prf just as AvrPtoB binding does. How Pto regulates Prf remains unclear, as does the precise mechanism of its disruption, but this work provides new insight into how one protein can recognize two sequence-divergent proteins to achieve the same downstream effect.

In another nice example of a structural approach to plant–pathogen interactions, **Aparna et al. (pages 1860–1873)** report on a novel conserved domain found in a virulence factor secreted by members of genus *Xanthomonas*.

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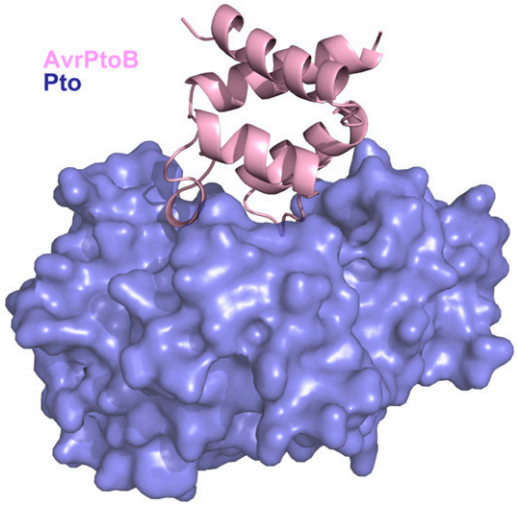

The structure of the AvrPtoB-Pto complex. $AvrPtoB_{121-205}$ and a surface representation of Pto are shown in pink and blue, respectively.

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