**IN BRIEF**

**Effector XopD Suppresses Tissue Degeneration in *Xanthomonas*-Infected Tomato Leaves**

Some plant pathogens deploy dozens of effector proteins through the type III secretion apparatus into host cells. These effectors manipulate host basal defenses and metabolism to the advantage of the pathogen, a process essential for pathogenesis (reviewed in Mudgett 2005; Grant et al., 2006). Effectors can also betray the presence of the pathogen, triggering robust plant defense responses that other proteins in the suite of effectors attempt to suppress at several key points. For example, several effectors diminish salicylic acid (SA) signaling by activating jasmonic acid signaling, which is antagonistic to the SA pathways. Pathogen type III effectors also affect host programmed cell death and ethylene signaling, suppress cell wall defenses, and perturb other defense signaling.

In the absence of pathogen recognition and effective host defense responses, plants are susceptible to pathogen colonization, which results in tissue degeneration, including chlorosis and necrotic cell death. Pathogen effectors are key to controlling the severity of host responses to infection. Kim et al. (pages 1915–1929) examined the type III effector XopD in pathogenesis of tomato by *Xanthomonas campestris* pathovar vesicatoria (Xcv). XopD is a Cys protease that can process the precursor of SUMO, the small ubiquitin-related modifier, or remove SUMO from other proteins. SUMO regulation is implicated in multiple responses, including repression of transcription. Consistent with this, XopD localizes to foci within the plant nucleus. The authors examined the role of XopD in Xcv pathogenesis by studying the phenotype of an Xcv strain carrying a deletion of XopD (Xcv ΔxopD). Tomato leaves infected with Xcv ΔxopD showed reduced pathogen growth, earlier onset of chlorosis and necrosis (see figure), and upregulation of senescence-associated genes. Genetic removal of SA from the host plant allowed Xcv ΔxopD to grow as well as wild-type Xcv, but did not change the premature onset of symptoms. Structure-function analysis showed that XopD nonspecific DNA binding activity, SUMO protease activity, and EAR motifs (associated with transcriptional repression) are essential to suppress symptom development late in infection. Consistent with XopD’s role in modulating senescence-associated gene expression, transient expression of XopD in *Nicotiana benthamiana* showed that XopD could repress induction from pathogenesis-related promoters by SA and methyl jasmonate.

In summary, Xcv appears to use the effector XopD to prolong the life of infected tissue, suppressing the onset of disease symptoms and thereby allowing for greater pathogen growth. The mechanism of SUMO protease, DNA binding, and EAR repressor activities in pathogenesis, and any connection with age-induced senescence, will be interesting to examine.

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