Friday, September 26, 10:30-11:00 a.m.

"Rewiring of host transcription promotes Xanthomonas euvesicatoria *growth and symptom development"*

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XopD is the only non-TAL (transcription activator-like) T3S effector that directly represses host transcription during *Xanthomonas euvesicatoria* (Xe) infection. XopD encodes a small ubiquitin-like modifier (SUMO) protease that suppresses ethylene (ET) and salicylic acid (SA) production to interfere with host immunity. XopD represses ET-stimulated transcription by cleaving SUMO from the tomato transcription factor (TF) ERF4. SUMO modification of ERF4 is required for its stabilization and transcription. XopD also stabilizes and sequesters the TF MYB30, impacting SA responses. These studies highlight that pathogen-dependent manipulation of host TFs is a key virulence strategy to dampen hormone-mediated immune signaling. How plants recognize XopD perturbation and activate defense is unknown. Given that XopD manipulates transcription complexes, we hypothesized that host transcription triggered by XopD, but not repressed by XopD, may define defense pathways effective for anti-Xe immunity. Indeed, we discovered a putative tomato basic helix-loop-helix (bHLH) TF that is significantly upregulated by XopD by 3 hours post-Xe infection. The gene was designated *DH1* for differentially regulated bHLH1. Notably, activation of *DH1* transcription requires XopD's SUMO protease activity. This suggests that XopD may target a TF-module that controls *DH1* transcription or that XopD virulence activity is guarded by a TF-module that activates *DH1* transcription. Interestingly, silencing *DH1* resulted in enhanced susceptibility and symptom development in tomato, and a significant loss of vegetative growth. These data suggest that DH1 function is required for both immunity and growth programs in tomato. In addition to DH1, we found 6 unrelated bHLHs (DH2 to DH7) whose transcription is regulated by Xe in a T3S effector-dependent (XopD-independent) manner. By contrast to DH1, DH4 is required to suppress anti-bacterial defense and symptom development. Taken together, our work suggests that Xe targets bHLH controlled transcription circuits to dynamically alter tomato growth and/or immunity to promote pathogenesis. Recent progress in studying the role of these tomato bHLHs in growth-defense trade-offs will be discussed.