Modulation of host defenses by the Type VI Secretion System (T6SS) of Agrobacterium tumefaciens

The soil-borne phytopathogen Agrobacterium tumefaciens induces crown gall tumors on a wide range of host species; this neoplastic growth is a consequence of stable transformation with bacterially derived DNA. In A. tumefaciens, as in several other bacteria species, the recently discovered type VI secretion system (T6SS) releases effectors that act as toxins in inter- and intra-bacterial competition. However, the contribution of the A. tumefaciens T6SS to virulence is not yet well understood. We discovered that inoculation of Arabidopsis thaliana stems with agrobacteria lacking the T6SS resulted in the formation of smaller tumors than infection with wild type bacteria. Ablation of the homologous secretion system in the closely related alpha-proteobacterium Rhizobium leguminosarum was previously shown to extend the bacterium’s host range (MPMI 16: 53; 2003). Collectively, these data have led us to hypothesize that the T6SS may dampen A. thaliana defenses, but in so doing, may inadvertently elicit host defenses as well. Using bacterial mutants deficient in one or more of the known T6SS effectors, we have begun to characterize the contributions of individual effectors to the attenuation and/or triggering of host responses. We have found that the T6SS impacts transient transformation as well as tumorigenesis. Other host responses that are altered include defense-related gene expression and resistance to subsequent infection by Pseudomonas syringae pv. maculicola. Investigations with mutant plant lines have revealed insights into particular host defense pathways, including the abscisic acid (ABA) hormone pathway, that are modulated by the T6SS. Our data to date implicate the receptor kinase Lyk3, a mediator of late ABA responses as well as Nod factor-induced repression of host immunity, in T6SS recognition. Finally, consistent with our observation that T6SS-deficient bacteria exhibit altered biofilm formation, we have uncovered differential effects of specific T6SS effectors on bacterial motility.