Dusit Athinuwat 2009: Specificity of Avirulence Genes of *Xanthomonas axonopodis* pv. *glycines* on Different Soybean Cultivars. Doctor of Philosophy (Plant Pathology), Major Field: Plant Pathology, Department of Plant Pathology. Thesis Advisor: Associate Professor Sutruedee Prathuangwong, Ph.D. 201 pages.

Three races of *Xanthomonas axonopodis* pv. *glycines* (*Xag*) strains were identified on pustule disease resistance (Williams82) and susceptibility (SJ4, Spencer, and PI 520733) soybean cultivars based on virulence phenotype. Race 1 induced hypersensitive response: HR within 48 h and Race 2 induced disease on all cultivars tested. Race 3 elicited HR on specific pustule-resistant cultivar (Williams82). In Race 3, strain KU-P-SW005, an *avrBs3* homolog, *avrXg1* new-named by this study was carried on plasmid DNA. An *avrXg1* conferred resistance expressed as HR on resistant cultivar Williams82. Mutations in two predicted functional domains, the 4th central repeat and an acidic activation domain, of *avrXg1* resulted in enhanced virulence and bacterial population on resistant and susceptible cultivars. Expression of *avrXg1* in Race 1, that was predicted to confer a non-specific HR, led to virulence on susceptible cultivars. This *avrXg1* was also expressed in Race 2 resulting in increased virulence and additive pathogen fitness on resistant and susceptible cultivars. Race 2 was shown to carry *avrBs3*-like genes but apparently not *avrXg1*. The results demonstrate multi-functions for *avrXg1* dependent on pathogen and plant genetic backgrounds.

The proteome of 15-day old soybean cultivar Williams82 inoculated with KU-P-SW005 in Race 3 expressed various defense related proteins including catalase, lipoxygenase-4, and phenylalanine ammonia-lyase. Williams82 also showed enhanced expression of PR-2, PR-4, PR-6, PR-10, and lipoxygenase for HR induction following inoculation with KU-P-SW005. This is the first study to examine the interaction between soybean and *Xag* based on a gene-for-gene relationship. It provides insight into breeding strategies for pustule resistance.

To elucidate the mechanism by which the *avrXg1* mutant was more efficient than the wildtype strain in virulent initiation, genes affected pathogenicity and virulence, including *flgC*, *flgK*, and *pilD* deletion were constructed. These gene mutations produced a dominant-negative effect on *Xag* virulence on soybean. Deletion of *flgC* resulted in complete loss of disease initiation, where *flgK* and *pilD* mutants displayed a significant reduction in disease development on soybean. This result particularly confirmed that swimming and twitching motility functioned by bacterial flagellum and pillus was required for full expression of disease and severity initiation. Moreover, a regulatory cascade initiated by *avrXg1* that its mutant generated stronger pustule severity than wildtype led to activation of the downstream *flgC*, *flgK*, and *pilD* genes via *hrp* cluster, was found in this study. The swarming ability of *Xag* therefore, seemingly depended on a functional *avrXg1* system that is a key contributor to pathogenicity and virulence in bacterial pustule pathogen. A new aspect of *avrXg1* identification is a significant advancement in studying *pilD* that found to mediate a twitching role for virulence and biofilm formation on soybean in this study, is the first report and lays the foundation for *Xag* motility during pathogenicity.

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