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Role of flagella, flagellin glycosyation and flagella-mediated chemotaxis in virulence of *Pseudomonas syringae* pv. *tabaci* 6605 (*Pta*6605)

Understanding the mechanisms by which phytopathogens express their virulence is crucial for controlling plant diseases. Using Gram-negative phytopathogen *Pseudomonas syringae* pv. *tabaci* 6605 (*Pta*6605) as a model pathogen, we revealed that flagella-defective mutant (Δ*fliC*) lost surface motility and reduced virulence. Furthermore, flagellin-glycosylation-defective mutant (Δ*fgt1*) remarkably reduced surface motility and virulence. These results indicate that flagella and flagellin glycosylation are important for surface motility and virulence. To examine whether motility with directionality is required in virulence we generated several mutants for chemotaxis. Although there are two sets of chemotaxis gene cluster (*cheⅠ* and *cheⅡ*), we found that *cheⅡ* is required for virulence in *Pta*6605. In general, compared with animal pathogenic bacteria, plant pathogenic bacteria have a large number of chemotactic receptor genes, *mcp*. *Pta*6605 also has 53 *mcp* genes and among them we revealed that one *mcp* encoding the chemoreceptor for gamma-aminobutyric acid and two *mcp* encoding the chemorecepotrs for amino acids are important for bacterial infection. Analysis of role of other Mcp proteins in plant infection is now under investigation.