

Deciphering the virulence-related signaling and regulatory system of the bacterial plant pathogen *Burkholderia glumae*

Jong Hyun Ham

Department of Plant Pathology and Crop Physiology
Louisiana State University Agricultural Center

Burkholderia glumae, the causal agent of bacterial panicle blight, is a major threat to rice production worldwide, including in the southeastern United States. The phytotoxin toxoflavin and its derivatives are a key virulence factor of *B. glumae*, and a quorum-sensing system encoded by the *luxI/luxR* homologs *tofl/tofR* had been known to play an essential role in the regulation of bacterial pathogenesis of this pathogen. However, our genetic analyses revealed that the regulatory mechanisms governing pathogenesis of *B. glumae* vary among different strains. Notably, certain virulent strains, such as 411gr-6, retain their ability to produce toxoflavin and cause disease even when the *tofl/tofR* quorum-sensing system (Tofl/TofR QS) is disrupted, suggesting the existence of an alternative, QS-independent pathway for pathogenesis. In contrast, deletion of *qsmR*, which encodes an LclR-family regulatory protein, abolished virulence across all tested strains, highlighting its essential regulatory function in pathogenicity of *B. glumae*. Further supporting this notion, we identified a naturally avirulent strain 257sh-1 and traced the cause of its lost virulence to a single amino acid substitution (T50K) in the coding sequence of *qsmR*. These findings position *qsmR* as a promising molecular target for managing bacterial panicle blight through suppressing the pathogenicity of *B. glumae*. Interestingly, the naturally avirulent strain 257sh-1 also exhibited strong biological control efficacy against both bacterial panicle blight and sheath blight, another major rice disease. This disease-suppressing activity of 257sh-1 appears to result from defense-priming in rice rather than its direct antagonistic activity against pathogens. To prevent potential reversion to virulence via mutation in *qsmR*, we generated a *qsmR*-deleted derivative of 257sh-1. This mutant retained comparable biocontrol efficacy to its parent strain, suggesting it may serve as a safer and effective biological agent.