Quorum Sensing and Survival of *Burkholderia glumae* in Response to Environmental Stimuli

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Burkholderia glumae causes bacterial rice grain rot and bacterial wilt in many field crops due to phytotoxin toxoflavin production. Molecular and genetic analyses showed that quorum sensing (QS) regulates toxoflavin biosynthetic genes and its transporter genes. QS of the bacterium regulates other phenotypes including swimming and swarming motilities, catalase production, the type II secretion system, and universal stress proteins (UspA). *N*-octanoyl-homoserine lactone (C8-HSL) and its cognate receptor TofR is a primary regulator. C8-HSL deficient mutant of *B. glumae* was non-motile and did not possess flagella at 37℃. We identified an IclR-type transcriptional regulator, called QsmR, affecting flagella formation by Tn*5rescue* mutagenesis. The gene *qsmR* belongs to a TofR/C8HSL regulon, which was determined by direct binding of TofR/C8HSL to the promoter region of *qsmR* and gene expression analysis. Nucleotide sequence analysis of one of the flagella gene clusters identified *flhDC* homologs whose expression is directly activated by QsmR. As if FlhDC is the master regulator of lateral flagella gene expression, FlhDC activated expression of genes involved in flagella biosynthesis, motor functions, and chemotaxis in *B. glumae*. Non-motile mutants except the QS-deficient mutant produced toxoflavin but lost pathogenicity in rice. Expression of *katG* and *katB* was regulated by QS not directly by TofR/C8-HSL but by QsmR whereas *ahpCF* expression was directly activated by TofR/C8-HSL. A gel mobility shift assay confirmed that QsmR directly activates *katG* expression. The *katG* mutant produced toxoflavin but exhibited less severe disease than the wild-type strain on rice panicles. Under visible light conditions and a photon flux density of 61.6 μmols^{-1}m^{-2}, the survival rate of the *katG* mutant was 10^5-fold lower than that of the wild-type strain. This suggests that KatG is a major catalase that protects bacterial cells from visible light, which probably results in less disease severity caused by the *katG* mutant. Among thirteen *uspA* homologs of *B. glumae*, expression of *uspA1* and *uspA2* was regulated by QsmR. The UspA1 and UspA2 mutants were very sensitive to temperature shift from 37°C to 44°C. This indicated that UspA1 and A2 play important roles for survival of the bacterium against heat shock.