## Suppression mechanism of rice immune responses by plant pathogenic bacteria *Acidovorax avenae*

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Microbial pathogens deliver effectors into plant cells to suppress plant immune responses and modulate host metabolism in order to support infection processes. To determine whether the Acidovorax avenae ricevirulent K1 strain suppress pathogen-associated molecular pattern (PAMP) -triggered immunity (PTI) induced by flagellin isolated from rice-avirulent N1141 strain, cultured rice cells were inoculated with K1 strain and then treated with the flagellin. The flagellin-triggered PTI, including H<sub>2</sub>O<sub>2</sub> generation, callose deposition, and expression of several immune-related genes were strongly suppressed in K1 pre-inoculated cultured rice cells in a Type III secretion system (T3SS) -dependent manner. By screening 4,562 transposon-tagged mutants based on their suppression ability, 156 transposon-tagged K1 mutants were identified as strain lacking the ability to suppress PTI induction. Mutant sequence analysis, comprehensive expression analysis using RNA-sequencing, and the prediction of secretion through T3SS showed that a protein named A. avenae K1 suppression factor 1 (AKSF1) suppresses flagellin-triggered PTI in rice. Translocation of AKSF1 protein into rice cells is dependent on T3SS during infection, an AKSF1-disruption mutant lost the ability to suppress PTI responses, and reintroduction of AKSF1 into AKSF1-disruption mutant complemented the suppression activity. When AKSF1 disruption mutant was inoculated into the host rice plant, reduction of the disease symptoms and suppression of the bacterial growth was observed. Taken together, our results demonstrate that AKSF1 is a novel effector-triggered susceptibility (ETS) effector that can suppress the PTI in host rice plant. AKSF1 localizes to the cytoplasm in rice cells where it interacts specifically with protein kinase Ser/Thr/Tyr (STY) 46. Since STY46 and MAPKKK were classified into the tyrosine kinase-like family, AKSF1 may blocks the flagellin recognition signaling through the inhibition of the MAPK cascade.