論文の内容の要旨

Regulatory Mechanism of Plant Innate Immunity

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Plants recognize pathogen-derived molecules known as pathogen-associated molecular patterns (PAMPs) through surface-localized pattern recognition receptors (PRRs), which leads to PAMP-triggered immunity (PTI). Plant pathogens, on the other hand, infect plants by using virulent effectors that target PRRs and PTI signaling components to suppress PTI. In PTI, production of reactive oxygen species (ROS) by the NADPH oxidase Respiratory burst oxidase homologue D (RBOHD) plays pivotal roles. ROS have direct antimicrobial properties, but also serve as signaling molecules to activate further immune outputs. ROS production has to be tightly controlled to avoid detrimental effects on host cells. Nonetheless, the regulatory mechanism of PAMP-inducible ROS production is still largely unclear. The purpose of my research is to understand the regulatory mechanism of PAMP-inducible ROS production and pathogen virulence at the molecular level. In addition, to utilize plant immune systems to reduce disease in the agricultural field, I aimed to clarify the mechanisms of a novel plant immunity inducer.

To understand the regulatory mechanism of PAMP-inducible ROS production, I identified and characterized a novel component of the PRR-RBOHD complex. RBOHD-EFR ASSOCIATED LEUCINE-RICH REPEAT RECEPTOR-LIKE PROTEIN KINASES 1 (REAL1) was identified by co-immunoprecipitation of RBOHD as well as Elongation Factor-Tu (EF-Tu) receptor (EFR), which is a PRR for peptides derived from bacterial EF-Tu (elf18). Analyses of *Arabidopsis* knockout mutants or overexpressers of *REAL1* revealed that REAL1 reduces PRR protein levels at the plasma membrane and negatively regulates PTI responses. Interestingly,

accumulating evidence showed that a bacterial type III secretion effector HopF2 $_{Pto}$, which encodes mono-ADP-ribosyltransferase, might directly interact with REAL1 when HopF2 $_{Pto}$ was expressed in plants. These results suggest the possibility that HopF2 $_{Pto}$ functions via REAL1 to reduce PRRs to inhibit PTI responses.

To utilize plant immune systems for disease control, I also analyzed the mechanisms of a novel plant immunity inducer, glutamate (Glu). I found that exogenous treatment of *Arabidopsis* with Glu enhances resistance against bacterial and fungal pathogens. Consistently, transcriptome analyses of *Arabidopsis* seedlings treated with Glu showed that Glu significantly activates the expression of genes induced by PAMPs at much later time points than PAMPs normally do. Moreover, activation of Glu-inducible genes does not require known components of the PAMP receptor complex, glutamate receptors, the salicylic acid-biosynthesis enzyme, or glutamate decarboxylase. Glu also enhances PAMP-inducible immune responses. These results show that Glu activates the PAMP-triggered immunity signaling pathway in a novel manner.