Public Abstract

First Name:Sang Hee

Middle Name:

Last Name:Kim

Adviser's First Name:Walter

Adviser's Last Name:Gassmann

Co-Adviser's First Name:

Co-Adviser's Last Name:

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Title:INNATE IMMUNITY IN ARABIDOPSIS: MOLECULAR MECHANISMS OF HOPA1 AND AVRRPS4 - SPECIFIC DISEASE RESISTANCE SIGNALING PATHWAYS

Plant immune responses are determined in part by specific interactions between a host resistance (R) gene and a pathogen avirulence (avr) gene. avr genes encode effectors for pathogen fitness, so this immune response is now named effector trigger immunity (ETI). ETI needs to be controlled both positively and negatively, because excessive ETI is detrimental to the host. hopA1 and avrRps4 from bacteria Pseudomonas syringae was identified as avr genes for Arabidopsis. Our research is aimed at understanding the function of R genes to their cognate avr genes. To understand the genetic basis for ETI, we used the mutants screen and map based cloning approaches. Using a gain of resistance screen, we cloned hopA1-specific RPS6 R gene which is a member of Toll/Interleukin-1 receptor (TIR) nucleotide-binding site (NBS) leucine-rich repeat (LRR) class R genes. Using a loss of resistance screen, we cloned SRFR1 (Suppressor of rps4-RLD 1), which reactivates avrRps4-triggered immunity and encodes a pioneer TPR (Tetratricopeptide Repeat) protein conserved between plants and animals. Mutation in the EDS1 (Enhanced Disease Susceptibility1) gene abolished resistance dependent on RPS4, RPS6 and SRFR1, suggesting EDS1 is a downstream key regulator of TIR-NBS-LRR R gene dependent ETI. Plant innate immunity controlled by plant resistance genes is highly effective and valuable in the war against pathogens. Our genetic resources from RPS6 and SRFR1, and also the knowledge from these researches can be utilized by other research groups who study plant-microbe interaction in plant sciences. In addition, information from cloning, characterization, and manipulation of resistance genes in the model plant Arabidopsis can be applied to crop plants for durable pathogen resistance which can reduce our reliance on antibiotic disease control and improve the agricultural safety and crop yield.