(05-1-58)

Isolation and characterization of *Arabidopsis ner* mutants impaired in AvrRpt2-mediated RPM1 elimination

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The TTSS effecter, AvrRpt2, targets and eliminates RIN4 that is not only a positive regulator for basal defense but also a negative regulator for RPS2-mediated resistance. Therefore, if *P. syringae* expressing avrRpt2 infects host plant lacking resistance gene RPS2, AvrRpt2-mediated elimination of RIN4 suppresses a basal defense and results in a hospital environment for propagation of pathogen. However, when RPS2 is present in the host plants, the elimination of RIN4 triggers RPS2-mediated effective defenses including hypersensitive response (HR) and lead to resistance against pathogens. Kinetics of RPS2-mediated HR reveals that AvrRpt2-mediated elimination of RIN4 occurs in 3 - 5 hrs post infiltration of *P. syringae* (avrRpt2), which sequentially destabilized RPM1 and eliminated RPM1 in 12 - 20 hrs independent on RPS2. When RPS2 is present, the elimination of RPM1 is tightly linked with RPS2-mediated HR time point. We wondered that what kinds of plant factors are involving in AvrRpt2-mediated RPM1 and what their functions are in the plant defense mechanism. To answer these questions, we developed a novel screening method and identified mutants that are deficient in AvrRpt2-mediated RPM1 degradation. At present, 4 different *ner* (no elimination of RPM1 by AvrRpt2) mutants were isolated. Each mutant phenotype is recessive and resulted from mutations in a single genetic locus. Specially, *ner1* and *ner2* mutations inhibited not only AvrRpt2-mediated elimination of RPM1 but also AvrRpt2-mediated elimination of RIN4. More interestingly, AvrRpm1-mediated phosphorylation of RIN4 by which RPM1-mediated resistance was activated was also abolished in these mutants. In consistent with these biochemical data, *ner1* and *ner2* mutants did not show HR against infiltration of either *P. syringae* (avrRpt2) or *P. syringae* (avrRpm1). We hypothesize that *ner1* and *ner2* mutants are mutated in the genes commonly involving in modification of RIN4 by AvrRpm1 and AvrRpt2 and their further characterization might help elucidate the molecular mechanism by which TTSS effectors regulate the host targets and suppress the basal defense.

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