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

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Kinetics of RPS2-mediated HR reveals that AvrRpt2-mediated elimination of RIN4 occurs in 3 - 5 hpi 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 wonder that what plant factors are involving in the AvrRpt2-mediated RPM1 elimination process 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 are isolated. Each mutant phenotype is recessive and resulted from mutations in a single genetic locus. Specially, *ner1*, *ner2* and *ner3* mutations inhibitnot 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 is activated is also abolished in these mutants. In consistent with these biochemical data, these mutants do not show HR against infiltration of either *P. syringae* (avrRpt2) or *P. syringae* (avrRpm1). We hypothesize that *ner1 ner2* and *ner3* 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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In Vitro expression of protoporphyrin IX induced by 5-aminolevulinic acid (ALA) in human cancer and normal cells

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We estimated the expression of protoporphyrin IX (PpIX) induced by ALA in human cancer and normal cells. These cells were incubated with various concentration of ALA (0～800㎍/mL) for 24hr, and observed at the levels of PpIX accumulation in intra and extra cellular by using the fluorescence spectroscopy. The accumulation of PpIX induced by ALA was observed in cancer cells(HeLa, A549, NCl-H460) and normal liver and lung cells(Chang and Hel299). Also the cell viability was assessed by MTT assay. The viability of HeLa, A549, Chang and Hel299 cells exposed to ALA at 800㎍/mL measured 65, 74, 70 and 76% respectively. The optimal concentration of ALA that induced maximum levels of extra cellular-PpIX was 50～100㎍/mL in HeLa, Chang and A549 cells, and was 200㎍/mL in NCl-H460 and Hel299. Also the optimal concentration of ALA that induced maximum levels of intra cellular-PpIX was was 50～100㎍/mL in cancer cells, and 400㎍/mL in normal liver cells (Chang). But the accumulation of intra cellular-PpIX in normal lung cells (Hel299) showed very low levels. Especially, formation of PpIX in cancer cells was higher than normal cells. This study suggests that the difference of PpIX induced in normal and cancer cells treated with ALA may use by means of fluorescence diagnosis for cancer.