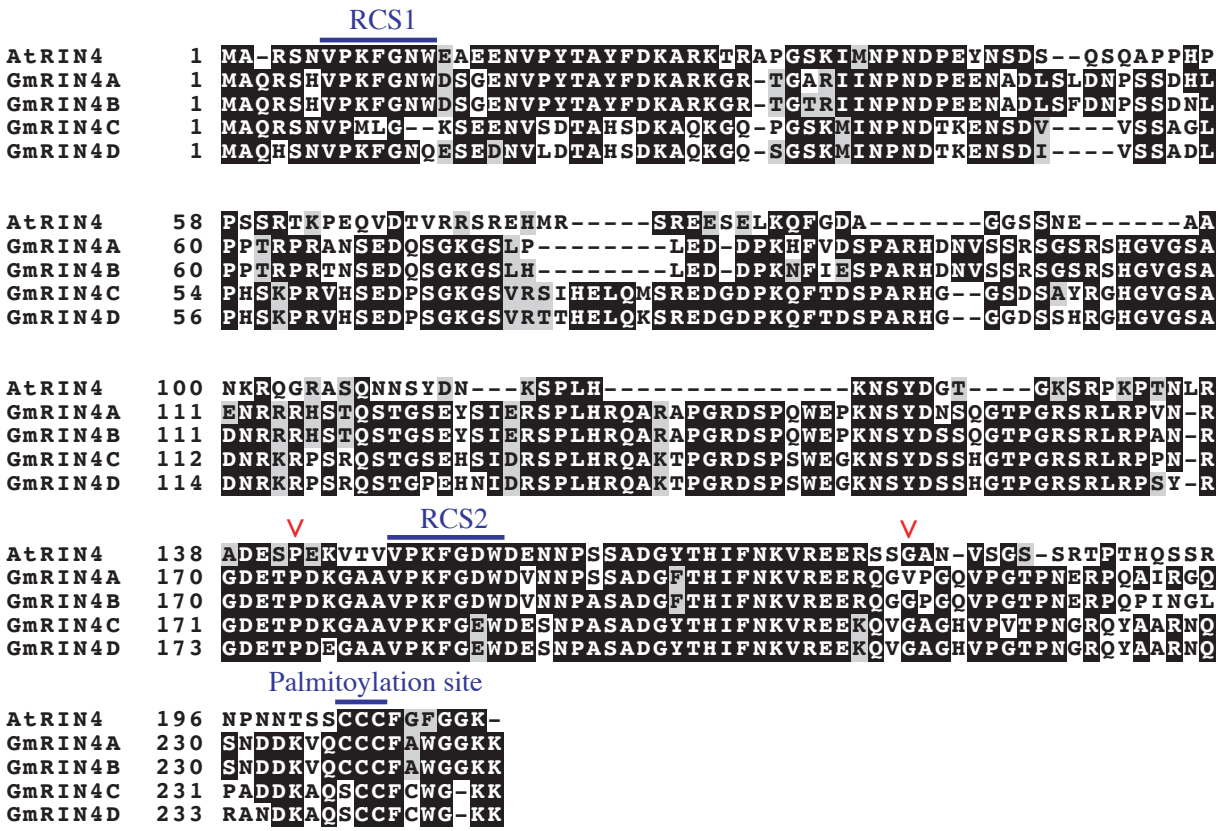
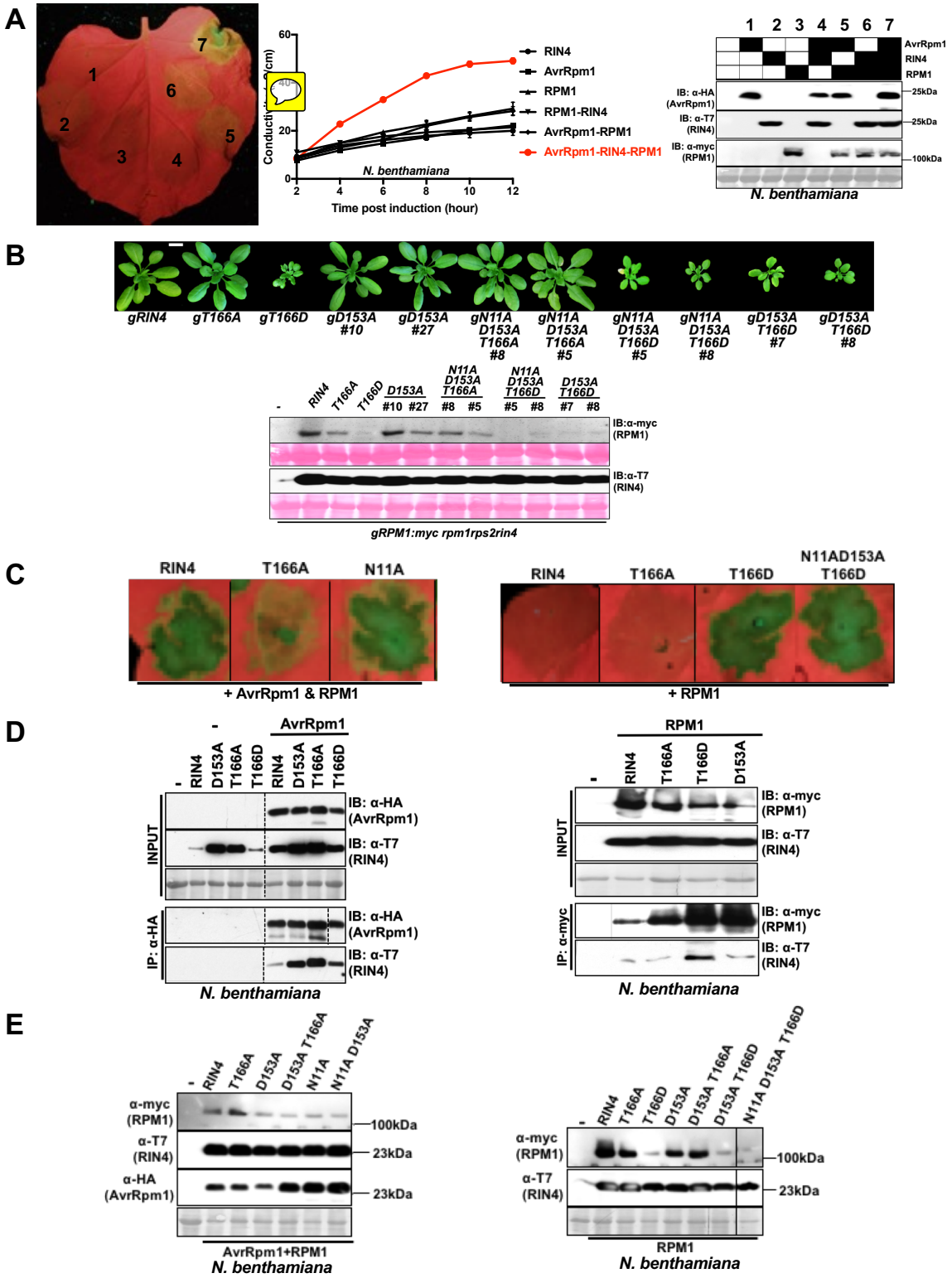


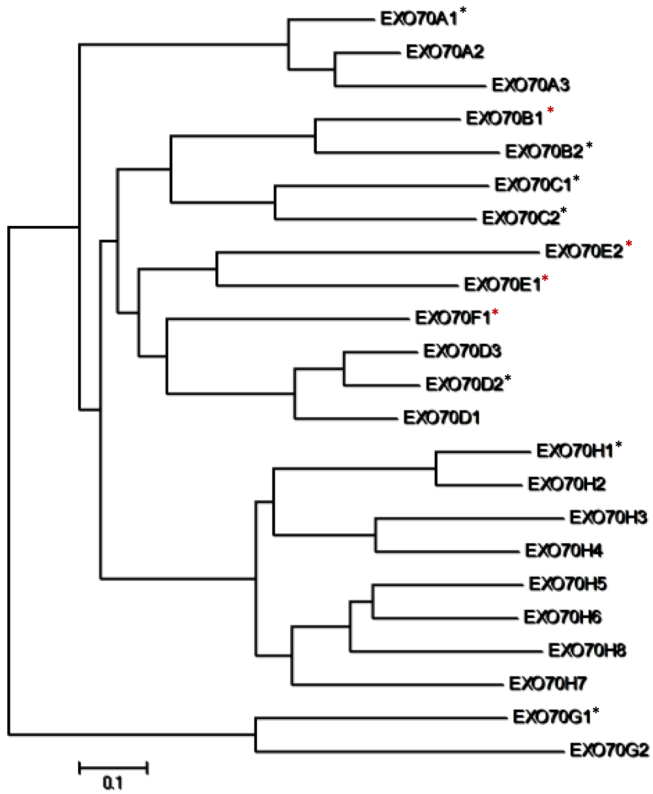
Supplemental Figure 1. RIN4 is Highly Conserved Between Species.

A box shade alignment illustrates the level of amino acid identity between soybean and Arabidopsis RIN4 proteins. RCS1 and RCS2 indicate the AvrRpt2 cleavage sites conserved among all RIN4 proteins. The region between the red arrow-heads span the region of RIN4 shown to interact with AvrB.

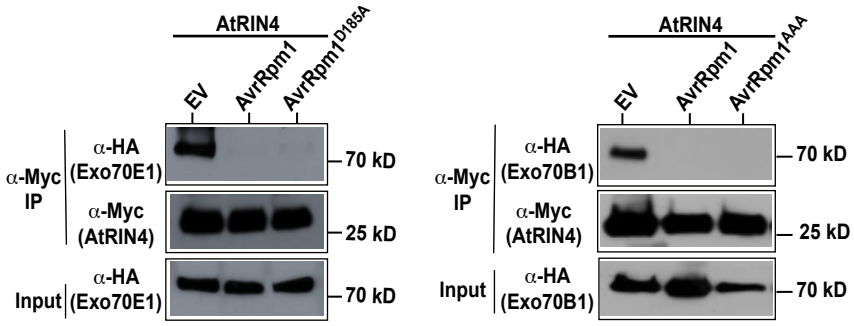


Supplemental Figure 2. ADP-ribosylation of *At*RIN4 on D153 Promotes Phosphorylation on T166.

(A) Reconstruction of AvrRpm1-dependent RPM1 activation in *N. benthamiana*. AvrRpm1, *At*RIN4 or RPM1 constructs were infiltrated alone or in combination with each other. The picture was taken 12 hours post induction of AvrRpm1 with 20 μM estradiol treatment. This confirms that RPM1 can only be activated following co-expression of AvrRpm1 and *At*RIN4 (left). Conductivity measurements for AvrRpm1-mediated RPM1 activation in this reconstruction system (middle). Expression of each protein was confirmed by immunoblots with α-HA for AvrRpm1, α-T7 for RIN4 and α-myc for RPM1 (right).
(B) Phenotypes of Arabidopsis transgenic lines for *At*RIN4^{D153A}, *At*RIN4^{N11AD153AT166A}, *At*RIN4^{N11AD153AT166D} and *At*RIN4^{D153AT166D} confirms that the phospho-mimic *At*RIN4^{T166D} is epistatic to loss of ADP ribosylation in triggering effector-independent cell death. Two independent T2 transgenic lines of each *At*RIN4 mutant allele were selected and protein expression of RPM1 and *At*RIN4 mutant alleles was monitored by immunoblot with α-myc for RPM1 or α-T7 for *At*RIN4, respectively.
(C) ADP-ribosylation at *At*RIN4^{N11} is not required for AvrRpm1-mediated RPM1 activation (left) and the phospho-mimic *At*RIN4^{T166D} is epistatic to loss of ADP ribosylation in triggering effector-independent cell death (right) in *N. benthamiana*. The visible cell death phenotypes are shown 12 hours post-induction of AvrRpm1 with 20μM of estradiol treatment (left) and 2.5 days post-inoculation (right).
(D) Co-immunoprecipitation (CoIP) of AtRIN4 mutant proteins with AvrRpm1 or RPM1 in *N. benthamiana* was performed by immunoprecipitation with α-HA for AvrRpm1 (left) or α-myc for RPM1 (right) followed by immunoblots with α-T7 for *At*RIN4. Samples were taken 12 hours post-estradiol treatment for CoIP of *At*RIN4 with AvrRpm1 or 2.5 days post inoculation for CoIP of *At*RIN4 alleles with RPM1, respectively.
(E) Expression of *At*RIN4 alleles, AvrRpm1 and RPM1 used to test AvrRpm1-mediated or effector-independent RPM1 activation. Total proteins were extracted 6 hours post-estradiol (20μM) treatment for AvrRpm1-mediated RPM1 activation and expression was confirmed by immunoblots with α-HA for AvrRpm1, α-T7 for *At*RIN4 alleles and α-myc for RPM1 (left). Protein expression of *At*RIN4 alleles and RPM1 during effector-independent activation were verified by immunoblots with α-T7 for *At*RIN4 alleles and α-myc for RPM1 from the samples taken 2 days post-infiltration in *N. benthamiana* (right). Note that RPM1 activation by the phospho-mimic *At*RIN4^{T166D} results in diminution of RPM1 levels.



Supplemental Figure 3. Phylogenetic Tree of Arabidopsis EXO70 Family Members.
Evolutionary relationships among proteins of the Arabidopsis EXO70 family (Synek et al., 2006) were inferred using the Neighbor-joining method (Saitou and Nei, 1987) implemented in MEGA6 (Tamura et al., 2013). The scale bar indicates an evolutionary distance of 0.1 substitutions per amino acid position. The asterisks indicate EXO70 proteins that were tested for interaction with AtRIN4 through a targeted yeast two-hybrid assay, with the red asterisks indicating those shown to interact with AtRIN4 (Figure 7A).



Supplemental Figure 4. AvrRpm1 Blocks Association of *At*RIN4 with Arabidopsis EXO70 Proteins. The indicated proteins were transiently expressed in *N. benthamiana* and then *AtRIN4* was immunoprecipitated using anti-myc agarose beads. EXO70B1 and EXO70E1 co-precipitated with *At*RIN4, but this interaction was blocked in the presence of both wild-type and catalytic site mutant AvrRpm1 proteins.