DNA REPAIR DEFICIENCIES INCREASE THE RESISTANCE OF *ARABIDOPSIS THALIANA* TO *HYALOPERONOSPORA PARASITICA*

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We have found that UV-C treatment of *Arabidopsis thaliana* induces resistance to the biotrophic pathogen *Hyaloperonospora parasitica*, and our data suggest UV induced DNA photoproducts are involved (see accompanying abstract by K.G. McKenzie et al.). To address the potential role of DNA damage, we have examined the effect of mutations in nucleotide excision repair (*uvr1-1*), photoreactivation of cyclobutane pyrimidine dimers (*uvr2-1*) or flavonoid production (*tt5*) on the resistance of Arabidopsis to the pathogen with or without pre-inoculation treatment with UV-C. In the mutant backgrounds, UV-C induced pathogen resistance (as measured by decreased conidiophore formation) to the same degree as in the wildtype plants, but much lower UV doses were required (e.g., 100 Jm^{-2} in the mutant vs. 400 Jm^{-2} in the wildtype). This is the result expected if damage to DNA rather than a non DNA target is involved. Interestingly, in the absence of UV-C, the *tt5* mutation alone resulted in a slight increase in resistance. However, when coupled with *uvr1-1*, resistance was enhanced to an even greater extent. Remarkably, the *tt5 uvr1-1 uvr2-1* triple mutant was completely resistant to the pathogen. Since *tt5* mutants are sensitive to reactive oxygen species, which can cause DNA damage susceptible to nucleotide excision repair, our results suggest that in addition to UV photoproducts, an accumulation of endogenous oxidative DNA damage may also trigger resistance to the pathogen. We are currently examining pathogen resistance in other DNA repair deficient mutants, and quantifying UV-C-induced DNA damage in Arabidopsis in order to assess the relationship between damage levels and the extent of resistance.