The effect of a bacterial derived UV-endonuclease on *C. elegans* nematodes displaying the Xeroderma Pigmentosum phenotype

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UV-induced DNA lesions are normally repaired by nucleotide excision repair (NER). The genetic human condition Xeroderma Pigmentosum (XP) results when this DNA repair is non-functional. Patients with XP are extremely photosensitive with 1000-fold increased chance of developing skin cancers from the unrepaired lesions. The *Caenorhabditis elegans* (*C. elegans*) mutants *xpa-1* and *xpf-1* are useful to model the XP condition as they are also deficient in NER. Without DNA repair, L1 stage larvae irradiated with UV undergo severe developmental arrest. Both humans and *C. elegans* rely on NER alone to reverse UV damage. However, the bacterium *Micrococcus luteus* expresses UV-endonuclease, an enzyme that repairs UV-induced DNA lesions irrespective of NER functionality. I sought out to determine if UV-endonuclease would repair DNA lesions in *C. elegans* enough to allow NER deficient L1 larvae to continue growth after UV exposure. When delivered in liposomes to *xpa-1* and *xpf-1* strains, the UV-endonuclease caused a slight, nonsignificant, increase in development of L1's to the L3 stage or larger following 5 J/m^2 UV-C radiation. Increases in development of 11.7 and 5.2% were observed for *xpa-1* and *xpf-1*, respectively ($P > 0.05$ for *xpa-1* and *xpf-1*; $q_{crit} = 4.21291$).