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Abstract: NEILs 1-3, part of the MutM/Nei family of DNA glycosylases, excise lesions from single stranded DNA and bubble structures. This unique activity makes them an important part of the base excision repair (BER) pathway in non-dividing, transcribing cells. Our goal was to determine if any of the NEIL genes containing single nucleotide polymorphisms (SNPs) were deleterious compared to the wild-type (WT) protein. We created variants of the NEIL1 and NEIL2 genes in expression vectors using site-directed mutagenesis and electroporated them into *Escherichia coli* (*E. coli*) cells with a reduced capacity to repair oxidative damages. These variants were compared to the WT protein by means of a rifampicin assay. Rifampicin is an antibiotic that inhibits RNA polymerase in *E. coli* cells. A mutation in the beta subunit of this protein prevents rifampicin binding and allows the cell to grow. Higher numbers of mutations yield a greater number of surviving colonies and indicate a deleterious variant. A spontaneous mutation frequency was calculated for each variant by comparing the number able to grow on rifampicin-containing plates to the number of cells growing on plain LB plates. These numbers were then compared between the wild-type cells and variants. The *E. coli* cells used had knockouts of *fpg*, *nei*, and *mutY* DNA glycosylase genes. Deleterious variants are now being further examined in mammalian cells for differences in cell survival and damage response compared to the wild-type.