

## **DDB2 INTERACTIONS WITH NUCLEOTIDE EXCISION REPAIR PROTEINS**

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Nucleotide excision repair (NER) is the principal pathway for removal of a broad spectrum of structurally unrelated lesions. In human cells, it is primarily responsible for repair of UV- induced cyclobutane pyrimidine dimers and (6-4)-photoproducts. DNA damaged by ultraviolet light (UV) is recognized by heterodimer complex UV-DDB which comprises two subunits DDB1 and DDB2. Functional defect in UV-DDB activity has a direct relationship to decreased NER efficiency and increased susceptibility to cancer. In particular, DDB2 plays an important role in the recognition step of UV-induced DNA damage in non- transcribed regions (GG-NER), and it is mutated in Xeroderma pigmentosum (group E) patients.

In this study, we have investigated the localization and the interaction between DDB2 and some of the proteins involved in NER process. DDB2 localization was determined in HeLa cells, transiently transfected with pcDNA3.1-DDB2 construct, before and 30 minutes after UV-C irradiation. Confocal analysis showed that DDB2 co-localized with PCNA and p21 proteins recruited to DNA-damaged sites. To study a possible interaction between DDB2 and both these proteins, solubilised chromatin fractions were immunoprecipitated with DDB2 antibody. The results demonstrated that the DDB2-p21 interaction is mediated by PCNA.