

concentration dependent. In addition protection against cell killing was very similar in magnitude to protection against induction of mutations. The protective effect could be detected only when anti-oxidants were added to the cells before irradiation. No protection was afforded upon addition immediately after irradiation.

REFERENCE:

1. Ben-Hur, E., Green, M., Prager, A. Rosenthal, I. and Riklis, E., J. Radiat. Res. 22, 250 (1981)

DNA REPAIR SYNTHESIS IN CARROT ROOT TISSUE IRRADIATED WITH ULTRAVIOLET LIGHT

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Plant DNA damage by ultraviolet (UV) light can be repaired by the light dependent photoreactivation process and by dark excision repair, which is a multienzyme process. In the initial steps of the excision repair process, pyrimidine dimers, which are the UV light photoproducts, are excised from the damaged DNA. Pyrimidine dimers were found in hydrolizates of labeled damaged DNA from actively growing tissues and plants such as carrot protoplasts, grass pea seedlings, *Wolfia microscopica* and the alga *chlamydomonas*. The subsequent filling of the gap in the damaged site of the DNA is done by repair synthesis. It is difficult to demonstrate this step of the excision repair process in actively growing tissue, because the incorporation of labeled thymidine due to scheduled DNA synthesis superimposes that which is due to repair replication. So far, repair synthesis in plants has been detected in *Petunia* pollen. In the present work repair synthesis was demonstrated in UV light irradiated ( $500-1000 \text{ erg/mm}^2$ ) carrot root slices which incorporated 50 to 100% more labeled thymidine than the non-irradiated control during 22 h of postirradiation dark incubation. In higher UV light fluences ( $1500-5000 \text{ erg/mm}^2$ ), the results were inconsistent.