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VITAMIN E AND UV RELATED SKIN DAMAGE IN MICE

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Biochemically, two of the early events following exposure of skin to ultra-violet (UV) light are the induction of lipid peroxidation and a suppression of replicative DNA synthesis due to DNA damage. UV induced lipid peroxidation and DNA damage both appear to be caused by reaction with free radicals generated within the epidermal cells. In the past, several groups have studied the potential protective effect of systemic antioxidants including vitamin E against UV exposure. De Rios et al. found no protective effect of vitamin E on its own against UV-induced erythema, however a mixture of several antioxidants did afford protection. Khettab et al. reported that topical applications of vitamin E prior to UV irradiation diminished the production of malondialdehyde (MDA) when applied immediately before exposure. In addition Igarishi et al. found that MDA levels in the skin of vitamin E deficient animals was greater than that in control animals, and showed a further increase following UV exposure.

The purpose of the current study was to determine whether variations in the dietary, and hence tissue, vitamin E levels could influence the cellular response following modest exposure to simulated solar radiation and to compare any such effects with topical application of the vitamin.

Hairless mice were fed diets containing different levels of vitamin E or received topical applications of the vitamin for three weeks prior to a single exposure to an artificial sunlight source. Production of malondialdehyde, an estimate of peroxidative damage and suppression of incorporation of thymidine into DNA were used to estimate the degree of damage caused by the radiation. Dietary vitamin E deficiency together with (UV) irradiation increased epidermal (MDA) levels but had little effect on suppression of thymidine incorporation into DNA. High levels of dietary vitamin E tended to reduce the production of MDA, but restored the incorporation of thymidine to similar levels to those of unirradiated animals. Topical administration of 1% vitamin E 1 or 24hr prior to irradiation also restored the incorporation of thymidine into DNA, and reduced the production of MDA. In a further study the skins of similarly treated animals were examined by light and transmission electronmicroscopy. No pathology was observed, however the mitotic index was substantially reduced following UV irradiation. The results suggest that dietary and topical vitamin E can protect against some, but not all of the potentially carcinogenic effects of UV irradiation.

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