Induction of the putative protective protein ferritin by infrared radiation: implications in skin repair

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Abstract

The modification of ferritin in human skin cells in vitro and in vivo following infrared-A irradiation by immunohistochemical analysis and ELISA were evaluated. In addition, we observed that IR-A is not capable of inducing frank damage to DNA (pyrimidine dimers, p53), induction of oxidative stress proteins (heme oxygenase, nitric oxide, superoxide dismutase, heat shock proteins) or proteases (collagenase, stromelysin, gelatinase) involved in carcinogenesis and photoaging of the skin. in vivo, basal levels of ferritin were heterogeneous for all individuals tested but all showed ferritin to stain precisely in the basal layer of unirradiated epidermis. Following IR-A radiation, the ferritin increase was localized to epidermal tissue and showed an increase from 120 to 220%. Parallel to the in vivo analysis, dermal fibroblasts were cultured from six individuals. Quantitative analysis for ferritin in cultured fibroblasts was assessed by ELISA and increases were seen to be dose-dependent and up to 130% of basal levels of ferritin following infrared-A. Our findings indicate that the putative defense system of ferritin that exists in human skin in vivo can be induced by infrared-A radiation and that these wavelengths may prove to be beneficial for human skin. Importantly, following the same doses of IR-A that induced ferritin levels, there was no alteration seen for nuclear DNA type damage, oxidative stress proteins or proteases involved in the degradation of skin. The increased concentrations of this antioxidant in human skin following acute UV radiation could afford increased protection against subsequent oxidative stress.