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Anti-photoaging effect of Crocin: Its molecular mechanism in UVB-irradiated keratinocytes and human dermal fibroblasts

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Solar ultraviolet (UV) radiation, particularly UVB radiation, is the major cause of photoaging, the most damaging effect of which is skin carcinogenesis. UVB-induced oxidative stress plays a crucial role in initiating and promoting cell signaling involved in aging. Specifically, photoaging results from the up-regulation of metalloproteinases (MMPs) via the activation of activator protein-1 (AP-1) and subsequent collagen breakdown in the skin. This study demonstrates the protective effect of Crocin, an active carotenoid component of *Crocus sativus* L and *Gardenia jasminoides* E, on UVB-induced photoaging. Using HaCaT human keratinocytes and human dermal fibroblasts (HDFs), we evaluated Crocin's anti-photoaging effect by conducting 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide (MTT) assays, western blots, enzyme assays and fluorescence-activated cell sorting. In keratinocytes, crocin significantly inhibited UVB-induced oxidative stress and apoptosis by suppressing reactive oxygen species (ROS) generation. Furthermore, it significantly increased the activity of antioxidant enzymes regulated by the transcription factor, Nrf2. In fibroblasts, Crocin significantly attenuated UVB-enhanced MMP expression by inhibiting AP-1 activity in a dose-dependent manner. In addition, it significantly promoted the synthesis of collagen and elastin in HDF cells. Taken together, these results suggest that Crocin prevents UVB-induced photoaging in keratinocytes and fibroblasts by suppressing ROS generation and regulating gene expression.

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