

C14 IP protects against photoaging by inhibiting the expression of MAPK/MMPs pathway and by promoting type I procollagen synthesis

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ABSTRACT

Skin provides an effective barrier between the organism and the environment; however, overexposure of the skin to ultraviolet (UV) radiation results in the development of inflammation, oxidative stress, immune suppression, and DNA damage, which can lead to premature aging of the skin. In this process, UV irradiation leads to the formation of reactive oxygen species (ROS) that activate the mitogen-activated protein (MAP) kinase pathway, subsequently stimulate the expression and activation of matrix metalloproteinases (MMPs) which degrade type I procollagen, and then leads to photoaging. Polyphenols rich IPE exhibited antioxidant activity and reducing UVB-induced intracellular reactive oxygen species production. Results of the photoaging screening experiments revealed that IPE at 1000 $\mu\text{g}/\text{mL}$ reduced the activity of bacterial collagenase by $92.7 \pm 4.2\%$ and reduced the activity of elastase by $32.6 \pm 1.4\%$. Therefore, we investigated the mechanisms by which IPE exerts its anti-photoaging activity. IPE at 1 $\mu\text{g}/\text{mL}$ led to an increase in type I procollagen expression and increased total collagen synthesis in fibroblasts at 5 $\mu\text{g}/\text{mL}$. We found that IPE inhibited MMP-1, MMP-3, and MMP-9 expression at doses of 1, 5, and 10 $\mu\text{g}/\text{mL}$, respectively, in fibroblasts exposed to UV irradiation ($40 \text{ mJ}/\text{cm}^2$). Gelatin zymography assay showed that IPE at 50 $\mu\text{g}/\text{mL}$ inhibited MMP-9 secretion/activity in cultured fibroblasts after UVB exposure. In addition, IPE inhibited the phosphorylation of p38, ERK, and JNK induced by UVB. According to these results, IPE can be a good functional material in cosmetics based on anti-photoaging activities.

Keywords: photoaging, matrix metalloproteinases (MMPs), mitogen-activated protein kinase (MAPK), type I procollagen