Synopsis of Original Research Paper

Cell biological and biochemical studies on antioxidative regulation of photoaging by cosmetics

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Photoaging is a term that denotes the gross and microscopic cutaneous changes induced by chronic sun exposure, which is quite different from intrinsic chronological aging both qualitatively and quantatively. Among the various insults brought by ultraviolet (UV) irradiation, reactive oxygen species (ROS) and lipid peroxides are one of the most reasonable candidates for explaining actinic injuries in photoaging.

In the present study, using an experimental animal model of photoaging, we investigated the time dependent antioxidant enzyme changes as well as dermal glycosaminoglycan levels by disaccharide analysis. We have found that SOD activity was increased by repeated UVB irradiation, but catalase was not, indicating that the skin SOD and catalase activities are not coordinately regulated by a long-term UV irradiation and that the SOD activity, which has been reported to decrease after acute actinic injury, is induced by chronic photo-oxidative stress.

We have also demonstrated that the total amount of main disaccharide units increased by UV irradiation confirming the previous histochemical findings.

Another important progress made during the present investigation was the development of the three-dimensional culture system supplemented with L-ascorbic acid 2-phosphate using dermal fibroblasts from photoaged murine skin. This method should provide a promising technique to evaluate the capacity for regulating the photoaging process by cosmetics in the near future.