Synopsis of Original Research Paper

Phtoaging −Studies on the pathogeneisis of actinic elastosis and the diagnostic method for UV^{s} syndrome−

Tomomichi Ono

Department of Dermatology, Kumamoto University School of Medicine

Long-term incubation of proteins with glucose leads to the formation of advanced glycation end products (AGEs), which are characterized by fluorescence, brown color, and cross-linking. AGEs are thought to be involved in aging and age-enhanced diseases. Chronic exposure of the skin to sunlight induces hyperplasia of the elastic tissue in the upper dermis known as actinic elastosis.

Herein we used a monoclonal anti-AGE antibody (6D12) and a rabbit polyclonal anti-AGE antibody, which are shown to recognize Nε-(carboxymethyl)lysine (CML) as those epitope. Fifty-seven skin samples of sun-exposed and -unexposed areas were obtained from 51 patients aged 9−100 years. Immunohistochemically, CML deposition was observed in the elastic materials from all these specimens showing actinic elastosis with marked elastic-tissue changes. In contrast, CML deposition was not found in the specimens from sun-unexposed areas without significant actinic elastosis. Immunoelectron microscopic examination demonstrated that CML accumulated predominantly in elastic fibers especially in the amorphous electron-dense materials corresponding to photo-induced degenerated area rather than the electron-lucent region. Immunochemical analyses with enzyme-linked immunosorbent assay (ELISA) of elastase-soluble fractions demonstrated that the CML levels of the sun-exposed area were significantly higher than those of the sun-exposed area.

These results indicate that elastin in actinic elastosis lesions of photoaged skin is modified by CML which has been proposed as a potential biomarker of oxidative damage of tissue proteins in vivo. Although it is not clear whether AGE accumulation in the skin per se is a cause of the disease or simply its effect, it could at least serve as a biomarker for the duration and severity of oxidative damage to the skin in vivo. To support this idea, further studies are needed to elucidate direct effects of UV irradiation on the formation of AGEs in vivo and *in vitro*.