〈原　著〉

紫外線による皮膚アスコルビン酸の酸化機構

北澤　学*，岩崎　敬治*，PACKER, Lester**

The Mechanism for Ascorbate Oxidation in UV-Irradiated Mouse Skin Homogenates

Manabu KITAZAWA,* Keiji IWASAKI,* Lester PACKER**

Abstract

The mechanism of oxidation of ascorbic acid in mouse skin homogenates by ultraviolet light was investigated by measuring ascorbate free radical formation using electron spin resonance signal formation. The rate of ascorbate oxidation was enhanced by a photosensitizer (riboflavin), but was not influenced by reactive oxygen radical quenchers, superoxide dismutase or 5,5-dimethyl-1-pyrroline-N-oxide. These experimental results suggest that the ultraviolet irradiation-induced ascorbate oxidation in murine skin homogenates is caused by photoactivated reactions rather than reactive oxygen radical reactions. The similar hydrophilicity of ascorbate, vitamin E short chain homologue [2,2,5,7,8-pentamethyl-6-hydroxychromane (PMC)] increased their interaction, thus accelerated ascorbate oxidation. When dihydrolipoic acid (DHLA) was added simultaneously with the vitamin E homologues, the accelerated ascorbate oxidation was prevented. This was due to the regeneration of ascorbate and PMC from their free radicals by a recycling mechanism between ascorbate, vitamin E homologues, and DHLA. Potentiation of antioxidant recycling may be protective against ultraviolet irradiation-induced damage.

Key words: ultraviolet light, ascorbate, electron spin resonance, vitamin E, riboflavin.