Several Animal Models of Oxidative Stress to the Skin

Y. Miyachi a
A. Yoshioka a
S. Suzuki b
S. Imamura a
Y. Niwa c

Departments of Dermatology a and Plastic Surgery b, Kyoto University Faculty of Medicine, Kyoto, Niwa Institute for Immunology c, Kochi, Japan

Yoshiki Miyachi, MD, Department of Dermatology, Faculty of Medicine, Kyoto University, Sakyou-ku, Kyoto 606 (Japan)

Since skin is always exposed to oxygen, several kinds of stimuli, physical, chemical or whatever, may generate reactive oxygen species in the skin with resultant harmful effects directly and/or through lipid peroxide formation [1]. These reactive oxidants are also produced at the site of cutaneous inflammation by activated phagocytes. However, it still remains unclear how oxidative stress to the skin and cutaneous antioxidant activity correlate with each other in the inflammatory process. The purpose of this study is to investigate the effect of acute oxidative stress on cutaneous superoxide dismutase (SOD) activity using several animal models.

We have attempted to induce several animal models of oxidative stress to the skin by the following methods: (1) irradiation of ultraviolet B [2]; (2) application of a chemical irritant (DNCB) [3]; (3) injection of xanthine oxidase plus hypoxanthine [4]; (4) exposure to hyperthermia [5], and (5) making ischemic skin flaps.

We assessed the skin SOD activity and also examined the effect of SOD pretreatment and aging on cutaneous SOD levels. A significant decrease in SOD activity after a single exposure of these procedures was readily observed which was more remarkable and prolonged in aged animals. Also a possible protective effect of liposomal SOD pretreatment was suggested. Further questions to be answered are as follows: (1) What is the mechanism of the decreased SOD activity? Is it due to consumption, leakage or destruction, or simply because of reduced DNA synthesis?; (2) Is there an appropriate dose of stimuli to induce SOD activity?; (3) How about other cutaneous antioxidants such as catalase or glutathione peroxidase?; (4) Is SOD activity induced after chronic and repeated oxidative stress?; and (5) Is there a possible disadvantage of exog-enously given antioxidants?

In any event, these experiments provide a clue to the question how oxidative stress and antioxidant activity correlate with each other in cutaneous inflammation, aging and even carcinogenesis.

References


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