〈シンポジウム〉
(紫外線と皮膚を考える)

光　免　疫
——抑　制　機　構——

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Photoimmunology
—Mechanisms of immunosuppression induced by ultraviolet irradiation—

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Abstract

Ultraviolet (UV) radiation affects cells of the skin, resulting in the modulation of immunoreaction. *Trans*-urocanic acid (UCA) in the stratum corneum isomerizes to its *cis* form on UV radiation. *Cis*-UCA can induce immunosuppression that mimics a UV-induced one. The number of epidermal Langerhans cells is decreased and their antigen presenting function is impaired by low-dose UV radiation. UV radiation induces keratinocytes to produce many cytokines, such as IL-10 and TNFα. Dermal cell components are also influenced by UV radiation, including overproduction of cytokines and expression of adhesion molecules in endothelial cells, inhibition of macrophage migration and NK activity, and induction of lymphocyte apoptosis. There are two models of suppression of T cell mediated immune responses by UV radiation: local suppression, and systemic suppression. In local suppression, an antigen is applied at an irradiated site with low-dose UV. This phenomenon is related to impaired Langerhans cells, as well as keratinocyte-derived cytokines. On the other hand, systemic suppression occurs when an antigen is applied at a non-irradiated site, and is mediated by soluble factors produced by UV-irradiated keratinocytes or other cells. Understanding the mechanism by which UV radiation induces immunosuppression of contact hypersensitivity and delayed-type hypersensitivity in mice may help to understand how the immune system is related to photocarcinogenesis and UV-induced skin disorders, as well as how phototherapy is effective for immunological skin diseases.

Key words: ultraviolet radiation, immunosuppression, contact hypersensitivity, Langerhans cell, cytokines.