## **(Regular Article)**

## A Potential of α-Tocopherol Fatty Acid Ester as an Anti-Pigmentation Agent

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## Abstract

Solar lentigos (SLs) are pigmented spots that typically appear in photoaged skin depending on its solar exposure history. It has been reported that ultraviolet (UV) light is a major cause of SLs because UV is a strong promoter of reactive oxygen species (ROS). The process of skin pigmentation has been demonstrated to be divided into three major steps: melanocyte proliferation, the maturation of melanosomes (MSs) according to increases in melanin synthesis and the transfer of MSs to keratinocytes. The maturation of MSs and their transfer from melanocytes to keratinocytes following UV exposure are accelerated via the effects of soluble factors such as  $\alpha$ -melanocyte stimulating hormone, endothelin-1, stem cell factor and prostaglandin E<sub>2</sub> (PGE<sub>2</sub>), produced by UV-exposed keratinocytes in autocrine and paracrine fashions. Among those soluble factors, PGE,, which is synthesized by keratinocytes in response to UV exposure, has been reported to promote dendrite development and melanin synthesis of melanocytes. The purpose of this study was to characterize the potential anti-pigmentation effects of  $\alpha$ -tocopherol fatty acid ester (VE-FA), which was synthesized with a natural fatty acid mixture enriched with linoleic acid, focusing on the activation of melanocytes and acceleration of MS incorporation to keratinocytes by the conditioned medium of UVB-exposed keratinocytes. VE-FA suppressed the incorporation of fluorescent beads, which were used as pseudo-MSs, into keratinocytes treated with the conditioned medium (CM) from UVB-exposed keratinocytes. In addition, treatment with VE-FA abolished the increased proliferation and dendrite elongation of melanocytes treated with CM from keratinocytes exposed to UVB. Furthermore, VE-FA reduced the level of PGE, in the CM from keratinocytes exposed to UVB. In summary, the results of this study demonstrated the possibility that VE-FA has an anti-pigmentation effect through its suppression of MS transfer to keratinocytes and of melanocyte activation following UVB exposure.

Key words: UVB, ROS, melanosome transfer, prostaglandin E<sub>2</sub>.