Available online at www.sciencedirect.com
SCIENCE DIRECT®
Cancer Letters 196 (2003) 169–177
CANCER Letters
www.elsevier.com/locate/canlet

Cancer chemopreventive effects of oral feeding α-tocopherol on ultraviolet light B induced photocarcinogenesis of hairless mouse

Masashi Kuchide[a,*], Harukuni Tokuda[a], Junko Takayasu[a], Fumio Enjo[a], Takeshi Ishikawa[b], Eiichiro Ichiishi[b], Yuji Naito[b], Norimasa Yoshida[b], Toshikazu Yoshikawa[b], Hoyoku Nishino[a]

[a]*Department of Biochemistry, Kyoto Prefectural University of Medicine, Kwaramachi Hirokouji, Kamigyo-ku, Kyoto 602-0841, Japan*
[b]*First Department of Internal Medicine, Kyoto Prefectural University of Medicine, Kwaramachi Hirokouji, Kamigyo-ku, Kyoto 602-0841, Japan*

Received 28 February 2003; received in revised form 28 March 2003; accepted 31 March 2003

Abstract

Ultraviolet light is the most common cause of skin cancers in humans and several effects of ultraviolet light B (UVB: 290–320 nm) are thought to contribute to skin photocarcinogenesis. The generation of free radicals and related oxidants produced by UVB exposure, result in photocarcinogenesis by directly damaging DNA. On the other side, activating of transcription factor, activator protein 1 (AP-1) induced by UVB exposure causes tumor promotion. α-tocopherol has two principal physiological activities and one is an antioxidant activity through which α-tocopherol protects unsaturated fatty acids, protein and DNA from oxidation. The other activity is to stabilize the structure of the biomembrane. In addition to these two activities, it has been recently established that α-tocopherol plays important roles in cell signal transduction. In course of these studies, we examined such effects of α-tocopherol on UVB induced skin photocarcinogenesis in hairless mice. These results indicate that oral feeding of α-tocopherol including diet exhibited a marked inhibitory effects on both tumor incidence and multiplicity in UVB induced mouse skin photocarcinogenesis.

Keywords: Ultraviolet light B; α-tocopherol; Hairless mice; Two-stage carcinogenesis test

1. Introduction

Tumors produced by exposure to ultraviolet (UV) light constitute nearly 50% of cancers diagnosed in the USA today [1]. In fact, most of about 1,000,000 new cases of skin cancers each year are attributable to UV light irradiation [2]. UV radiation causes the largest risk factor associated with the development of skin cancer [3]. The ultraviolet light B (UVB) wavelengths (280–320 nm) of UV radiation is the most common environmental factor in the pathogenesis of skin cancers, particularly induce both tumor initiation and promotion of basal and squamous cell carcinomas [4,5]. The generation of reactive oxygen species (ROS) by UVB exposure cause to carcinogenesis by directly damaging DNA and activating several

* Corresponding author. Tel.: +81-75-251-5317; fax: +81-75-213-2746.
E-mail address: ma-kuchi@abox3.so-net.ne.jp (M. Kuchide).

0304-3835/03/$ - see front matter © 2003 Elsevier Science Ltd. All rights reserved.
doi:10.1016/S0304-3835(03)00278-7