Carotenoids and carotenoids plus vitamin E protect against ultraviolet light–induced erythema in humans[1–3]

Wilhelm Stahl, Ulrike Heinrich, Holger Jungmann, Helmut Sies, and Hagen Tronnier

ABSTRACT

Background: Carotenoids and tocopherols, known to be efficient antioxidants and capable of scavenging reactive oxygen species generated during photooxidative stress, may protect the skin from ultraviolet light–induced erythema. β-Carotene is widely used as an oral sun protectant but studies on its protective effects are scarce.

Objective: The objective of this study was to investigate the protective effects of oral supplementation with carotenoids and a combination of carotenoids and vitamin E against the development of erythema in humans.

Design: A carotenoid supplement (25 mg total carotenoids/d) and a combination of the carotenoid supplement and vitamin E [335 mg (500 IU) *RRR*-α-tocopherol/d] were given for 12 wk to healthy volunteers. Erythema was induced by illumination with a blue-light solar simulator. Serum β-carotene and α-tocopherol concentrations and skin carotenoid levels were assessed by HPLC and reflection photometry.

Results: Serum β-carotene and α-tocopherol concentrations increased with supplementation. Erythema on dorsal skin (back) was significantly diminished ($P < 0.01$) after week 8, and erythema suppression was greater with the combination of carotenoids and vitamin E than with carotenoids alone.

Conclusion: The antioxidants used in this study provided protection against erythema in humans and may be useful for diminishing sensitivity to ultraviolet light. *Am J Clin Nutr* 2000;71:795–8.

KEY WORDS Carotenoids, tocopherol, sunburn, skin, erythema, healthy adults, ultraviolet light

INTRODUCTION

β-Carotene supplements are widely used as so-called oral sun protectants. However, studies on the protective effect of oral β-carotene supplements against skin responses to sun exposure are scarce. The protective effects are thought to be related to the antioxidant properties of the carotenoid. With ultraviolet (UV) irradiation, skin is exposed to photooxidative damage induced by the formation of reactive oxygen species such as singlet molecular oxygen ($^1O_2$), superoxide radical anion ($O_2^{\cdot -}$), and peroxyl radicals (1). Photooxidative damage affects cellular lipids, proteins, and DNA and is considered to be involved in the pathobiochemistry of erythema, premature aging of the skin, photodermatoses, and skin cancer (2). β-Carotene, other carotenoids, and tocopherols are efficient scavengers of reactive oxygen species (3).

In vitro studies showed that carotenoids are among the most effective naturally occurring quenchers of $^1O_2$, with bimolecular rate constants in the range of 1×10^9 to 1×10^{10} $mol \cdot L^{-1} \cdot s^{-1}$ (4–6). In addition, carotenoids interact with peroxyl radicals, thus inhibiting lipid peroxidation (7, 8). α-Tocopherol is less active as a quencher of $^1O_2$ but occurs at a correspondingly higher plasma concentration (9); it is among the most effective lipid-soluble inhibitors of lipid peroxidation in human blood (10). Tocopherol and carotenoids interact with each other in the scavenging process (11, 12), and a synergism was found in multilamellar liposomal systems in lipid peroxidation induced by 2,2′-azobis(2,4-dimethylvaleronitrile) (13).

Several animal studies and in vitro experiments provided evidence that carotenoids and tocopherols prevent UV light–induced skin lesions and protect against skin cancer. Several human studies showed that plasma and skin carotenoid concentrations decrease with UV irradiation; lycopene is lost preferentially over other carotenoids (14, 15). Thus, beneficial effects of supplementation have been postulated.

Garmyn et al (16) found no protective effect of β-carotene given to subjects for 23 d at a dosage of 90 mg/d, although plasma and skin β-carotene concentrations were higher than control values. The subjects were exposed to a dose of solar simulated light that was 3 times the individually determined minimal erythema dose (MED), but there was no clinically or histologically detectable protection during β-carotene supple-

[1] From the Institut für Physiologische Chemie I and Biologisch-Medizinisches Forschungszentrum, Heinrich-Heine-Universität Düsseldorf, Germany; the Institut für Experimentelle Dermatologie, Universität Witten-Herdecke, Witten, Germany; and Krebsforschung Herdecke eV, Herdecke, Germany.

[2] Supported by the Henkel Nutrition and Health Group, the Institut Danone für Ernährung eV, Munich, and the Werner Richard–Carl Dörken-Stiftung, Herdecke, Germany.

[3] Address reprint requests to W Stahl, Institut für Physiologische Chemie I, Heinrich-Heine-Universität Düsseldorf, PO Box 101007, D-40001 Düsseldorf, Germany. E-mail: wilhelm.stahl@uni-duesseldorf.de.

Received March 11, 1999.

Accepted for publication September 23, 1999.