## Lycopene-rich products and dietary photoprotection

Wilhelm Stahl, Ulrike Heinrich, Olivier Aust, Hagen Tronnier and Helmut Sies\*a

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Plant constituents such as carotenoids and flavonoids are involved in the light-protecting system in plants and contribute to the prevention of UV damage in humans. As micronutrients they are ingested with the diet and are distributed into light-exposed tissues where they provide systemic photoprotection. β-Carotene is an endogenous photoprotector, and its efficacy to prevent UV-induced erythema formation has been demonstrated in intervention studies. Lycopene is the major carotenoid of the tomato and is a very efficient singlet oxygen quencher in the group of carotenoids. Following ingestion of lycopene or tomato-derived products rich in lycopene, photoprotective effects have been demonstrated. After 10-12 weeks of intervention a decrease in the sensitivity towards UV-induced erythema was observed in volunteers. Dietary carotenoids may contribute to life-long protection against harmful UV radiation.

## Introduction

UV exposure of the skin leads to chemical and biological reactions either damaging or adaptive to light-induced stress.<sup>1,2</sup> Primarily, light of an appropriate wavelength interacts with a suitable chromophore which may be directly damaged or act as a photosensitizer. Short-lived electronically excited species initiate subsequent reactions. In the presence of oxygen, secondary reactive oxygen species are generated extending the range

"Institut für Biochemie und Molekularbiologie I, Heinrich-Heine-Universität Düsseldorf, P.O. Box 101007, D-40001, Düsseldorf, Germany. E-mail: sies@uni-duesseldorf.de; Fax: +49 211 811 3029; Tel: +49 211 811 2707 <sup>b</sup>Institut für Experimentelle Dermatologie, Universität Witten-Herdecke, Alfred-Herrhausen-Str. 44, D-58455, Witten, Germany

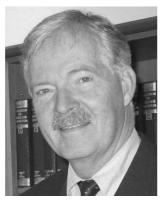
of photodamage. Photooxidative damage affects cellular lipids, proteins and DNA and is involved in the pathobiochemistry of erythema formation, premature aging of the skin, development of photodermatoses, and skin cancer.

Sunburn is a visible dermal reaction following excessive exposure to sunlight, called UV-induced or solar erythema and is characterized by tenderness, sometimes painful blistering and second degree burns.3 Direct and indirect damage resulting from photochemical reactions leads to vasodilation of dermal vessels and edema and causes increased blood flow in the affected area. Damage to proteins and DNA accumulates within skin cells, and morphological changes occur in keratinocytes and other skin cells. When a cell becomes irreversibly damaged by UV exposure, cell death follows via apoptotic mechanisms, leading to the appearance of so-called sunburn cells in the epidermis.4,5

Dr Stahl (b. 1955) is Professor of Biochemistry at the Heinrich Heine University of Düsseldorf in Germany. He studied chemistry at the University of Heidelberg and received his PhD from the University of Kaiserslautern. From 1987 to 1990 he worked in pharmaceutical industry as a research scientist for E. Merck in the field of pharmacokinetics and metabolism. He joined the Institute of Biochemistry and Molecular Biology I at the Heinrich Heine University of Düsseldorf in 1990. Dr Stahl has over 15 years of experience in the field of antioxidant research related to preventative effects of dietary antioxidants against degenerative diseases. His research addresses the biological effects and biokinetics of carotenoids, retinoids and tocopherols.







**Helmut Sies** 

Dr Helmut Sies (b. 1942) is a Professor at the Heinrich Heine University of Düsseldorf. He is chairman of the Department of Biochemistry and Molecular Biology I at the Faculty of Medicine. He received a MD degree from the Ludwig Maximilian University at Munich, Germany, in 1967. He received an honorary PhD degree from the University of Buenos Aires, Argentina, in 1996. He was president of the International Society for Free Radical Research from 1998 to 2000. He is president of the North-Rhine-Westphalian Academy of Sciences. His research interests include oxidative stress: oxidants and antioxidants as well as micronutrients and vitamins