Beta carotene and Skin Health

Photochem Photobiol. 2008 Mar-Apr;84(2):284-8. Epub 2007 Dec 15.

Protection from sunburn with beta-Carotene--a meta-analysis.

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Nutritional protection against skin damage from sunlight is increasingly advocated to the general public, but its effectiveness is controversial. In this meta-analysis, we have systematically reviewed the existing literature on human supplementation studies on dietary protection against sunburn by beta-carotene. A review of literature until June 2007 was performed in PubMed, ISI Web of Science and EBM Cochrane library and identified a total of seven studies which evaluated the effectiveness of beta-carotene in protection against sunburn. Data were abstracted from these studies by means of a standardized data collection protocol. The subsequent meta-analysis showed that (1) beta-carotene supplementation protects against sunburn and (2) the study duration had a significant influence on the effected size. Regression plot analysis revealed that protection required a minimum of 10 weeks of supplementation with a mean increase of the protective effect of 0.5 standard deviations with every additional month of supplementation. Thus, dietary supplementation of humans with beta-carotene provides protection against sunburn in a time-dependent manner.

Publication Types:

- Meta-Analysis

PMID: 18086246 [PubMed - indexed for MEDLINE]

J Invest Dermatol. 2005 Feb;124(2):428-34.

beta-Carotene interferes with ultraviolet light A-induced gene expression by multiple pathways.

Wertz K, **Hunziker PB**, **Seifert N**, **Riss G**, **Neeb M**, **Steiner G**, **Hunziker W**, **Goralczyk R**.

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Ultraviolet light A (UVA) exposure is thought to cause skin aging mainly by singlet oxygen ((1)O(2))-dependent pathways. Using microarrays, we assessed whether pre-treatment with the (1)O(2) quencher beta-carotene (betaC; 1.5 microM) prevents UVA-induced gene regulation in HaCaT human keratinocytes. Downregulation of growth factor signaling, moderate induction of proinflammatory genes, upregulation of immediate early genes including apoptotic regulators and suppression of cell cycle genes were hallmarks of the UVA effect. Of the 568 UVA-regulated genes, betaC reduced the UVA effect for 143, enhanced it for 180, and did not interact with UVA for 245 genes. The different interaction modes imply that betaC/UVA interaction involved multiple mechanisms. In unirradiated keratinocytes, gene regulations suggest that betaC reduced stress signals and extracellular matrix (ECM) degradation, and promoted keratinocyte differentiation. In irradiated cells, expression profiles indicate that betaC inhibited UVA-induced ECM degradation, and enhanced UVA induction of tanning-associated protease-activated receptor 2. Combination of betaC-promoted keratinocyte differentiation with the cellular "UV response" caused synergistic induction of cell cycle arrest and apoptosis. In conclusion, betaC at physiological concentrations interacted with UVA effects in keratinocytes by mechanisms that included, but were not restricted to (1)O(2) quenching. The retinoid effect of betaC was minor, indicating that the betaC effects reported here were predominantly mediated through vitamin A-independent pathways.

PMID: 15675964 [PubMed - indexed for MEDLINE]

Eur J Pharm Biopharm. 2009 May 12. [Epub ahead of print]

Cutaneous lycopene and beta-carotene levels measured by resonance Raman spectroscopy: High reliability and sensitivity to oral lactolycopene deprivation and supplementation.

Blume-Peytavi U, **Rolland A**, **Darvin ME**, **Pineau I**, **Voit C**, **Zappel K**, **Schäfer G**, **Meinke M**, **Sterry W**, **Lademann J**.

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Carotenoids, naturally occurring lipophilic micronutrients, possess an antioxidant activity associated with protection from damage induced by free radicals. The present study investigated an innovative non-invasive method to measure cutaneous levels of lycopene and beta-carotene and to monitor the distribution of orally administered lactolycopene in human skin and plasma. A double-blind placebo-controlled randomized study was performed in 25 volunteers, who were under a lycopene-deprived diet (4weeks prior to study until end of the study) and orally received either lactolycopene or placebo for 12weeks. Skin and plasma levels of lycopene and beta-carotene were monitored monthly using Raman spectroscopy and HPLC, respectively. Cutaneous levels of lycopene and beta-carotene monitored by resonance Raman spectroscopy showed high reliability. Irrespective of the investigated area, cutaneous levels were sensitive to lycopene deprivation and to oral supplementation; the forehead showed the closest correlation to lycopene variation in plasma. Plasma and skin levels of lycopene were both sensitive to oral intake of lactolycopene and, interestingly, also skin levels of beta-carotene. Thus, oral supplementation with lycopene led to an enrichment of beta-carotene in human skin, possibly due to the fact that carotenoids act in the skin as protection chains, with a natural protection against free radicals.

PMID: 19442725 [PubMed - as supplied by publisher]

Acta Dermatovenerol Alp Panonica Adriat. 2008 Dec;17(4):160-2, 164-6.

Beta-carotene in dermatology: Does it help?

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UV irradiation of the skin leads to the induction of free radicals, carcinogenesis, and skin aging, and thus the use of beta-carotene in humans as a chaperoning agent is discussed. In the photohemolysis model, beta-carotene protects against the phototoxic effects of porphyrins. Beta-carotene should be used in erythropoietic protoporphyria, photosensitive diseases, and to reduce the effects of phototoxic drugs. Its effects on aging skin and on actinic keratosis have not yet been sufficiently studied.

Publication Types:

- Review

PMID: 19104740 [PubMed - indexed for MEDLINE]

Planta Med. 2008 Oct;74(13):1548-59. Epub 2008 Aug 11.

Phytochemicals as protectors against ultraviolet radiation: versatility of effects and mechanisms.

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Ultraviolet (UV) radiation is one of the most abundant carcinogens in our environment, and the development of non-melanoma skin cancers, the most common type of human malignancy worldwide, represents one of the major consequences of excessive exposure. Because of growing concerns that the level of UV radiation is increasing as a result of depletion of the stratospheric ozone and climate change, the development of strategies for protection of the skin is an urgent need. Many phytochemicals that belong to various families of secondary metabolites, such as alkaloids (caffeine, sanguinarine), flavonoids [(-)-epigallocatechin 3-gallate, genistein, silibinin], carotenoids (beta-carotene, lycopene), and isothiocyanates (sulforaphane), offer exciting platforms for the development of such protective strategies. These phytochemicals have been consumed by humans for many centuries as part of plant-rich diets and are presumed to be of low toxicity, an essential requirement for a chemoprotective agent. Mechanistically, they affect multiple signalling pathways and protect against UV radiation-inflicted damage by their ability to act as direct and indirect antioxidants, as well as anti-inflammatory and immunomodulatory agents. Such "pluripotent character" is a critical prerequisite for an agent that is designed to counteract the multiple damaging effects of UV radiation. Especially attractive are inducers of the Keap1/Nrf2/ARE pathway, which controls the gene expression of proteins whose activation leads to enhanced protection against oxidants and electrophiles. Such protection is comprehensive, long-lasting, and unlikely to cause pro-oxidant effects or interfere with the synthesis of vitamin D.

Publication Types:

- Research Support, Non-U.S. Gov't
- Review

PMID: 18696411 [PubMed - indexed for MEDLINE]

J Nutr Biochem. 2009 May;20(5):389-98. Epub 2008 Jul 24.

Peroxidized cholesterol-induced matrix metalloproteinase-9 activation and its suppression by dietary beta-carotene in photoaging of hairless mouse skin.

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The activation of matrix metalloproteinase (MMP)-9 leading to the formation of wrinkle and sagging of skin is an essential step in the skin photoaging on exposure to ultraviolet A (UVA). This study attempted to elucidate the role of peroxidized cholesterol including cholesterol hydroperoxides (Chol-OOHs), primary products of lipid peroxidation in biomembranes, in MMP-9 activation and the effect of dietary beta-carotene in MMP-9 activation. Hairless mice were subjected to periodic UVA irradiation for 8 weeks. The amount of peroxidized cholesterol detected as total hydroxycholesterol in the skin was increased significantly by the exposure. The activity and protein level of MMP-9 were elevated with wrinkling and sagging formation. MMP-9 activity was also enhanced by the intracutaneous injection of Chol-OOHs into the mouse skin. Adding beta-carotene to the diet of the mice during the period of irradiation suppressed the activity and expression of MMP-9 as well as the wrinkling and sagging formation. The amount of cholesterol 5alpha-hydroperoxide, a singlet molecular oxygen oxygenation-specific peroxidized cholesterol, was significantly lowered by the addition of beta-carotene to the diet. These results strongly suggest that Chol-OOHs formed on exposure to UVA contribute to the expression of MMP-9, resulting in photoaging. Dietary beta-carotene prevents the expression of MMP-9, at least partly, by inhibiting photodynamic action involved in the formation of Chol-OOHs.

Publication Types:

- Research Support, Non-U.S. Gov't

PMID: 18656335 [PubMed - in process]

Mol Biotechnol. 2007 Sep;37(1):26-30.

Carotenoids and flavonoids contribute to nutritional protection against skin damage from sunlight.

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The concept of photoprotection by dietary means is gaining momentum. Plant constituents such as carotenoids and flavonoids are involved in protection against excess light 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, such as skin or the eye where they provide systemic photoprotection. beta-Carotene and lycopene prevent UV-induced erythema formation. Likewise, dietary flavanols exhibit photoprotection. After about 10-12 weeks of dietary intervention, a decrease in the sensitivity toward UV-induced erythema was observed in volunteers. Dietary micronutrients may contribute to life-long protection against harmful UV radiation.

Publication Types:

- Research Support, Non-U.S. Gov't
- Review

PMID: 17914160 [PubMed - indexed for MEDLINE]

Skin Pharmacol Physiol. 2006;19(4):224-31. Epub 2006 May 4.

Antioxidant supplements improve parameters related to skin structure in humans.

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In the present study we investigated the influence of two different antioxidant supplements composed of carotenoids, vitamin E and selenium on parameters related to skin health and skin aging. Thirty-nine volunteers with healthy, normal skin of skin type 2 were divided into 3 groups (n = 13) and supplemented for a period of 12 weeks. Group 1 received a mixture of lycopene (3 mg/day), lutein (3 mg/day), beta-carotene (4.8 mg/day), alpha-tocopherol (10 mg/day) and selenium (75 microg/day). Group 2 was supplemented with a mixture of lycopene (6 mg/day), beta-carotene (4.8 mg/day), alpha-tocopherol (10 mg/day) and selenium (75 microg/day). Group 3 was the placebo control. Upon supplementation serum levels of selected carotenoids increased in both verum groups. Skin density and thickness were determined by ultrasound measurements. A significant increase for both parameters was determined in the verum groups. Roughness, scaling, smoothness and wrinkling of the skin were determined by Surface Evaluation of Living Skin (Visioscan). Roughness and scaling were improved by the supplementation with antioxidant micronutrients. In the placebo group no changes were found for any of the parameters.

Publication Types:

- Controlled Clinical Trial

PMID: 16679825 [PubMed - indexed for MEDLINE]

Hautarzt. 2006 Apr;57(4):286, 288-90.

[Functional food and bioavailability in the target organ skin]

[Article in German]

Darwin M, **Schanzer S**, **Teichmann A**, **Blume-Peytavi U**, **Sterry W**, **Lademann J**.

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Reactive free radicals can be produced in the skin by the action of environmental factors, such as sun radiation and toxins. These radicals can damage the DNA, proteins and lipids of the living cells. The consequences can be skin aging, immune suppression and even skin cancer. Humans have developed a protective mechanism against the action of free radicals in the form of antioxidant substances. Several of these antioxidants cannot be produced by humans and have to be acquired via food, such as carotenoids. Optical, non-invasive methods, like resonance Raman spectroscopy, allow a qualitative and quantitative online detection of the kinetics of antioxidants such as carotenoids in the skin. By employing this method it has been shown that the uptake of carotenoids in food can lead to an accumulation in the skin. On the other hand, stress, illness and UV-radiation can reduce the concentration of antioxidant substances in the skin. A high concentration of antioxidant substances is protective and associated with a reduction in skin wrinkling.

Publication Types:

- English Abstract

PMID: 16485123 [PubMed - indexed for MEDLINE]

Hautarzt. 2006 Apr;57(4):281-5.

[Systemic photoprotection through carotenoids]

[Article in German]

Stahl W, **Krutmann J**.

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Nutritional supplements are increasingly used to protect human skin against environmentally-induced damage, most importantly as a consequence of ultraviolet radiation exposure. beta-carotene is a major constituent of comercially available products administered for systemic photoprotection. Studies on the systemic use of beta-carotene provide evidence that 15-30 mg/d over a period of about 10-12 wk produces a protective effect against UV-induced erythema. Similar effects have been attributed to mixtures of carotenoids or after long-term intake of dietary products rich in carotenoids. Supplementation with carotenoids contributes to basal protection of the skin but is not sufficent to obtain complete protection against severe UV irradiation.

Publication Types:

- English Abstract
- Review

PMID: 16463037 [PubMed - indexed for MEDLINE]

Int J Vitam Nutr Res. 2005 Jul;75(4):227-34.

Effects of administration of beta-carotene, ascorbic acid, persimmons, and pods on antioxidative ability in UV-irradiated ODS rats.

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To evaluate the effects of supplementing diets with carotenoid and ascorbic acid (AsA) on the antioxidative ability of Osteogenic Disorder-Shionogi (ODS) rats, we added synthetic beta-carotene (betaC), AsA, and powders of persimmon (Ka) and pods (Po) containing betaC and AsA to the diet and obtained the following results. The urinary 8-hydroxydeoxyguanosine (8-OHdG) concentration was low in the -betaC.AsA and +AsA groups but high in the +betaC.AsA, +Ka, and +Po groups. The thiobarbituric acid-reactive substances (TBARS) in both the liver and skin were higher in the -betaC.AsA group than in the +betaC.AsA group and were low in the +Ka and +Po groups. As antioxidant enzymes, glutathione peroxidase (GSH-Px) activity was high in the +betaC.AsA group, low in the -beta3C.AsA group in both the skin and liver, and also high in the + Ka and +Po group in the liver. Superoxide dismutase (SOD) activity was high in the -betaC.AsA group and low in the +betaC.AsA and +Ka groups in both the skin and liver. Catalase (CAT) activity in the liver was low in the -betaC.AsA, +AsA, and +betaC groups and high in the +betaC.AsA and +Po groups. These results confirmed that the administration of betaC, AsA, and persimmons and pods increases antioxidative ability in the skin and liver of ultraviolet-b(UV-B)-irradiated ODS rats.

PMID: 16229338 [PubMed - indexed for MEDLINE]

Biochim Biophys Acta. 2005 May 30;1740(2):101-7. Epub 2004 Dec 28.

Bioactivity and protective effects of natural carotenoids.

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Carotenoids comprise a class of natural fat-soluble pigments which are found in numerous fruits and vegetables. The consumption of a diet rich in carotenoids has been epidemiologically correlated with a lower risk for several diseases. The antioxidant activity of carotenoids and biochemical properties influencing signaling pathways have been discussed as basic mechanisms of prevention. Conflicting data from intervention studies with beta-carotene to prevent cancers and cardiovascular disorders have challenged the concept. However, there is convincing evidence that carotenoids are important components of the antioxidant network. Photooxidative damage is suggested to be involved in the pathobiochemistry of several diseases affecting the skin and the eye, and carotenoids may protect light-exposed tissues. Lutein and zeaxanthin are the predominant carotenoids of the retina and are considered to act as photoprotectants preventing retinal degeneration. The unique distribution, localization and high levels of both carotenoids within the macula lutea as well as their physicochemical properties make them suitable candidates for photoprotection. beta-Carotene is used as an oral sun protectant for the prevention of sunburn and has been shown to be effective either alone or in combination with other carotenoids or antioxidant vitamins. Protective effects are also achieved with a diet rich in lycopene.

Publication Types:

- Research Support, Non-U.S. Gov't
- Review

PMID: 15949675 [PubMed - indexed for MEDLINE]

Free Radic Biol Med. 2004 Dec 1;37(11):1854-63.

Participation of singlet oxygen in ultraviolet-a-induced lipid peroxidation in mouse skin and its inhibition by dietary beta-carotene: an ex vivo study.

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Dietary beta-carotene acts as a photoprotective agent in the skin, but the exact mechanism of protection is unknown. This ex vivo study is focused on determining the mechanism of action of beta-carotene against UV-A-induced skin damage by characterizing peroxidized phosphatidylcholine (PC) and beta-carotene oxidation products. BALB/c mice were fed with basal or a beta-carotene-supplemented diet, and homogenates from their dorsal skin were prepared after 3 weeks for UV-A irradiation. Analyses revealed that the degree of lipid peroxidation in the beta-carotene group was significantly lower than that in the controls. The isomeric composition of hydroperoxy fatty acids, constituting peroxidized PC, was determined by thin-layer chromatography-blotting followed by gas chromatography/mass spectrometry (MS)/selected ion monitoring analysis. The 9- and 10-isomers of peroxidized PC, resulting from the reaction of singlet molecular oxygen ((1)O(2)) with oleic acid, were elevated in the UV-A-exposed control group compared to the experimental group. Similar results were obtained from methylene-blue-sensitized photooxidation of mouse skin lipids in vitro. Liquid chromatography/MS analysis of the homogenates confirmed the formation of beta-carotene 5,8-endoperoxide, a specific marker for the (1)O(2) reaction. These results indicate that dietary beta-carotene accumulates in the skin and acts as a protective agent against UV-A-induced oxidative damage, by quenching the (1)O(2).

Publication Types:

- Research Support, Non-U.S. Gov't

PMID: 15528044 [PubMed - indexed for MEDLINE]

Free Radic Biol Med. 2004 Sep 1;37(5):654-70.

Beta-carotene inhibits UVA-induced matrix metalloprotease 1 and 10 expression in keratinocytes by a singlet oxygen-dependent mechanism.

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UVA exposure causes skin photoaging by singlet oxygen (1)O(2)-mediated induction of, e.g., matrix metalloproteases (MMPs). We assessed whether pretreatment with beta-carotene, a (1)O(2) quencher and retinoic acid (RA) precursor, interferes with UVA-induced gene regulation. HaCaT keratinocytes were precultured with beta-carotene at physiological concentrations (0.5, 1.5, and 3.0 microM) prior to exposure to UVA from a Hönle solar simulator (270 kJ/m(2)). HaCaT cells accumulated beta-carotene in a time- and dose-dependent manner. UVA irradiation massively reduced the cellular beta-carotene content. Beta-carotene suppressed UVA-induction of MMP-1, MMP-3, and MMP-10, three major matrix metalloproteases involved in photoaging. We show that regulation by not only MMP-1, but also MMP-10, involves (1)O(2)-dependent mechanisms. Beta-carotene dose-dependently quenched (1)O(2)-mediated induction of MMP-1 and MMP-10. Thus, as in chemical solvent systems, beta-carotene quenches (1)O(2) also in living cells. Vitamin E did not cooperate with beta-carotene to further inhibit MMP induction. HaCaT cells produced weak retinoid activity from beta-carotene, as demonstrated by mild upregulation of RAR beta and activation of an RARE-dependent reporter gene. Beta-carotene did not regulate the genes encoding other RARs, RXRs, or the two beta-carotene cleavage enzymes. These results demonstrate that beta-carotene acts photoprotectively, and that this effect is mediated by (1)O(2) quenching.

PMID: 15288123 [PubMed - indexed for MEDLINE]

Cancer Epidemiol Biomarkers Prev. 2004 Jun;13(6):1042-51.

Diet and melanoma in a case-control study.

Millen AE, **Tucker MA**, **Hartge P**, **Halpern A**, **Elder DE**, **Guerry D 4th**, **Holly EA**, **Sagebiel RW**, **Potischman N**.

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BACKGROUND: Malignant melanoma has been one of the most rapidly increasing cancers within the United States with few modifiable risk factors. This study investigates risk related to dietary factors, which are potentially modifiable. METHODS: Newly diagnosed patients with melanoma (n = 502) were recruited from pigment lesion clinics and controls (n = 565) were recruited from outpatient clinics. To investigate the relationship between melanoma and dietary factors in this case-control study, study subjects were requested to complete a food frequency questionnaire, which assessed diet over the previous year. Using logistic regression, odds ratios (ORs) for melanoma were computed for nutrient and alcohol intake. RESULTS: Persons in high versus low quintiles of energy-adjusted vitamin D, alpha-carotene, beta-carotene, cryptoxanthin, lutein, and lycopene had significantly reduced risk for melanoma (ORs < or = 0.67), which remained after adjustment for presence of dysplastic nevi, education, and skin response to repeated sun exposure. Addition of micronutrients from supplements did not add an additional reduction in risk. High alcohol consumption was associated with an increased risk for melanoma, which remained after adjustment for confounders [OR (95% confidence interval) in highest versus lowest quintiles, 1.65 (1.09-2.49)]. CONCLUSIONS: Diets consisting of foods rich in vitamin D and carotenoids and low in alcohol may be associated with a reduction in risk for melanoma. These analyses should be repeated in large, prospective studies.

PMID: 15184262 [PubMed - indexed for MEDLINE]

Photochem Photobiol Sci. 2003 Jun;2(6):655-9.

Betacarotene supplementation protects from photoaging-associated mitochondrial DNA mutation.

Eicker J, **Kürten V**, **Wild S**, **Riss G**, **Goralczyk R**, **Krutmann J**, **Berneburg M**.

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Mutations of mitochondrial DNA accumulate during normal aging and can be detected at elevated levels in skin prematurely aged by chronic exposure to ultraviolet (UV) light (photoaging). In normal human fibroblasts, we have previously demonstrated that mtDNA deletions are induced by repetitive exposure to sublethal doses of UVA radiation mediated through singlet oxygen. Betacarotene is a known quencher of ROS and singlet oxygen in particular, and it is widely applied in photoprotective compounds. Therefore we investigated whether in our in vitro system, betacarotene is capable of protecting from the induction of photoaging-associated mtDNA deletions. All-E (trans) betacarotene was tested at doses from 0.25 to 3.0 microM for uptake into cells as well as its protective capacity. Assessment of cellular uptake of all-E betacarotene measured by HPLC revealed a dose dependent increase of intracellular concentrations, as well as an increase in oxidative metabolites. UVA-exposure led to a decrease of all-E-betacarotene, its Z-isomers and oxidative metabolites. Assessment of mtDNA deletions by PCR revealed reduced levels of mtDNA mutagenesis in cells coincubated with betacarotene at concentrations of 0.5 microM and higher. Taken together, these results indicate that betacarotene (i) is taken up into the cell in a dose dependent manner, (ii) interacts with UVA radiation in the cell and (iii) shows protective properties from the induction of a photoaging-associated mtDNA mutation.

Publication Types:

- Research Support, Non-U.S. Gov't

PMID: 12859149 [PubMed - indexed for MEDLINE]

Free Radic Biol Med. 2003 Feb 15;34(4):456-64.

Beta-carotene suppresses UVA-induced HO-1 gene expression in cultured FEK4.

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The ultraviolet region of sunlight causes a significant oxidative stress to human skin cells and modulates expression of a series of genes in dermal fibroblasts and other cell types. The human heme oxygenase 1 (HO-1) gene is strongly activated within the first hours that follow UVA irradiation of normal human dermal fibroblasts (FEK4) and this response is being used as a marker of oxidative stress in cells. It has been shown that the induction of this gene occurs via singlet oxygen ((1)O(2)) produced upon interaction of UVA radiation with an as yet undefined cellular chromophore. Carotenoids, as the most potent singlet oxygen quenchers in nature, are expected to effectively suppress the UVA-induced HO-1 gene activation in human cells. In this study, we measured the suppression of UVA-induced levels of HO-1 mRNA after the addition of a series of six all-trans-beta-carotene concentrations (0.07, 0.2, 0.8, 2.3, 8.0, and 21 microM) to the culture medium of exponentially growing FEK4 cells. The corresponding levels of beta-carotene uptake and apo-carotenal formation were measured following HPLC separation. The results of this study show a concentration-dependent suppression of UVA- (250 kJ/m(2)) induced transcriptional activation of HO-1 in exponentially growing FEK4 cells by beta-carotene. Suppression occurred at concentrations that have been observed in human plasma after dietary supplementation with beta-carotene.

PMID: 12566071 [PubMed - indexed for MEDLINE]

J Nutr. 2003 Jan;133(1):98-101.

Supplementation with beta-carotene or a similar amount of mixed carotenoids protects humans from UV-induced erythema.

Heinrich U, **Gärtner C**, **Wiebusch M**, **Eichler O**, **Sies H**, **Tronnier H**, **Stahl W**.

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Carotenoids are useful oral sun protectants, and supplementation with high doses of beta-carotene protects against UV-induced erythema formation. We compared the erythema-protective effect of beta-carotene (24 mg/d from an algal source) to that of 24 mg/d of a carotenoid mix consisting of the three main dietary carotenoids, beta-carotene, lutein and lycopene (8 mg/d each). In a placebo-controlled, parallel study design, volunteers with skin type II (n = 12 in each group) received beta-carotene, the carotenoid mix or placebo for 12 wk. Carotenoid levels in serum and skin (palm of the hand), as well as erythema intensity before and 24 h after irradiation with a solar light simulator were measured at baseline and after 6 and 12 wk of treatment. Serum beta-carotene concentration increased three- to fourfold ($P < 0.001$) in the beta-carotene group, whereas in the mixed carotenoid group, the serum concentration of each of the three carotenoids increased one- to threefold ($P < 0.001$). No changes occurred in the control group. The intake of either beta-carotene or a mixture of carotenoids similarly increased total carotenoids in skin from wk 0 to wk 12. No changes in total carotenoids in skin occurred in the control group. The intensity of erythema 24 h after irradiation was diminished in both groups that received carotenoids and was significantly lower than baseline after 12 wk of supplementation. Long-term supplementation for 12 wk with 24 mg/d of a carotenoid mix supplying similar amounts of beta-carotene, lutein and lycopene ameliorates UV-induced erythema in humans; the effect is comparable to daily treatment with 24 mg of beta-carotene alone.

Publication Types:

- Clinical Trial
- Randomized Controlled Trial

PMID: 12514275 [PubMed - indexed for MEDLINE]

Carcinogenesis. 2002 Aug;23(8):1263-5.

The effect of beta-carotene on lung and skin carcinogenesis.

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The induction of pre-cancerous squamous metaplasia in lungs of ferrets by high doses of dietary beta-carotene (BC) and cigarette smoke is compared with and contrasted to the different effects of high doses of dietary BC on skin papilloma and carcinoma induction by the two-stage carcinogenesis protocol. Whereas high dietary BC can inhibit the conversion of skin papillomas to carcinomas, such treatment would not be expected to inhibit smoke-induced lung tumors.

Publication Types:

- Comparative Study

PMID: 12151342 [PubMed - indexed for MEDLINE]

FEBS Lett. 2001 Dec 7;509(2):186-90.

The effect of beta-carotene on the expression of interleukin-6 and heme oxygenase-1 in UV-irradiated human skin fibroblasts in vitro.

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beta-Carotene is discussed as an anti-oxidant micronutrient and singlet oxygen quencher in human skin, protecting against UV light-induced damage. However, we recently demonstrated that beta-carotene has a pro-oxidant potential in cultured human skin fibroblasts because it enhances the UVA induction of heme oxygenase-1 (HO-1). Herein, we further show that beta-carotene also strongly promotes the UVA induction of pro-inflammatory interleukin-6 (IL-6) in skin fibroblasts in vitro. Singlet oxygen quencher sodium azide abrogated up-regulation of IL-6, and likewise also of HO-1. In UVB-irradiated cells, beta-carotene did not modulate levels of IL-6 and HO-1. The observed effects might be relevant for UV-induced inflammatory processes.

PMID: 11741586 [PubMed - indexed for MEDLINE]