Flavonoids and Skin Health

Summary

Due to first-pass metabolism ([/glossary#first-pass-metabolism]) in the digestive tract and liver, dietary flavonoids are extensively modified before reaching the skin. ([More information])

Topical ([/glossary#topical]) application of flavonoids is a successful way to achieve pharmacological levels of parent compounds in the skin; however, chemical composition of topical formulations greatly influences stability and bioavailability ([/glossary#bioavailability]). ([More information])
Green tea polyphenols exert photoprotective effects when obtained from both dietary and topical sources. (More information)

Animal and in vitro (../../../glossary#in-vitro) experiments suggest that topically applied genistein exerts photoprotective effects. (More information)

Some flavonoids may protect the skin by absorbing UVB and thus functioning as sunscreen. (More information)

Various flavonoids inhibit enzymes (../../../glossary#enzyme) involved in the inflammatory (../../../glossary#inflammation) response and may counteract UV-induced inflammation in the skin. (More information)

Flavonoids can influence endogenous (../../../glossary#endogenous) defense mechanisms in the skin, potentially modulating the response to environmental agents, such as UVR and procarcinogens (../../../glossary#procarcinogen). (More information)

Flavonoids may influence wound healing and blood vessel health, but further research in humans is necessary before clinical efficacy can be determined. (More information)

Overview

Flavonoids are dietary factors that belong to the general class of compounds known as phytochemicals (../../../glossary#phytochemical), or plant chemicals. More than 5,000 varieties of flavonoids have been identified, and hundreds of flavonoids can exist in a single food item (1). Flavonoids consist of a basic polyphenolic ring structure (see Figure 1 (../../../dietary-factors/phytochemicals/flavonoids#figure-1) in the article on Flavonoids) with different side chains attached, thus imparting different properties to the compound (see Table 1 (../../../dietary-factors/phytochemicals/flavonoids#table-1) in the article on Flavonoids). Although often touted for their antioxidant (../../../glossary#antioxidant) properties, the ability of flavonoids to absorb ultraviolet (UV) light and modulate signaling pathways that influence cellular function appears to underlie their beneficial effects in skin health.

Content and availability

Dietary flavonoids are subjected to first-pass metabolism (../../../glossary#first-pass-metabolism) by the gastrointestinal (../../../glossary#gastrointestinal) tract and liver, which results in extensive modification of the ingested compound (see the article on Flavonoids (../../../dietary-factors/phytochemicals/flavonoids)) (2, 3). The biological actions of flavonoid metabolites (../../../glossary#metabolite) likely differ from the parent compound; thus, the flavonoid that reaches the skin may have different effects when obtained from ingested versus topical (../../../glossary#topical) sources (see Topical application).

Human epidermal (../../../glossary#epidermis) keratinocytes (../../../glossary#keratinocyte) express specific members of the organic anion transporting polypeptides (OATP) family, transporters responsible for the uptake of a variety of xenobiotics (../../../glossary#xenobiotic), drugs, and large amphipathic (../../../glossary#amphipathic) molecules (4). It is postulated that flavonoids enter epidermal cells via this route. Once inside the cell, flavonoids can bind catalytic (../../../glossary#catalyze) ATP (../../../glossary#ATP)-binding sites on a diversity of proteins (../../../glossary#protein), thus exerting influence on a wide range of cellular processes (5, 6).
example, genistein and quercetin are known to inhibit tyrosine kinase and PI3 kinase, respectively. Specific interactions between certain flavonoids and cellular proteins also occur. Soy isoflavones (../../../dietary-factors/phytochemicals/soy-isoflavones) for example bind to estrogen (../../../glossary#estrogen) receptor (../../../glossary#receptor)-β (ER-β), the ER isoform expressed in skin cells and the cardiovascular system (7, 8).

**Topical application**

Topically (../../../glossary#topical) applied flavonoids are not subjected to first-pass metabolism (../../../glossary#first-pass-metabolism). Pharmacological doses (../../../glossary#pharmacologic-dose) can be achieved with topical delivery, although solubility, stability, and permeation issues are all concerns. Some specific flavonoid formulations have been evaluated in *in vitro* (../../../glossary#in-vitro) experiments for their ability to permeate excised human skin samples. Penetration enhancers reversibly modify skin barrier properties in order to influence the diffusion of topical agents through the skin. When dissolved in acetone, the flavanones naringenin and hesperitin successfully permeated excised human skin and pretreatment with penetration enhancers (D-limonene and lecithin) increased their permeation (9). The flavonol quercetin, on the other hand, had very low skin permeation under all experimental conditions. In a small sampling of human volunteers (six men and women, aged 25 to 35 years old), Saija et. al. (9) confirmed that topical application of naringenin and hesperitin in the presence of penetration enhancers protected against UVB-induced erythema (../../../glossary#erythema).

The absorption and permeability of epigallocatechin-3-gallate (EGCG) in a hydrophilic ointment was tested in excised human skin (10). Firstly, EGCG in hydrophilic ointment was highly unstable unless stored at low temperature or supplemented with the antioxidant (../../../glossary#antioxidant) BHT. Secondly, EGCG in hydrophilic (../../../glossary#hydrophilic) ointment successfully permeated, but did not exit, the human dermis (../../../glossary#dermis), suggesting minimal accessibility to the systemic circulation. Another *in vitro* study used excised human skin to evaluate the penetration of EGCG and quercetin from a cosmetic formulation (11). After 24 hours, topical EGCG was retained in the stratum corneum (../../../glossary#stratum-corneum), epidermis (../../../glossary#epidermis), and dermis but did not leave the skin sample; quercetin accumulated in the epidermis and stratum corneum but was not detected in the dermis.

Several additional *in vitro* experiments have evaluated the solubility and permeability of flavonoids from a variety of formulations (12-15). Although there is potential for flavonoids as topical photoprotective agents, suitable delivery vehicle, chemical additives, and pH (../../../glossary#pH) significantly influence the permeation of bioactive product. The efficacy of various topical formulations requires further testing in humans.

**Deficiency**

Flavonoids are not considered essential nutrients; therefore, there are no established dietary reference intakes (../../../glossary#DRI) (DRIs) or clinical markers of deficiency. The potential health benefits of consuming a flavonoid-rich diet are discussed more extensively in the article on Flavonoids (../../../dietary-factors/phytochemicals/flavonoids).

**Functions in Healthy Skin**
Photoprotection

Exposure to UV-radiation (UVR) has many negative effects on skin, including erythema, edema, sunburned cells, hyperplasia, inflammation, immunosuppression, photoaging, and photocarcinogenesis (16). Studies performed in cell culture, animals, and humans demonstrate that treatment with certain flavonoids can minimize adverse skin reactions caused by UVR.

Green tea polyphenols

Heinrich et.al. (17) conducted a 12-week, placebo-controlled trial in 60 healthy women (40-65 years of age) to evaluate the effect of dietary green tea polyphenol consumption on skin photoprotection, structure, and function. Subjects consumed 1L of beverage throughout the day, containing 1,402 mg of green tea catechins or inert ingredients matched for additives and flavor. Erythema formation in response to 1.25X MED, skin elasticity and structure (roughness, scaling, volume, and wrinkles), transepidermal water loss (TEWL), cutaneous blood flow, and serum flavonoid concentration were measured at 0, 6, and 12 weeks of treatment. Serum levels of the green tea polyphenols EGCG, ECG, and epicatechin significantly increased in the treatment group at both six and 12 weeks of intake. Additionally, all skin variables measured were significantly improved in those consuming green tea beverage compared to placebo at both time points (17).

Recognizing the challenges of consuming 1L of beverage over the course of a day, Heinrich et.al. performed a small dosing study to assess the effects of green tea extract ingested in capsule form (17). Fifteen healthy women received placebo or 0.5, 1, or 2 g of encapsulated green tea extract. Serum flavonoid content and capillary blood flow to the dermis were measured over the course of four hours in order to assess the short-term effects of a single dose of encapsulated green tea extract. Dermal microcirculation was measured since increased cutaneous blood flow may contribute to enhanced delivery of oxygen and nutrients to the skin, a proposed impact of flavonoids on skin health.

There was an equivalent quick and brief increase in dermal microcirculation (15-30 minutes) at all doses of green tea extract ingested compared to placebo. Serum epicatechin levels increased in a dose-dependent manner, with a maximum concentration two hours post-ingestion.

Ingestion of high-flavanol cocoa powder, rich in epicatechin and catechin, for 12 weeks also improved photoprotection and skin structure in healthy female subjects (18). In this double-blind intervention, 24 female volunteers (18-25 years of age) were randomly assigned to ingest a high (326 mg) or low (27 mg) flavanol-containing cocoa beverage daily for 12 weeks. As with green tea beverage, high-flavanol cocoa powder diminished UV-induced erythema formation, increased microcirculation, improved skin structure (as roughness, scaling, volume, and wrinkles), and reduced TEWL; none of these parameters changed in the low-flavanol group. Additionally, a single dose of high-flavanol (329 mg) cocoa beverage quickly and transiently increased plasma epicatechin levels and dermal microcirculation compared to low-flavanol cocoa beverage (19). The effects of catechins on skin structure, texture, and water homeostasis may be due to their ability to increase cutaneous blood flow (17-19). Vasodilatory mechanisms, which have also been shown for cocoa polyphenols in non-skin vessels, may underlie the vascular...
benefits associated with flavanols (20, 21). For more information on chocolate and cardiovascular health, see the article in the Spring/Summer 2012 LPI Research Newsletter (../../../../files/pdf/newsletters/ss12.pdf#page=14).

Katiyar et al. studied the effects of topically applied green tea extract on several UV-mediated skin responses in humans (22-24). In each study, four to six volunteers (both male and female, aged 25 to 55 years old) received a topical application of purified green tea extract (1 mg/cm² dissolved in acetone to a sun-protected skin) containing a mixture of the four major polyphenols in green tea: epigallocatechin-3-gallate (EGCG), epicatechin (EC), epigallocatechin (EGC), and epicatechin-3-gallate (ECG). Twenty or 30 minutes after topical application, skin sites were exposed to UVB (4X MED). Twenty-four or 48 hours later, punch biopsies (i.e., both epidermis (../../../glossary#epidermis) and dermis) were collected and various endpoints measured. In each case, pretreatment with purified green tea polyphenols inhibited UV-induced inflammation (23), DNA (../../../glossary#DNA) damage (24), and formation of reactive oxygen species (../../../glossary#reactive-oxygen-species) (ROS) (22) compared to vehicle-treated sites on the same individual.

In a similar study design, Elmets et. al. (25) also observed a protective effect of topically applied purified green tea extract on UV-induced photodamage (../../../glossary#photodamage), assessed as erythema formation, presence of sunburn cells, DNA damage, and number of Langerhans cells (../../../glossary#Langerhans-cell), a marker of immunosuppression. A 5% solution that contained a mixture of the four major green tea polyphenols (0.5 g of purified extract dissolved in ethanol/water) was the most effective at minimizing UV-damage compared to vehicle-treated sites in the same subjects. EGCG and ECG were also protective, although to a lesser extent than the mixed extract.

Camouse et. al. (26) performed a double-blind treatment in ten volunteers to compare topical green tea and white tea extracts as photoprotective agents. Pretreatment with either tea extract (2.5 mg/cm² in organic solvent) protected against depletion of Langherhans cells and oxidative DNA damage (../../../glossary#oxidative-damage) caused by UVB exposure (2X MED) compared to vehicle-treated sites on the same individual.

UV damages DNA by causing strand breaks or creating cyclobutane pyrimidine dimers (CPDs), a photoproduct formed when energy derived from UVR is absorbed by DNA, forming an unwanted covalent bond (../../../glossary#covalent-bond) between pyrimidine bases. A cell can either repair the damage or sacrifice itself (apoptosis (../../../glossary#apoptosis)) as a way to protect the organism from mutations (../../../glossary#mutation) and malignant (../../../glossary#malignant) transformation. The mechanism by which green tea polyphenols combat UV-induced cellular damage appears to be due primarily to their induction of DNA repair pathways in the skin (24, 25, 27) and influence on certain immune mediators known as cytokines (../../../glossary#cytokine) (23, 28-31). Because DNA damage initiates immunosuppression, a risk factor for skin carcinogenesis (../../../glossary#carcinogenesis), green tea polyphenols appear to function early in the UV-damage response in the skin.

**Genistein**

The photoprotective effect of genistein has been investigated in animals and in in vitro (../../../glossary#in-vitro) models of human skin. Pretreatment with topical genistein (5 µM, 60 minutes prior to UV exposure) reduced skin roughness and wrinkling and epidermal hyperproliferation in hairless mice that were exposed to daily doses of acute and chronic UVB irradiation (32). As observed
with EGCG, the photoprotective effects of genistein may result from its impact on UV-induced DNA damage as topical genistein decreased CPD formation and restored proliferating cell nuclear antigen (PCNA) expression, a marker of proliferation and DNA repair (32). The authors performed a small study in six men to extend their observations to humans: topical genistein (5 μM/cm²) applied 30 minutes before UVB exposure (1X MED) blocked erythema formation as evaluated photographically 24 hours after treatment (32). Moreover, pretreatment with topical genistein dose-dependently reduced CPD formation and increased PCNA expression in human reconstituted skin samples (33).

**Other flavonoids**

Silymarin is a special type of flavonoid classified as a flavonolignan, part flavonoid and part lignan. Silymarin is present in the seeds of milk thistle (Silybum marianum), and its major bioactive flavonoid is called silibinin. Like green tea polyphenols, topical silymarin minimizes UV-induced photodamage and photocarcinogenesis in animal studies (34). Experiments using primary cultures of normal human epidermal keratinocytes (NHEKs) and transgenic mice indicate that topical silymarin inhibits UV-induced apoptosis and reduces CPD formation in the skin (35). By using cells and animals deficient in nucleotide excision repair, the authors further demonstrated that topical silymarin contributes to photoprotection by upregulating DNA repair processes.

**Sunscreen effect**

Topical application of certain flavonoids may protect skin by absorbing UVR before it can interact with and damage cellular components, thereby providing a sunscreen effect. Major epidermal chromophores (molecules that absorb UV light) include melanin, urocanic acid, amino acids, and nucleic acids (36). Likewise, topically applied flavonoids may protect skin by absorbing UV light and blocking UV penetration. Pycnogenol® (a registered mixture of naturally occurring mono- and oligomeric procyanidins) and honeybush extract (containing the flavanone hesperidin and xanthone mangiferin) absorb light in the UVB range (37, 38). Thus, topical application of these flavonoids would function as sunscreens when applied prior to UV exposure.

**Prevention versus suppression**

The timing of flavonoid administration dictates if the intervention is being used as a preventive or treatment strategy. The majority of information reports on flavonoid administration prior to UV exposure, as a means to prevent UV-induced photodamage. However, flavonoid administration following UV exposure has been evaluated for several flavonoids. Genistein or EGCG dissolved in acetone was applied to hairless mouse skin one or four hours post-irradiation (2X MED), and 24 hours later, epidermal sections were collected and analyzed (39). Both flavonoids decreased the number of sunburn cells, epidermal hyperplasia, and immune suppression even when applied subsequent to UV exposure. Widyarini et al. applied isoflavone extracts (20 μM) from red clover (Trifolium pretense) to the skin of hairless mice immediately following UV exposure in order to evaluate their ability to protect against acute effects induced by UVR (40). Genistein and the isoflavone metabolites equol, isoequol, and dehydroequol significantly reduced inflammation, edema, and immunosuppression caused by UV exposure. In a randomized, double-blind, placebo-controlled trial, Casetti et al. (41)
compared a luteolin-rich Reseda extract (RE) to hydrocortisone, a standard anti-inflammatory agent, for its efficacy following UV exposure. Forty healthy volunteers (both sexes, 18 years of age and older) were exposed to UVB (1.5X MED) followed by immediate application of a topical nanoparticle formulation of RE (2.5%), hydrocortisone (0.1%), or vehicle (glycerol). Compared to vehicle, both RE and hydrocortisone significantly reduced UVB-induced erythema to a similar extent (41).

The benefit of suppressing the sunburn response in order to minimize skin damage is a subject of debate. Preventing sun damage in the first place is advised as primary protection against the damaging effects of UVR.

**Photoaging**

*Green tea polyphenols*

The long-term influence of oral supplementation with green tea polyphenols on clinical and histological (\[\text{histology}\]) signs of photoaging was evaluated in a two-year, double-blind (\[\text{double-blind}\]), placebo-controlled trial (42). Fifty-six healthy female volunteers (aged 25-75 years old) received either 250 mg green tea polyphenols or placebo twice daily for two years. Photodamaged (\[\text{photodamage}\]) facial skin appearance and histology were evaluated by a dermatologist at 0, 6, 12, and 24 months for wrinkling, hyperpigmentation, depigmentation, lentigines (liver spots), pore size, roughness, erythema (\[\text{erythema}\]), telangiectasias (permanent dilation of superficial blood vessels), and overall solar damage. Although some skin parameters were improved in green tea over placebo at 12 months, no significant differences were observed between the groups after 24 months of treatment, with both groups showing improvements in overall solar damage and elastosis (abnormal accumulation of elastin) (42).

A small double-blind, placebo-controlled pilot study evaluated the impact of combined oral and topical (\[\text{topical}\]) treatment with green tea extract on skin appearance and histology in female subjects with moderate photoaging (43). Forty healthy women were randomly assigned to green tea treatment (10% green tea extract cream applied to the face and arms plus 300 mg green tea extract in an oral supplement) or placebo cream and supplement, both received twice daily for eight weeks. Self-reported grading of wrinkles and roughness were the same in placebo and treatment groups, while several patients in the green tea group complained of irritation, drying, and sun sensitivity at the site of application. Physician assessment of skin appearance found no significant differences between treatment and placebo groups. Histological examination revealed an improvement only in elastic tissue content in the green tea group compared to placebo (43).

**Genistein**

Because estrogen (\[\text{estrogen}\]) has a significant effect on skin aging (7), the isoflavone genistein has been investigated for its potential to counteract signs of photoaging in postmenopausal women. In a pilot study, 30 postmenopausal women who ingested a concentrated soy-extract (100 mg daily for six months) showed a significant increase in skin thickness, elastic fiber content, collagen (\[\text{collagen}\]) fiber content, and vasculature in a gluteal skin biopsy after six months of treatment compared to baseline (44). Moraes et. al. performed a randomized (\[\text{randomized}\]), double-blind estrogen-controlled trial to evaluate the effect of topical isoflavones on morphological parameters in postmenopausal facial skin (45). Forty subjects applied either estrogen (0.01% 17-β-estradiol) or isoflavone (4% genistein) gel to their facial skin daily...
for 24 weeks. Topical estrogen significantly improved all parameters measured compared to baseline and to isoflavone treatment. Isoflavones significantly improved epidermal thickness and blood vessel number after 24 weeks of treatment, though to a lesser extent than that of estrogen treatment.

Xenobiotic metabolism

Skin is both a physical and biochemical barrier (46). Inactivation of potentially harmful compounds via xenobiotic metabolism in the skin serves as a second line of defense against substances that penetrate the skin surface (46-48). Xenobiotic metabolism involves a series of enzymatic reactions that convert a foreign chemical compound into an inert substance that can be safely excreted from the body (49, 50). In phase I, also referred to as activation, oxygen is used to form a reactive site on the xenobiotic compound; members of the cytochrome P450 (cytochrome-P450) family of enzymes participate in phase I metabolism. Phase II, or conjugation, involves the addition of a water-soluble functional group to the reactive site of the phase I metabolite. And finally, in phase III, the solubilized compound is expelled from the cell.

Monoinduction of phase I enzymes without the concomitant induction of phase II enzymes can lead to the production of “activated” compounds that may cause cellular damage. Epidermal CYP 1A1 and 1B1 are induced in response to UVB exposure in a time- and dose-dependent manner (51). CYP 1A1 and 1B1 activate numerous compounds from exogenous substrates, including polycyclic aromatic hydrocarbons (PAH) a well-known class of procarcinogens. Thus, through its induction of phase I enzymes, UVB could enhance the activation of environmental pollutants, further increasing the mutagenic load in the epidermis (53). Flavonoid modulation of enzymes involved in xenobiotic metabolism may thus represent another mechanism for counteracting UV-induced photodamage.

Different flavonoids have variable effects on xenobiotic metabolism in the skin by targeting phase I or phase II components of the cellular detoxification pathway. The flavonols myricetin and quercetin can inhibit aryl hydrocarbon hydrolase (a phase I enzyme) activity when applied topically to mouse skin, potentially preventing the metabolic activation of procarcinogens and the formation of DNA adducts (54) and the formation of DNA adducts (55). On the other hand, flavonoids that induce phase II enzymes could facilitate the inactivation of CYP-generated metabolites. Oral administration of silibinin, the active component of silymarin, for 15 days significantly induced phase II enzyme activity (glutathione S-transferase and quinone reductase) in mouse skin compared to vehicle-treated control mice (56).

Wound healing

Onion extract, rich in the flavonoids quercetin and kaempferol, has been used to reduce scar formation, particularly keloid scars. Cho et al. demonstrated that onion extract and quercetin induce matrix metalloproteinase-1 (MMP-1) expression in cultured human skin fibroblasts and hairless mouse skin. MMPs are enzymes secreted by epidermal keratinocytes and dermal fibroblasts in response to various stimuli, including UVR, oxidative stress, and inflammatory cytokines.
UVR induces three MMPs: MMP-1 (collagenase), MMP-3, (stromelysin), and MMP-9 (gelatinase) that cleave and degrade skin collagen and contribute to photoaging. In the case of wound healing, a balance between MMP-1 and tissue inhibitor matrix metalloproteinase-1 (TIMP-1) enzymatic activity affects the amount of extracellular matrix (including collagen) formed at the wound site. Thus, quercetin may influence extracellular matrix deposition during wound healing in order to reduce hypertrophic scarring.

Other functions

Blood vessel health

Flavonoids, especially rutin and its derivatives, can benefit skin by influencing blood vessel permeability and fragility. Their protective effect on blood vessels may reduce the formation of telangiectasias (small dilated blood vessels near the surface of the skin) and petechiae (small red spots caused by broken capillaries or blood vessels). It appears that flavonoid binding of metals leads to inhibition of enzymes involved in blood clotting and inflammation, which in turn influence capillary permeability and platelet aggregation. However, clinical experimentation is lacking and more human studies are needed to conclusively establish a role for specific flavonoids on blood vessel health.

Conclusion

The majority of information on flavonoids and skin health relates to photoprotective effects of green tea polyphenols, catechins, and genistein. Both oral supplementation and topical administration of the flavanol subclass in particular have demonstrated photoprotective effects in humans. Experimentation with topically applied flavonoids typically test purified compounds or concentrated plant extracts dissolved in organic solvent; although they show promise as photoprotective agents, delivery is an issue that can influence how commercially available formulations penetrate and function in human skin. Flavonoids exert a wide range of influence due to their specific and nonspecific affinity for a diversity of proteins throughout the cell. The precise mechanisms by which flavonoids protect skin from the damaging effects of UVR are still being investigated, but there is evidence that flavonoids physically block UV penetration, influence DNA repair, reduce oxidative damage, attenuate the inflammatory response, preserve immune function, and induce cytoprotective pathways.

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References


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Bone Health (/mic/micronutrients-health/bone-health)

Cognitive Function (/mic/micronutrients-health/cognitive-function)

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Inflammation (/mic/micronutrients-health/inflammation)

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Skin Health (/mic/micronutrients-health/skin-health)

Nutrient Index (/mic/micronutrients-health/skin-health/nutrient-index)

Vitamin A (/mic/micronutrients-health/skin-health/nutrient-index/vitamin-A)

Vitamin C (/mic/micronutrients-health/skin-health/nutrient-index/vitamin-C)

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