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Review

A comprehensive approach to skin protection against ultraviolet radiation: Integrating sunscreens, antioxidants, DNA repair agents, oral supplements, and probiotics

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Abstract

As melanoma rates continue to rise, there is an increasing need for comprehensive strategies to mitigate the harmful effects of ultraviolet (UV) radiation. This review explores an integrative approach to photoprotection that combines traditional mineral and chemical UV filters with emerging protective strategies, including antioxidants, oral supplementation, DNA repair agents, and probiotics. By addressing UV-induced damage at multiple levels, this multifaceted approach may enhance skin protection and help reduce the long-term risk of photoaging and skin cancer.

Introduction

The damaging effects of sun exposure and ultraviolet (UV) radiation have been extensively studied in dermatology, with a growing understanding of their role in both cutaneous malignancies and premature skin aging. UV radiation is a primary causative agent of melanoma and nonmelanoma skin cancers, including basal cell carcinoma and cutaneous squamous cell carcinoma (SCC), as well as a significant contributor to photoaging.[1] Sunlight, the most common source of UV radiation, can cause both acute and chronic skin damage, including erythema, pigmentary changes, and DNA mutations, which may culminate in skin cancer and other long-term dermatologic consequences.

The skin has evolved several natural defense mechanisms to combat the harmful effects of UV radiation, including epidermal thickening, DNA repair processes, antioxidant enzymes, and the synthesis of skin pigments such as melanin.[1] These mechanisms are essential for maintaining skin homeostasis and protecting against the mutagenic effects of UV exposure. However, prolonged or intense UV radiation can overwhelm these protective systems, leading to irreversible damage and an increased risk of malignancy.[2]

UV radiation is categorized into 3 primary wavelengths: UVA (320–400 nm), UVB (290–320 nm), and UVC (200–290 nm), each with distinct biologic effects on the skin. Midday sunlight consists of approximately 95% UVA and 5% UVB, with UVC and much of UVB absorbed by the stratospheric ozone layer before reaching Earth's surface.[3] UVB is primarily absorbed by the epidermis and superficial dermis, whereas UVA penetrates more deeply into the dermis, exerting longer-lasting effects on connective tissue and cellular structures. Approximately 70% of UVB radiation is absorbed by the stratum corneum, 20% penetrates the viable epidermis, and 10% reaches the superficial dermis. In contrast, 20% to 30% of UVA radiation penetrates into deeper dermal layers.[4]

The irradiance of UVA and UVB depends on multiple geo-orbital and environmental factors, including latitude, season, and weather; however, UVA is less affected by these variables than UVB. Additionally, UVA can penetrate glass, allowing for significant indoor exposure, whereas UVB is largely blocked. UVA plays a central role in photoaging and photocarcinogenesis by modulating genetic pathways, generating reactive oxygen species, and inducing oxidative damage to lipids, proteins, and DNA. This damage contributes to dyschromia, wrinkling, loss of elasticity, sagging, and dryness.[4-8] In contrast, UVB and UVC induce mutagenic damage by forming cy-

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clobutane pyrimidine dimers and 6–4 photoproducts, which interfere with cellular replication and repair, increasing skin cancer risk.[5,6,9] UVB is also more erythematogenic, causing acute effects such as sunburn and tanning.[1,8]

Despite widespread sunscreen use, many individuals lack sufficient understanding of effective UV protection. This is compounded by improper application practices, including inadequate coverage, insufficient reapplication, and failure to account for full-spectrum UV exposure.[10] This knowledge gap highlights the need for improved public education. Herein, we review the current literature on sun protection, focusing on an integrative approach that includes sunscreen, dietary strategies, antioxidant supplementation, and complementary methods that may synergistically enhance skin defense against photoaging. These approaches may help mitigate photooxidative stress and reduce the risk of melanoma and other UV-induced skin cancers, promoting long-term skin health.

Discussion

Overview of Sunscreens and UV Filters

Sunscreens are over-the-counter products designed to prevent skin cancer, sunburn, and photoaging. Two primary metrics are used to determine sunscreen efficacy: sun protection factor (SPF) and substantivity. SPF is a ratio that measures the relative amount of time it takes for sunscreen-protected skin to begin reddening compared with unprotected skin. Substantivity refers to the ability of a formulation to remain efficacious despite removal of the vehicle through external factors such as perspiration.[11]

Sunscreens with an SPF of 15 or higher have been associated with a reduced risk of both melanoma and nonmelanoma skin cancers, and daily use of UV-protective agents prevents photodamage.[12-15] Consistent use of broad-spectrum sunscreen, which protects against both UVA and UVB rays, with an SPF of 30 or higher, as recommended by the American Academy of Dermatology, should be incorporated into a comprehensive photoprotection regimen. This regimen also includes wearing protective clothing, hats, and sunglasses, as well as practicing sun avoidance and seeking shade.[16]

Despite these recommendations, surveys indicate that a substantial proportion of individuals do not apply sunscreen regularly, and many apply amounts below recommended guidelines.[17-19] In the United States, only 33.7% of adults report routine sunscreen use.[20] The American Academy of Dermatology recommends applying 1 ounce of sunscreen to cover the entire body, with reapplication every 2 hours or more frequently with perspiration or water exposure. However, studies show that consumers typically apply insufficient quantities, ranging from 0.39 to 1.0 mg/cm², well below the recommended 2 mg/cm², and fail to reapply as frequently as needed.[10,21] Common issues contributing to inadequate protection include missed areas and application after UV exposure, both of which reduce efficacy.[10]

Sunscreen ingredients are typically categorized as mineral (inorganic) or chemical (organic), with most formulations primarily absorbing UV radiation.[22] Each type has distinct characteristics in terms of efficacy, safety, and user experience. The following sections review the ingredients, mechanisms of action, advantages, and limitations of both mineral and chemical sunscreens before examining the emerging role of combination sunscreens and synergistic formulations.

Mineral Sunscreens

Mineral sunscreens are composed of metal oxide nanoparticles, most commonly zinc oxide (ZnO) and titanium dioxide (TiO_2). Mechanistically, these inorganic nanoparticles reduce UV burden through 2 primary methods: absorption and scattering.[23] Absorption accounts for approximately 90% of UV attenuation, with the remaining 10% attributed to the scattering, or refractive, effect of UV photons.[22] These inorganic sunscreens offer several advantages, including broad-spectrum UV protection, high SPF, increased photostability, limited skin penetration, and reduced irritancy owing to their chemically inert properties. However, because of the large particle size of their active ingredients, mineral sunscreens often leave a white cast on the skin, particularly in individuals with skin of color, which may reduce user compliance.[23,24]

Despite their similarities, ZnO and TiO_2 differ in their protective properties. TiO_2 primarily attenuates UVB radiation and blocks shorter UVA wavelengths (320–340 nm), whereas ZnO provides broader UVA protection. For this reason, combination formulations containing both TiO_2 and ZnO are often used to leverage their complementary UV-protective properties.[23,25] Both nanoparticles have been deemed safe for topical human use by the US Food and Drug Administration; however, concerns remain regarding their photocatalytic properties, which may induce cytotoxicity and genotoxicity.[25,26]

Tinted mineral sunscreens use compounds such as iron oxides and pigmentary TiO_2 to protect against both visible and UV light. These formulations are especially helpful for individuals with hyperpigmentation disorders or visible light–induced dermatoses.[27]

Chemical Sunscreens

Chemical sunscreens are composed of carbon-based compounds and can be divided into 5 main types: para-aminobenzoic acid (PABA) derivatives, benzophenones, salicylates, cinnamates, and others.[28] These organic filters act exclusively by absorbing UV radiation and converting it into thermal energy. Compared with mineral sunscreens, chemical sunscreens are less visible when applied to the skin. However, they do not provide visible light protection, often lack complete UVA coverage unless specifically formulated as broad-spectrum, and are associated with an increased risk of systemic absorption, skin irritation, and photodegradation.[24,29] To improve stability, manufacturers may add photostabilizers, including diethylhexyl 2,6-naphthalate, bumetrizole, and benzotriazolyl dodecyl *p*-cresol, to sunscreen formulations.[30]

The type of UV coverage provided by chemical sunscreens depends on the specific organic filter. PABA and its derivatives, such as padimate O, primarily provide UVB protection. Benzophenones, including dioxybenzone and oxybenzone, provide UVB and UVA2 (320–340 nm) coverage. Salicylates and cinnamates consistently protect against UVB and may provide UVA2 coverage depending on the specific compound. Other chemical filters, including methyl anthranilate, provide UVA1 (340–400 nm) protection.[31]

Combination Sunscreens

Protection from harmful UV radiation can be enhanced by combining multiple UV filters, both organic and inorganic, into a single formulation. Such combination sunscreens improve photostability, achieve broader UV coverage, and allow higher SPF levels to be reached.[32,33] For example, Ma et al[32] demonstrated that encapsulating TiO_2 and diethylamino hydroxybenzoyl hexyl benzoate (DHHB) in mesoporous silica nanoparticles and coating them with silicon dioxide (SiO_2) created a synergistic effect in UV blocking. The SiO_2 coating minimized skin absorption of DHHB and reduced TiO_2 photocatalysis, providing complementary UV protection while limiting adverse effects.[32]

Similarly, El-Boury et al[33] found that certain combinations of organic and inorganic filters produced a synergistic increase in SPF. TiO_2 combined with anisotriazine or octyldimethyl PABA enhanced SPF, and ZnO combined with 11 organic filters, including phenylbenzimidazole sulfonic acid, octocrylene, octyl methoxycinnamate, PEG-25 PABA, isoamyl *p*-methoxycinnamate, octyltriazone, diethylhexylbutamidotriazone, 3-benzylidene camphor, octyldimethyl PABA, benzophenone-5, and polysilicone-15, produced similar synergistic effects. These findings highlight how multiple filters can be combined to develop sunscreens with improved efficacy and broader protection.

Overview of Antioxidants

Photodamage occurs when free radicals generated by UV light cause degenerative changes in the skin. Characteristic manifestations of photodamage include telangiectasias, lentigines and ephelides, rhytides, volume loss, and cutaneous malignancies.[2] To combat photodamage, many dermatologists recommend supplementing with exogenous antioxidants, as the body's intrinsic antioxidants may become overwhelmed by UV-induced free radicals.[2] Antioxidants act by neutralizing reactive oxygen species (ROS).[34] Commonly recommended antioxidants include vitamin C, vitamin E, nicotinamide (vitamin B3 derivative), ferulic acid, carotenoids, retinoids, and polyphenols (**Table 1**).

Vitamin C

Vitamin C is the most abundant antioxidant in human skin and plays a key role as a cofactor for collagen biosynthesis. Vitamin C may be administered orally or topically; however, its role in dermatology as a topical agent has been more thoroughly described in the literature. Common topical formulations of vitamin C include L-ascorbic acid, ascorbyl-6-palmitate, and magnesium ascorbyl phosphate.[35] Importantly, vitamin C is equally effective against oxidation caused by both UVB and UVA radiation.[57] Mechanistically, vitamin C acts by inhibiting activator protein-1 (AP-1), a transcription factor upregulated by UV-generated ROS. The inhibition of AP-1 by vitamin C leads to a reduction in the activity of the skin's matrix metalloproteinases (MMPs)—enzymes chiefly responsible for collagen damage and elastin production.[35]

One clinical trial involving 10 patients found that individuals who used 10% topical vitamin C experienced significant reductions in photoaging scores ($P \leq .01$) and improvements in wrinkling when compared to the placebo.[36] Moreover, many studies have demonstrated histological improvements in the density and organization of collagen networks after treatment with vitamin C.[36,37] In addition to protection against photoaging, vitamin C has been shown to protect against UV-induced immunosuppression by preventing the reduction of antigen-presenting Langerhans cells in the epidermis. Similarly, both laboratory and clinical studies have established the role vitamin C plays in reducing UV-induced erythema and thymine dimer mutations, thereby decreasing the risk of photocarcinogenesis.[35]

Vitamin E

Vitamin E is the body's major lipid-soluble antioxidant, and it is especially abundant in the stratum corneum. There are 8 molecular forms of vitamin E, which may be broken down into 2 groups: the tocopherols and the tocotrienols. The difference between the tocopherols and the tocotrienols lies in their phenyl tail, as each group is identified by their linear, saturated tails and nonlinear, unsaturated tails, respectively. During times of oxidative stress, ROS from UV radiation may attack membrane lipids in the stratum corneum, leading to a chain reaction involving the production of more free radicals and further lipid membrane damage. Vitamin E's primary role as an antioxidant is to prevent lipid peroxidation and maintain the structural integrity of the membrane lipids at the level of the stratum corneum.[58]

Earlier laboratory studies in animals revealed that the topical application of vitamin E was successful in protecting the skin's natural antioxidant enzymes (superoxide dismutase and glutathione peroxidase) and in inhibiting the formation of dimeric DNA-damaging photoproducts (cyclobutene pyrimidine dimers).[59,60] In another more recent clinical trial, Pedrelli et al[38] demonstrated vitamin E's significantly superior ability to protect against UVB-induced damage when compared to the vehicle-only control or retinol. In this study, areas of skin treated with

Table 1. *Summary of the Photoprotective Effects of Antioxidants.*

Antioxidants	Formulation/Types	Mechanism of Action	Key Findings
Vitamin C	Oral/topical (L-ascorbic acid, ascrobyl-6-palmitate, magnesium ascorbyl phosphate, etc)	Inhibits AP-1, reduces MMP activity, decreases UV-induced erythema, inhibits formation of DNA-damage photoproducts, and enhances collagen biosynthesis[35–37]	Protects against both UVB- and UVA-induced oxidation, reduces photoaging, improves collagen density, protects against UV-induced erythema, and lowers photocarcinogenesis risk[35-37]
Vitamin E	Oral/topical (tocopherols, tocotrienols)	Prevents lipid peroxidation, stabilizes cell membranes, protects body's natural antioxidant enzymes, and inhibits the formation of DNA-damaging photoproducts[39–41]	Reduces symptom intensity across erythema, edema, itch, vesiculation, lesion extension, and total symptom burden; and improves sunscreen efficacy (eg, as soybean oil)[38,39]
Nicotinamide (Vitamin B3 derivative)	Oral/topical	Delays stem cell differentiation, enhances proliferation, and protects keratinocytes from UVB-induced damage[40,41]	Reduces nonmelanoma skin cancer risk, delays tumor onset, and lowers SCC and AK rates[42,43]
Ferulic acid	Topical	Stabilizes phenoxy radicals, protects against thymine dimer generation, and inhibits UVB-induced MMPs[44,45]	Reduces erythema intensity when combined with vitamins C and E and decreases signs of UVB damage (collagen fiber degradation, abnormal elastin fiber accumulation, epidermal hyperplasia)[49–51]
Curcumin	Oral/topical (plant-based)	Activates antioxidant pathways, neutralizes UV-induced free radicals, and reduces inflammation[46]	Reverses UVA- and UVB-induced damage by reducing catalase production, increasing cell apoptosis, and inducing Nrf2 transcription[46]
Other polyphenols	Oral/topical (green tea, propolis, chlorophyllin)	Antioxidant, anti-inflammatory, and immunomodulatory properties[47]	Improves photodamage (eg, lentigines) and enhances sunscreen efficacy via antiradical properties[53–55]
Astaxanthin (carotenoid)	Oral/topical	Scavenges superoxide radicals, enhances DNA repair via the protein kinase B pathway, and prevents MMP activation[48]	Improves skin elasticity, decreases skin pigmentation, and lessens overall UV-induced damage[49]
Lutein/ zeaxanthin (carotenoid)	Oral/topical	Reduces UV-induced immunosuppression and hyperplasia, and decreases lipid peroxidation[60–62]	Improves skin hydration and elasticity by protecting against skin lipid damage[50,51]
Lycopene (carotenoid)	Oral	Reduces MMP activity and decreases mtDNA damage[52]	Decreases erythema formation[52]
Retinoids	Topical (tretinoin, all-trans retinoic acid)	Increases cell turnover and collagen production[53]	Reduces fine and coarse wrinkle surface area, improves skin texture, increases collagen deposition, and decreases hyperpigmentation[66–68]
Caffeine	Oral/topical	Activates autophagy, removes free radicals, enhances UVB-induced apoptosis, and prevents DNA photodamage[54, 55]	Reduces risk of nonmelanoma and melanoma skin cancers and decreases sunburn lesions[55,56]

Abbreviations: AK, actinic keratosis; AP-1, activator protein-1; MMP, matrix metalloproteinase; mtDNA, mitochondrial DNA; Nrf2, nuclear factor erythroid 2–related factor 2; SCC, squamous cell carcinoma; UV, ultraviolet.

vitamin E revealed significantly lower mean values describing symptom intensity across the following 6 domains: extension of lesion ($P < .001$), erythema ($P < .001$), edema ($P < .001$), itch ($P < .001$), vesiculation ($P < .05$), and total symptom burden ($P < .001$).[38]

Other studies have detailed the photoprotective effects of certain products with naturally high vitamin E levels, such as soybean oil. Soybean oil has been found to prevent 30%–50% of DNA damage in UVC-exposed cells, as measured by the Comet assay.[39] A study also indicated that sunscreen containing soybean oil had a significantly higher SPF value (21.57 ± 1.21) compared to sunscreen without soybean oil, suggesting the importance of vitamin E supplementation in sunscreen formulations.[5]

Nicotinamide (Vitamin B3 Derivative)

Nicotinamide delays stem cell differentiation and enhances proliferation.[40] It has been shown to combat the effects of UVB radiation–induced keratinocyte differentiation, DNA damage, and metabolic decline.[40] Nicotinamide exhibits photoprotective characteristics against cancer and UV-induced immune suppression in both humans and mice, making it a useful defense against UV-induced damage to keratinocytes.[41] A study involving mice demonstrated that oral nicotinamide delayed tumor onset and reduced tumor burden ($P \leq .015$) compared to control groups.[42] In another study, participants with a history of nonmelanoma skin cancer were given 500 mg of nicotinamide daily for 12 months, which resulted in a 23% reduction in new nonmelanoma skin cancers compared to a placebo group.[40] Additionally, actinic keratosis rates were 13% lower ($P = .001$), and new SCC rates were reduced by 30% ($P = .05$).[43] These benefits were not observed after the cessation of nicotinamide supplementation.[43]

Polyphenols

Polyphenols are a large, diverse group of naturally occurring compounds derived from vegetables, fruits, grains, and beverages. They are frequently recommended by dermatologists for the prevention of UV-induced photodamage owing to their anti-inflammatory, immunomodulatory, and antioxidant properties.[47] Examples of polyphenols include ferulic acid, curcumin, chlorophyllin, green tea extract, *Eclipta prostrata*, and propolis extract.

Ferulic acid's effectiveness as an antioxidant may be attributed to its molecular structure, consisting of a phenolic nucleus and a long side chain that stabilizes phenoxy radicals. Staniforth et al[44] described ferulic acid's inhibitory effect on UVB-induced MMPs in mouse skin using immunohistochemical analyses. Ferulic acid successfully reduced UVB damage, including collagen fiber degradation, abnormal elastin fiber accumulation, and epidermal hyperplasia. Other studies have investigated ferulic acid in combination with vitamins C and E in a topical antioxidant complex.[45,61] Murray et al[45] found that this combination significantly reduced erythema intensity and the number of sunburn cells compared to vehicle-only control and provided near-complete protection against thymine dimer formation and p53 induction.

Curcumin, a naturally occurring polyphenolic compound from *Curcuma longa* (turmeric), has been used for centuries in traditional medicine and as a dietary spice.[46] It is best known as the primary bioactive component responsible for turmeric's yellow color and exhibits potent anti-inflammatory, antioxidant, and anticancer properties. Curcumin reverses UVA- and UVB-induced damage in HaCaT keratinocytes by reducing catalase production, increasing apoptosis, and promoting Nrf2 (nuclear factor erythroid 2–related factor 2) transcription.[46]

Other polyphenols, including chlorophyllin, green tea extract, *Eclipta prostrata*, and propolis extract, have been investigated for photoprotective properties. Chlorophyllin extract improves photodamage and solar lentigines, enhancing skin elasticity.[62] Green tea and *Eclipta prostrata* act as sunscreen boosters when combined with octocrylene.[7] Propolis extract promotes Nrf2 nuclear translocation after UVA exposure, demonstrating antioxidant potential.[63,64]

Carotenoids

Carotenoids are a subset of phytonutrients that enhance the skin's photoprotective ability against UVB-induced erythema, UVA-induced pigmentation, and UVA-induced oxidative stress. Humans cannot synthesize carotenoids intrinsically and therefore must obtain them through a carotenoid-rich diet or supplementation.[65,66]

Astaxanthin is a carotenoid with superoxide-scavenging activity and anti-inflammatory properties.[48] It has immune-modulating and DNA-repair characteristics, potentially influencing the protein kinase B (AKT) pathway, which is vital for DNA repair.[48] Astaxanthin also prevents the activation of MMPs.[48] Oral supplementation of astaxanthin in guinea pigs resulted in improved skin elasticity, decreased pigmentation, and reduced UV-induced damage.[49]

Lutein and zeaxanthin, both non-provitamin A carotenoids from the xanthophyll subclass, are found in the skin after consumption of foods such as poultry and green leafy vegetables.[67] These carotenoids possess anti-inflammatory properties and reduce UV-induced immune suppression and hyperplasia in a dose-dependent manner.[67,68] In elderly subjects with photoaged skin, supplementation with 6 mg lutein and 0.18 mg zeaxanthin daily for 8 weeks significantly decreased skin lipid peroxidation and improved skin hydration.[50] Lutein and zeaxanthin's antioxidant properties are particularly significant in the skin and eyes, the 2 organs most exposed to light.[67] Combined oral and topical carotenoid treatments resulted in a 63% increase in skin lipids over 12 weeks, with notable improvements in skin hydration, elasticity, and photoprotective activity.[51]

Lycopene, found in tomato paste, is another powerful antioxidant classified as a carotenoid. Oral supplementation with lycopene reduced MMP activity and the mitochondrial DNA 3,895-bp deletion, markers of UV damage, in experimental groups compared to controls.[69] After 8 weeks, a diet rich in lycopene decreased erythema formation following solar light exposure.[52] A 10-week regimen of 16 mg lycopene per day also increased carotenoid levels in the blood and skin, leading to reduced erythema.[52]

Retinoids

Retinoids are another class of antioxidants derived from vitamin A. They increase both the rate of cell turnover

Table 2. *Summary of the Photoprotective Effects of DNA-Repair Additives.*

DNA-Repair Additive	Formulations/ Types	Mechanism of Action	Key Findings
T4 Endonuclease V	Topical (Liposomes with T4 Endonuclease V)	Repairs UV-induced DNA damage and reduces inflammation by preventing IL-10 and TNF-α upregulation[73]	Promotes DNA repair and anti-inflammatory effects at treated sites[73]
Photolyases	Topical (enzyme-based)	Repairs UV-induced CPDs via monomerization using blue light/UVA energy[9,74]	Reduces UVB-induced DNA damage and enhances resistance to UV-induced mutations[75]
Acetyl zingerone	Topical	Reduces CPD formation, neutralizes UVA-induced ROS, and mitigates downstream effects of IL-17A[76,77]	Reduces UVA-induced ROS, improves anti-aging effects, and decreases expression of pro-inflammatory genes/ cytokines in skin cells[76,77]
Centella asiatica	Topical (titrated extract, TECA)	Removes ROS, restores fibroblast viability, and modulates gene expression related to proliferation, differentiation, and apoptosis[78,79]	Enhances skin elasticity, collagen expression, reduces UVB toxicity, and improves hydration and skin texture over time[80]

Abbreviations: CPD, cyclobutane-pyrimidine dimer; IL, interleukin; ROS, reactive oxygen species; TECA, titrated extract of *Centella asiatica*; TNF-α, tumor necrosis factor-alpha; UV, ultraviolet.

and collagen production in the skin and have been shown to significantly reduce wrinkle surface area and hyperpigmentation.[53] Tretinoin is considered the gold standard for skin rejuvenation.[70] It has been shown to reduce fine and coarse wrinkling, improve skin texture, and decrease hyperpigmentation.[6,70] All-trans retinoic acid significantly increases type I collagen deposition in artificial skin-resembling cultures, further demonstrating its potential for skin repair.[71]

Caffeine

Caffeine is a naturally occurring antioxidant that has inhibitory effects on UVB-induced carcinogenesis and cell senescence.[54,55] Mechanistically, caffeine activates autophagy to remove free radicals, with its primary target being the adenosine A2a receptor.[54] Additionally, caffeine enhances UVB-induced apoptosis via both p53-dependent and p53-independent mechanisms, promoting the elimination of UV-damaged keratinocytes before they become cancerous.[55]

Both oral and topical caffeine formulations have demonstrated success in preventing and treating UVB-induced skin damage.[55,56] Consumption of caffeinated beverages has been associated with a significantly reduced risk of basal cell carcinomas in both retrospective (OR, 0.60; 95% CI, 0.38–0.96) and prospective studies (RR, 0.84; 95% CI, 0.80–0.87). Likewise, the Nurses' Health Study II reported an inverse association between daily caffeine intake and melanoma rates; individuals with higher caffeine consumption had significantly lower rates of melanoma in sun-exposed areas (HR, 0.71; 95% CI, 0.59–0.86; $P = .0001$).[56]

Topical caffeine has also been investigated for its photoprotective effects in both pre- and post-radiation states. Mice treated with topical caffeine before UV exposure demonstrated fewer thymine dimer–positive cells and sunburn lesions. Similarly, topical application of caffeine was associated with a dose-dependent increase in UVB-induced apoptosis immediately after radiation exposure.[55]

Overview of DNA-Repair Additives

Conventional mineral and chemical sunscreens serve as prophylactic measures against UV damage. However, these sunscreens cannot reverse DNA damage once it has occurred. Recent studies have investigated supplementing sunscreen formulations with DNA-repair additives.[72] In this section, we discuss the following DNA-repair supplements: T4 endonuclease V, photolyases, acetyl zingerone, and *Centella asiatica*. These DNA-repair supplements are summarized in **Table 2**.

T4 Endonuclease V

T4 endonuclease V has been studied for its potential to repair UV-induced DNA damage. In 1 study, liposomes containing T4 endonuclease V were applied to the skin of patients irradiated with UV radiation at different time intervals.[73] The enzyme successfully entered cells, and immunohistochemical stains demonstrated a trend toward DNA repair at treated sites. It also nearly prevented the upregulation of interleukin-10 and tumor necrosis factor-alpha in affected areas, suggesting significant anti-inflammatory effects in the context of UV-induced damage.[73]

Photolyases

Photolyases are another group of enzymes crucial for DNA repair. UV radiation induces cyclobutane-pyrimidine

Table 3. *Summary of the Photoprotective Effects of Probiotics.*

Probiotics	Formulations/ Types	Mechanism of Action	Key Findings
Lactobacillus	Oral/topical (eg, *Lactobacillus plantarum* HY7714 and *Lactobacillus johnsonii*)	Produces EPS that strengthen intestinal cells, downregulate MMPs, and reduce ROS[82,83]	Protects against UV-induced cytotoxicity, improves skin hydration, reduces DNA damage, and enhances immune response of host keratinocytes[82,83]
Streptococcus thermophilus	Oral/topical	Enhances structural integrity of the epidermis and protects against ROS[83,84]	Increases hydration of the epidermal barrier by reducing transepidermal water loss[83,84]
Bifidobacterium breve	Oral/topical	Enhances structural integrity of the epidermis and reduces IL-1β expression[85]	Increases hydration of the epidermal barrier and reduces UV-induced epidermal thickening[85]

Abbreviations: EPS, exopolysaccharides; IL-1β, interleukin-1 beta; MMP, matrix metalloproteinase; ROS, reactive oxygen species; UV, ultraviolet.

dimers (CPDs) and other mutagenic photoproducts, which photolyases repair via photoreactivation.[9,74] Photolyases use blue light/UVA energy to monomerize dimers, removing the bulk of UV-induced CPDs through absorption of light in the 300–600 nm range.[75] Overexpression of photolyases has been shown to induce resistance to UVB-induced DNA damage, making them key mediators of UVB sensitivity.[75]

Acetyl Zingerone

Acetyl zingerone (AZ) reduces ongoing CPD formation in melanocytes, even after UV exposure has ceased.[76] When applied after UVA exposure, AZ reduces UVA-induced ROS in keratinocytes and provides multiple photostable benefits by scavenging free radicals.[76,77] It also exhibits anti-aging properties by reversing and mitigating UVA-induced damage.[76,77] Additionally, AZ modulates the inflammatory response by decreasing the expression of genes and chemokines involved in inflammation, particularly countering interleukin-17A–mediated pathways.[77]

Centella Asiatica

Centella asiatica is a natural DNA-repair additive with antioxidant properties.[78] Titrated extracts contain 4 centelloids (asiatic acid, madecassic acid, asiaticoside, and madecassoside) collectively referred to as the titrated extract of *Centella asiatica* (TECA).[78] TECA actively removes ROS and prevents senescence. Fibroblasts treated with 25–50 µg/mL of TECA before and after UVB exposure showed restored cell viability.[78] TECA also influenced gene expression related to development, differentiation, proliferation, and apoptosis.[78] Treatment decreased UVB toxicity and modulated microRNAs involved in apoptosis and cell proliferation.[79]

Madecassoside, extracted from *Centella asiatica*, induces collagen expression, modulates inflammation, and improves skin elasticity.[80] One randomized double-blind study compared a combination cream containing 5% stabilized vitamin C and 0.1% madecassoside to a placebo cream containing 6% glycerin.[80] The treatment significantly improved hydration, roughness, laxity, suppleness, wrinkles, radiance, and pigmented spots as assessed by dermatologists and participants.[80] After 3 months, hydration increased ($P < .0001$) and "crow's feet" size decreased ($P < .002$).[80] After 6 months, skin elasticity improved ($P < .0001$), and histology revealed partial restoration of the elastic network, with more organized collagen fibers observed on transmission electron microscopy.[80]

Overview of Probiotics

Probiotics play a key role in modulating the cutaneous microbiome and regulating the effects of UV radiation on the skin. Both dietary and topical probiotics have been studied for their ability to attenuate UV-induced damage. Specific probiotic strains have demonstrated benefits in skin health and are summarized in **Table 3**.[81]

Certain *Lactobacillus* strains help maintain skin homeostasis and protect against UV-induced damage. For example, *Lactobacillus plantarum* HY7714 produces exopolysaccharides (EPS), which upregulate genes encoding tight junction proteins, such as occludin-1, strengthening epithelial cells and countering inflammation.[82] These EPS also downregulate MMPs and ROS, improving UVB-induced cytotoxicity and hydration in human dermal fibroblasts.[82] Other *Lactobacillus* strains, such as *Lactobacillus johnsonii*, restore immune function following UV-induced immunosuppression via anti-inflammatory effects on keratinocytes.[83]

Streptococcus thermophilus has been shown to enhance epidermal structural integrity, increase hydration by reducing transepidermal water loss (TEWL), and protect against UVB- and H_2O_2-induced DNA damage.[83,84] Oral probiotics such as *Bifidobacterium breve* B-3 also decrease TEWL, improve tight junction integrity, reduce UV-induced epidermal thickening, and lower IL-1B production, highlighting their protective role against UV damage.[85]

Conclusion

Despite the rapid growth of the sunscreen industry, the incidence of UV–related skin cancers continues to rise. This review highlights the protective role of UV filters and the cellular repair functions of various compounds. Further studies, including human trials with topical additives and oral supplementation, are needed to evaluate the efficacy of these compounds. An integrative approach that incorporates conventional UV filters, topical antioxidants, and dietary supplementation is recommended to more comprehensively minimize the harmful effects of UV radiation.

Potential conflicts of interest

The authors declare no conflicts of interest.

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