Photoprotection in ethnic skin

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ABSTRACT

Although cutaneous photodamage is partially mitigated by darker skin pigmentation, sun-induced aging, dyspigmentation, sunburns, and skin cancers are reported worldwide in all skin types and races. The severity of photodamage varies from individual to individual, and is predominantly based upon genetic differences altering the body’s response or susceptibility to sun damage. In addition, non-Caucasian patients are less likely to perform skin self-examinations, attend dermatologic follow-ups, and seven times less likely to apply sunscreen than Caucasian patients. Therefore, the remainder of this article will discuss the categories of photoprotective agent [environmental, biologic, physical, and UV filters, i.e., sunscreens] as well as the topics of photoaging, dyspigmentation, photocarcinogenesis, and the controversy surrounding vitamin D deficiency from photoprotection in the context of ethnic skin.

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Introduction

Ethnicity and race are frequently interchanged in both conversation and in literature, despite having subtle differences in their definitions. Ethnicity refers to a subgroup of the population sharing a common “cultural heritage, ancestry, history, homeland, language (dialect), ideology…religion, mythology and ritual[s], cuisine, [and] dressing style.”1 By contrast, race categorizes the population by genetic origins. Historically, there are five main racial groupings (Table 1). As these groups intermix, new racial groups can form (e.g., the American Negroid).1 Since categorizing dermatologic conditions solely by race or skin phenotype will account for dermal and epidermal differences between races, or an individual’s reactivity to UV radiation (UVR), respectively, additional classification systems, such as the Kawada Skin Classification System for Japanese Individuals, have been developed to attempt to encompass both features in specific populations.2

Exposure to sunlight causes acute and chronic changes to the skin, as well as deleterious effects on the eyes.3 Although skin pigmentation assists in the mitigation of these effects, melanin is not entirely protective against UV rays.4 This fact is reflected by the non-Caucasian population exhibiting signs of photodamage: sunburn, dyspigmentation, photoaging, and cutaneous malignancy in a sun-exposed distribution.3 To minimize these effects, public education usually includes staying in the shade during peak UVR hours (from 10 AM to 2 PM), applying sunscreens, and wearing sunglasses and protective head/body coverings. In addition, physicians and other care providers have long recommended self and physician-performed skin exams. Although some ethnicities value fair and unblemished skin by practicing rigorous photoprotection, epide-

miologic studies show non-Caucasian patients are less likely to perform skin self-examinations, attend dermatologic follow-ups, and seven times less likely to apply sunscreen than Caucasians.5 Owing to these concerns and increased documentation on UV-induced morbidities in ethno-racial populations, efforts to increase awareness of proper photoprotection in people of color (poc) have taken place.6,7

Assessment of photoprotection

Sun protection factor

Humanity has sought protection from the erythemogenic effects of UVR, commonly known as sunburn, since at least 400 B.C.8 This focus on the erythemogenic spectra of UVR (UVB and UVA-2, the latter being 320–340 nm) has evolved in modern times to an abundance of products possessing a broad range of UVB photoprotection.

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The United States Food and Drug Administration (FDA) standardized the definition of sun protection factor (SPF) and adopted this test as the gold standard for quantifying the erythrogenic photoprotection of a product. SPF, as defined by the FDA, is the “numerical ratio between the minimal erythema dose (MED) of skin protected by 2 mg/cm² of the tested [substrate] and the MED of the unprotected skin after exposure to standard doses of a standardized solar simulated radiation.”

The FDA’s definition of SPF has received international acceptance. However, studies demonstrate that few consumers use sunscreen at the tested concentration, 2 mg/cm²; the average consumer applies approximately 25–50% of the recommended concentration creating a discrepancy between the in-use and labeled SPF. The FDA also mandated the methods for testing for water resistance. For sunscreens to be allowed to have a standardized solar simulated radiation.

Table 2 | Photoprotection properties of different natural agents.

<table>
<thead>
<tr>
<th>Agent</th>
<th>Photoprotection</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ozone</td>
<td>Attenuate all UVC light emitted by the sun, 90% of UVB light, and minimal to no attenuation on solar UVA light and VL.</td>
</tr>
<tr>
<td>Latitude and altitude</td>
<td>The transmission of UVB light decreases by 3% for every degree increase in latitude. For every 1000 feet increase in elevation, there is an increase of 4% to 10% of UVB light that reaches the earth’s surface.</td>
</tr>
<tr>
<td>Time of day and season</td>
<td>UVB light is strongest from 10 AM to 2 PM. UVA light is relatively constant throughout daylight hours. Terrestrial UVR is strongest in the summer.</td>
</tr>
<tr>
<td>Clouds</td>
<td>Cloud cover minimally affects UVA light. On cloudy days, the reduction of solar IR is much greater than UVR. Clear skies allow virtually 100% of UV transmission of solar UVR, scattered clouds 89%, broken clouds 73%, and overcast skies 31%.</td>
</tr>
<tr>
<td>Pollutants</td>
<td>Significant reductions in UVR radiation can occur with dust, volcanic ash, and wild-fire aerosols. Snow, ice, sand, glass, and metal can reflect up to 85% of UVB light. Reflection of UVR from most terrestrial surfaces is usually &lt;10%.</td>
</tr>
<tr>
<td>Shade</td>
<td>Fifty percent of exposure to UVA light occurs in the shade. Umbrellas provide only low UV light protection. SPF of single trees range from 4 to &gt;50.</td>
</tr>
<tr>
<td>Water</td>
<td>Swimmers can get significant UVR because it penetrates to a depth of 60 cm without significant attenuation.</td>
</tr>
<tr>
<td>Skin thickness</td>
<td>Human skin absorbs UVB radiation, scatters most VL, and reflects 5% to 10% of all solar radiation from 250 nm to 3000 nm. UVB light is mostly absorbed in the epidermis. UV light reaches the dermis.</td>
</tr>
<tr>
<td>Melanin</td>
<td>There is a five-fold lower penetration of UVA through epidermis obtained from dark-skinned compared to that from light-skinned individuals. The epidermis of dark skin has an intrinsic SPF of 13.4, whereas light skin has an SPF of 3.3.</td>
</tr>
</tbody>
</table>

IR = infrared; SPF = sun protection factor; UVA/UVB/UBC = ultraviolet A/B/C; UVR = ultraviolet radiation; VL = visible light.

**Assessment of UVA protection**

The focus on UV photoprotection beyond the erythrogenic spectrum did not evolve until the late 20th century. Like UVB, UVA-1 (340–400 nm) has biologic effects on immunosuppression, photoaging, and tumorigenesis. Because UVA is 1000-fold less erythrogenic than UVB, two biologic indices of UVA exposure were employed: immediate pigment darkening (IPD) and persistent pigment darkening (PPD). IPD appears within a few minutes of UVA exposure; it resolves in 2 hours. This is then followed by PPD, which lasts between 2 hours and 24 hours. Both IPD and PPD result from the oxidation of pre-existing melanin; no neo-melanogenesis occurs. While both permit a colorimetric assessment at 15 minutes and 2 hours following irradiation, respectively, the chemically-altered melanin seen in IPD is unstable making it a less reliable assessment method than PPD. Therefore, comparing the numerical ratio of the minimal pigmenting dose (MPD) necessary to cause PPD for “protected” skin (skin coated with 1.3 mg/cm² of the sunscreen tested) to the MPD of “unprotected skin” (normal, unadulterated skin) yields the UVA protection factor (UVA-PF) of a product. This methodology results in a four-point scale of UVA absorbance rating. Due to concerns over carcinogenicity from UVA-PF assessments, a few in vitro models, such as Australia’s ISO 24443:2012 and the FDA’s critical wavelength method, have been developed.

The Australian and FDA UVA-PF in vitro methods compare the photoprotective qualities of substances through the analysis of transmittance data collected by a UV spectrophotometer with and without an overlying film of the tested substrate. If the mathematical algorithm determines that 90% of the substrate’s absorbency ranges from 290 nm to 400 nm, the substance is declared to possess a broad-spectrum protective effect in the Australian method. The FDA “critical wavelength” method calls for the in vitro determination of the absorbance of the product; only products that have 90% of the absorbance at >370 nm (i.e., a critical wavelength of >370 nm) are allowed to have the broad-spectrum claim. While efforts are on-going, to date, there has not been worldwide harmonization of UVA protection assessment.

**UV protection factor**

UV protection factor (UPF) was coined to confer UV photoprotection on fabrics. Using a spectrophotometer and methodology similar to in vitro UVA-PF, UPF quantifies the UV transmittance of a fabric. If the fabric permits >5% of UVA transmittance and has an overall UPF score of 30+, the European Committee for Standardization has declared these fabrics to provide “broad-spectrum” protection.

**Biologic protection factors**

Several biologic protection factors exist: free radical skin protection factor (RSF), p53 protection factor, genotoxic protecting factor (GPF), and immune protection factor. These methods quantify the protective effects a sunscreen has against photo-induced production of reactive oxygen species (ROS), p53 activation, number of sunburn cells, and UV-induced suppression of local contact hypersensitivity and delayed-type hypersensitivity, respectively. These markers generally correlate well with UVA-PF but poorly with SPF ratings. Currently, these markers are primarily used for research purposes and have not been adopted by any regulatory agencies.
The intensity of sun-related UV irradiation is influenced by environmental factors. These factors include the concentration of reflective or absorptive atmospheric molecules, intensity of radiance as determined by time of day, terrestrial coverage, and reflective qualities of the surrounding surfaces. Over the past 40 years, scientists have noted an annual decline in the overall concentration of atmospheric ozone. This decline has mainly been attributed to chlorofluorocarbons, which undergo a photo-induced chemical reaction with UVR and ozone to yield chlorine monoxide and oxygen. It has been estimated that each percent of ozone lost correlates with a 3–4.6% increased risk for developing squamous cell carcinoma and an additional 1.7–2.7% additional risk for basal cell carcinoma secondary to higher levels of UVB irradiation. This loss has also been estimated to correlate with an increased risk of melanoma mortality by 1–2%. In an effort to protect the ozone, several countries have enacted emission regulations on chemicals known to damage the ozone.

### Types of photoprotective agents

#### Innate photoprotective agents

The photoprotection properties of different natural agents are shown in Table 2.

#### Environmental photoprotective agents

The intensity of sun-related UV irradiation is influenced by environmental factors. These factors include the concentration of reflective or absorptive atmospheric molecules, intensity of radiance as determined by time of day, terrestrial coverage, and reflective qualities of the surrounding surfaces. Over the past 40 years, scientists have noted an annual decline in the overall concentration of atmospheric ozone. This decline has mainly been attributed to chlorofluorocarbons, which undergo a photo-induced chemical reaction with UVR and ozone to yield chlorine monoxide and oxygen. It has been estimated that each percent of ozone lost correlates with a 3–4.6% increased risk for developing squamous cell carcinoma and an additional 1.7–2.7% additional risk for basal cell carcinoma secondary to higher levels of UVB irradiation. This loss has also been estimated to correlate with an increased risk of melanoma mortality by 1–2%. In an effort to protect the ozone, several countries have enacted emission regulations on chemicals known to damage the ozone.

#### Biological photoprotective agents

One of the many barrier functions of the skin is to protect underlying structures from the degradative effects of solar UVR. Utilizing chromophores, which are molecules that absorb solar photons, the skin can absorb, reflect, or scatter wavelengths across a large spectrum, 250–3000 nm. The main identified chromophores in the skin are melanin, hemoglobin, bilirubin, nucleic acids, aromatic amino acids (e.g., phenylalanine, tyrosine), and urocanic acid. Melanin confers significant photoprotection. Depending on skin phototype-dependent differences in melanosome size and density, UV transmission ranges between 17% and 55% for UVA wavelengths to between 6% and 29% for UVB wavelengths. Additional chromophores, such as NADH, flavins, and unsaturated fatty acids, are also known to influence photo-induced ROS formation or reduction in the skin. Rays absorbed by chromophore defenses will either be dissipated as heat or transferred to neighboring molecules forming photoproducts (e.g., cyclobutamine pyrimidine dimers and ROS). While the body quickly repairs or neutralizes many of these insults, the summative accumulation of atypical intra- and extra-cellular repairs can be seen as the formation of wrinkles, dyspigmentation, and skin cancers.

The eye incorporates many of the same photoprotective methods. However, several of these mechanisms are not mature at birth or adolescence. For example, a higher amount of UVA radiation is transmitted to deep eye structures until approximately 10 years of age. Blue light, which is associated with an increased risk of developing macular degeneration, declines from 80% transmission...
during teenage years to 40% for those aged 60 years.25 The accumulation of photo-induced insults has been strongly correlated with acute and chronic ophthalmologic disease, including photokeratoconjunctivitis, retinopathy, pterygium, pinguecula, and corneal cataracts, among others.14,26 Therefore, it is extremely important to incorporate photoprotective measures beyond these innate agents throughout all stages of life.

**Physical photoprotective agents beyond UV filters**

Clothes and clothing accessories, such as gloves, hats, and sunglasses, provide another layer of protection from solar rays. The combination of fabric thickness, fabric type, fiber size, dye, and construction are vital to determining the overall UPF of the item. In general, thicker fabrics composed of wool, denim, or polyester material in dark colors with fine threads, tightly woven together, will provide maximum photoprotection.5,21,27,28 Additional chemical processing may either increase the protective qualities (i.e., inclusion of UV filters) or reduce them (i.e., bleach). Hydration, by contrast, may increase or decrease UPF depending upon the fabric used.14,29 Although sunglasses could theoretically be described in UPF, regulatory agencies have not yet utilized this terminology for eye photoprotection. A full description of specific clothing properties as they relate to UPF is described in Table 3. A similar description of photoprotective properties in glass and eyewear can also be found in Table 3.

**UV filters**

UV filters are topically applied organic and inorganic substances that absorb, disperse, and reflect UV rays. While reflection and dispersion (scattering of solar rays) can account for up to 10% of the overall photoprotection, the primary mechanism in commercially available sunscreens is absorption. Once a UV photon is absorbed, the energy must be released as fluorescence, phosphorescence, heat, vibration, energy transference to another molecule, or a photo-reaction prior to accepting another photon. The faster the rate of return to the original ground state, the more advantageous the filter is for sunscreens. However, if the substrate undergoes an irreversible photoreaction, UV degradation of the substrate will occur. Specific combinations of UV filters, such as avobenzone and octinoxate, may enhance the photodegradation of individual filters.30

Owing to the initial focus of sunscreen ingredients toward erythemogenic protection, the number of commercially available sunscreen agents is skewed toward UVB protection. Currently, there are around 15 agents with a significant UVA–1 absorption spectrum approved or under review in Australia, Japan, Europe, and the United State (Table 4).

**Antioxidants**

Antioxidants are substances that inhibit the oxidation of other molecules via electron donation while remaining chemically stable to prevent the formation of free radicals. Enzymes, such as superoxide dismutase, catalase, and peroxidase, remove free radicals and thus are considered a type of antioxidant. The remainder of this section will discuss only exogenous, nonenzymatic antioxidants.

Exogenous antioxidants can be administered either orally or topically. Oral, in comparison to topical administration, provides the added benefits of systemic distribution and resilience to dissipation following topical trauma or water. The limitation of oral administration is poor accumulation of significant concentrations in the skin. Both methods of administration have been limited by premature oxidation of the antioxidant, rendering it biologically inactive.

Carotenoids, vitamin C, vitamin E, and polyphenol are among the most heavily investigated antioxidants in the literature. Each of these antioxidants has shown photoprotective benefits in one role or another. For example, carotenoids are naturally occurring organic pigments utilized in the human eye to absorb blue and near-UV light preventing photo-induced retinal damage and age-related macular degeneration.31 No significant reduction in erythema nor the development of nonmelanoma skin cancer in humans was identified.12,23 Similarly, topical vitamin E has not been shown to reduce UV-induced cutaneous edema despite suppressing contact hypersensitivity, UV-related cyclobutamine pyrimidine dimers, and skin cancer in mouse models.14 However, when combined with vitamin C as either a topical or oral formulation, vitamin E increases the measured minimal erythemal dose.32 Because vitamin C has shown no equivalent effect when used alone, it is likely that this synergistic effect may in part be due to the role of vitamin C in regenerating oxidized vitamin E. In contrast to vitamins C and E, polyphenols have shown significant photoprotective benefits following oral supplementation of this ubiquitous plant-derived molecular family. In a double-blind, placebo-controlled human trial, oral administration of green tea-derived polyphenols reduced UV-induced erythema.36 Similar findings were displayed in an animal study where oral supplementation of polyphenols reduced UV-induced skin cancer.2 Polypodium leucotomos is a tropical fern used by Native American Indians for centuries to treat inflammatory and cutaneous disorders. Human trials reveal that oral and topical extracts protect against UV-mediated phototoxic reactions, histologic changes, and biomarker expression changes through antioxidant and anti-inflammatory properties.38–40 These effects do not appear to be due to UV-absorptive qualities.40

**Additional photoprotective agents**

Other agents that have been reported to have photoprotective properties are listed in Table 5.

**Topics related to photoprotection**

**Vitamin D**

Because sufficient levels of vitamin D3 can be obtained through dietary ingestion or suberythemic UVB-related conversion of D2 to D3, concern has arisen over the risk of vitamin D deficiency in photoprotective practices. Epidemiologic studies indicate that the increased pigmentation in a dark-skinned patient may predispose them to develop vitamin D deficiency in geographic locations of higher latitudes. Extrapolating this theory to photoprotective measures has prompted studies revealing that patients who practice rigorous sun avoidance due to photosensitivity are at an increased risk of developing vitamin D deficiencies compared to controls.41–43 However, population-based studies investigating regular use of sunscreen failed to show a similar correlation.44–46 It is hypothesized that this lack of vitamin deficiency may be due to inadequate application of sunscreens.

A recent study from the United States showed that black Americans, compared with whites, had low levels of 25-hydroxyvitamin D and vitamin D-binding protein, resulting in similar levels of bioavailable 25-hydroxyvitamin D.47 Therefore, determining the need of vitamin D supplementation should be assessed on an individual basis, taking into account the duration of outdoor exposure, geographic location, and skin type. If supplementation is deemed necessary, the Institute of Medicine...
Academy American Note US ¼ AU

The Control (CDC) has shown that the melanoma incidence rate for

and soles. Epidemiologic data published by the Center for Disease

exam annually, which should include examination of palms

should be made to encourage the patient to undergo a full body

Because photodamage has been reported worldwide in all races

populations. Photodamage, dyspigmentation, and photocarcinogenesis

For those aged between 1 year and 70 years, and 800 IU for in-

ternational units (IU) of vitamin D for infants up to 1 year of age, 600 IU

A list of common UV light filters approved in Australia, Europe, Japan, and the United States.®

Coverage spectrum USAN INCI Trade name© Concentration limits in sunscreen (%) Additional comments

Broad-spectrum and UVAI (340–400 nm)

Bemotrizinol® Bis-ethylhexyloxyphenol

methoxyphenyl triazined® Tinosorb S — 10 3 10 Photolabile; enhance the photodegradation of octinoxate

Avobenzone Butyl methoxydibenzoylmethane Parisol 1789 3 5 10 5

— Diethylaminohydroxybenzyl 10 10 10

hexyl benzoate

Bisdisulizole disodium — Disodium phenyl dibenzimidazole tetrasulfonate NEO Heliosap AP — 10 — 10

Meradimate — Menthol anthranilate Mexoryl XL — 15 — 15

Bisoctrizole® Methylene bis-benzotriazolyl tetramethylbutylphenol® Tinosorb M — 10 10 10

Ecamsule® TECPHILYLYIDENE DICAMPHOR SULFONIC ACID® Mexoryl SX — 10 10 10

Zinc oxide Zinc oxide ZnO (Nanox) 25 25 No limit No limit Photostable; commonly coated with dimethicone or silica to minimize generation of ROS upon UV exposure

UVB (290–320 nm) and UVAII (320–340 nm)

Enzacamene® 4-Methylbenzylidine camphor® Eusolex 6300 — 4 — 4

Oxybenzone Benzophenone-3 — 6 10 5 10

Suliso-benzene Benzophenone-4 — Uvinul MS40 10 5 10 10

— Polysilicone-15 Parsol SLX — 10 10 10

Padimate O® Ethanylhexyl rimethyl PABA® Eusolex 6007 8 8 10 8 Most commonly used PABA derivative

Octinoxate Ethanylhexyl methoxyccinnamate Uvinul MC 80 7.5 10 20 10 Most widely used UVB filter; photostable

Octisalate Ethylhexyl salicylate Neo Heliosap OS 5 5 10 5

Octytriazole® Ethylhexyl triazine® Uvinul T 150 — 5 3 5

Homosalate Homomenthyl salicylate Eusolex HMS 15 10 10 15

Amiloxate® Isoamyl p-methoxyccinnamatre Neo Heliosap E1000 — 10 — 10

Octocrylene Octocrylene Uvinul N 539 T 10 10 10 10

Ensulizole Phenylbenzimidazole sulfinic acid Eusolex 232 4 8 3 4

Titanium dioxide Titanium dioxide Eusolex T2000 25 25 No limit 25 No report of sensitization reaction


Trade names are the property of their respective manufacturers. Some have more than one trade name

Submitted for US Food and Drug Administration approval through time and extent application

Approved in certain formulations up to 3% via the new drug application

Not supported in the EU and may be delisted

Not yet approved in the EU or anywhere else but positive opinion by Scientific Committee on Consumer Safety.

recommends a dietary intake or supplementation of 400 international units (IU) of vitamin D for infants up to 1 year of age, 600 IU for those aged between 1 year and 70 years, and 800 IU for individuals aged >70 years.57

Photodamage, dyspigmentation, and photocarcinogenesis

Because photodamage has been reported worldwide in all races and is a frequent cause of dermatologic presentations, efforts should be made to encourage the patient to undergo a full body skin exam annually, which should include examination of palms and soles. Epidemiologic data published by the Center for Disease Control (CDC) has shown that the melanoma incidence rate for

Caucasoid males is over three times greater than that for Mongoloids and over 10 times higher than Negroids.58 Similar ratios of racial variation in melanoma are reported by the United Kingdom.59 When comparing mortality, however, the Caucasoid mortality rate is only four times that of Mongoloid and Negroid rates.60 However, this number may not be accurate, because many melanoma cases are diagnosed in nonhospital settings, which historically are not included in central cancer registry data collections.61,62 This point would explain why multiple studies that monitored 5-year survival of melanoma report a lower survival for black compared to white populations.51–53 This worse prognosis persists despite adjusting for sex, age, stage, histology, anatomic site, treatment, and socioeconomic status.54 Oddly, UVR does not appear to be associated


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with melanoma in black and Asian populations. In fact, most of these melanomas are discovered on the palms, soles, mucous membranes, and subungual sites.\(^{55}\) In the Asian population, NMSC appears to be partially related to solar UV exposure and skin phototype, but genetic and additional factors are felt to play an important role as well.\(^{56}\) In the black population, BCCs predominantly present in females, to seborrheic keratosis, which is the major pigmentary disorder in sun-exposed skin of men.\(^{9}\) Patients of African descent will often develop dermatosis papulosa nigra also.

Contrary to initial theories, darker pigmentation in Asians and Africans does not provide impenetrable protection from non-melanoma skin cancer (NMSC).\(^{55}\) NMSC still occurs in these populations, but at lower reported rates than in the Caucasian population. However, the exact incidence rate of NMSC varies depending on race, geographic location, and latitude across the globe.\(^{55}\) In the Asian population, NMSC appears to be partially related to solar UV exposure and skin phototype, but genetic and additional factors are felt to play an important role as well.\(^{56}\) In the racial genre, basal cell carcinoma (BCC) is the most frequent NMSC comprising up to 60% of skin cancers in the Chinese population.\(^{57,58}\) Asians maintain greater barrier function with decreased TEWL than Caucasian counterparts despite manifesting higher frequencies of photosensitivity in erythropoietic protoporphyria and solar urticaria patients\(^{71,72}\); current status by the FDA: investigational drug.

### Table 5 Additional photoprotective agents.

<table>
<thead>
<tr>
<th>Agent</th>
<th>Source</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>N-Acetylcyesteine</td>
<td>Synthetic</td>
<td>Endogenous antioxidant: increase of glutathione level</td>
</tr>
<tr>
<td>Butyroyl hydroxytyolene</td>
<td>Preservatives, additives</td>
<td>Synthetic antioxidant</td>
</tr>
<tr>
<td>Cadmium chloride</td>
<td>Synthetic</td>
<td>Induction of metallothionein (scavenger of free radicals)</td>
</tr>
<tr>
<td>Calcitriol</td>
<td>Plants and vegetables</td>
<td>Antioxidant and free radical scavenging</td>
</tr>
<tr>
<td>(1,25-dihydroxyvitamin D3)</td>
<td>Synthesized in kidneys</td>
<td>Induction of metallothionein (scavenger of free radicals)</td>
</tr>
<tr>
<td>Cistus 2-Furidoxime</td>
<td>Mediterranean shrubs</td>
<td>COX-1 and COX-2 inhibitors: may reduce SCC, BCC, and melanoma risk by inhibition of COX-2 pathways involved in carcinogenesis</td>
</tr>
<tr>
<td>Isoflavones</td>
<td>Plants</td>
<td>Free radical scavenging</td>
</tr>
<tr>
<td>Melatonin</td>
<td>Synthetic</td>
<td>Iron chelator; topical combinations with sunscreen enhances SPF properties in animal models from SPF 4 to 30</td>
</tr>
<tr>
<td>Xyloglucans</td>
<td>Tamarind seeds</td>
<td>Plant-derived oligosaccharide with evidence of preventing UVB-induced systemic immunosuppression</td>
</tr>
<tr>
<td>Aloe poly/oligosaccharide</td>
<td>Aloe barbadensis</td>
<td>Plant-derived oligosaccharide with evidence of preventing UVB-induced systemic immunosuppression</td>
</tr>
<tr>
<td>Omega-3 polysaturated fatty acid</td>
<td>Fish oil</td>
<td>Decrease of sunburn cell formation, anti-inflammation</td>
</tr>
<tr>
<td>Zinc</td>
<td>Mineral</td>
<td>Antioxidant</td>
</tr>
<tr>
<td>T4 endonuclease V</td>
<td>Bacteria</td>
<td>Bacterial DNA excision enzyme shown to repair cyclobutane pyrimidine dimer</td>
</tr>
<tr>
<td>Thymidine dinucleotide</td>
<td>Synthetic</td>
<td>Enhancement of melanogenesis, increase of DNA repair</td>
</tr>
<tr>
<td>Selenium</td>
<td>Essential element</td>
<td>Topical application decreases MED in humans</td>
</tr>
<tr>
<td>Nicotinamide</td>
<td>Amide form of vitamin B3</td>
<td>Antioxidant with possible benefits in reducing UV-induced ROS; topical administration reduces UV immunosuppression</td>
</tr>
<tr>
<td>Phytomelanin</td>
<td>Date/palm fruit derivative</td>
<td>Common sunscreen ingredient for topical application that is intended to mimic melanin</td>
</tr>
<tr>
<td>Silmarin</td>
<td>Milk thistle seeds</td>
<td>Antioxidant demonstrating topical UVB filtering properties in in vivo studies</td>
</tr>
<tr>
<td>Senma alata</td>
<td>Plant native to Central America</td>
<td>Antioxidant commonly added to sunscreen formulations</td>
</tr>
<tr>
<td>DHA</td>
<td>Synthetic</td>
<td>Sunless tanning active ingredient; provides superficial orange/brown pigmentation to the stratum corneum and in turn yields an SPF of 2; the inhalation effects of aerosolized DHA “spray tans” have not yet been determined.(^{60})</td>
</tr>
<tr>
<td>Afamelanotide</td>
<td>Synthetic</td>
<td>Alpha melanocyte stimulating hormone analog; induces epidermal melanin, melanocyte proliferation, tyrosinase activity, and enhances DNA photoprotect repair; subcutaneous administration decreases photosensitivity in erythropoietic protoporphyria and solar urticaria patients(^{1,72}); current status by the FDA: investigational drug</td>
</tr>
</tbody>
</table>

BCC – basal cell carcinoma; COX-1 – cyclooxygenase 1; COX-2 – cyclooxygenase 2; DHA – dihydroxycetone; FDA – Food and Drug Administration; NSAID – nonsteroidal anti-inflammatory drugs; ROS – reactive oxygen species; SCC – squamous cell carcinoma; SPF – sun protection factor; UVB – ultraviolet B.

dyspigmentation, additional etiologic mechanisms may account for these differences.\(^5\)\(^6\)\(^7\) Although uneven pigmentation occurs with greater frequency than skin wrinkling in Asian populations compared to Caucasians, skin wrinkling does occur.\(^6\)\(^9\)

For poc, moderate to severe wrinkling of skin becomes apparent one to two decades later than in age-matched Caucasians.\(^1\)\(^67\) Koreans, for example, rarely have wrinkles prior to the age of 30 years and are frequently not noticed until after the age of 50 years.\(^1\) Korean women have also been observed to develop more wrinkling compared to their male counterparts.\(^6\)\(^8\) Utilizing assessments of skin wrinkling from a multicenter study, researchers have discovered that Mongoloid populations display less wrinkling than Negroid populations.\(^1\) While the intensity of sun exposure and photoprotection practice may differ between these two populations, this report suggests that there are other contributory factors in the development of wrinkles in addition to constitutive skin color.\(^6\)\(^10\)

**Conclusion**

While poc have more effective mechanisms of preventing injuries from solar irradiation than their Caucasian counterparts, photodamage and photocarcinogenesis do occur in all skin types and races. The implementation of photoprotective measures, such as avoiding the sun during peak hours of UVR, utilizing UV-protective clothing, hats, and sunglasses, and applying sunscreen (when outdoors, every 2 hours), will reduce both the acute and chronic effects of solar rays. For select populations, such as those living at higher latitudes, those with limited sun exposure due to work or outdoors, every 2 hours), will reduce both the acute and chronic effects of solar rays. For select populations, such as those living at higher latitudes, those with limited sun exposure due to work or

**References**
