Photochemical & Photobiological Sciences (2023) 22:441–456
https://doi.org/10.1007/s43630-022-00314-z

REVIEWS

Photoprotection in skin of color

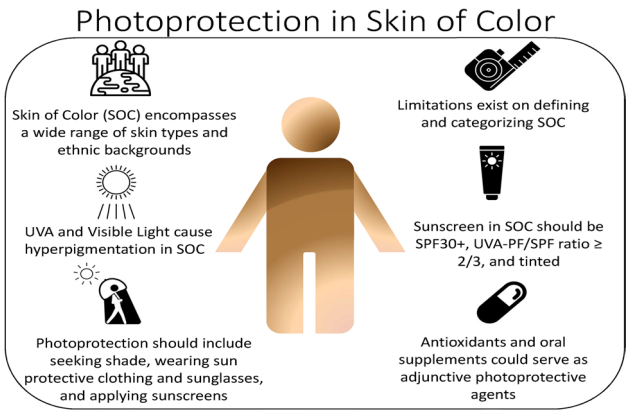
Sokhna Seck[1] · Judy Hamad[2] · Sergio Schalka[3] · Henry W. Lim[2]

Received: 9 July 2022 / Accepted: 25 September 2022 / Published online: 13 October 2022

Abstract

As populations in many parts of the world are projected to become more racially diverse over the coming decades, we must better understand the unique characteristics of the skin of populations with skin of color (SOC). This review aims to highlight important physiologic and clinical considerations of photoprotection in SOC. Ultraviolet radiation and visible light affect dark and light skin differently. SOC populations have historically not been informed on photoprotection to the same degree as their light skinned counterparts. This has exacerbated dermatologic conditions in which SOC populations are disproportionately affected, such as hyperpigmentary disorders. Patients should be encouraged to utilize multiple methods of photoprotection, ranging from avoidance of sunlight during peak intensity hours, seeking shade, wearing sun-protective clothing and wide-brimmed hat, and applying sunscreen. Ideal sunscreens for SOC populations include those with UVA-PF/SPF ratios $\geq 2/3$ and tinted sunscreens to protect against VL. Although there have been increased efforts recently, more research into photoprotection for SOC and targeted public education are required to disseminate photoprotection resources that are patient-centered and evidence-based.

Graphical abstract



Abbreviations

FST	Fitzpatrick skin typing
ITA	Individual typology angle
MED	Minimal erythema dose
MMP	Matrix metalloproteinase
PIH	Post-inflammatory hyperpigmentation
ROS	Reactive oxygen species
SOC	Skin of color
SPF	Sun-protection factor
SPT	Skin phototypes
UPF	Ultraviolet protective factor
UV	Ultraviolet

Sokhna Seck and Judy Hamad are co-first authors.

✉ Henry W. Lim
hlim1@hfhs.org

[1] Cleveland Clinic Lerner College of Medicine, Case Western Reserve University, Cleveland, OH, USA

[2] Photomedicine and Photobiology Unit, Department of Dermatology, Henry Ford Health, Henry Ford Medical Center – New Center One, 3031 West Grand Blvd, Suite 800, Detroit, MI 48202, USA

[3] Medcin Skin Research Center, São Paulo, Brazil

UVR Ultraviolet radiation
VL Visible light

1 Introduction

Increased education and awareness of skin conditions disproportionately seen in pigmented skin is crucial to championing equitable care and optimizing patient outcomes [1]. As populations in many parts of the world are projected to become more racially diverse over the coming decades, we must better understand the unique characteristics of the skin of populations with skin of color (SOC) [2].

SOC has many unique biological considerations. Ultraviolet (UV) radiation and visible light (VL) do not affect dark and light skin in the same ways [3]. Understanding the science behind effects of UV and VL on dark skin will help clinicians better inform patients—and the public at large—on photoprotective recommendations.

SOC populations have historically not been counseled on photoprotection to the same degree as their light skinned counterparts [4]. This has exacerbated the prevalence and severity of dermatologic conditions in which SOC populations are disproportionately affected, such as hyperpigmentary disorders. The scientific community has thus increased efforts to dispel misconceptions and provide practice recommendations on photoprotection for SOC [5].

The aim of this review is to highlight important physiologic and clinical considerations of photoprotection in SOC. We will discuss reaction to UV and VL, biological response to UVA and UVB protection, and advise on photoprotection for SOC populations.

2 Methods

We conducted a narrative review of the literature. A literature search of PubMed and GoogleScholar using the terms "photoprotection," "skin of color," "dark skin, "black skin," "ethnic skin," "sunscreen," "visible light," "ultraviolet light," "photoaging," and "photodamage" in various combinations was performed. This literature search was limited to articles in English. The search was not limited by year of publication. One hundred fifty-four articles were identified on PubMed and 2590 articles on GoogleScholar. Articles were selected for inclusion depending on subjective relevance. One hundred thirty-four articles were included in this review.

2.1 Assessing skin phototype

The racial and ethnic diversity of populations in many parts of the world is changing substantially. In the United States, for example, population projections suggest more than one-half of the U.S. population will be non-white by the year 2050 [6]. A similar phenomenon have been described in the UK [7]. These changing demographics underscore the importance of increased education and awareness of concerns in pigmented skin to ensure optimal health outcomes in the future [8]. In North America, SOC generally refers to individuals of African, Asian, Native American, Middle Eastern, and Hispanic (or Latino) descent [9]. Fitzpatrick skin typing (FST), the most commonly used classification system in dermatology, was originally designed to categorize Caucasian skin into four Fitzpatrick skin phototypes (SPT), I–IV [10]. SPT V–VI were later added to enable classification of darker skin tones. For SPT I–IV, classification is made by the propensity of the skin to burn or tan following sun exposure. For dark skin types (SPT V–VI), determination is made by the color of the skin (Table 1). There are some variations in the definition of SOC among articles, with some including SPT III as SOC. For this article, we did not limit SOC to SPT IV–VI given the diversity of ethnic skin types across the SPT scale, and thus used the varying classifications used in each of the reviewed articles. Additionally, many studies use ethnicity or skin color rather than SPT to describe subjects, making translation of all cited studies to SPT values impossible given the limited information provided in these articles.

Skin phototyping is useful in predicting the risk of acute and chronic photodamage, photocarcinogenesis and the outcome of esthetic procedures. FST is often misinterpreted; a survey of academic dermatologists and dermatology trainees in the U.S. found that approximately one-third to half of these providers use FST as a means of describing race/ethnicity and constitutive skin color [11]. This misuse occurs more frequently among physicians who do not identify as having SOC [8, 11]. Although individuals with SOC are often regarded as having SPT III, IV, V, and VI, ethnic skin colors span the entire spectrum of phototypes and do not always match the FST categories [12–14]. While SPT has been widely used in clinical settings for phototherapy, classification systems that more objectively assess skin UV sensitivity/response are needed to accurately determine cancer risk in racially and ethnically diverse patients [13, 15–17].

Table 1 Fitzpatrick skin phototypes

Skin phototype	Description
I	Always burns, never tans
II	Burns easily, sometimes tans
III	Sometimes burns, always tans
IV	Rarely burns, tans easily
V	Rarely burns, tans easily, moderately pigmented skin
VI	Rarely burns, tans well, highly pigmented skin

Diffuse reflectance spectrophotometry objectively measures the melanin index and is the most reliable way to assess the color of the skin, however, it is not practical to use in routine clinical settings due to the expertise, time and associated cost needed [16]. A colorimeter-based assessment of skin color, individual typology angle (ITA), has increasingly been accepted as an objective and reproducible method to assess skin color, especially in photodermatology research [10]. The equipment however is costly, and its use requires specialized training. Therefore, for use in the clinics, FST is still the widely used method.

2.2 Electromagnetic radiation

The sun emits energy in a wide range of electromagnetic wavelengths classified into different spectral regions (Fig. 1). The shorter the wavelength, the more energetic the radiation and the greater potential for adverse biological effects [18]. UV radiation (UVR) is in wavelengths between 100 and 400 nm; while there are several variations on subdivision of UV spectrum, in photodermatology, the most widely used one is UVC (200–290 nm), UVB (290–320 nm), and UVA (320–400 nm). Visible light (VL) is characterized by wavelengths ranging from 400 to 700 nm. Melanin, one of the naturally occurring chromophores in the skin, absorbs throughout the UVB, UVA, and visible wavelengths [19].

2.3 Effects of ultraviolet radiation

The biological processes associated with exposure to UVR can be divided in to acute and chronic effects. Acute UV exposure induces the production of inflammatory, vasoactive and neuroactive mediators that together result in an inflammatory response and cause erythema (sunburn) [20]. Physiologic responses to protect the skin against subsequent UV damage occur including adaptive melanization (tanning) and epidermal hyperkeratosis. If the dose of UV exceeds a threshold damage response, keratinocyte death occurs via apoptosis. UVB is more damaging per unit of physical dose (ie, J/cm^2) compared to UVA. It dominates the carcinogenic effects of sunlight by causing direct DNA damage, while UVA causes indirect damage mainly through the production of reactive oxygen species (ROS) [3]. Erythema (sunburn) is mainly caused by UVB radiation, although UVA and visible light can also contribute [21, 22]. Chronic UV exposure results in photoaging and photocarinogens, most prominently seen in light-skinned individuals. Although these acute and chronic effects are well understood, the differences in UV effects between light skinned and dark-skinned subjects are beginning to be elucidated. In addition to being more resistant to DNA damage, dark skin has been suggested to be better adept at DNA damage repair compared to light skin [23]. Sheehan et al. compared biopsies of individuals with phototype II and IV using monoclonal antibodies for thymine dimers; this study reported a greater loss of thymine dimers in skin type IV, suggesting that rates of DNA repair are greater in the dark skin [24].

2.3.1 Photoprotective role of melanin

Melanin functions as a physical barrier that scatters UVR and reduces penetration through the epidermis. The epidermis of dark skin is estimated to have an intrinsic SPF of 13.4, whereas light skin has an SPF of 3.3 [23]. This is primarily thought to be a result of differences in melanocyte size and melanosome distribution and packaging [25]. Melanocytes produce two main types of melanin from dihydroxyphenylalanine precursors: yellow–red pheomelanin and black-brown eumelanin. Eumelanin is superior in its photoprotective properties, and the proportion of eumelanin to pheomelanin has implications on skin type. Differences in size, number and packaging of melanosomes, the type of melanin produced, and the melanin content of melanosomes, which can range from 17.9 to 72.3%, determines skin pigmentation [3]. SOC has comparatively larger melanocytes, higher eumelanin/ pheomelanin ratio, and melanosomes that are distributed individually in keratinocytes rather than in aggregates, allowing for more efficient absorption [26]. This increased epidermal melanin and distribution provides an inherent photoprotection and allows for absorption and scattering of two to five times more UV photons. For a given exposure to UVR, individuals with less epidermal melanin will exhibit greater erythema and less tanning than persons with more melanin [27]. There are also skin type differences in melanosome degradation. Melanosomes in dark skin are resistant to degradation by lysosomal enzymes and remain intact throughout the epidermal layers [3, 28]. Melanosomes in fair skin are degraded and only persist as "melanin dust" in the suprabasal layers.

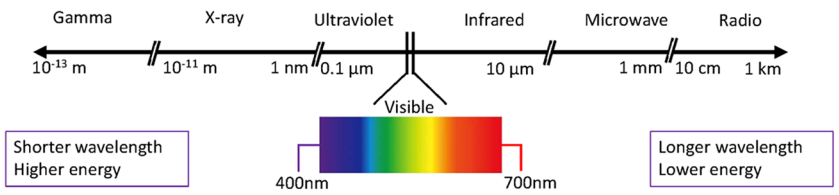


Fig. 1 Electromagnetic radiation spectrum

Tadokoro et al. explored the relationship between melanin content and DNA damage in individuals from different racial/ethnic origins and different phototypes [29]. After exposure to 1 minimal erythema dose (MED) of UVR (60% UVA and 40% UVB), subjects from all groups suffered significant DNA damage and this damage was greatest immediately following UV exposure. The extent of DNA damage was inversely correlated with skin pigmentation. Del Bino et al. assessed the relationship between UV sensitivity and skin color type by analyzing DNA damage, apoptosis and p53 accumulation in skin samples after UV exposure. Rather than using SPT, they classified skin samples into five categories (light, intermediate, tanned, brown and dark) according to their ITA value. The lower the ITA value, the darker the skin color type. They found that in light, intermediate and tanned skin types, DNA damage was observed to be distributed throughout the epidermis and superficial dermis while in brown and dark skin types, they were present only in the suprabasal layers [30].

2.3.2 Photoaging

Long-term exposure to UVR causes premature aging through a sequence of specific molecular responses that damage skin connective tissue. UVR is directly absorbed by DNA, giving rise to genomic alterations varying from point mutations to chromosomal rearrangements. Photochemical generation of ROS also causes deleterious chemical modifications to DNA and other cellular components through photosensitizing reactions [18]. Accumulation of protein and DNA damage leads to delayed effects and characteristic morphological changes in keratinocytes and other skin cells. UV penetrates the skin in a wavelength-dependent manner. While both UVA and UVB contribute to photoaging, the longer wavelength of UVA is able to reach the deep dermis and thus plays a greater role in premature aging; UVB, in contrast, is almost completely absorbed by the epidermis [18]. Skin of SPT III–VI is better protected against UV-induced damage than skin of SPT I–II because it is more effective in inhibiting UV penetration, typically allowing penetration of 7.4% UVB and 17.5% UVA, compared to 29.4% and 55.5% respectively, for white skin [31]. Melanin not only acts as a physical barrier to UV, it also has antioxidant and radical scavenging properties, so response to oxidative stress also differs between skin types [3].

UVR induces alterations in the collagenous extracellular matrix of connective tissue. These alterations include fragmentation of collagen fibrils and accumulation of abnormal elastin-containing material. UV stimulates activator protein 1, which leads to upregulation of matrix metalloproteinase (MMP) and inhibition of transforming growth factor (TGF)-β signaling. MMPs are primarily responsible for the structural degradation of extracellular matrices and sustained elevations lead to a breakdown of the structural proteins that confer strength and resilience in skin. Furthermore, by blocking the effects of TGF-β, there is a reduction of type I procollagen gene expression [27]. These derangements in the epidermis and dermis are hallmarks of photoaging and clinically present as coarse wrinkles, solar lentigines, mottled hyperpigmentation, skin dullness, and telangiectasias [32, 33]. Fisher et al. explored how skin pigment protects against UV-induced responses that lead to collagen degradation [27]. In subjects with SPT I and II, exposure to 2 MED of a UVB/UVA2 source resulted in substantial induction of MMP-1 (collagenase) messenger RNA (mRNA) levels and formation of thymine dimers. In contrast, twice the average exposure of this group produced only modest MMP-1 mRNA induction and DNA damage in subjects with SPT V and VI.

Prominent features of photoaging vary depending on skin type. Individuals with SPT I–III have reduced melanin content and small aggregates of melanosomes, making them more susceptible to UV damage and earlier signs of photoaging. In this group, photoaging presents as lines and wrinkles. Melanosomes in SOC are larger and contain more melanin thus allowing less UV penetration, reducing the impact of photoaging, and delaying the development of wrinkles.

Other properties in the epidermal and dermal architecture of SOC that contribute to differences in features of photoaging include a thicker and more compact dermis, increased fibrillary collagen, and larger, more numerous, and multinucleated fibroblasts [34, 35]. In those with SOC, photoaging is most associated with pigmentary changes, including uneven skin tone, post-inflammatory hyperpigmentation, and melasma [5, 36]. To highlight the unique features of photoaging across skin types and ethnic groups, photonumeric scales for evaluation of photodamage have been developed for Caucasians [37, 38], Asians [39], and African Americans [40]. Photodamage accumulates in all skin types and all individuals will at some point exhibit signs of photoaging. Therefore, photoprotection is the most important method for preventing photoaging.

2.3.3 Photocarcinogenesis

UVR is classified as a complete carcinogen as it has properties of both a tumor initiator and a tumor promoter [18]. Skin cancer is one of the most commonly occurring cancers in the world [41]. A combination of experimental and epidemiological data suggests that the risk of skin cancer is heavily influenced by cumulative UV exposure and skin pigmentation. The incidence of melanoma and keratinocyte carcinoma has been strongly correlated with history of sunburns and tanning bed use, SPT I–II with blonde or red hair, and a personal or family history of skin cancer [42–45].

The most important modifiable risk factor for skin cancer in non-Hispanic white populations is exposure to UV. In contrast, in SOC, UV exposure is not believed to be as important an etiologic factor in the pathogenesis of skin cancer. A systematic review assessing the association between UV exposure and risk of melanoma development in SOC found that in thirteen studies, 11 showed no association, 1 showed a small positive relationship in Black males, and 1 showed a weak association in Hispanic males [46]. Another review evaluating the association between UV exposure and development of keratinocyte carcinoma in SOC found that the association may depend on the type of UV exposure. UV exposure through phototherapy showed no association while cumulative sun exposure demonstrated a positive association primarily in East Asian populations [47]. There were no studies among Black individuals and only 1 study among a Hispanic population. In both reviews, all the studies analyzed were rated as moderate to low quality (Oxford Centre ratings 2b to 4) suggesting that further research is required to fully elucidate this association with dark skin types.

The incidence rate of melanoma and keratinocyte carcinoma in different ethnic populations has been well reported in several review articles [14, 48–51]. Non-Hispanic Whites are reported to have 30 times higher incidence rates than non-Hispanic Blacks or Asian/Pacific Islanders [52]. Incidence in individuals with SOC is about 5% in Hispanic, 4% in Asian, and 2% in Black populations [14, 26]. A study by Yamaguchi et al., assessed DNA damage in the epidermal layers and subsequent apoptosis and phosphorylation of p53 at different time points following exposure to 1 MED of UV (60% UVA and 40% UVB) [53]. This study classified patients as fair or dark skin based on their race/ethnicity: White subjects were grouped as "fair skin" and Black subjects as "dark skin". They found that fair skin was less efficient at UV filtration, allowing for DNA damage in the lower epidermis, including keratinocyte stem cells and melanocytes. Fair skin was also less efficient at removing UV-damaged cells with less than 1% of the damaged cells becoming apoptotic. At the same UV dose, dark skin acquired less UV-induced DNA damage and had an increased rate of apoptotic cell formation, greatly reducing the risk of carcinogenesis.

Although the incidence rate of skin cancer in SOC is substantially lower than that of individuals who are not SOC, when skin cancer does occur in SOC patients, they face worse overall outcomes with increased morbidity and mortality [14, 31, 54, 55]. It has been previously demonstrated that African Americans have a higher utilization of Mohs Micrographic Surgery (MMS) compared to Caucasian populations (44.2% vs 10.0%) [56] which would further suggest that skin cancers within this population may be more aggressive at time of treatment than in white populations. When skin cancer occurs in SOC, there is a greater propensity for inherently aggressive cancers with a higher risk for tumor invasion and metastasis [50]. However, the poor prognosis is likely multifactorial and due at least in part to atypical clinical presentations resulting in delayed diagnosis [57]. Structural inequalities in medicine and socioeconomic factors, such as lack of access to health care (including dermatologic care), inadequate insurance coverage, and lack of transportation, also contribute to the higher morbidity and mortality seen in patients with SOC [58–60]. There is also clinician bias and decreased index of suspicion among both patients and providers due to lack of representation and data in clinical research [61].

2.4 Effects of visible light

VL (400–700 nm) is a spectrum of electromagnetic radiation to which the rods and cones of the human eye will respond. Sunlight reaching the earth's surface is composed approximately 50% VL; VL is also emitted by artificial sources include light bulbs, computers, and cell phones, among others [62, 63].

Over the last several decades, studies in photodermatology focused mainly on the deleterious effects of UVR on human skin. VL was regarded as having no photobiologic effect; we only recently have begun to understand the cutaneous photobiologic effects of VL [62, 64, 65]. Most of the currently available sunscreens only block wavelengths up to 380 nm and thus do not protect the skin from VL-induced responses, highlighting the need for more effective photoprotection strategy [63].

2.4.1 Photoaging

Similar to UVR, VL may induce photoaging through alterations in extracellular matrix components. In a 2008 study, skin samples of 16 human volunteers were taken 24 h post-exposure to VL. Results showed significantly increased MMP-1 and MMP-9 expression and decreased type I procollagen expression, comparable to the effects of UV irradiation [66]. VL has also been shown to induce mitochondrial DNA damage and free-radical production in epithelial cells [67]. An ex vivo study investigated ascorbate radical production by solar-simulated light in human skin biopsies. Selective filters were used to assess the relative contributions of UV and visible wavelengths. Radical production by UV was found to be about 67% while that of the visible component accounted for about 33% [68].

Studies exploring skin response to isolated components of solar light or a combination of these components suggest that UV, visible, and infrared light have synergistic effects [22]. Hudson et al. studied the effects of individual and interacting components of solar light on human donor primary dermal fibroblasts and epidermal keratinocytes [69]. ROS generation, mitochondrial DNA damage, and nuclear DNA

damage were significantly increased in dermal fibroblasts when exposed to complete solar-simulated radiation compared to each wavelength individually. Studies like this have important implications as the interaction of the numerous wavelengths is representative of the normal physiological condition in sunlight.

2.4.2 Photocarcinogenesis

Although the role of UVR in photocarcinogenesis has been well studied, there is paucity in the literature on the role of and potential pathways for photocarcinogenesis induced by VL. Published data suggest that the mechanism responsible for the genotoxic effects of VL differs from that in the UV range. While direct damage through UV-induced dipyrimidine photoproducts is the major class of DNA lesion involved in photocarcinogenesis, indirect damage through the generation of ROS, especially in the presence of endogenous photosensitizers, is the most reported mechanism of VL-induced DNA damage [70].

Kielbassa et al. analyzed DNA damage induced by UV and VL by exposing mammalian cells to filtered monochrome or broad-band radiation. They found that while oxidative DNA damage formation was observed extending from the UVA1 into VL range, beyond UVB (315 nm), the yield of pyrimidine dimers per unit dose decreased exponentially. DNA damage in the VL range was attributed to oxidative damage induced by the excitation of endogenous photosensitizers [71]. On the other hand, Liebel et al. reported a lack of thymine dimer formation in VL-irradiated tissues even at concentrations sufficient to induce significant increases in ROS [64].

2.4.3 Hyperpigmentation

Hyperpigmentation is a result of an increased amount of melanin within the epidermis, the dermis, or both. Epidemiological studies show that hyperpigmentation is one of the most common reasons why individuals with SOC visit a dermatologist [72]. Post-inflammatory hyperpigmentation (PIH) is a common sequelae of inflammatory dermatoses that present with greater severity in SOC [73]. The most common cause of hyperpigmentation is a result of a post-inflammatory response to UV damage to skin [74]. Several studies have reported the impact of VL in inducing immediate erythema in light and dark-skinned subjects, and immediate pigment darkening, persistent pigment darkening and delayed tanning in dark-skinned subjects [75, 76]. These observations explain the prominent clinical manifestations of pigmentary changes seen in SOC, ranging from post-inflammatory hyperpigmentation to melasma, which is considered by some as part of the manifestation of photoaging process in SOC. This further highlights the importance of broad-spectrum photoprotection beyond UV wavelengths in this population.

When compared to UVA, VL has been found to induce a more prominent and long-lasting pigmentation in skin type III and higher. Mahmoud et al. compared the effect of VL-induced and UVA-induced pigmentary change on immediate pigmentation and delayed tanning [77]. Volunteers were irradiated and cutaneous pigmentary changes, erythema, and edema were assessed by visual examination, digital photography, and spectroscopy over a 2-week period. Results were dose-dependent with higher irradiation doses resulting in darker pigmentation responses in subjects with skin types IV–VI. However, no pigmentation was induced in SPT II. Notably, VL-induced pigmentation was more intense and persistent, lasting for the 2-week study period compared to UV-induced pigmentary change, which quickly faded during the study period. Additionally, erythema was noted immediately after VL irradiation, whereas UVA1 (340–400 nm) caused no erythema at any time point after irradiation.

Specific wavelengths within the VL spectrum have been found to induce different skin responses. Dutiel et al. examined the photobiological effects of wavelengths located at both extremities of the VL spectrum on pigmentation in skin types III and IV [78]. Monochromatic radiation with blue/violet light ($\lambda = 415$ nm) and red light ($\lambda = 630$ nm) were compared to non-exposed and UVB-exposed skin. Blue/violet light induced a dose-dependent hyperpigmentation response whereas red light induced no hyperpigmentation. Furthermore, blue/violet light-induced hyperpigmentation was more pronounced and long-lasting than UVB-induced pigment change.

The mechanism of VL-induced pigmentation has been shown by Regazzetti et al. to be through activation of the Opsin-3 (OPN3), a key sensor for visible light pigmentation in melanocytes [79]. Stimulation of OPN3 by blue light leads to the phosphorylation of microphthalmia-associated transcription factor (MITF) and ultimately increased melanogenesis enzymes: tyrosinase and dopachrome tautomerase.

2.5 Photoprotection in SOC

2.5.1 Photoprotective practices in SOC

Photoprotective measures can mitigate and prevent the damaging effects of UV and VL radiation highlighted above. As outlined in Table 2, such measures can include avoiding sunlight during peak hours from 10 a.m. to 2 p.m., staying in the shade while outdoors, wearing sun-protective clothing, wide-brimmed hat, sunglasses, and applying sunscreen (SPF $\geq$ 30, broad spectrum, and especially for dark-skinned individuals, tinted) [80]. Photoprotective practices differ widely among SPT I–VI populations. A study found that in a large group of individuals participating in a skin cancer

Table 2 Options for photoprotection in individuals with SOC

Behavioral modifications 	Seeking shade Avoiding peak sunlight hours (10am-2pm) Sun protective items: Sunglasses Wide brimmed hats Sun gloves Long sleeve shirts, pants
Topical 	Sunscreen with SPF30+, UVA-PF/SPF ratio ≥ 2/3, and tinted Sunscreens containing antioxidants
Oral 	Antioxidants

screening program, White and Hispanic individuals were more likely to report having used sunscreens in the previous year compared with Black individuals [81]. SOC populations have historically not been counseled to photoprotect to the same degree as light skinned populations [4]. Although the benefits of sun protection in defending against sunburns, photoaging, and skin cancer are well studied in SPTs I–III, the same is not true in SPTs IV–VI.

The underlying reasons for differences between photoprotection among patients with SPT I–III versus those with SPT IV–VI have been studied mostly in the US. There is a lack of representation of SPTs greater than III in medical student resources [82] and dermatology textbook photos [83]. One survey of dermatology trainees and board-certified dermatologists found that 42.9% of respondents reported that they "never, rarely, or only sometimes" based sunscreen recommendations on their patients' skin type [84]. In a National Ambulatory Medical Care Survey, dermatologists recorded mentioning sunscreen in only 1.6% of all dermatology visits, and most commonly mentioned sunscreen to white patients [85]. Recently, researchers have taken efforts to dispel the many misconceptions that exist with regards to photoprotection for SOC to bridge the photoprotection gap SOC populations face [5]. Furthermore, expert guidelines on phototherapy for SOC have been published to aid clinicians in providing high-quality, evidence-based recommendations for their SOC patients [86].

2.5.2 Role of sunscreen

a. *Protection against UVB, UVA and VL*

Sunscreens are made from active ingredients called filters and are labeled based on their sun protection factor (SPF) and UVA protection factor (UVA-PF). SPF is determined by calculating the ratio of minimum erythematous dose (MED), i.e., the minimum dose of UV radiation that elicits erythema, on sunscreen-protected vs not sunscreen-protected skin. It is therefore a reflection of primarily the effect of UVB, and to a lesser extent, UVA2 (320–340 nm) [87]. Though there are different methods to calculate UVA-PF, one common technique involves determining the level of UVA exposure leading to persistent pigment darkening (PPD) [88]. UVA-PF is then a ratio of PPD on sunscreen-protect skin vs not sunscreen-protected skin. Regulatory agency in Japan introduced categorization of PPD measurements into a range from PA+ (some protection) to PA++++ (excellent protection) [89]. European guidelines dictate a minimum UVA-PF/SPF ratio of 1:3 for sunscreens and a critical wavelength of ≥ 370 nm [90, 91]. In the United States, the Food and Drug Administration (FDA) utilizes a critical wavelength determination and classifies sunscreens as "broad spectrum" should their 90% UV absorbance occur at ≥ 370 nm [92]. Currently published VL protection assessment methods have been reviewed, and a VL protection method has been proposed [93]. However, there is no standardized test for VL protection in sunscreen.

The biological differences in skin of color response to UVA, UVB, and VL highlighted earlier in this review allows for tailoring sunscreen recommendations for SOC. There is a stronger need among SOC to protect against UVA and VL due to greater propensity for hyperpigmentary disorders. Thus, sunscreens worn by SOC populations should be chosen not only for the protection against UVB and UVA2 (320–340 nm) to prevent erythema, but also against UVA1 and VL to prevent hyperpigmentation. Skin phototype can be used to guide selection of sunscreen with different spectral absorption profiles and protection factors (PF) (Fig. 2) [94]. For SPT IV-VI, sunscreen with SPF30+ and an UVA-PF/SPF ratio ≥ 2/3 addresses the unique photoprotective needs of SOC [94]. In contrast, SPT I–III requires SPF-50+ (given the relatively greater protection against UVB) and an UVA-PF/SPF ratio ≥ 1/3 [94]. While such "broad spectrum" sunscreens by definition protect against UVA and UVB, they do not protect against VL. Tinted sunscreens offer the VL protection lacking in broad-spectrum sunscreens by leveraging iron oxides and pigmentary titanium dioxides in their formulations [95]. Of note, titanium dioxides must be pigmentary and not micronized to offer VL protection [96].

Recently, three new filters have been developed that extend coverage to the longwave UVA and VL spectra. An in vitro study evaluated the addition of methoxypropylamino cyclohexenylidene (MCE), a UVA1 filter which absorbs UV rays at a peak wavelength of 385 nm [97]. Addition of 0.7% MCE resulted in UVA1 absorption up to 385 nm and addition of 1.5%

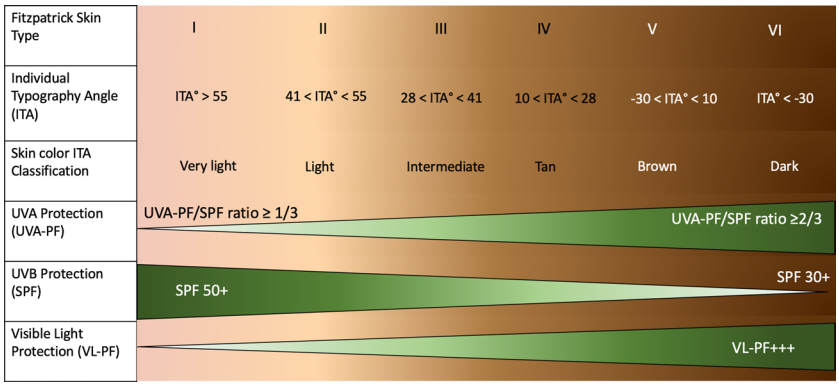


Fig. 2 SPF/UPF for different skin phototypes. Sunscreen should be chosen based on the SPT; for example, individuals with SPT IV–VI should opt for a sunscreen with a UVA-PF/SPF ratio of > 2/3 and with an SPF minimum of 30 or higher. Individuals with SPT I–II should choose a sunscreen with SPF-50+ but a UVA-PF-SPF ratio of > 1/3 is typically sufficient. Individuals with SPT III may opt for more intermediary SPF such as 40+. Adapted from Passeron et al. [94]

MCE resulted in absorption up to 400 nm in a three-dimensional human skin model, while also decreasing UVA1-induced hyperpigmentation compared to the control sunscreen. Another filter, TriAsorB, from the 1,2,4-triazine family of compounds, was found to provide coverage up to the blue light wavelength in the visible light spectrum and protected skin from VL-induced oxidative skin damage [98]. A third organic filter, bis-(diethylaminohydroxybenzoyl benzoyl) piperazine (BDBP), absorbs between 350 and 425 nm; it has been shown to protect against pigmentation induced by 385–405 nm radiation [99]. All of these filters have now been approved by EU regulatory agency and incorporated in commercial sunscreens available in EU.

A common concern with daily sunscreen use is the potential for compromised vitamin D synthesis; however, there has been no definitive evidence that regular sunscreen use decreases vitamin D synthesis, even when sunscreen is applied under optimal conditions [100, 101]. Based on a study of over 3400 individuals in the US, routine practice of photoprotection (shade, clothing, sunscreen) was not associated with decreased bone mineral density or increased in osteoporotic fracture [102].

b. *Treatment and prevention of pigmentary disorders*

The use of sunscreen has been shown to help reduce and prevent pigmentary disorders including PIH and melasma [103]. Patients undergoing laser therapies are often advised to avoid sun exposure and to apply sunscreen regularly for several weeks following treatment to prevent new hyperpigmentation [103, 104]. A study showed that daily sunscreen use can prevent development of PIH post-procedurally [105]; and another study demonstrated that it can also lighten existing hyperpigmentation, with greater improvement seen with application of SPF 60 compared to SPF30 sunscreens [106].

While the pathogenesis of melasma is multifactorial, an important cause is sun exposure. In a study of 185 Moroccan women using SPF-50 UVA-PF 28 sunscreen every two hours, assessments were made based on patient self-reported pigmentation outcomes and colorimetry measurements at 3-, 6-, and 12-month intervals. With consistent sunscreen use, only 5 new cases of melasma (2.7%) occurred, as compared to an incidence of 53% in a study by the same investigators with a similar population, geographic region, and time frame. Additionally, 8 of 12 patients (67%) with pre-existing melasma noted improvement in their disease. This study demonstrated that regular sunscreen use was effective at reducing incidence of melasma, though the validity of the study is limited by lack of control group [107]. Regular sunscreen use has also been shown to improve preexisting melasma. A study of 100 patients (80 women, 20 men) with SPT III-IV and melasma primarily of the cheeks and nose were instructed to apply SPF 19 PA+++ sunscreen three times daily. Melasma area severity index (MASI) scores were assessed at baseline and at 12 weeks after daily sunscreen use. MASI scores decreased from 12.38 to 9.15, a statistically significant change, with concurrent improvement in Melasma Quality of Life (MELASQOL) scores [108]. The researchers concluded that regular sunscreen use is effective at improving existing melasma and improving melasma-related quality of life [108]. Very few studies have been done on the efficacy of tinted sunscreen in melasma. This represents an area in need of further research. One open-label single-site study of 10 female subjects found that use of a 30% tetrahexyldecyl (THD) ascorbate serum plus a mineral-based tinted SPF 45 sunscreen resulted in improvement in skin tone evenness and erythema in 7 out of 10 (70%) subjects [109].

c. *Sunscreen with depigmenting agents*

Sunscreens that contain depigmenting agents can be particularly helpful for patients with pigmentary disorders. Commonly used depigmenting agents in the treatment of melasma include hydroquinone, niacinamide, vitamin E, and thiamidol. A randomized controlled trial of 35 Hispanic FST III–V women undergoing twice daily application of a sunscreen (SPF was not stated) containing 4% hydroquinone plus 10% glycolic acid, vitamin C and vitamin E versus sunscreen-only control for 12 weeks found significantly improved pigmentation in the hydroquinone containing sunscreen group [110]. A randomized controlled trial of 27 SPT IV–V women treated with 4% hydroquinone versus 4% niacinamide did not find significant differences in clinical outcomes between the two treatments, as assessed by MASI, Physicians Global Assessment and colorimetric assessment [111]. A randomized controlled trial evaluating 4% hydroquinone versus 0.2% thiamidol (a tyrosine kinase inhibitor) in the treatment of melasma amongst a group of SPT IV–V found no statistically significant difference in MASI, MELASQOL, colourimetric contrast, or Global esthetic Improvement Scale scores. Both groups saw statistically significant reductions in all outcomes with use of the depigmenting agents [112].

d. *Sunscreen with antioxidants*

Sunscreens may also contain other additives such as antioxidants to combat the free radicals that occur from exposure to UVR and VL. Free radicals on the skin are known to cause DNA damage, hyperpigmentation, and melanogenesis [113]. Exogenous antioxidants thus play a role in supporting the skin's own endogenous antioxidants that minimize the damaging effects of free radicals and reactive oxygen species [113]. Such antioxidants can include ascorbate (vitamin C), tocopherols, carotenoids, polyphenols, and flavenoids [114].

Vitamin C is normally found in large, plentiful levels in the skin, aiding the skin's natural defenses against UV-induced photodamage and stimulating collagen synthesis to improve the elasticity of skin [115]; however, older or photodamaged skin has been found to have depleted levels of vitamin C [116]. Numerous studies have evaluated the use of topical vitamin C in protecting against erythema and sunburn [117–119], as well as improving existing photodamage [120]. Vitamin E also protects keratinocytes from UVA1-induced pyridine dimer and oxidatively- generated DNA damage [121]. The combination of topical vitamin C and vitamin E provides superior protection, especially when used daily, as compared to the use of vitamin C or vitamin E alone [118].

Antioxidants, and sunscreen containing antioxidants, have been shown to decrease VL and UVA1-induced pigmentation in dark-skinned subjects [122, 123]. In an open-label, single-center, 12-week study, sunscreen containing photolyase and antioxidants have been shown to improve photoaging changes [124]. It should be noted that while antioxidants are beneficial as adjuvants, they should not replace the sunscreen and other photoprotective measures as sole agents.

e. *Tinted sunscreen & cosmetic elegance*

Improving cosmetic appearance of sunscreen can help improve compliance of regular sunscreen use. The transparency of the sunscreen application and reduction of white residue is important especially given the potential contrast on dark skin [95]. To reduce the whiteness and chalkiness of sunscreens, formulations often utilize mineral (inorganic) filters composed of nanoparticles of zinc oxide or titanium dioxide; however, in order for sunscreens to provide VL protection, they must be visible on the skin, i.e., tinted [95]. Tinted sunscreens, containing iron oxides and/or pigmentary titanium dioxide, can also be leveraged to improve adherence by color-matching to dark skin while also evening out the appearance of hyperpigmented areas. A 2020 survey of dermatology trainees and board-certified dermatologists found that respondents regarded the cosmetic elegance of a sunscreen the least important factor when forming recommendations for their patients [84]. This, in combination with the finding that dermatologists counseled their SOC patients less on sunscreen use compared to other patients, highlights the need for more culturally competent training and care by dermatologists [73]. Having a familiarity with the breadth of sunscreen formulations and which are more suited to SOC—not only in regards to UV and VL protection but also considering the cosmetic elegance–will hopefully boost patient adherence to sunscreen application. A practical guide to tinted sunscreens has been recently published [125].

2.6 Role of sun-protective clothing

Aside from sunscreens, other photoprotective measures include sunglasses, wide-brimmed hats, and articles of clothing such as long-sleeved shirts and pants. Although sun-protective clothing has been found to block UVR better than sunscreen, use of sun-protective clothing lags behind other photoprotection measures [126]. A cross-sectional analysis of the National Health and Nutrition Examination survey of respondents aged 20–60 from 2003 to 2006 found that sunscreen was the most common sun-protective measure Americans utilized (30% of respondents), while even less sought shade frequently (24%), wore a hat (16%), or wore long sleeves (4%).

Interestingly, they found that the odds of sunburn was decreased in individuals seeking shade or frequently wearing

long sleeves, and higher in those frequently wearing sunscreen [127]. This is likely related to the fact that consumers rarely apply sunscreen as appropriately or frequently as recommended (2 mg/cm^2 and reapplied every two hours when outdoors) whereas wearing photoprotective clothing is less likely to be subject to user error [128]. Additionally, the use of sunscreen has been associated with longer intentional sun exposure leading to sunburn [129]. A major limitation of this study is that it was limited to non-Hispanic White respondents. Other articles have demonstrated that non-Hispanic Black Americans are more likely to practice sun avoidance and wear sun-protective clothing as opposed to sunscreen [130]. Hispanics have demonstrated decreased likelihood of seeking shade and wearing sun-protective clothing with acculturation to US norms [131]. An important benefit of sun avoidance and sun-protective clothing is a decreased odds of sunburn [132], and the resultant photoaging and hyperpigmentation in SOC.

Clothing provides protection from the sun by scattering or absorbing UVR. Commercially available clothing does not necessarily protect well against UVA and UVB, with data suggesting that one-third of summer clothing articles have an Ultraviolet Protective Factor (UPF) of less than 15 [133]. Patients should seek UPF labeled clothing, which is manufactured with tightly woven fibers and tested to determine the UPF values [133]. Various factors affect the degree of UPF protection, including the color, material, and fiber quality used [134, 135]. Other factors that affect the performance of UPF clothing include stretching, shrinking, wetting, washing, or laundering; because of this, the minimum UPF level recommended is 40 to 50, or higher [136].

Limitations to sun-protective clothing include undesirability of being fully clothed in hot or humid environments, inability to cover all exposed skin surfaces with clothing at all times, and finding UPF clothing that is lightweight and moisture-wicking [126]. Cost could be a barrier to accessing high-quality, effective UPF clothing, although this does not appear to have been examined yet in the literature.

2.7 Role of oral agents

a. *Vitamins*

Oral photoprotective agents include antioxidants, anti-inflammatories, and immunomodulators [137]. One of the better studied classes of oral photoprotective agents is vitamins. Carotenoids-pigments that occur naturally in fruits and vegetables-work by decreasing ROS [138]. Vitamin E (α-tocopherol) prevents production of ROS during lipoxidation and during the free-radical reaction, mitigating UVA1-induced keratinocyte damage [121]. Vitamin E, when taken together with vitamin C [139] or carotenoids [140], has synergistic protection against UV-mediated skin damage. Oral vitamin C supplementation alone, however, has not been shown to reduce UV-mediated erythema [141].

b. *Polypodium leucotomos*

Polypodium leucotomos is a fern found in Central and South America that is harvested for *Polypodium leucotomos* extract (PLE), an over-the-counter UV protectant [142]. Among oral photoprotective agents, PLE has been most well studied. A review on the effect of PLE highlighted that PLE exerts photoprotective properties by preventing UV and VL-induced extracellular matrix degradation, while it wields antiinflammatory properties by inhibiting UV- induced immunosuppression [142]. Studies on PLE photoprotection have been done in vitro, in animal models and in human subjects [142]. The human studies have small sample sizes; Mohammad et al. conducted a prospective clinical trial of 22 volunteers with FST IV-VI who were treated with PLE then irradiated with VL (up to 480 J/cm^2). They found a decrease in VL- induced pigmentation, and a statistically significant decrease in inflammatory marker COX-2 and MART-2 (melanocytic marker of pigmentation) in the PLE treated skin [143]. Truchuelo et al. report a study of 7 volunteers whose gluteal skin was irradiated with a single dose of VL (200 J/cm^2) and IR (600 J/cm^2) and who took oral PLE supplementation for 21 days. They found a statistically significant decrease of 52% in MMP-1 expression in participant's skin, demonstrating PLE's ability to reduce extracellular matrix degradation [144]. Goh et al. conducted a double-blind, placebo-controlled trial of 40 patients with melasma and receiving treatment with topical 4% hydroquinone cream and sunscreen with a SPF of 50+. The subjects were randomized to receive either oral PLE supplementation or placebo for 12 weeks. The modified Melasma Area and Severity Index (mMASI) scores of the PLE group at 12 weeks were significantly lower than those of the placebo group [145].

In contrast, Ahmed et al.'s study evaluating the effectiveness of PLE UVR amongst a group of Hispanic women showed no statistically significant benefit to PLE supplementation. Thirty-three patients were randomized to oral PLE or placebo three times daily for 12 weeks; all subjects were instructed to apply broad-spectrum SPF 55 sunscreen every morning. Researchers assessed the change in melanin index from baseline at 6 and 12 weeks. They found statistically significant improvements in melanin index scores between weeks 0 and 12 for both groups (28.8% improvement in PLE group; 13.8% improvement in placebo group), with no statistically significant difference in melanin index between groups [146]. Though the results did not support the hypothesis that PLE would result in significant improvement in melanin index, the sample size was small and

limited the power to detect a significant difference. The study also reinforces the effectiveness of sunscreen in improving existing melasma, as discussed earlier in this review paper.

c. *Pinus pinaster*

Pinus pinaster is an extract from the French maritime pine bark containing proanthocyanidins and other antioxidants which has been formulated into a standardized extract called Pycnogenol. A prospective study of 30 women who took Pycnogenol 25 mg three times daily with meals for 30 days assessed melasma area indices and pigment intensity indices after treatment. Upon completion of the study, participants had an average melasma area index decrease of 25.86 mm^2 [147]. Another study by Aladrén et al. of 30 women with FST I–V and melasma investigated the use of *Pinus pinaster* and grapeseed oil containing supplements. Participants took two supplement capsules every morning and wore SPF-50 sunscreen daily. Investigators measured MASI scores, Physician Global Assessment, Patient Global Assessment, and Melasma Assessment by VISIA Complexion Analysis Images at Days 28, 56, and 84. Participants had a statistically significant decrease in MASI scores at all three follow-up intervals (−28% at Day 28, −33.7% at Day 56, −41.1% at Day 84). VISIA Complexion analyses found a statistically significant reduction in melasma spots of 15.3% at Day 28, 28.6% at Day 56, and 46.2% at Day 84. Remarkably, 96.7% of participants reported self-improvement and 93.3% of physicians noted improvement of their hyperpigmentation [148]. This study is limited by lack of control group, with unknown contribution of the *Pinus pinaster* versus sunscreen in reduction of the hyperpigmentation.

Numerous other oral photoprotective botanicals exist, including green tea, pomegranate, reservatrol, turmeric, and silmarin [149]. In summary, data is emerging that oral photoprotective agents are potentially useful adjuvant in comprehensive photoprotection strategy.

3 Conclusion

Though dark skin individuals are less afflicted by UV-erythema, photoaging and photocarcinogenesis given natural protection from melanin in the skin, they are disproportionately affected by hyperpigmentary disorders. Understanding the unique considerations of SOC reaction to sun exposure helps clinicians tailor photoprotective recommendations for their patients. Healthcare providers should have a good basic understanding of the effects of UVA, UVB and VL on the skin, so that they would be comfortable in counseling SOC patients on sun exposure, risks for photodamage and pigmentary disorders, and methods for photoprotection. The general public should be encouraged to utilize multiple methods of photoprotection, ranging from avoidance of sunlight during peak intensity hours, seeking shade, wearing sun-protective clothing and wide-brimmed hat, and applying sunscreen. Sunscreens that are most appropriate for SOC populations are those with SPF30+, UVA-PF/SPF ratios ≥ 2/3 and tinted. Emerging data shows that topical antioxidants and oral photoprotective agents might be useful adjuvants in photoprotection. Although there have been increased efforts recently, more research into photoprotection for SOC and targeted public education is required to disseminate photoprotection resources that are patient-centered and evidence-based.

Declarations

Conflict of interest SSeck and JH have no conflict to disclose. SSchalka has served as consultant for ISDIN and FQM Brasil and as a speaker for Pierre Fabre, La Roche Posay and Bioderma. HWL has served as investigator for Incyte, La Roche-Posay, Pfizer, and PCORI, as a consultant for Pierre Fabre, ISDIN, Ferndale, La Roche-Posay, and Beiersdorf, and as a speaker on general educational session sponsored by La Roche-Posay, Pierre Fabre and Bioderma.

References

1. Heath, C. R., DeLeo, V. A., & Taylor, S. C. (2021). Increasing skin of color publications in the dermatology literature: A call to action. *Cutis, 108*(3), 155–157.
2. Kimball, A. B. (2008). Skin differences, needs, and disorders across global populations. *Journal of Investigative Dermatology Symposium Proceedings, 13*(1), 2–5.
3. Brenner, M., & Hearing, V. J. (2008). The protective role of melanin against UV damage in human skin. *Photochemistry and Photobiology, 84*(3), 539–549.
4. Kumar, P., & Kim, N. (2021). Age-based gaps between awareness of photoaging and photoprotective practices among African American adults. *Photodermatology, Photoimmunology & Photomedicine, 37*, 428–430.
5. Taylor, S. C., Alexis, A. F., Armstrong, A. W., Fuxench, Z. C. C., & Lim, H. W. (2022). Misconceptions of photoprotection in skin of color. *Journal of the American Academy of Dermatology, 86*(3), S9–S17.
6. Colby, S. L., & Ortman, J. M. (2015). Projections of the size and composition of the US Population: 2014 to 2060. Population estimates and projections. Current Population Reports. P25-1143. *US Census Bureau*.
7. Rees, P., Wohland, P., Norman, P., & Boden, P. (2012). Ethnic population projections for the UK, 2001–2051. *Journal of Population Research, 29*(1), 45–89.
8. Taylor, S. C. (2002). Skin of color: Biology, structure, function, and implications for dermatologic disease. *Journal of the American Academy of Dermatology, 46*(2), S41–S62.
9. Kundu, R. V., & Patterson, S. (2013). Dermatologic conditions in skin of color: Part I. Special considerations for common skin disorders. *American Family Physician, 87*(12), 850–856.
10. Osto, M., Hamzavi, I. H., Lim, H. W., & Kohli, I. (2022). Individual typology angle and fitzpatrick skin phototypes are not equivalent in photodermatology. *Photochemistry and Photobiology, 98*(1), 127–129.

11. Ware, O. R., Dawson, J. E., Shinohara, M. M., & Taylor, S. C. (2020). Racial limitations of Fitzpatrick skin type. *Cutis, 105*(2), 77–80.
12. Onalaja, A. A., & Taylor, S. C. (2021). Defining skin of color. In B. S. Li & H. I. Maibach (Eds.), *Ethnic skin and hair and other cultural considerations* (pp. 3–18). Springer.
13. Sommers, M. S., Fargo, J. D., Regueira, Y., Brown, K. M., Beacham, B. L., Perfetti, A. R., Everett, J. S., & Margolis, D. J. (2019). Are the Fitzpatrick skin phototypes valid for cancer risk assessment in a racially and ethnically diverse sample of women? *Ethnicity & Disease, 29*(3), 505.
14. Agbai, O. N., Buster, K., Sanchez, M., Hernandez, C., Kundu, R. V., Chiu, M., Roberts, W. E., Draelos, Z. D., Bhushan, R., Taylor, S. C., & Lim, H. W. (2014). Skin cancer and photoprotection in people of color: A review and recommendations for physicians and the public. *Journal of the American Academy of Dermatology, 70*(4), 748–762.
15. Wilkes, M., Wright, C. Y., du Plessis, J. L., & Reeder, A. (2015). Fitzpatrick skin type, individual typology angle, and melanin index in an African population: Steps toward universally applicable skin photosensitivity assessments. *JAMA Dermatology, 151*(8), 902–903.
16. Eilers, S., Bach, D. Q., Gaber, R., Blatt, H., Guevara, Y., Nitsche, K., Kundu, R. V., & Robinson, J. K. (2013). Accuracy of self-report in assessing Fitzpatrick skin phototypes I through VI. *JAMA Dermatology, 149*(11), 1289–1294.
17. Karsten, A. E., Singh, A., Karsten, P. A., & Braun, M. W. (2013). Diffuse reflectance spectroscopy as a tool to measure the absorption coefficient in skin: South African skin phototypes. *Photochemistry and Photobiology, 89*(1), 227–233.
18. D'Orazio, J., Jarrett, S., Amaro-Ortiz, A., & Scott, T. (2013). UV radiation and the skin. *International Journal of Molecular Sciences, 14*(6), 12222–12248.
19. Austin, E., Geisler, A. N., Nguyen, J., Kohli, I., Hamzavi, I., Lim, H. W., & Jagdeo, J. (2021). Visible light. Part I: Properties and cutaneous effects of visible light. *Journal of the American Academy of Dermatology, 84*(5), 1219–1231.
20. Clydesdale, G. J., Dandie, G. W., & Muller, H. K. (2001). Ultraviolet light induced injury: Immunological and inflammatory effects. *Immunology and Cell Biology, 79*(6), 547–568.
21. Sklar, L. R., Almutawa, F., Lim, H. W., & Hamzavi, I. (2012). Effects of ultraviolet radiation, visible light, and infrared radiation on erythema and pigmentation: A review. *Photochemical & Photobiological Sciences, 12*(1), 54–64.
22. Kohli, I., Chaowattanapanit, S., Mohammad, T. F., Nicholson, C. L., Fatima, S., Jacobsen, G., Kollias, N., Lim, H. W., & Hamzavi, I. H. (2018). Synergistic effects of long-wavelength ultraviolet Al and visible light on pigmentation and erythema. *British Journal of Dermatology, 178*(5), 1173–1180.
23. Kaidbey, K. H., Agin, P. P., Sayre, R. M., & Kligman, A. M. (1979). Photoprotection by melanin—A comparison of black and Caucasian skin. *Journal of the American Academy of Dermatology, 1*(3), 249–260.
24. Cragg, N., Chadwick, C. A., Potten, C. S., Sheehan, J. M., & Young, A. R. (2002). Repeated ultraviolet exposure affords the same protection against DNA photodamage and erythema in human skin types II and IV but is associated with faster DNA repair in skin type IV. *Journal of Investigative Dermatology, 118*(5), 825–829.
25. Jimbow, K., Quevedo, W. C., Jr., Fitzpatrick, T. B., & Szabo, G. (1976). Some aspects of melanin biology: 1950–1975. *Journal of Investigative Dermatology, 67*(1), 72–89.
26. Bradford, P. T. (2009). Skin cancer in skin of color. *Dermatology nursing/Dermatology Nurses' Association, 21*(4), 170.
27. Fisher, G. J., Kang, S., Varani, J., Bata-Csorgo, Z., Wan, Y., Datta, S., & Voorhees, J. J. (2002). Mechanisms of photoaging and chronological skin aging. *Archives of Dermatology, 138*(11), 1462–1470.
28. Szabó, G., Gerald, A. B., Pathak, M. A., & Fitzpatrick, T. B. (1969). Racial differences in the fate of melanosomes in human epidermis. *Nature, 222*(5198), 1081–1082.
29. Tadokoro, T., Kobayashi, N., Zmudzka, B. Z., Ito, S., Wakamatsu, K., Yamaguchi, Y., Korossy, K. S., Miller, S. A., Beer, J. Z., & Hearing, V. J. (2003). UV-induced DNA damage and melanin content in human skin differing in racial/ethnic origin. *The FASEB Journal, 17*(9), 1177–1179.
30. Del Bino, S., Sok, J., Bessac, E., & Bernerd, F. (2006). Relationship between skin response to ultraviolet exposure and skin color type. *Pigment Cell Research, 19*(6), 606–614.
31. Halder, R. M., & Bridgeman-Shah, S. (1995). Skin cancer in African Americans. *Cancer, 75*(S2), 667–673.
32. Han, A., Chien, A. L., & Kang, S. (2014). Photoaging. *Dermatologic Clinics, 32*(3), 291–299.
33. Taylor, S. C. (2005). Photoaging and pigmentary changes of the skin. In C. M. Burgess (Ed.), *Cosmetic dermatology* (pp. 29–51). Springer.
34. Iwuala, C., & Taylor, S. C. (2022). Structural and functional differences in skin of colour. *Clinical and Experimental Dermatology, 47*(2), 247–250.
35. Badreshia-Bansal, S., Patel, M., & Taylor, S. C. (2016). Structure and function of skin. In A. Kelly, S. C. Taylor, H. W. Lim, & A. Serrano (Eds.), *Taylor and Kelly's dermatology for skin of color, 2e.* McGraw Hill.
36. Fisher, G. J., Wang, Z., Datta, S. C., Varani, J., Kang, S., & Voorhees, J. J. (1997). Pathophysiology of premature skin aging induced by ultraviolet light. *New England Journal of Medicine, 337*(20), 1419–1429.
37. Griffiths, C. E., Wang, T. S., Hamilton, T. A., Voorhees, J. J., & Ellis, C. N. (1992). A photonumeric scale for the assessment of cutaneous photodamage. *Archives of Dermatology, 128*(3), 347–351.
38. Larnier, C., Ortonne, J. P., Venot, A., Faivre, B., Béani, J. C., Thomas, P., Brown, T. C., & Sendagorta, E. (1994). Evaluation of cutaneous photodamage using a photographic scale. *British Journal of Dermatology, 130*(2), 167–173.
39. Chung, J. H., Lee, S. H., Youn, C. S., Park, B. J., Kim, K. H., Park, K. C., Cho, K. H., & Eun, H. C. (2001). Cutaneous photodamage in Koreans: Influence of sex, sun exposure, smoking, and skin color. *Archives of Dermatology, 137*(8), 1043–1051.
40. Chien, A. L., Qi, J., Grandhi, R., Kim, N., César, S. S., Harris-Tryon, T., Jang, M. S., Olowoyeye, O., Kuhn, D., Leung, S., & Rainer, B. M. (2018). Effect of age, gender, and sun exposure on ethnic skin photoaging: Evidence gathered using a new photonumeric scale. *Journal of the National Medical Association, 110*(2), 176–181.
41. Stern, R. S. (2010). Prevalence of a history of skin cancer in 2007: Results of an incidence-based model. *Archives of Dermatology, 146*(3), 279–282.
42. Dennis, L. K., Vanbeek, M. J., Freeman, L. E. B., Smith, B. J., Dawson, D. V., & Coughlin, J. A. (2008). Sunburns and risk of cutaneous melanoma: Does age matter? A comprehensive meta-analysis. *Annals of Epidemiology, 18*(8), 614–627.
43. Wu, S., Han, J., Laden, F., & Qureshi, A. A. (2014). Long-term ultraviolet flux, other potential risk factors, and skin cancer risk: A cohort study. *Cancer Epidemiology, Biomarkers & Prevention, 23*(6), 1080–1089.
44. An, S., Kim, K., Moon, S., Ko, K. P., Kim, I., Lee, J. E., & Park, S. K. (2021). Indoor tanning and the risk of overall and early-onset melanoma and non-melanoma skin cancer: Systematic review and meta-analysis. *Cancers, 13*(23), 5940.
45. Colantonio, S., Bracken, M. B., & Beecker, J. (2014). The association of indoor tanning and melanoma in adults: Systematic

review and meta-analysis. *Journal of the American Academy of Dermatology, 70*(5), 847–857.
46. Lopes, F. C., Sleiman, M. G., Sebastian, K., Bogucka, R., Jacobs, E. A., & Adamson, A. S. (2021). UV exposure and the risk of cutaneous melanoma in skin of color: A systematic review. *JAMA Dermatology, 157*(2), 213–219.
47. Kolitz, E., Lopes, F. C., Arffa, M., Pineider, J., Bogucka, R., & Adamson, A. S. (2022). UV Exposure and the risk of keratinocyte carcinoma in skin of color: A systematic review. *JAMA Dermatology, 158*, 542.
48. Gloster, H. M., Jr., & Neal, K. (2006). Skin cancer in skin of color. *Journal of the American Academy of Dermatology, 55*(5), 741–760.
49. Ridky, T. W. (2007). Nonmelanoma skin cancer. *Journal of the American Academy of Dermatology, 57*(3), 484–501.
50. Higgins, S., Nazemi, A., Chow, M., & Wysong, A. (2018). Review of nonmelanoma skin cancer in African Americans, Hispanics, and Asians. *Dermatologic Surgery, 44*(7), 903–910.
51. Gloster, H. M., Jr., & Brodland, D. G. (1996). The epidemiology of skin cancer. *Dermatologic Surgery, 22*(3), 217–226.
52. American Cancer Society. (2022). *Cancer facts & figures 2022*. American Cancer Society.
53. Yamaguchi, Y., Takahashi, K., Zmudzka, B. Z., Kornhauser, A., Miller, S. A., Tadokoro, T., Berens, W., Beer, J. Z., Hearing, V. J., Yamaguchi, Y., & Takahashi, K. (2006). Human skin responses to UV radiation: Pigment in the upper epidermis protects against DNA damage in the lower epidermis and facilitates apoptosis. *The FASEB Journal, 20*(9), 1486–1488.
54. Gupta, A. K., Bharadwaj, M., & Mehrotra, R. (2016). Skin cancer concerns in people of color: Risk factors and prevention. *Asian Pacific Journal of Cancer Prevention: APJCP, 17*(12), 5257.
55. Battie, C., Gohara, M., Verschoore, M., & Roberts, W. (2013). Skin cancer in skin of color: An update on current facts, trends, and misconceptions. *Journal of Drugs in Dermatology: JDD, 12*(2), 194–198.
56. Reeder, V. J., Gustafson, C. J., Mireku, K., Davis, S. A., Feldman, S. R., & Pearce, D. J. (2015). Trends in Mohs surgery from 1995 to 2010: An analysis of nationally representative data. *Dermatologic Surgery, 41*(3), 397–403.
57. Kaufman, B. P., & Alexis, A. F. (2017). Skin cancer mortality in patients with skin of color. *Cutis, 99*(5), 307–308.
58. Dawes, S. M., Tsai, S., Gittleman, H., Barnholtz-Sloan, J. S., & Bordeaux, J. S. (2016). Racial disparities in melanoma survival. *Journal of the American Academy of Dermatology, 75*(5), 983–991.
59. Conic, R. Z., Cabrera, C. I., Khorana, A. A., & Gastman, B. R. (2018). Determination of the impact of melanoma surgical timing on survival using the National Cancer Database. *Journal of the American Academy of Dermatology, 78*(1), 40–46.
60. Tripathi, R., Archibald, L. K., Mazmudar, R. S., Conic, R. R., Rothermel, L. D., Scott, J. F., & Bordeaux, J. S. (2020). Racial differences in time to treatment for melanoma. *Journal of the American Academy of Dermatology, 83*(3), 854–859.
61. Davis, D. S., Robinson, C., & Callender, V. D. (2021). Skin cancer in women of color: Epidemiology, pathogenesis and clinical manifestations. *International Journal of Women's Dermatology, 7*(2), 127–134.
62. Frederick, J. E., Snell, H. E., & Haywood, E. K. (1989). Solar ultraviolet radiation at the earth's surface. *Photochemistry and Photobiology, 50*(4), 443–450.
63. Cohen, L., Brodsky, M. A., Zubair, R., Kohli, I., Hamzavi, I. H., & Sadeghpour, M. (2020). Cutaneous interaction with visible light: What do we know. *Journal of the American Academy of Dermatology*. https://doi.org/10.1016/j.jaad.2020.03.115
64. Liebel, F., Kaur, S., Ruvolo, E., Kollias, N., & Southall, M. D. (2012). Irradiation of skin with visible light induces reactive oxygen species and matrix-degrading enzymes. *Journal of Investigative Dermatology, 132*(7), 1901–1907.
65. Ezekwe, N., Maghfour, J., & Kohli, I. (2022). Visible light and the skin. *Photochemistry and photobiology*. https://doi.org/10.1111/php.13634
66. Cho, S., Lee, M. J., Kim, M. S., Lee, S., Kim, Y. K., Lee, D. H., Lee, C. W., Cho, K. H., & Chung, J. H. (2008). Infrared plus visible light and heat from natural sunlight participate in the expression of MMPs and type I procollagen as well as infiltration of inflammatory cell in human skin in vivo. *Journal of Dermatological Science, 50*(2), 123–133.
67. Pourang, A., Tisack, A., Ezekwe, N., Torres, A. E., Kohli, I., Hamzavi, I. H., & Lim, H. W. (2022). Effects of visible light on mechanisms of skin photoaging. *Photodermatology, Photoimmunology & Photomedicine, 38*(3), 191–196.
68. Haywood, R. (2006). Relevance of sunscreen application method, visible light and sunlight intensity to free-radical protection: A study of ex vivo human skin. *Photochemistry and Photobiology, 82*(4), 1123–1131.
69. Hudson, L., Rashdan, E., Bonn, C. A., Chavan, B., Rawlings, D., & Birch-Machin, M. A. (2020). Individual and combined effects of the infrared, visible, and ultraviolet light components of solar radiation on damage biomarkers in human skin cells. *The FASEB Journal, 34*(3), 3874–3883.
70. Mahmoud, B. H., Hexsel, C. L., Hamzavi, I. H., & Lim, H. W. (2008). Effects of visible light on the skin. *Photochemistry and Photobiology, 84*(2), 450–462.
71. Kielbassa, C., Roza, L., & Epe, B. (1997). Wavelength dependence of oxidative DNA damage induced by UV and visible light. *Carcinogenesis, 18*(4), 811–816.
72. Callender, V. D., Baldwin, H., Cook-Bolden, F. E., Alexis, A. F., Stein Gold, L., & Guenin, E. (2022). Effects of topical retinoids on acne and post-inflammatory hyperpigmentation in patients with skin of color: A clinical review and implications for practice. *American Journal of Clinical Dermatology, 23*(1), 69–81.
73. Davis, E. C., & Callender, V. D. (2010). Postinflammatory hyperpigmentation: A review of the epidemiology, clinical features, and treatment options in skin of color. *The Journal of Clinical and Aesthetic Dermatology, 3*(7), 20.
74. Ortonne, J. P., & Bissett, D. L. (2008). Latest insights into skin hyperpigmentation. *Journal of Investigative Dermatology Symposium Proceedings, 13*(1), 10–14.
75. Porges, S. B., Kaidbey, K. H., & Grove, G. L. (1988). Quantification of visible light-induced melanogenesis in human skin. *Photo-dermatology, 5*(5), 197–200.
76. Rosen, C. F., Jacques, S. L., Stuart, M. E., & Gange, R. W. (1990). Immediate pigment darkening: Visual and reflectance spectrophotometric analysis of action spectrum. *Photochemistry and Photobiology, 51*(5), 583–588.
77. Mahmoud, B. H., Ruvolo, E., Hexsel, C. L., Liu, Y., Owen, M. R., Kollias, N., Lim, H. W., & Hamzavi, I. H. (2010). Impact of long-wavelength UVA and visible light on melanocompetent skin. *Journal of Investigative Dermatology, 130*(8), 2092–2097.
78. Duteil, L., Cardot-Leccia, N., Queille-Roussel, C., Maubert, Y., Harmelin, Y., Boukari, F., Ambrosetti, D., Lacour, J. P., & Passeron, T. (2014). Differences in visible light-induced pigmentation according to wavelengths: A clinical and histological study in comparison with UVB exposure. *Pigment Cell & Melanoma Research, 27*(5), 822–826.
79. Regazzetti, C., Sormani, L., Debayle, D., Bernerd, F., Tulic, M. K., De Donatis, G. M., Chignon-Sicard, B., Rocchi, S., & Passeron, T. (2018). Melanocytes sense blue light and regulate pigmentation through opsin-3. *Journal of Investigative Dermatology, 138*(1), 171–178.
80. Jansen, R., Wang, S. Q., Burnett, M., Osterwalder, U., & Lim, H. W. (2013). Photoprotection: Part I. Photoprotection by naturally

occurring, physical, and systemic agents. *Journal of the American Academy of Dermatology, 69*(6), 8531.
81. Friedman, L. C., Bruce, S., Weinberg, A. D., Cooper, H. P., Yen, A. H., & Hill, M. (1994). Early detection of skin cancer: Racial/ethnic differences in behaviors and attitudes. *Journal of Cancer Education, 9*(2), 105–110.
82. Perlman, K. L., Williams, N. M., Egbeto, I. A., Gao, D. X., Siddiquee, N., & Park, J. H. (2021). Skin of color lacks representation in medical student resources: A cross-sectional study. *International Journal of Women's Dermatology, 7*(2), 195.
83. Lester, J. C., Taylor, S. C., & Chren, M. M. (2019). Under-representation of skin of colour in dermatology images: Not just an educational issue. *The British Journal of Dermatology, 180*(6), 1521–1522.
84. Song, H., Beckles, A., Salian, P., & Porter, M. L. (2021). Sunscreen recommendations for patients with skin of color in the popular press and in the dermatology clinic. *International Journal of Women's Dermatology, 7*(2), 165–170.
85. Akamine, K. L., Gustafson, C. J., Davis, S. A., Levender, M. M., & Feldman, S. R. (2014). Trends in sunscreen recommendation among US physicians. *JAMA Dermatology, 150*(1), 51–55.
86. Rigel, D. S., Taylor, S. C., Lim, H. W., Alexis, A. F., Armstrong, A. W., Fuxench, Z. C., Draelos, Z. D., & Hamzavi, I. H. (2022). Photoprotection for skin of all color: Consensus and clinical guidance from an expert panel. *Journal of the American Academy of Dermatology, 86*(3), S1-8.
87. Schalka, S., & Reis, V. M. S. D. (2011). Sun protection factor: Meaning and controversies. *Anais brasileiros de dermatologia, 86*, 507–515.
88. Hedayat, K., Nasrollahi, S. A., Firooz, A., Rastegar, H., & Dadgarnejad, M. (2020). Comparison of UVA protection factor measurement protocols. *Clinical, Cosmetic and Investigational Dermatology, 13*, 351.
89. ISO 24442: 2011. (2011). *Cosmetics—Sun protection test methods—In vivo determination of sunscreen UVA protection*. ISO.
90. Wang, S. Q., Xu, H., Stanfield, J. W., Osterwalder, U., & Herzog, B. (2017). Comparison of ultraviolet A light protection standards in the United States and European Union through in vitro measurements of commercially available sunscreens. *Journal of the American Academy of Dermatology, 77*(1), 42–47.
91. European Commission (n.d.) Sunscreen products. Retrieved June 21, 2022, from https://ec.europa.eu/growth/sectors/cosmetics/cosmetic-products-specific-topics/sunscreen-products_en
92. Food and Drug Administration, HHS. (2012). Labeling and effectiveness testing; sunscreen drug products for over-the-counter human use; delay of compliance dates. Final rule; delay of compliance dates; request for comments. *Federal Register, 77*(92), 27591–27593.
93. Lim, H. W., Kohli, I., Granger, C., Trullàs, C., Piquero-Casals, J., Narda, M., Masson, P., Krutmann, J., & Passeron, T. (2021). Photoprotection of the skin from visible light-induced pigmentation: Current testing methods and proposed harmonization. *Journal of Investigative Dermatology, 141*(11), 2569–2576.
94. Passeron, T., Lim, H. W., Goh, C. L., Kang, H. Y., Ly, F., Morita, A., Ocampo Candiani, J., Puig, S., Schalka, S., Wei, L., & Dréno, B. (2021). Photoprotection according to skin phototype and dermatoses: Practical recommendations from an expert panel. *Journal of the European Academy of Dermatology and Venereology, 35*(7), 1460–1469.
95. Lyons, A. B., Trullas, C., Kohli, I., Hamzavi, I. H., & Lim, H. W. (2021). Photoprotection beyond ultraviolet radiation: A review of tinted sunscreens. *Journal of the American Academy of Dermatology, 84*(5), 1393–1397.
96. Guan, L. L., Lim, H. W., & Mohammad, T. F. (2021). Sunscreens and photoaging: A review of current literature. *American Journal of Clinical Dermatology, 22*(6), 819–828.
97. Marionnet, C., de Dormael, R., Marat, X., Roudot, A., Gizard, J., Planel, E., Tornier, C., Golebiewski, C., Bastien, P., Candau, D., & Bernerd, F. (2022). Sunscreens with the new MCE filter cover the whole UV spectrum: Improved UVA1 photoprotection in vitro and in a randomized controlled trial. *JID Innovations, 2*(1), 100070.
98. Bacqueville, D., Jacques-Jamin, C., Lapalud, P., Douki, T., Roullet, N., Sereno, J., Redoulès, D., Bessou-Touya, S., & Duplan, H. (2022). Formulation of a new broad-spectrum UVB+ UVA and blue light SPF50+ sunscreen containing Phenylene Bis-Diphenyltriazine (TriAsorB), an innovative sun filter with unique optical properties. *Journal of the European Academy of Dermatology and Venereology, 36*, 29–37.
99. Lawrence, K. P., Sarkany, R. P., Acker, S., Herzog, B., & Young, A. R. (2022). A new visible light absorbing organic filter offers superior protection against pigmentation by wavelengths at the UVR-visible boundary region. *Journal of Photochemistry and Photobiology B: Biology, 227*, 112372.
100. Neale, R. E., Khan, S. R., Lucas, R. M., Waterhouse, M., Whiteman, D. C., & Olsen, C. M. (2019). The effect of sunscreen on vitamin D: A review. *British Journal of Dermatology, 181*(5), 907–915.
101. Passeron, T., Bouillon, R., Callender, V., Cestari, T., Diepgen, T. L., Green, A. C., van der Pols, J. C., Bernard, B. A., Ly, F., Bernerd, F., & Marrot, L. (2019). Sunscreen photoprotection and vitamin D status. *British Journal of Dermatology, 181*(5), 916–931.
102. Afarideh, M., Sartori-Valinotti, J. C., & Tollefson, M. M. (2021). Association of sun-protective behaviors with bone mineral density and osteoporotic bone fractures in US adults. *JAMA Dermatology, 157*(12), 1437–1446.
103. Fatima, S., Braunberger, T., Mohammad, T. F., Kohli, I., & Hamzavi, I. H. (2020). The role of sunscreen in melasma and postinflammatory hyperpigmentation. *Indian Journal of Dermatology, 65*(1), 5.
104. Chan, H. H., Alam, M., Kono, T., & Dover, J. S. (2002). Clinical application of lasers in Asians. *Dermatologic surgery, 28*(7), 556–563.
105. Wanitphakdeedecha, R., Phuardchantuk, R., & Manuskiatti, W. (2014). The use of sunscreen starting on the first day after ablative fractional skin resurfacing. *Journal of the European Academy of Dermatology and Venereology, 28*(11), 1522–1528.
106. Halder, R., Rodney, I., Munhutu, M., Foltis, P., Battie, C., Verschoore, M., & Oresajo, C. (2015). Evaluation and effectiveness of a photoprotection composition (sunscreen) on subjects of skin of color. *JAMA Dermatology, 72*(5), AB215.
107. Lakhdar, H., Zouhair, K., Khadir, K., Essari, A., Richard, A., Seité, S., & Rougier, A. (2007). Evaluation of the effectiveness of a broad-spectrum sunscreen in the prevention of chloasma in pregnant women. *Journal of the European Academy of Dermatology and Venereology, 21*(6), 738–742.
108. Sarkar, R., Ghunawat, S., Narang, I., Verma, S., Garg, V. K., & Dua, R. (2019). Role of broad-spectrum sunscreen alone in the improvement of melasma area severity index (MASI) and Melasma Quality of Life Index in melasma. *Journal of Cosmetic Dermatology, 18*(4), 1066–1073.
109. Kelm, R. C., Zahr, A. S., Kononov, T., & Ibrahim, O. (2020). Effective lightening of facial melasma during the summer with a dual regimen: A prospective, open-label, evaluator-blinded study. *Journal of Cosmetic Dermatology, 19*(12), 3251–3257.
110. Guevara, I. L., & Pandya, A. G. (2003). Safety and efficacy of 4% hydroquinone combined with 10% glycolic acid, antioxidants, and sunscreen in the treatment of melasma. *International Journal of Dermatology, 42*(12), 966–972.
111. Navarrete-Solís, J., Castanedo-Cázares, J. P., Torres-Álvarez, B., Oros-Ovalle, C., Fuentes-Ahumada, C., González, F. J.,

Martínez-Ramírez, J. D., & Moncada, B. (2011). A double-blind, randomized clinical trial of niacinamide 4% versus hydroquinone 4% in the treatment of melasma. *Dermatology Research and Practice, 2011*, 1–5.
112. Lima, P. B., Dias, J. A. F., Cassiano, D. P., Esposito, A. C. C., Miot, L. D. B., Bagatin, E., & Miot, H. A. (2021). Efficacy and safety of topical isobutylamido thiazolyl resorcinol (Thiamidol) versus 4% hydroquinone cream for facial melasma: An evaluator-blinded, randomized controlled trial. *Journal of the European Academy of Dermatology and Venereology, 35*(9), 1881–1887.
113. Lim, H. W., Kohli, I., Ruvolo, E., Kolbe, L., & Hamzavi, I. H. (2022). Impact of visible light on skin health: The role of antioxidants and free radical quenchers in skin protection. *Journal of the American Academy of Dermatology, 86*(3), S27–S37.
114. Solano, F. (2020). Photoprotection and skin pigmentation: Melanin-related molecules and some other new agents obtained from natural sources. *Molecules, 25*(7), 1537.
115. Pullar, J. M., Carr, A. C., & Vissers, M. C. (2017). The roles of vitamin C in skin health. *Nutrients, 9*(8), 866.
116. Rhie, G. E., Shin, M. H., Seo, J. Y., Choi, W. W., Cho, K. H., Kim, K. H., Park, K. C., Eun, H. C., & Chung, J. H. (2001). Aging-and photoaging-dependent changes of enzymic and non-enzymic antioxidants in the epidermis and dermis of human skin in vivo. *Journal of Investigative Dermatology, 117*(5), 1212–1217.
117. Lin, F. H., Lin, J. Y., Gupta, R. D., Tournas, J. A., Burch, J. A., Selim, M. A., Monteiro-Riviere, N. A., Grichnik, J. M., Zielinski, J., & Pinnell, S. R. (2005). Ferulic acid stabilizes a solution of vitamins C and E and doubles its photoprotection of skin. *Journal of Investigative Dermatology, 125*(4), 826–832.
118. Lin, J. Y., Selim, M. A., Shea, C. R., Grichnik, J. M., Omar, M. M., Monteiro-Riviere, N. A., & Pinnell, S. R. (2003). UV photoprotection by combination topical antioxidants vitamin C and vitamin E. *Journal of the American Academy of Dermatology, 48*(6), 866–874.
119. Murray, J. C., Burch, J. A., Streilein, R. D., Iannacchione, M. A., Hall, R. P., & Pinnell, S. R. (2008). A topical antioxidant solution containing vitamins C and E stabilized by ferulic acid provides protection for human skin against damage caused by ultraviolet irradiation. *Journal of the American Academy of Dermatology, 59*(3), 418–425.
120. Traikovich, S. S. (1999). Use of topical ascorbic acid and its effects on photodamaged skin topography. *Archives of Otolaryngology–Head & Neck Surgery, 125*(10), 1091–1098.
121. Delinasios, G. J., Karbaschi, M., Cooke, M. S., & Young, A. R. (2018). Vitamin E inhibits the UVAI induction of "light" and "dark" cyclobutane pyrimidine dimers, and oxidatively generated DNA damage, in keratinocytes. *Scientific Reports, 8*(1), 1–12.
122. Lyons, A. B., Zubair, R., Kohli, I., Nahhas, A. F., Braunberger, T. L., Mokhtari, M., Ruvolo, E., Lim, H. W., & Hamzavi, I. H. (2022). Mitigating visible light and long wavelength UVA1-induced effects with topical antioxidants. *Photochemistry and Photobiology, 98*(2), 455–460.
123. Ruvolo, E., Boothby-Shoemaker, W., Kumar, N., Hamzavi, I. H., Lim, H. W., & Kohli, I. (2022). Evaluation of efficacy of antioxidant-enriched sunscreen prodcuts against long wavelength ultraviolet al and visible light. *International Journal of Cosmetic Science, 44*, 394–402.
124. Kern, J., Wood, E., Almukhtar, R., Angra, K., Lipp, M., & Goldman, M. (2022). Evaluation of an SPF50 sunscreen containing photolyase and antioxidants for its anti-photoaging properties and photoprotection. *Journal of Drugs in Dermatology: JDD, 21*(5), 517–520.
125. Torres, A. E., Awosika, O., Maghfour, J., Taylor, S., & Lim, H. W. (2022). Practical guide to tinted sunscreens. *Journal of the American Academy of Dermatology, 87*(3), 656–657.
126. Berry, E. G., Bezecny, J., Acton, M., Sulmonetti, T. P., Anderson, D. M., Beckham, H. W., Durr, R. A., Chiba, T., Beem, J., Brash, D. E., & Kulkarni, R. (2022). Slip versus Slop: A head-to-head comparison of UV-protective clothing to sunscreen. *Cancers, 14*(3), 542.
127. Linos, E., Keiser, E., Fu, T., Colditz, G., Chen, S., & Tang, J. Y. (2011). Hat, shade, long sleeves, or sunscreen? Rethinking US sun protection messages based on their relative effectiveness. *Cancer Causes & Control, 22*(7), 1067–1071.
128. Thieden, E., Philipsen, P. A., Sandby-Møller, J., & Wulf, H. C. (2005). Sunscreen use related to UV exposure, age, sex, and occupation based on personal dosimeter readings and sun-exposure behavior diaries. *Archives of Dermatology, 141*(8), 967–973.
129. Autier, P., Boniol, M., & Doré, J. F. (2007). Sunscreen use and increased duration of intentional sun exposure: Still a burning issue. *International Journal of Cancer, 121*(1), 1–5.
130. Tsai, J., & Chien, A. L. (2022). Photoprotection for skin of color. *American Journal of Clinical Dermatology, 23*, 1–11.
131. Andreeva, V. A., Unger, J. B., Yaroch, A. L., Cockburn, M. G., Baezconde-Garbanati, L., & Reynolds, K. D. (2009). Acculturation and sun-safe behaviors among US Latinos: Findings from the 2005 Health Information National Trends Survey. *American Journal of Public Health, 99*(4), 734–741.
132. Patel, S. P., & Chien, A. L. (2021). Sun protective clothing and sun avoidance: The most critical components of photoprotection in patients with melanoma. *Dermatologic Surgery, 47*(3), 333–337.
133. Gambichler, T., Altmeyer, P., & Hoffmann, K. (2002). Role of clothes in sun protection. In R. Dummer, F. O. Nestle, & G. Burg (Eds.), *Cancers of the skin* (pp. 15–25). Springer.
134. Louris, E., Sfiroera, E., Priniotakis, G., Makris, R., Siemos, H., Efthymiou, C., & Assimakopoulos, M. N. (2018). Evaluating the ultraviolet protection factor (UPF) of various knit fabric structures. *IOP Conference Series: Materials Science and Engineering, 459*(1), 012051.
135. Boothby-Shoemaker, W. T., Mohammad, T. F., Ozog, D. M., & Lim, H. W. (2022). Photoprotection by clothing: A review. *Photodermatology, Photoimmunology & Photomedicine, 38*, 478–488.
136. Adam, J. (1998). Sun-protective clothing. *Journal of Cutaneous Medicine and Surgery, 3*(1), 50–53.
137. Parrado, C., Philips, N., Gilaberte, Y., Juarranz, A., & Gonzalez, S. (2018). Oral photoprotection: Effective agents and potential candidates. *Frontiers in Medicine, 5*, 188.
138. Bendich, A., & Olson, J. A. (1989). Biological actions of carotenoids 1. *The FASEB Journal, 3*(8), 1927–1932.
139. Eberlein-König, B., Placzek, M., & Przybilla, B. (1998). Protective effect against sunburn of combined systemic ascorbic acid (vitamin C) and d-α-tocopherol (vitamin E). *Journal of the American Academy of Dermatology, 38*(1), 45–48.
140. Cesarini, J. P., Michel, L., Maurette, J. M., Adhoute, H., & Bejot, M. (2003). Immediate effects of UV radiation on the skin: Modification by an antioxidant complex containing carotenoids. *Photodermatology, Photoimmunology & Photomedicine, 19*(4), 182–189.
141. Fuchs, J., & Kern, H. (1998). Modulation of UV-light-induced skin inflammation by D-alpha-tocopherol and L-ascorbic acid: A clinical study using solar simulated radiation. *Free Radical Biology and Medicine, 25*(9), 1006–1012.
142. Choudhry, S. Z., Bhatia, N., Ceilley, R., Hougeir, F., Lieberman, R., Hamzavi, I., & Lim, H. W. (2014). Role of oral polypodium leucotomos extract in dermatologic diseases: A review of the literature. *Journal of Drugs in Dermatology, 13*(2), 148–153.
143. Mohammad, T. F., Kohli, I., Nicholson, C. L., Treyger, G., Chaowattanapanit, S., Lim, H. W., & Hamzavi, I. (2019). Oral polypodium leucotomos extract and its impact on visible

light-induced pigmentation in human subjects. *Journal of Drugs in Dermatology, 18*(12), 1198-I203.
144. Truchuelo, M. T., Jiménez, N., Días, I. J., Gallego-Rentero, M., & Alonso-Juarranz, M. (2019). A pilot study to assess the effects of an oral photo protector of botanical origin against visible and infrared radiations in human volunteers. *The Journal of Dermatology, 6*(282), 2.
145. Goh, C. L., Chuah, S. Y., Tien, S., Thng, G., Vitale, M. A., & Delgado-Rubin, A. (2018). Double-blind, placebo-controlled trial to evaluate the effectiveness of Polypodium leucotomos extract in the treatment of melasma in Asian skin: A pilot study. *The Journal of Clinical and Aesthetic Dermatology, 11*(3), 14.
146. Ahmed, A. M., Lopez, I., Perese, F., Vasquez, R., Hynan, L. S., Chong, B., & Pandya, A. G. (2013). A randomized, double-blinded, placebo-controlled trial of oral *Polypodium leucotomos* extract as an adjunct to sunscreen in the treatment of melasma. *JAMA Dermatology, 149*(8), 981–983.
147. Ni, Z., Mu, Y., & Gulati, O. (2002). Treatment of melasma with pycnogenol®. *Phytotherapy Research: An International Journal Devoted to Pharmacological and Toxicological Evaluation of Natural Product Derivatives, 16*(6), 567–571.
148. Aladrén, S., Garre, A., Valderas-Martínez, P., Piquero-Casals, J., & Granger, C. (2019). Efficacy and safety of an oral nutritional (dietary) supplement containing pinus pinaster bark extract and grape seed extract in combination with a high SPF sunscreen in the treatment of mild-to-moderate melasma: A prospective clinical study. *Cosmetics, 6*(1), 15.
149. Torres, A. E., Luk, K. M., & Lim, H. W. (2020). Botanicals for photoprotection. *Plastic and Aesthetic Research, 7*, 57.