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The Skin's Sun Story: Risks and Precautions

Wiktoria Julia Krzesłowska *
Central Clinical Hospital of Medical University of Lodz, ul. Pomorska 251, 92-213 Łódź, Poland
https://orcid.org/0009-0009-6368-1572

Kamila Szewczyk
Regional Specialist Hospital, Czerwona Góra 10, 26-060 Chęciny**,** Poland
https://orcid.org/0009-0008-4493-9593

Paulina Pytel
Regional Specialist Hospital, Czerwona Góra 10, 26-060 Chęciny**,** Poland
https://orcid.org/0009-0001-6959-4666

Bartłomiej Szewczyk
St. Lucas Hospital,Gimnazjalna 41B, 26-200 Końskie, Poland
https://orcid.org/0000-0001-8743-5186

Szymon Wiśniewski
Collegium Medicum, Jan Kochanowski University of Kielce, Al.IX Wieków Kielc 19A, 25-317 Kielce, Poland
https://orcid.org/0009-0000-9474-821X

Weronika Hołownia
Medical University of Warsaw ul. Żwirki i Wigury 61, 02-091 Warsaw, Poland
https://orcid.org/0009-0005-5771-6725

***Corresponding author:**
+48 509 138 942, wiktoria.julia.krzeslowska@gmail.com

ABSTRACT

Background: The skin, representing the largest organ in the human body, serves numerous funtions crucial for homeostasis, as well as acts as the primary physical barrier protecting the organism from external environmental factors such as microorganisms, mechanical damage, heat or cold exposure, toxins, as well as solar radiation, posing various risks to the skin, including sunburn, photoageing and skin malignancies.

Objectives: This study aims to explore the effects of solar radiation on the skin and to discuss preventive measures against its negative influence on the skin.

Methods: An exhaustive search of references related to sun exposure and skin published in PubMed was undertaken, using the search terms: "solar exposure and skin, ultraviolet radiation and skin, visible light and skin, sunscreen, photoprotection".

Conclusions: Solar radiation, particularly ultraviolet radiation (UVR), induces acute and chronic damage to the skin, leading to conditions such as sunburn, photoaging, and skin malignancies. However, adequate exposure to sunlight is crucial for the synthesis of vitamin D3, which plays essential roles in calcium homeostasis, immune function, and potentially other physiological processes. Strategies such as proper sunscreen use and other photoprotection methods can mitigate the adverse effects of solar radiation while allowing for sufficient vitamin D synthesis.

Keywords: solar exposure and skin; solar radiation; vitamin D; sunscreen; photoprotection; photodermatoses

INTRODUCTION

The skin, comprising three layers - the epidermis, dermis, and subcutaneous tissue, represents the largest organ in the human body. It serves numerous crucial functions related to maintaining homeostasis, including sensation, endocrine and exocrine activity, temperature regulation, as well as immunity [1]. Furthermore, the skin acts as the primary physical barrier protecting the organism from external environmental factors such as microorganisms, mechanical damage, heat or cold exposure, toxins, as well as solar radiation [1,2].

Solar radiation primarily consists of optical radiation, covering a wide range of the electromagnetic spectrum including ultraviolet radiation (UVR), described as the most harmful exogenous factor, visible light (VL), and infrared radiation (IR). UVR, comprising 5% of solar radiation reaching Earth's surface, spans wavelengths from 100 to 400 nm and is divided into UVA (315–400 nm), UVB (280–315 nm), and UVC (100–280 nm).

The sunlight's UVR component during midday consists of approximately 95% UVA and 5% UVB. UVC and the majority of UVB are filtered out by the stratospheric ozone layer, thus reducing their presence in terrestrial radiation from space [3,4,5]. UVA negatively affects skin epidermis, as well as dermis, while changes attributed to UVB predominantly occur within the epidermis. VL, comprising approximately 50% of solar radiation, encompasses wavelengths ranging from 400 to 700 nm that are visible to the human eye and can be further subdivided based on color and wavelength: blue light, green light, yellow light and red light, with the wavelength of 400–490 nm, 490–570 nm, 570–595 nm, and 630–770 nm, respectively. Approximately 4-7% of VL is reflected by the skin surface, regardless of incident wavelength, pigmentation, or structure. The depth of penetration depends on the wavelength, with red light exhibiting the greatest penetration, reaching most, if not all, dermal layers. IR, spanning from 700 nm to 1 mm, constitutes the residual 45% of solar irradiance [5]. It penetrates to deeper layers of the skin compared to other forms of optical radiation, with up to 17% of incident IR capable of directly penetrating into the subcutaneous tissue. IR is absorbed by tissue chromophores, such as water, and converted into heat, resulting in the heating of deep tissues. This thermal effect can potentially cause pathological changes like skin or corneal burns.

The skin presents various mechanisms to protect itself and the interior of human's body from adverse effects of solar radiation. The outermost layer, epidermis, characterized by layered structure and distinctive lipid composition of its cells' membranes, serves as the primary defense against external threats. This defense is further reinforced by immune cells such as Langerhans cells and T lymphocytes. Additionally, melanocytes play a pivotal role by producing melanin, a pigment that absorbs UVR and inhibits its penetration into deeper skin layers. Furthermore, UVR-induced DNA damage activates complex molecular repair pathways, encompassing nucleotide repair, base excision, and the activation of apoptotic responses and cell cycle checkpoints [4,6].

However, despite protective functions, solar radiation, primarily UVR, negatively impacts skin, leading to disturbances classified into two groups. Acute damages encompass conditions such as sunburn, erythema, pain, edema, and photodermatoses. Conversely, more severe chronic damages involve photoaging and the formation of precancerous skin lesions like actinic keratosis (AK), as well as the development of skin cancers such as basal cell carcinoma (BCC), squamous cell carcinoma (SCC), and cutaneous malignant melanoma (CMM) [2].

PHOTOAGEING

Skin aging is influenced by a combination of inherited genetics and external environmental factors, predominantly driven by UVR, which accounts for 80-90% of morphological, structural, and biochemical alterations, termed chronic cutaneous photoaging [2,7]. Photoaged skin displays changes in epidermal thickness and pigment heterogeneity, as well as alterations in the dermis, including elastosis, collagen degradation, and the development of ectatic vessels. Clinically, this manifests as an increase in wrinkles (rhytides), telangiectasias, dyspigmentation, loss of volume, and cutaneous malignancies [8,9].

UVB has been shown to induce the expression of matrix metalloproteinases (MMPs), reactive oxygen species (ROS), and elastases, all of which contribute to the process of photoaging. UVA exposure leads to apoptosis in dermal fibroblasts and increases MMPs levels, enzymes responsible for collagen degradation.

Moreover, repeated exposure to UVA in vivo has been found to elevate ferritin and lysozyme levels, which participate in oxidative stress response and elastin degradation, respectively [8,9]. VL and IR can also contribute to skin photoaging. Studies suggest that VL, especially blue light, can potentiate damage caused by UVR and independently generate ROS and proinflammatory cytokines, while also increasing MMPs levels. Moreover, in vivo studies involving skin irradiation with IR and VL demonstrated increased MMPs expression and decreased type I procollagen expression, suggesting a role for IR and VL in dermal collagen degradation. Longer wavelengths of VL, particularly those in the yellow and red spectrum, are considered protective against photoaging. However, it's crucial to consider radiation dose, regardless of wavelength [9,10].

HYPERPIGMENTATION

Hyperpigmentation, defined as the darkening of the skin, arises from various factors. Typically, it stems from an increased deposition of melanin in the epidermis or/and dermis. Additionally, it can be a result of the accumulation of endogenous or exogenous pigments such as hemosiderin, iron, as well as heavy metals. Factors like carotenoids, vascularity, and variations in skin thickness may also contribute to the manifestation of hyperpigmentation [11].

UVR serves as a distinctive regulator of skin pigmentation, influencing pathways associated with tanning. There are two pathways involved in tanning: immediate and delayed. The immediate pathway, transient and driven by UVA, consists of two phases. The initial phase is immediate pigment darkening (IPD), occurring within minutes and lasting for a few hours. The subsequent phase is persistent pigment darkening (PPD), which can persist for up to a day. Both IPD and PPD are believed to result from the dispersion of larger pigment granules and the oxidation of existing melanin, rather than from melanogenesis itself. Delayed tanning, occurring within days and lasting up to weeks or months, is primarily driven by UVB. However, there is some ambiguity regarding the terminology "delayed tanning" in the current literature. Additionally, a recently identified phenomenon called long-lasting pigmentation (LLP) involves persistent skin pigmentation in UV-exposed areas of certain individuals, lasting for months to years [12]. Visually, individuals exhibiting long-term LLP demonstrated heightened pigmentation and a hyperpigmented basal layer when compared to both their unexposed control group and LLP-negative counterparts [13].

In pigmentary disorders associated with solar radiation, VL can also play a role. Blue or green light is commonly regarded as a primary contributor to hyperpigmentation disorders, particularly in skin of color (Fitzpatrick’s type IV-VI) and yellow or red light may act as important facilitators for the development of hypopigmentary diseases [5,6,14].

SUNBURN

Sunburn, an acute inflammatory response triggered by prolonged exposure to UV, either from sun exposure of artificial sources like tanning beds, affecting all skin layers, particularly the epidermis and dermis. UVA and UVB exposure induces DNA damage. UVA rays cause oxidative DNA damage, while UVB rays are directly absorbed by DNA, leading to the formation of thymine-thymine cyclobutane dimers. Consequently, DNA repair response is initiated, including cell apoptosis and the release of inflammatory markers like prostaglandins, ROS, and bradykinin.

This cascade results in sunburn symptoms, such as vasodilation, edema and pain. Erythema tends to manifest between 3 to 5 hours post-exposure to sunlight, achieving maximum intensity within 12 to 24 hours. The condition typically resolves itself within a period of 3 to 7 days, whereas blistering, associated with more severe partial-thickness sunburn, often resolves within 7 to 10 days. Desquamation may continue for approximately 7 to 10 days following exposure. General fatigue, nausea, chills and fever can also be observed [2].

Individuals with lighter skin tones (Fitzpatrick skin types I to III), younger age (18 to 29 years old), non-Hispanic white ethnicity, and certain behavioral factors such as the use of tanning lotions, engagement in physical activity, binge drinking, and overweight, are at a higher risk of experiencing sunburn [15]. Interestingly, a cohort study involving Norwegian women demonstrates that frequent sunburns, particularly during childhood, are significantly correlated with heightened risks of CMM and SCC, which emphasize the critical need for early and consistent sunburn prevention to mitigate future skin cancer risks [16].

PHOTOSENSITIVITY

Photosensitivity is a dermatological phenomenon characterized by the skin's reaction to solar UVR exposure, triggered by the presence of endogenous or exogenous chromophores within the dermal or epidermal layers. This reaction occurs when such chromophores accumulate in the skin and are selectively activated predominantly upon UVA, although UVB, VL, or IR may also play a role. There are two distinct reactions associated with photosensitivity: phototoxicity, a non-specific inflammation and photoallergy, a specific immune reaction. The main differences between these two reactions have been presented in Table 1 [17].

Characteristics	**Phototoxicity**	**Photoallergy**
Frequency	High	Low
Latency period/sensitisation	No	Yes
Doses of UV/ photosensitizer	High	Low
Cross-reactions	No	Yes
Sharp limits	Yes	No
Involvement of covered skin areas	No	Yes

Table 1. Distinction between phototoxicity and photoallergy.

Phototoxicity typically occurs more frequently and manifests in every individual given sufficient simultaneous presence of a photosensitizer (typically plant or systemic drug), as well as sun exposure. Its onset is dependent on the dose of UV radiation and the concentration of the photosensitizing agent.

Initial reactions can arise after the first exposure without subsequent flare-ups or cross-reactions upon further exposures. Clinically, it typically presents as sharply demarcated erythema localized to sun-exposed areas, resembling sunburn, and often resolves spontaneously. However, clinical patterns of phototoxic reactions may also include pseudoporphyria, photoonycholysis, hyperpigmentation, phytophotodermatitis, dyschromia, telangiectasia, as well as pellagra-like reactions [17,18]. Photoallergy develops in a limited subset of individuals, requires prior sensitization, and may occur with chemically similar agents (cross-reactions). It is not strictly dose-dependent and can manifest even with low UV exposure doses. UV-absorbers, as well as topical medications are the major contributors to photoallergic reactions. Clinically, they usually present as eczema that may extend to non-UV-exposed areas. However, clinical patterns of photoallergy reactions may also include urticaria in sun-exposed areas, erythema multiforme-like lesions, lichenoid reactions, as well as subacute or chronic lupus erythematosus [17,19-20].

Three primary groups of photosensitizers stand out for their contribution to photosensitivity reactions: plants, systemic drugs, and UV absorbers. Among plants that may contribute to these reactions are: bishop's weed, celery, parsley, giant hogweed, bergamot, lime, lemon, common rue, burning bush, fig and Saint John's wort. Systemic medications known to induce photoallergic or phototoxic reactions include antimicrobials (tetracyclines, sulfonamides, fluoroquinolones, voriconazole, efavirenz), nonsteroidal anti-inflammatory drugs, phenothiazines, cardiovascular drugs (amiodarone, quinidine, amlodipine, diltiazem, furosemide, indapamide, thiazide diuretics), anticancer drugs and miscellaneous (clomipramine, imipramine, sertraline, flutamide, fenofibrate, pirfenidone, simvastatin). UV absorbers responsible for such reactions include classical UV absorbers, such as octocrylene, or homosalate, as well as "newer" UV absorbers [17].

ACTINIC KERATOSIS

AK, also known as solar keratosis, is the most frequently recognized skin disorder in North America. The risk of AK development correlates strongly with cumulative UVR exposure [21]. It appears predominantly in elderly men with Fitzpatrick phototype I and II skin, and has higher prevalence rate in regions with greater solar UVR exposure, with advanced age groups (>60 years) exhibiting notably increased prevalence compared to younger cohorts (<40 years) [21,22]. AK is widely recognized as a precursor to SCC. Researches suggest that in a 10-year period, about 16% of AKs can progress to invasive SCC and up to 59% of SCCs are a result of malignant transformation from AK. Therefore monitoring and potentially treating solar keratosis is very important [22].

SKIN MALIGNANCIES

Long term exposure to solar radiation induces alterations in the molecular, pigmentary and morphological features of the skin, contributing to carcinogenic progression observed in skin malignancies. The three main types of skin neoplasm associated with sun exposure are non-melanoma skin cancer (NMSC): BCC, SCC, as well as CMM, called after the type of skin cell from which they develop [2]. A recent study conducted by Hammond et al. identified specific professions, such as builders, gardeners and road workers, as particularly susceptible to skin malignancy [23].

BCC stands as the predominant form of skin malignancy globally [24]. Its incidence escalates notably post the age of 40, yet a recent surge in occurrence has been observed among younger individuals, particularly females, as a result of increased UVR exposure, considered the major environmental risk factor for BCC [25,26]. In particular, acute intermittent exposure, especially during childhood or adolescence, represents a significant contributor to BCC. However, factors such as cumulative exposure effect and sun skin's ability to tan also influence the risk [27]. SCC is the second most prevalent cutaneous malignancy following BCC and exhibiting a rising incidence on a global scale. While numerous factors may elevate the risk of SCC, cumulative sun exposure, particularly during childhood holds paramount significance. Additionally, recent years have investigated the role of immunosuppression, including that associated with organ transplantation, as a significant contributor to tumorigenesis. Furthermore, the emergence of SCC in regions of chronic inflammation should be also kept in mind [28]. CMM is one of the deadliest tumors, with sunlight undoubtedly representing the primary risk factor.

VITAMIN D SYNTHESIS

The majority of vitamin D produced in the human body, approximately 80%, is synthesized endogenously within the skin through exposure to UVR. 7-dehydrocholesterol, the precursor of vitamin D present in epidermal cells, undergoes transformation into previtamin D upon exposure to UVB radiation by opening the B ring of the steroid molecule. Subsequently, through thermal isomerization, provitamin D is converted into cholecalciferol, called vitamin D. However, for vitamin D to become active in the human body, two hydroxylation reactions occur. The first one occurs in the liver, yielding 25-hydroxyvitamin D (25(OH)D), which serves as a measurable indicator of vitamin D status in serum. The second hydroxylation reaction, primarily occurring in the kidneys but also in various other cell types including skin cells, results in the production of 1,25-dihydroxyvitamin D (1,25(OH)D) [30]. Vitamin D plays a pivotal role in maintaining calcium homeostasis by facilitating calcium absorption in the intestine and promoting its reabsorption in the kidneys. Additionally, it contributes to innate and adaptive immunity by inducing antimicrobial peptides and modulating inflammatory responses. Emerging research suggests potential roles for vitamin D in conditions like SARS-CoV-2 infection and cancer, where it may impact viral infectivity and inhibit cancer cell growth. Moreover, vitamin D regulates keratinocyte function in skin conditions such as psoriasis. However, further studies are needed to fully understand the extent of previtamin D and its photoproducts, as well as vitamin D's, in these areas [31].

Interresigly, cutaneous vitamin D production under sunlight exposure appears to have superiority over dietary supplementation. UVR-induced synthesis of vitamin D generates lumisterol3, converted further to 1,25-dihydroxylumisterol3 that seems to have anti-tumor effects in the skin. Furthermore, suprasterols and toxisterols, produced with prolonged UVR exposure potentially aiding in tumor prevention [32].

PHOTOPROTECTION

Photoprotection aims to mitigate the harmful impact of UVR on the skin and reduce the risk of sunburn, as well as skin malignancies. It should be used by people at every age, however it is extremely important in children under the age of 14, individuals with immunocompetence, as well as with Fitzpatrick skin phototypes I and II [33].

It should be emphasized that sunscreen use alone is not considered sufficient photoprotection strategy, therefore avoidance of excessive sun-exposure (especially between 10 am and 4 pm), seeking shade while outdoors and the use of sun-protective clothes are essential [33,34,35]. According to European standards, protective clothing should have an ultraviolet protection factor (UPF) of at least 40, with UVA transmission below 5%. This ensures adequate protection against UVB and sunburn. Special laundry detergents containing UV-absorbing additives can significantly enhance the UPF of clothing without altering its texture or color. Sunglasses with lenses absorbing ultraviolet radiation up to 400 nm are recommended to prevent eye damage from UVR. Wide-brimmed hats, especially those with a brim size exceeding 7.5 cm, provide effective skin protection against UVR. Hats with smaller brims offer varying degrees of protection based on their size, while those with brims below 2.5 cm offer minimal protection [33].

Photoprotective products can contain primary and secondary protective factors. Primary factors encompass sunscreens, which can be physical or chemical barriers. Physical agents function by reflecting and scattering UV photons. They also have the ability to protect skin from negative effects of VL. Chemical filters absorb photons from UVA and UVB and convert them into heat. The effectiveness of UVB sunscreen can be measured with a use of international sun protection factor (SPF), representing the ratio of the minimum dose of radiation producing erythema (minimal erythema dose - MED) on the skin protected with a specific agent to the minimal dose of radiation causing erythema on skin with no protection: SPF = MED of photoprotected skin/ MED of skin with no protection [33,37,40]. Erythema induced by UVA develops later, therefore there are other methods used for assessing the degree of UVA protection, such as PPD, IPD, protection factor in the UVA or critical wavelength. To ensure effectiveness of the sunscreen, the current guidelines from the American Academy of Dermatology recommend a broad-spectrum (offering protection against UVA and UVB radiation), water-resistant sunscreen with the sun protection factor (SPF) at least 30, reapplied every 2 hours or after swimming or sweating [34, 35]. Moreover, according to Recommendations of the Polish Dermatological Society, proper initial application of sunscreen occurs 20 minutes before going outside. The recommended dose is 2 mg/cm2 of the skin surface, equivalent to approximately 30 ml or a handful of sunscreen for the entire body. The “teaspoon rule” where one teaspoon should be applied to the face, hand and neck, one teaspoon to each arm and forearm and two teaspoons to the trunk and each leg [33,36,37].

Sunscreens play a crucial role in preventing skin malignancies and other UV-related skin damage, such as photoageing as evidenced by a significant study in Australia, which showed that its regular use reduces the risk of MM and SCC [33,38]. Moreover, although recent literature has raised questions about the impact of sunscreen on the synthesis of vitamin D in the skin, proper application of sunscreen in adequate amounts (≥2 mg/cm2) does not significantly affect vitamin D levels. Research involving Polish participants on a sunny week-long holiday in the Canary Islands demonstrated that those adhering to recommended sunscreen application practices avoided sunburn and still showed notable increases in serum 25(OH)D levels. These findings indicate that when sunscreen is correctly used, it can prevent sunburn effectively while still allowing for sufficient vitamin D synthesis, illustrating the complex interplay between sunscreen use and vitamin D production in the skin [39,40].

CONCLUSIONS

Solar radiation poses a dual role in skin health, as it is essential for vitamin D3 synthesis yet can cause significant damage if exposure is excessive or unprotected. While UVR contributes to photoaging, hyperpigmentation, sunburn, and skin malignancies, appropriate photoprotection measures, including sunscreen use and protective clothing, can mitigate these risks. Proper application of sunscreen, with an SPF of at least 30 and broad-spectrum protection, ensures effective prevention of sunburn and skin cancer while allowing for sufficient vitamin D levels. Additionally, other photoprotection methods such as seeking shade and wearing UV-protective clothing are crucial for minimizing sun-induced skin damage. Balancing the benefits of cholecalciferol synthesis with the risks of solar exposure underscores the importance of adopting holistic approaches to sun protection to maintain skin health and prevent sun-induced skin damage and diseases. However, further research focusing on the effects of IR and VL on the skin is necessary to fully understand their impact and if needed to develop comprehensive photoprotection strategies.

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