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MODERN SUNSCREEN PHOTOPROTECTION: MECHANISMS, CLINICAL BENEFITS, AND EMERGING TECHNOLOGIES

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ABSTRACT

The rising global incidence of cutaneous malignancies and the aesthetic impacts of photoaging underscore cutaneous photoprotection as a crucial pillar of clinical dermatology and oncological prevention. This narrative review analyzes the efficacy of the Sun Protection Factor (SPF) metric, the biophysical mechanisms of organic and inorganic ultraviolet (UV) filters, additional antioxidants, pathophysiology of photoaging, epidemiology of skin malignancies. By analyzing current clinical evidence, this paper explains the role of regular sunscreen use in the active prevention of photoaging and skin cancer prophylaxis. Furthermore, it addresses contemporary regulatory challenges, formulation photostability, and promising future innovations in topical photoprotection. Ultimately, optimizing clinical outcomes requires a synthesis of advanced formulation science and targeted public health education to overcome behavioral application deficits.

KEYWORDS

SPF, Sunscreens, Photoprotection, Skin Cancer, Photoaging

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1. Introduction

One of the most commonly diagnosed malignancies worldwide is skin cancer which creates growing socioeconomic burden on global healthcare systems. The incidence of melanoma (MSCs) and non-melanoma skin cancers (NMSCs) – also referred as keratinocyte carcinomas (KCs) - has risen significantly over recent decades. Epidemiological data indicate growth of NMSCs from 402 to 787 per 100,000 persons between 1990 and 2019 (Aggarwal et al., 2021).

This trend is correlated with increased human longevity, ozone depletion, sun-seeking behaviour. All these factors cumulate a higher lifetime dose of ultraviolet radiation (UVR). Besides photocarcinogenesis, UVR is also an etiologic factor responsible for up to 80% signs of skin aging such as wrinkles, dyspigmentation, coarse skin texture, and loss of elasticity (Iannaccone et al., 2014).

The solar electromagnetic spectrum consists mainly of infrared radiation (IR), visible light (VL), and UVR. Although UVR accounts for only 5% to 10% of terrestrial solar energy it is the most biologically active component. UVR is divided by wavelength into ultraviolet B (UVB; 290-320nm) and ultraviolet A (UVA; 320-400nm). UVB is approximately 5% to 10% of terrestrial UVR and is absorbed mainly within the epidermis. It acts as potent, direct carcinogen by exciting cellular DNA, leading to the formation of highly mutagenic cyclobutane pyrimidine dimers (CPDs). This triggers keratinocyte apoptosis, local immunosuppression, and the initiation of keratinocyte carcinomas (Guan et al., 2021; Sullivan et al., 2025).

UVA radiation penetrates deeper into the skin layers and acts as an indirect carcinogen. UVA excites chromophores to generate reactive oxygen species (ROS) and the oxidative stress upregulates the expression of matrix metalloproteinases (MMPs), specifically MMP1, MMP3, and MMP9. These enzymes degrade collagen and elastin fibers which is related to hallmarks of photoaging. Furthermore, modern research found that high-energy VL (HEVL) and IR act synergistically with UVR, exacerbating oxidative stress, stimulating melanogenesis via the opsin-3 (OPN3) pathway, contributing to pigmentary disorders independently of UVB (González et al., 2023; Guan et al., 2021).

In response to this biological threat, photoprotection has evolved into a scientific discipline connecting cell biology, optical physics, and dermatology. The regular application of broad-spectrum sunscreens provides protection against both acute and chronic solar damage.

Despite clinical recommendations, adherence remains low, which might be caused by barriers such as cosmetic factors, SPF metric confusion, ecotoxicity (Miller et al., 2021).

2. SPF efficacy

2.1. SPF ratings

SPF is the universal metric used to measure a sunscreen's effectiveness, primarily against UVB, which is highly erythemagenic. SPF is mathematically defined as the ratio of the minimal erythema dose (MED) on sunscreen-protected skin required to induce the same erythema on unprotected skin. SPF 15 products block approximately 93.3% of UVB rays, an SPF 30 product blocks 96.7% of UVB, SPF 50 products block 98% of UVB. The SPF rating system possesses limitations. Primarily it evaluates protection against UVB-induced erythema but fails to adequately quantify UVA absorption (Wilson et al., 2012). Advanced in vitro and in vivo protocols, such as persistent pigment darkening (PPD), are needed to assess UVA protection and broad-spectrum efficacy (Sullivan et al., 2025).

Another factor impacting SPF efficacy is the application deficit. In vivo testing protocols assessed an application density of 2mg/cm² needed to verify product's labeled SPF. Clinical observations have demonstrated that consumers typically apply approximately 0.5mg/cm². The relationship between application thickness and UV protection is not linear and suboptimal application significantly reduces the photoprotective efficacy (Latha et al., 2013; Schalka et al., 2009).

2.2. Organic and inorganic UV filters

Sunscreen formulations use active agents classified into organic (chemical) and inorganic (physical/mineral) filters (Rai et al., 2012).

Organic filters are carbon-based molecules and their chemical architecture allows them to absorb UV photons by transitioning their electrons to an excited state. When the molecule returns to its ground state, the absorbed energy is dissipated as thermal heat. Organic filters are broadly used, because they provide lightweight film. Examples include avobenzone, octinoxate, octocrylene and the spectral coverage depends on the specific compound (Rai et al., 2012).

Inorganic filters (e.g. zinc oxide (ZnO), titanium dioxide (TiO₂), magnesium oxide (MgO)) have different mechanisms of UV protection. They act as physical barriers that reflect and scatter UVR and they also directly absorb UV photons. ZnO provides a broad-spectrum absorption covering the entire UVB and UVA range, and TiO₂ possesses superior UVB absorption (Rai et al., 2012). MgO is an alternative option because it possesses UVR dispersion capability, antioxidant and anti-inflammatory properties. A historical disadvantage of inorganic filters was their tendency to leave an undesirable white cast on the skin. To minimize this side effect, manufacturers have reduced particle size to the nanoscale (10-50nm). However, decreasing the particle size reduces the chalky appearance, it also introduces shift in the absorption spectrum – UVB absorption is enhanced, but longer UVA wavelengths filtering is weaker. To maintain the optimal UVR protection, mixture of micro- and nanosized particles is needed (de Melo Fantato et al., 2026; Schneider & Lim, 2019; Smijs & Pavel, 2011; Thy et al., 2025).

Furthermore, standard filters fail to protect against HEVL and blue-violet light (400-450nm). HEVL deeply penetrates and strongly correlates long-lasting hyperpigmentation via OPN3 receptor activation, especially in persons with Fitzpatrick skin types III-VI. To address this matter, advanced sunscreens incorporate iron oxides. Clinical trials have demonstrated that the addition of these compounds synergistically enhances blue light attenuation and reduces the recurrence of melasma and hyperpigmentation, compared to formulations without iron oxides (Doan et al., 2026).

2.3. Safety and formulation

The toxicological profiles, systemic absorption, photostability are the main concerns of current dermatological research. A major challenge with certain organic filters is their susceptibility to photodegradation. For instance, avobenzone exhibits keto-enol tautomerism and is easily degraded under intense UV exposure, which reduces UVA filtering capacity and generates reactive byproducts that can cause allergic dermatitis. Furthermore, mixing inorganic filters with specific organic filters can cause photocatalytic reactions, leading to the destruction of the organic ingredients. This issue might be solved by coating inorganic nanoparticles with silica or dimethicone (de Melo Fantato et al., 2026; Kullavanijaya & Lim, 2005).

Systemic absorption is another issue. Several organic filters, including oxybenzone, octocrylene, and avobenzone, penetrate the skin barrier and reach significant plasma concentrations. Particularly, oxybenzone has been identified as a potential endocrine-disrupting chemical (EDC), demonstrating estrogenic activity and accumulation, and also posing an ecological threat to coral reef ecosystems. However, inorganic nanoparticles penetrate only to stratum corneum, without clinically relevant systemic penetration (de Melo Fantato et al., 2026; Santander Ballestín & Luesma Bartolomé, 2023; Smijs & Pavel, 2011).

To overcome mentioned challenges, modern formulations include next-generation, large-molecular-weight organic filters such as bemotrizinol and phenylene bis-diphenyltriazine. These molecules dissipate excitation energy at very fast rates, resulting in high photostability and broad-spectrum coverage without penetrating the dermal-epidermal junction (Bacqueville et al., 2021; Dobos, 2025; Sullivan et al., 2025).

3. Prevention of photoaging

3.1. Molecular mechanisms of photoaging

The extrinsic aging is driven by chronic exposure to solar radiation, which is responsible for up to 80% of visible facial aging signs. UVA accounts for 90 to 95% of the terrestrial UV spectrum and penetrates deep into the skin layers. Unlike UVB, which causes direct DNA mutagenesis, UVA acts as an indirect carcinogen and the primary driver of photoaging by generating ROS. This oxidative stress triggers a destructive cascade by chronically upregulating MMPs, particularly MMP-1 (interstitial collagenase), MMP-3 (stromelysin-1), and MMP-9 (gelatinase B). These enzymes degrade type I procollagen and elastin fibers, causing elastosis, deep wrinkling, and the total loss of structural integrity (Feng et al., 2024).

Oxidative stress generated by UVR also alters transduction pathways such as mitogen-activated protein kinase (MAPK), the nuclear factor kappa beta (NF- κ B), the janus kinase (JAK), signal transduction and activation of transcription (STAT) and the nuclear factor erythroid 2-related factor (Nrf2). UVR induces the expression of pro-inflammatory genes, and causes immunosuppression by reducing the number and activity of the Langerhans cells (Bosch et al., 2015).

Furthermore, UVA-induced oxidative stress generates large-scale deletions in mitochondrial DNA (mtDNA). The “common deletion” – 4977bp – is found in the dermis of photoaged skin. This shows that UVA significantly influences mitochondrial function and reduces cellular energy production (Latha et al., 2013; Sullivan et al., 2025).

3.2. The role of visible light and infrared-A radiation

Modern research has expanded the role of photoprotection beyond the UV spectrum to address the biological impact of HEVL (400-500nm) and IR-A (760-1440nm). HEVL activates the OPN3 receptor pathway. This mechanism acts through calcium-dependent signaling and operates independently of the p53/POMC pathway utilized by UVR. Similarly, IR-A causes dermal aging by initiating a retrograde signaling response in fibroblasts, and the production of mitochondrial ROS induces expression of MMP-1, and further degradation of extracellular matrix. Standard UV filters fail to successfully filter these wavelengths, and the addition of iron oxides is necessary to shield against HEVL, and also antioxidants to attenuate oxidative matrix degeneration (González et al., 2023; Sullivan et al., 2025).

3.3. Secondary photoprotection

UV filters alone are often insufficient to provide complete protection. Broad-spectrum sunscreens only reduce free radical generation by approximately 55%. To address this protection gap, modern formulations add non-filtering agents such as L-ascorbic acid, tocopherol, niacinamide, and botanical polyphenols. These antioxidants actively neutralize ROS and attenuate UVA and IR-A-induced MMP upregulation (Ghali et al., 2026; González et al., 2023; Jesus et al., 2023).

Furthermore, the topical application of exogenous DNA repair enzymes (photolyase, endonuclease, glycosylase) is a crucial addition to photoprotection. These enzymes demonstrate significant *in vitro* and *in vivo* efficacy in the excision of CPDs and in the reduction of apoptotic death in the UV-damaged keratinocytes. Together, these biological components act synergistically with standard UV filters to provide broad-spectrum protection against molecular pathology of photoaging (Ghali et al., 2026; Luze et al., 2020).

3.4. Clinical evidence in prevention and reversal of photoaging

The efficacy of daily sunscreen usage is supported by long-term, randomized clinical data. In the Nambour Skin Cancer Prevention Trial in Australia, cutaneous microtopography of participants was evaluated. The study has demonstrated that individuals who used broad-spectrum sunscreen daily experienced no significant increase in skin aging over a 4.5-year period (A. Green et al., 1994).

More recent, prospective clinical trials have suggested that the consistent use of broad-spectrum sunscreen may contribute to the active reversal of existing photodamage. In a 52-week prospective study, participants applying a daily SPF 30 broad-spectrum, stable formulation showed significant improvements in skin texture, clarity and pigmentation by week 12. By week 52, 100% of participants demonstrated measurable improvements in texture and clarity, with significant reduction in crow's feet fine lines. The proposed biological mechanism for this reversal states that regular UV attenuation stops the environmental activation of MMP transcription. Elimination of the UV-induced inflammation permits dermal fibroblasts to reattach to intact collagen system, restoring the mechanical tension required to induce the synthesis of new type I procollagen. This allows the dermal matrix to enter a regenerative phase (Randhawa et al., 2016).

4. Clinical evidence in skin neoplasm prophylaxis

4.1. The Nambour skin cancer prevention trial

This clinical trial is the source of high-level evidence establishing the efficacy of daily sunscreen use. It was a randomized controlled trial with 1621 participants aged 25 to 75 years who were assigned to either daily sunscreen application or occasional application. The participants were observed for 4.5 years, and then for over a decade to evaluate long-term cancer incidence. This trial provided the first prospective data demonstrating the ability of photoprotection to decrease incidence of cutaneous malignancies (A. Green et al., 1994).

4.2. The efficacy against NMSCs

The Nambour trial has proven that regular sunscreen application significantly reduces the incidence of cutaneous squamous cell carcinoma (cSCC). Daily sunscreen users achieved a 39% reduction in cSCC tumors compared to occasional users. Long-term follow-up confirmed that this protective effect lasted with 35 to 40% reduction in cSCC tumor burden up to 8 years post-intervention (A. C. Green et al., 2011).

Conversely the trial has proven no statistically significant reduction in the incidence of basal cell carcinoma (BCC), which is connected with different mutation mechanisms. The development of cSCC is directly connected to lifetime UVR exposure which daily sunscreen effectively decreases. BCC is driven by intense, intermittent sun exposure and sunburns during childhood and adolescence, establishing a latency period that adult intervention cannot significantly reverse. However, in susceptible and immunosuppressed individuals (like organ transplant recipients) the consistent use of SPF 50 sunscreen over 24 months has reduced the incidence of actinic keratoses and keratinocyte carcinomas (A. C. Green et al., 2011; Hughes et al., 2013).

4.3. Efficacy against cutaneous melanoma

The Nambour trial provided prospective data regarding sunscreen effectiveness against melanoma. In the 10-year follow-up period 11 new melanomas developed in the daily sunscreen group compared to 22 in the discretionary group, demonstrating a 50% reduction in overall melanoma risk, and specifically 73% risk reduction for invasive melanomas. This finding suggests that the regular use of broad-spectrum sunscreen in adulthood can significantly reduce the incidence of the most lethal form of skin cancer (A. C. Green et al., 2011).

4.4. The epidemiological paradoxes

Despite the clinical trials results, systematic reviews and meta-analyses, studies often failed to show a protective effect of sunscreen against melanoma, with some reports of an increased risk. Some evidence for preventing BCC and cSCC was graded as “low certainty” because of a lack of histopathological confirmation and unclear correlation between sunscreen and beta-carotene (Sánchez et al., 2016). The paradox (connection between sunscreen and increased skin cancer risk) observed in available data is now understood to be the result of some crucial factors. Individuals with high melanoma risk (e.g. fair skin, high nevus burden, tendency to sunburn) are strongly motivated to use sunscreen which might falsely correlate with cancer incidence. Observations indicate that sunscreen users often embrace riskier, prolonged sun-seeking behaviour. Furthermore, older formulations of sunscreens effectively filtered UVB but allowed UVA to penetrate skin layers preventing the sunburn while producing chronic oxidative stress and melanogenesis (Nijsten, 2016). However, modern, broad-spectrum filters circumvent these limitations, providing highly effective protection against solar-induced carcinogenesis.

It is also possible that rising patient and physician awareness of skin malignancies, including melanoma, increases reports and documentation, which might suggest more accurate incidence of these malignancies (Lapides et al., 2023).

5. Future trends

5.1. Advanced delivery systems

Modern formulations increasingly turn to advanced pharmacological delivery systems. Microencapsulation technology involves inserting unstable organic filters within silica polymers, liposomes, or synthetic polymer matrices. This architecture provides physical isolation between UV chromophores, skin, and incompatible co-filters, preventing photocatalytic degradation. All of it reduce the risk of allergic contact dermatitis and transdermal penetration into the systemic circulation (Cozzi et al., 2018; Safian et al., 2025).

The integration of solid lipid nanoparticles (SLNs) and nanostructured lipid carriers (NLCs) represents a major interest in formulation science. These lipid-based molecules are not only highly biocompatible but also possess UV-attenuating properties due to their crystalline structures. SLNs and NLCs combined with organic or inorganic filters demonstrate synergistic protective effect, allowing to achieve high SPF with significantly lower concentrations of active ingredients (Afonso et al., 2026; Khobragade et al., 2025; Sullivan et al., 2025).

5.2. Next-generation and bio-derived UV filters

Driven by the need for sustainable, non-toxic, and biodegradable options, modern biotechnology explores natural sunscreens obtained from extremophilic organisms. Mycosporine-like amino acids (MAAs), naturally synthesized by cyanobacteria and marine red algae, are highly promising biological UV filters. They are low-molecular-weight, water-soluble compounds which dissipate UVR as heat without reactive byproducts generation. Furthermore, MAAs possess secondary antioxidant properties. They neutralize free radicals and protect fibroblasts from oxidative stress. While some current biotechnological extractions (such as engineered *Saccharomyces cerevisiae* or *Corynebacterium glutamicum*) remain not accessible for commercial use, the MAAs hold potential for a new class of purely organic and ecologically harmless sunscreens (Lawrence et al., 2018; Sullivan et al., 2025).

5.3. Artificial intelligence in formulation modeling

The integration of artificial intelligence (AI) and machine learning into cosmetic chemistry is upgrading sunscreens desingm testing, and optimization. AI algorithms are used to predict the UV absorption spectra, photostability, and potential toxicity of new compounds before they are synthesized (Lapides et al., 2023; Shim et al., 2019; Sullivan et al., 2025).

6. Conclusions

The growing incidence of skin malignancies coupled with the aesthetic and structural impacts of photoaging makes photoprotection a preventative pillar in dermatology. Available data demonstrate that daily application of broad-spectrum sunscreen significantly alters the trajectory of cutaneous malignancies, causing significant reductions in the incidence of cSCC and invasive melanoma. Furthermore, clinical data confirm that consistent UV protection not only stops the activation of MMPs but also induces the active reversal of existing photodamage, allowing the dermal extracellular matrix to enter a regenerative phase.

Modern photoprotection evolves beyond UVA/UVB. The pathogenic roles of HEVL and IR-A which induce oxidative stress and melanogenesis make it necessary to integrate physical blockers like iron oxides and secondary antioxidants into modern formulations.

Furthermore, the dermatological community and regulatory bodies have to face the challenge of marine ecotoxicity.

The future of photoprotection is strongly connected with clinical medicine, synthetic biology, and AI. The NLCs, the regulatory approval of large-molecular-weight photostable filters, and the biotechnological synthesis of MAAs represent a crucial shift toward safer, highly efficacious and ecologically sustainable solutions.

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