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Herbal Sunscreen Formulations for Enhanced UV Protection: A Review of Aloe vera, Curcumin, and Green Tea polyphenol

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ABSTRACT
Excessive exposure to ultraviolet (UV) radiation is a major environmental factor responsible for oxidative stress, inflammation, photoaging, immunosuppression, DNA damage, and skin carcinogenesis. Although conventional synthetic sunscreens effectively provide broad-spectrum protection against UVA and UVB radiation, increasing concerns regarding skin irritation, photoallergic reactions, photoinstability, and environmental toxicity have encouraged the development of safer and more sustainable alternatives. Herbal sunscreen formulations have emerged as promising candidates because of their ability to provide multifunctional photoprotection while improving overall skin health. This review summarizes the biological effects of UV radiation, the mechanisms of action of conventional sunscreens, and recent advances in plant-based photoprotective formulations. Particular emphasis is placed on medicinal plants and bioactive phytochemicals, including Aloe vera, curcumin, green tea polyphenols, and other botanicals, which protect the skin through antioxidant, anti-inflammatory, immunomodulatory, and DNA repair mechanisms. The review also highlights recent progress in nanotechnology-enabled herbal sunscreen systems, including nanoemulsions, liposomes, niosomes, and solid lipid nanoparticles, which improve the stability, skin penetration, controlled release, and photoprotective efficacy of herbal bioactives. Furthermore, the advantages and limitations of synthetic and herbal sunscreen formulations are critically compared to identify current challenges and future opportunities. Despite encouraging experimental findings, further standardized phytochemical characterization, comprehensive safety evaluation, well-designed clinical studies, and regulatory validation are essential before widespread clinical application. Overall, integrating herbal bioactive compounds with advanced drug delivery technologies offers a promising strategy for developing safe, effective, and environmentally sustainable next-generation sunscreen formulations for the prevention of UV-induced skin disorders.
Keywords: *Herbal sunscreen, UV protection, Aloe vera, Turmeric, Zinc Oxide, antioxidant activity, photoprotection, herbal cosmetics.*

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INTRODUCTION

As the body's outermost protective organ, the skin is constantly exposed to a variety of environmental factors, including sunlight, pollution, dust, smoke, chemicals, and microbial contamination. In recent years, environmental pollution, ozone layer depletion, and changes in lifestyle are regarded as major elements that has the greatest negative impact on skin health. Ultraviolet (UV) radiation is one of the major environmental factors responsible for various skin disorders. Continuous exposure to UVA and UVB rays leads to numerous acute and chronic skin conditions, including sunburn, erythema, hyperpigmentation, oxidative stress, wrinkles, loss of skin elasticity, immunological suppression, cellular damage and skin cancer [1,2]. UV radiation's detrimental effects on human skin have greatly grown in recent years due to rising environmental pollution, ozone layer depletion, and altered lifestyle choices.

UVA, UVB, and UVC rays are the three main categories of ultraviolet radiation based on wavelength. UVA rays have longer wavelengths and penetrate deeply into the skin's dermal layer, causing photoaging, wrinkles, pigmentation, and oxidative damage; UVB rays primarily affect the epidermal layer and cause sunburn, inflammation, erythema, and DNA damage, which primarily impact the epidermal layer and may raise the risk of skin cancer. Although UVC rays are thought to be extremely hazardous, they typically do not reach the earth's surface since they are primarily absorbed by the ozone layer [3]. Reactive oxygen

species (ROS), which harm proteins, lipids, collagen fibers, and cellular DNA, are produced by prolonged exposure to UVA and UVB radiation. This oxidative stress impairs the skin's natural defenses and speeds up the aging process [4].

Sunscreen formulations are frequently employed as protective agents to lessen the negative effects of UV radiation. Chemical UV filters used in synthetic sunscreens are widely accessible and offer protection from UV radiation by either reflecting or absorbing damaging light. Synthetic sunscreen formulations demand increasing photoprotection day by days; however, long-term usage of synthetic sunscreen ingredients has been linked to negative outcomes like skin irritation, allergic responses, contact dermatitis, hormone imbalance, phototoxicity, and environmental and human toxicity, according to a number of published research [5]. Prolonged health issues could result from certain synthetic sunscreen chemicals penetrating deeper layers of the skin like cancer. These drawbacks have led to an increase in interest in herbal and natural sunscreen formulations as herbal sunscreen formulations with their natural origin reflecting superior safety profile, biocompatibility, and low side effects provides excellant antioxidant activity. Herbal ingredients such as Aloe vera and Turmeric possess significant antioxidants, anti-inflammatory, moisturizing, and photoprotective properties. Aloe vera helps in skin hydration, soothing, and repair, whereas Turmeric contains curcumin, a potent antioxidant that protects the skin against oxidative stress caused by ultraviolet radiation [6,7]. Zinc Oxide acts as a mineral sunscreen agent providing broad-spectrum protection against both UVA and UVB radiation [8].

Through this review we highlight the role of herbal ingredients and Zinc Oxide in sunscreen formulations, mechanisms of UV protection, phytochemical constituents responsible for photoprotection, advantages of herbal sunscreens over synthetic formulations, and findings reported in previous scientific studies [9]. Various reviewed studies suggest that herbal sunscreen formulations may serve as safe and effective alternatives to conventional sunscreens while also providing additional skin benefits [8].

Ultraviolet violet (UV) Radiation (Table 1)

Sunlight comprises a continuous spectrum of electromagnetic radiation that is broadly categorized into ultraviolet (UV), visible, and infrared (IR) regions [10]. Among these, ultraviolet radiation is the most biologically active component and is primarily responsible for photoaging, photodamage, and the initiation of skin cancer. UV radiation occupies the region of the electromagnetic spectrum between visible light and X-rays and is classified into three wavelength ranges: UV-A (320–400 nm), UV-B (280–320 nm), and UV-C (200–280 nm) (**Table 1**). UV-C possesses the shortest wavelength and highest energy, making it highly mutagenic and capable of causing extensive DNA damage, immune dysfunction, and carcinogenesis. However, under natural environmental conditions, nearly all UV-C radiation is absorbed by the stratospheric ozone layer and does not reach the Earth's surface. Consequently, UV-A and UV-B are the major forms of solar UV radiation responsible for biological effects in human skin. Of the solar UV radiation that reaches the Earth, UV-A accounts for approximately 90–99%, whereas UV-B contributes only 1–10%. Despite its lower abundance, UV-B exhibits greater biological activity because of its higher photon energy and stronger interaction with cellular DNA. In contrast, UV-A penetrates more deeply into the dermis, where it primarily induces oxidative stress through the generation of reactive oxygen species (ROS), thereby contributing to photoaging and indirect DNA damage.

Table 1: Classification of Ultraviolet Radiation and Its Biological Effects

UV Type	Wavelength (nm)	Penetration Depth	Major Biological Effects	Clinical Outcome
UVA	320–400	Dermis	ROS generation, oxidative stress, collagen degradation	Photoaging, pigmentation, indirect DNA damage
UVB	280–320	Epidermis	Direct DNA damage (CPDs), inflammation	Sunburn, photocarcinogenesis
UVC	200–280	Does not naturally reach skin	Highly mutagenic and germicidal	Absorbed by ozone layer

UV radiation promotes skin carcinogenesis through multiple interconnected molecular and cellular mechanisms. Individual susceptibility to UV-induced skin cancer is influenced by several genetic factors, particularly variations in genes involved in pigmentation, DNA repair, and immune regulation [11]. Individuals with inherited defects in DNA repair pathways are especially vulnerable to UV-mediated mutagenesis and subsequent skin cancer development. In addition, UV exposure modulates the cutaneous immune system by activating inflammatory signaling pathways, which may facilitate tumor initiation and progression through local immunosuppression. The skin possesses several intrinsic defense mechanisms that help limit UV-induced injury. Melanin, the principal epidermal pigment, serves as the primary

photoprotective barrier by absorbing ultraviolet radiation and dissipating the absorbed energy as heat, thereby reducing damage to underlying cellular components. Damaged keratinocytes can also undergo programmed cell death through activation of death receptors, including Fas, preventing the survival and proliferation of genetically compromised cells. Furthermore, the melanocortin-1 receptor (MC1R) plays a critical role in regulating melanogenesis, adaptive tanning responses, and resistance to UV-induced oxidative damage [12]. Genetic polymorphisms in MC1R are associated with reduced photoprotection and an increased susceptibility to skin cancer [13].

At the molecular level, UV radiation damages cellular DNA through both direct and indirect pathways. Direct absorption of UV photons, particularly UV-B, results in the formation of cyclobutane pyrimidine dimers (CPDs) and 6-4 photoproducts, which interfere with DNA replication and transcription if left unrepaired [14]. Indirect damage occurs predominantly through UV-A-mediated generation of ROS, which oxidize DNA bases, proteins, and membrane lipids, leading to genomic instability, cellular dysfunction, and mutagenesis. Persistent accumulation of these molecular lesions contributes to premature skin aging and malignant transformation. Although excessive UV exposure is harmful, controlled exposure provides important physiological benefits. UV-B radiation initiates the conversion of 7-dehydrocholesterol to pre-vitamin D_3 in the epidermis, representing the first step in vitamin D biosynthesis [15]. Additionally, UV-induced tanning and epidermal thickening confer a modest degree of natural photoprotection, corresponding to an estimated sun protection factor (SPF) of approximately 3-4. Therefore, UV radiation exerts both beneficial and detrimental effects on human skin [16]. While limited exposure is essential for maintaining normal physiological functions, prolonged or excessive exposure substantially increases the risk of oxidative stress, photoaging, immune suppression, DNA damage, and skin cancer. These contrasting biological effects underscore the importance of effective photoprotective strategies that reduce harmful UV exposure while preserving its beneficial physiological functions [17].

Types of UV Radiation

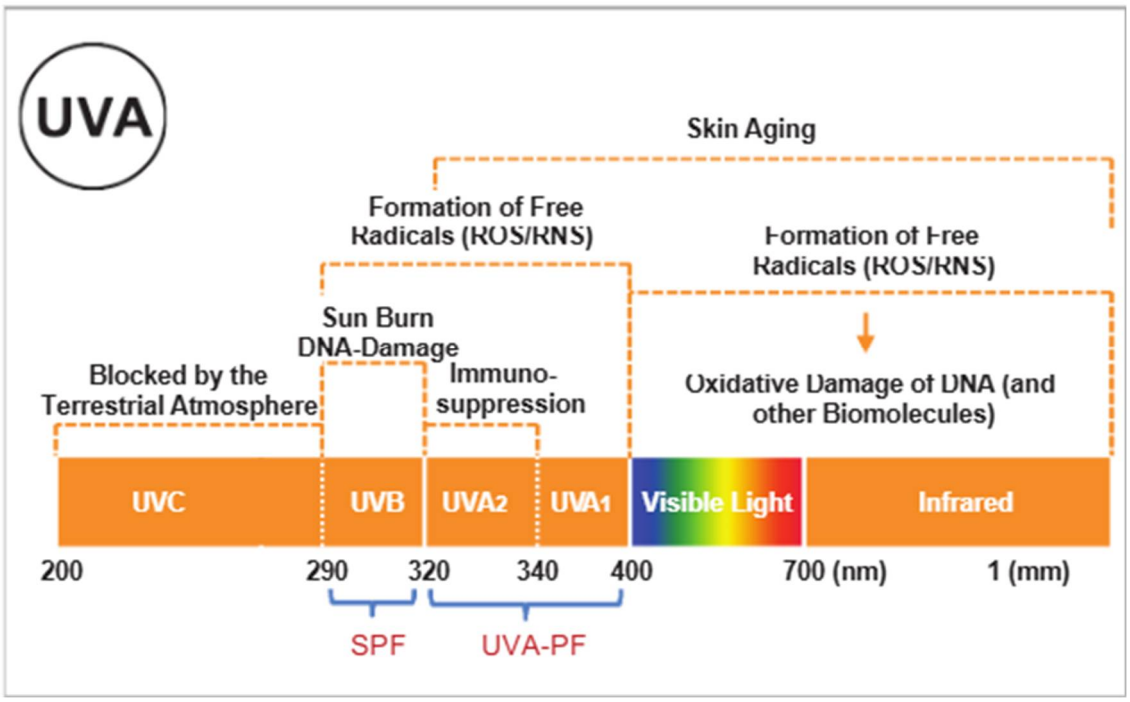
Based on wavelength and biological effects, UV radiation is classified into three categories: ultraviolet A (UVA), ultraviolet B (UVB), and ultraviolet C (UVC), each exhibiting distinct penetration depths and biological consequences in the skin [18]. UVA radiation (320–400 nm), also known as long-wave UV, constitutes the largest proportion of solar UV radiation reaching the Earth's surface [19]. Due to its longer wavelength, UVA penetrates deeply into the dermis, where it primarily induces oxidative stress through the generation of reactive oxygen species (ROS), leading to collagen degradation, photoaging, and indirect DNA damage. Although its carcinogenic potential is lower than that of UVB, chronic UVA exposure significantly contributes to cumulative skin damage [20].

UVB radiation, with wavelengths ranging from 290 to 320 nm, penetrates predominantly into the epidermis and possesses higher photon energy than UVA. Consequently, UVB is the primary cause of erythema (sunburn), direct DNA damage, and photodamage [21]. It induces the formation of cyclobutane pyrimidine dimers (CPDs) and other DNA photoproducts, making it a potent mutagen and a complete carcinogen. Therefore, UVB is considered the major contributor to photocarcinogenesis and many of the detrimental biological effects associated with excessive solar exposure.

UVC radiation comprises wavelengths between approximately 200 and 290 nm and is characterized by the highest energy among the three UV regions [22]. It exhibits potent germicidal activity and can cause severe cellular and DNA damage upon direct exposure. However, under normal atmospheric conditions, almost all UVC radiation is absorbed by the stratospheric ozone layer and therefore does not reach the Earth's surface or contribute significantly to natural skin exposure.

UV radiation exerts both beneficial and harmful effects on human health. The intensity of UV radiation reaching the Earth's surface varies according to several environmental factors, including latitude, altitude, season, time of day, cloud cover, and stratospheric ozone concentration. Controlled UV exposure is essential for the cutaneous synthesis of vitamin D and has therapeutic applications in the management of several dermatological disorders, including psoriasis vulgaris and vitiligo [23]. In addition, UV radiation possesses antimicrobial properties that are exploited in selected medical and environmental applications. Nevertheless, excessive or prolonged UV exposure disrupts extracellular matrix homeostasis by accelerating collagen and elastin degradation, ultimately leading to erythema, hyperpigmentation (tanning), photoaging, immune suppression, and an increased risk of skin cancer [24] (**Fig. 1**).

FIGURE 1: Effect of UV radiation on skin



UVA Radiation

Among all UV radiation, UVA photons have the longest wavelength and pierce the skin's dermal layer deeply. The primary causes of photoaging, wrinkles, skin elasticity loss, and pigmentation are these rays. Reactive oxygen species (ROS), which cause oxidative stress and cellular damage, are produced and collagen degradation is accelerated by prolonged exposure to UVA radiation. Even when exposed to indirect sunlight, UVA rays can still be damaging because they can pass through glass and clouds [25].

UVB Radiation

UVB rays mainly affect the epidermal or outermost layer of the skin. They are primarily responsible for sunburn, redness, inflammation, and erythema. UVB radiation causes direct DNA damage in skin cells and significantly increases the risk of skin cancer after prolonged exposure. Although UVB rays do not penetrate as deeply as UVA rays, they are considered more harmful in causing acute skin damage [26].

UVC Radiation

Among UV radiations, UVC rays have the maximum intensity and the shortest wavelength. The ozone layer entirely absorbs these rays under typical environmental conditions, preventing them from reaching the earth's surface despite their tremendous risk to living things. As a result, UVC radiation typically has little effect on normal skin damage [27].

EFFECT OF UV RADIATION ON SKIN

Human skin is significantly impacted by UV radiation, primarily from sunshine. Both short-term and long-term skin damage can result from prolonged or severe UV exposure [28]. The following are some primary effects:

a. Sunburn and Erythema

Sunburn, which is characterized by redness, discomfort, and inflammation of the skin, is brought on by excessive UV exposure. Erythema is another name for this disorder, which typically develops following acute UVB exposure. The extent of erythema is influenced by the level and duration of UVR exposure experienced by the skin [29].

b. UV-induced Skin Carcinogenesis

UV-induced skin carcinogenesis refers to the uncontrolled proliferation of skin cells resulting from prolonged exposure to ultraviolet radiation. Skin cancers are broadly classified into basal cell carcinoma (BCC), squamous cell carcinoma (SCC), collectively referred to as non-melanoma skin cancers (NMSCs), and cutaneous malignant melanoma [30]. Basal cell carcinoma originates from the basal layer of the epidermis and is typically characterized by slow growth with a low tendency to metastasize. In contrast, squamous cell carcinoma develops from keratinocytes within the upper layers of the epidermis and exhibits a greater potential for local invasion and metastasis than BCC. Cutaneous malignant melanoma arises from

melanocytes, the pigment-producing cells responsible for melanin synthesis, and is considered the most aggressive form of skin cancer, accounting for nearly 75% of skin cancer-related mortality [31]. The global burden of skin cancer has increased considerably over recent decades, making it one of the most common malignancies worldwide. Epidemiological studies indicate that approximately one in five individuals in the United States is expected to develop skin cancer during their lifetime. Furthermore, more than 23 million new cases of non-melanoma skin cancer are estimated to occur annually worldwide, with the highest incidence reported among fair-skinned (Caucasian) populations. The rising prevalence of skin cancer highlights the critical need for effective photoprotective measures and early preventive interventions.

c. Cutaneous Photoaging

Cutaneous photoaging refers to the premature aging of the skin resulting from prolonged exposure to solar ultraviolet radiation. Skin aging is a complex and inevitable biological process characterized by the gradual decline in the structural integrity and physiological functions of the skin [32]. It occurs through two major mechanisms: intrinsic aging and extrinsic aging. Intrinsic aging is a genetically regulated, time-dependent process that develops naturally with advancing age, whereas extrinsic aging is primarily driven by environmental factors, particularly chronic exposure to ultraviolet radiation, leading to accelerated deterioration of the skin [33,34].

The onset and progression of skin aging are significantly influenced by exposure to various environmental factors. Among these, ultraviolet radiation (UVR) is considered the primary external factor responsible for accelerating skin aging, a process known as cutaneous photoaging. Unlike intrinsic aging, which occurs naturally over time, the severity of photoaging largely depends on cumulative sun exposure and individual skin pigmentation, with lighter skin types generally exhibiting greater susceptibility to UV-induced damage [35]. Compared with intrinsic aging, photoaging produces far more pronounced changes in the skin and is estimated to account for nearly 90% of the visible signs of skin aging [36].

d. Premature aging and wrinkles

Premature skin aging is one of the most common consequences of chronic ultraviolet (UV) radiation exposure and is characterized by the early appearance of wrinkles, fine lines, reduced skin elasticity, uneven pigmentation, rough skin texture, and loss of skin tone. These visible alterations result primarily from progressive damage to the dermal extracellular matrix, particularly the degradation of collagen fibers, which are responsible for maintaining the structural integrity and mechanical strength of the skin [37]. The molecular basis of this process was demonstrated by Fisher et al., who reported that repeated exposure to ultraviolet radiation significantly increased the expression of matrix metalloproteinases (MMPs), including collagenase (MMP-1), stromelysin (MMP-3), and gelatinase (MMP-9). These collagen-degrading enzymes accelerated the breakdown of type I collagen within the dermal extracellular matrix, thereby promoting wrinkle formation and other manifestations of premature skin aging. Furthermore, the study showed that topical pretreatment with tretinoin markedly suppressed UV-induced MMP expression and activity, reducing collagen degradation and confirming that MMP-mediated extracellular matrix remodeling is a key mechanism responsible for UV-induced premature skin aging [38].

e. Hyperpigmentation and tanning

Chronic exposure to ultraviolet radiation activates melanocytes and enhances melanin biosynthesis, causing increased deposition of melanin granules in the upper epidermal layers and contributing to skin hyperpigmentation [39]. The accumulation of melanin in the epidermis promotes tanning and the development of hyperpigmented lesions, resulting in uneven skin pigmentation that is most evident in chronically sun-exposed regions [40].

f. Loss of skin elasticity

Skin elasticity is primarily maintained by the organized three-dimensional network of collagen and elastin fibers within the dermal extracellular matrix [41]. Chronic exposure to UVB radiation disrupts the structural organization of these matrix proteins, leading to a progressive decline in the skin's elastic properties. This mechanism was demonstrated in an experimental study using UVB-irradiated rat skin, where scanning electron microscopy (SEM) revealed marked alterations in the three-dimensional architecture of elastic fibers following UVB exposure. The disruption and fragmentation of the elastin network impaired the normal mechanical support of the dermis, resulting in reduced skin elasticity and contributing to wrinkle formation and other manifestations of premature skin aging. These findings provide evidence that UVB-induced damage to the dermal elastic fiber network is a key structural mechanism underlying the loss of skin elasticity associated with photoaging [42].

g. DNA damage and Oxidative Stress

UVR-induced generation of reactive oxygen species (ROS) promotes oxidative damage to DNA bases, leading to the formation of DNA dimers that are commonly used as biomarkers of UV-mediated oxidative DNA injury. Failure to repair UV-induced DNA damage activates programmed cell death through the apoptotic pathway. In contrast, the persistence of DNA lesions during the S phase of the cell cycle can result

in increased cell proliferation, promoting mutagenesis and the subsequent development of skin cancer [43]. UV radiation can directly damage the DNA of skin cells. This may lead to mutations and improper cell functioning, increasing the risk of skin disorders [44]. Exposure to UV rays generates free radicals in the skin, leading to oxidative stress. This imbalance damages skin cells and accelerates aging and tissue breakdown.

h. **Skin Inflammation**

Long-term, low-grade inflammation plays a significant role in the pathogenesis of skin photoaging by promoting continuous tissue damage and impairing normal skin homeostasis [45,46]. The persistent presence of a pro-inflammatory microenvironment contributes to an increased risk of skin carcinogenesis and tumor progression. UV radiation-induced DNA damage and degradation of the extracellular matrix (ECM) disrupt normal skin homeostasis, generating cellular stress that initiates inflammatory responses. These responses are primarily regulated through the activation of the nuclear factor-kappa B (NF-κB) and p38 mitogen-activated protein kinase (p38 MAPK) signaling pathways in both immune and non-immune skin cells. In addition, accumulating evidence suggests that UVB radiation further amplifies cutaneous inflammation by activating inflammasomes, thereby promoting the production of pro-inflammatory mediators [47]. Hasegawa et al. [48] Study have shown that UV-induced DNA damage in human keratinocytes activates the NLRP3 inflammasome, leading to the release of several pro-inflammatory cytokines, including IL-1β, IL-1α, IL-6, and TNF-α. In addition, UV radiation-induced cellular stress promotes the formation of senescent cells that acquire a senescence-associated secretory phenotype (SASP), characterized by the persistent secretion of inflammatory mediators that sustain chronic cutaneous inflammation [49,50]. Premature senescence has been observed in diverse cutaneous cell types, including keratinocytes, fibroblasts, melanocytes, and subcutaneous preadipocytes, contributing to the progression of skin aging and chronic inflammation [51]. Cellular senescence halts cell proliferation while promoting the production of numerous pro-inflammatory and anti-inflammatory mediators [52]. Evidence from experimental models indicates that inflammatory responses are characterized by the secretion of several pro-inflammatory mediators, including interleukins, chemokines (CCL2, CCL3, CXCL1, and CXCL8), and granulocyte-macrophage colony-stimulating factor (GM-CSF). Kondo early biochemical investigations of skin photoaging have demonstrated that chronic UV exposure enhances the expression of numerous interleukins and chemokines in both immune cells, including monocytes, macrophages, and natural killer (NK) cells, as well as various non-immune skin cells. Furthermore, genome-wide transcriptional profiling studies have shown that UVB irradiation markedly upregulates the expression of several inflammatory cytokines and chemokines, particularly CCL3, CXCL1, CXCL3, and CXCL5, highlighting their important role in mediating UV-induced cutaneous inflammation [53]. In addition, the robust increase in the expression of COX-2 indicates that prostanoids have a role in the photoaging process. Collectively, these findings suggest that UVR-induced skin damage activates protective defense mechanisms by enhancing cytokine-mediated transcriptional responses and promoting chemokine-directed recruitment of immune cells to the sites of injury [54]. UV exposure can trigger inflammatory responses in the skin, causing redness, swelling, and irritation.

i. Skin Cancer

Prolonged and repeated exposure to UV radiation is a major risk factor for skin cancer. It can lead to serious conditions such as melanoma and non-melanoma skin cancers. Skin cancer susceptibility is primarily governed by the degree of ultraviolet radiation exposure and individual differences in skin pigmentation [55]. Cutaneous malignant melanoma is the most aggressive and life-threatening form of skin cancer. It originates from melanocytes located within the epidermis and is characterized by a high metastatic potential and resistance to treatment. Over the past several decades, the incidence of melanoma has risen steadily, making it a major global public health concern [56]. Numerous epidemiological and molecular studies have consistently demonstrated a close relationship between UV exposure and the occurrence of all types of skin cancer [57]. Current estimates suggest that UV exposure accounts for nearly 65% of melanoma and approximately 90% of non-melanoma skin cancer cases [58,59]. UVR induces characteristic mutational signatures in several cancer-associated genes, including the **p53** tumor suppressor gene, which is frequently mutated in squamous cell carcinoma. In addition, exome sequencing studies of melanoma have provided compelling genetic evidence that UV radiation acts as a direct mutagen, playing a pivotal role in the initiation and progression of melanoma [60,61]. Given that UV-induced genetic mutations are key contributors to melanomas and other forms of skin cancer, resistance to UV-driven mutagenesis represents an important determinant of an individual's susceptibility to these malignancies.

ROLE OF SUNSCREENS AGAINST ULTRAVIOLET RADIATION

Sunscreens are the primary topical agents used to prevent UV-induced skin damage by reducing the amount of solar radiation that penetrates the skin. Depending on their mode of action, sunscreen

formulations contain either organic (chemical) UV filters, inorganic (mineral) UV filters, or a combination of both to achieve broad-spectrum protection against UVA and UVB radiation [62]. Organic sunscreens, also known as chemical UV filters, protect the skin by absorbing incident ultraviolet radiation. Their photoprotective activity is attributed to the presence of conjugated aromatic structures, typically consisting of aromatic rings linked to carbonyl groups, which readily absorb high-energy UV photons. Following absorption, these molecules enter an excited electronic state and subsequently return to their ground state by dissipating the absorbed energy as lower-energy heat or longer-wavelength radiation. This photophysical process prevents excessive UV energy from reaching viable skin cells and thereby reduces UV-induced cellular injury. Individual organic filters exhibit distinct absorption profiles; therefore, modern sunscreen formulations generally combine multiple UVA and UVB filters to provide broad-spectrum photoprotection [63].

The clinical efficacy of sunscreen depends not only on its formulation but also on proper application. Sunscreen should be applied uniformly to all exposed skin approximately 15–30 minutes before sun exposure at the recommended dose of 2 mg/cm^2, equivalent to approximately 30 mL for full-body coverage in an average adult. To maintain adequate photoprotection, reapplication every two hours is recommended, particularly after swimming, excessive perspiration, or towel drying, as these activities substantially reduce the protective film on the skin [64]. Regular sunscreen use plays a pivotal role in preventing both acute and chronic UV-induced skin damage. By limiting ultraviolet-induced melanogenesis, sunscreen effectively reduces the incidence of post-inflammatory hyperpigmentation, particularly in individuals with darker skin phototypes. Furthermore, consistent sunscreen application decreases DNA damage, oxidative stress, photoaging, and photocarcinogenesis associated with prolonged solar exposure.

In addition to organic filters, mineral UV filters such as zinc oxide and titanium dioxide are approved by regulatory authorities for use in broad-spectrum sunscreen formulations. These inorganic ingredients effectively attenuate both UVA and UVB radiation while exhibiting excellent photostability and a favorable safety profile, making them suitable for individuals with sensitive skin. Several professional organizations strongly advocate routine sunscreen use as an essential component of comprehensive photoprotection. The American Society of Clinical Oncology (ASCO) identifies regular application of broad-spectrum sunscreen, together with protective clothing and avoidance of excessive sun exposure, as an effective strategy for reducing the incidence of skin cancer. Similarly, the American Academy of Dermatology (AAD) recommends the routine use of broad-spectrum, water-resistant sunscreens with a minimum sun protection factor (SPF) of 30. These recommendations are complemented by additional protective measures, including wearing sun-protective clothing, seeking shade during peak sunlight hours, and minimizing unnecessary UV exposure [65].

The AAD further emphasizes the importance of photoprotection in patients with premalignant and malignant skin disorders. Individuals diagnosed with actinic keratosis, basal cell carcinoma, or cutaneous squamous cell carcinoma are advised to adopt lifelong sun-protective practices, including regular use of broad-spectrum sunscreens, avoidance of tanning beds, reduction of direct sunlight exposure, and consistent use of protective clothing. Collectively, these preventive measures substantially decrease cumulative UV exposure and reduce the likelihood of recurrent UV-induced skin damage and subsequent skin malignancies.

ADVANTAGES AND LIMITATIONS OF SYNTHETIC SUNSCREEN FORMULATIONS

Conventional sunscreen formulations employ both organic (chemical) and inorganic (mineral) UV filters to minimize the harmful effects of ultraviolet radiation. Inorganic sunscreens primarily utilize metal oxide-based filters, particularly titanium dioxide (TiO_2) and zinc oxide (ZnO), which provide broad-spectrum protection through a combination of reflection, scattering, and absorption of UV radiation. Their excellent photostability and broad UV coverage have established them as the most widely used mineral sunscreen ingredients [66].

Despite their effectiveness, conventional inorganic filters possess several formulation limitations. Micron-sized TiO_2 and ZnO particles strongly scatter visible light, producing a characteristic opaque white film on the skin that negatively affects cosmetic appearance and user acceptance. To overcome this limitation, these minerals are increasingly manufactured as nanoparticles. Nanosized TiO_2 and ZnO exhibit improved transparency while maintaining efficient UV attenuation, thereby enhancing both aesthetic appeal and sunscreen performance [67]. Although nanoparticle-based mineral sunscreens offer improved cosmetic properties, their long-term safety remains an active area of investigation. Concerns have been raised regarding the potential generation of reactive oxygen species under UV irradiation, particularly by titanium dioxide nanoparticles, as well as the possible biological and environmental consequences associated with chronic nanoparticle exposure. Current evidence generally supports the safety of approved mineral UV

filters when appropriately formulated; however, continued evaluation of their long-term toxicological profile remains necessary.

Organic UV filters are also associated with certain limitations. Some synthetic sunscreen ingredients have been linked to adverse dermatological reactions, including irritant and photoallergic contact dermatitis, with benzophenone-3 (oxybenzone) being among the most frequently reported sensitizing agents [68]. In addition to cutaneous safety concerns, increasing public awareness regarding the potential environmental impact of selected synthetic UV filters has further encouraged the search for safer and more sustainable alternatives. These limitations have stimulated considerable interest in herbal and plant-derived photoprotective agents. Numerous medicinal plants contain naturally occurring polyphenols, flavonoids, carotenoids, and other bioactive phytochemicals possessing ultraviolet-absorbing, antioxidant, and anti-inflammatory properties. Because these compounds may provide photoprotection while exhibiting favorable biocompatibility, they represent promising candidates for next-generation sunscreen formulations. Nevertheless, comprehensive *in vitro, in vivo*, and clinical investigations remain essential to establish their efficacy, stability, safety, and regulatory acceptability before widespread incorporation into commercial sunscreen products [69]. Conventional synthetic sunscreens and herbal sunscreen formulations differ in their source, mechanism of photoprotection, safety profile, and additional therapeutic benefits. A comparative overview of their key characteristics is presented in **Table 2**, highlighting the advantages and limitations of each approach in the prevention of UV-induced skin damage.

Table 2: Comparison of Synthetic and Herbal Sunscreens

Parameter	Synthetic Sunscreens	Herbal Sunscreens
Source	Synthetic UV filters	Plant-derived bioactive compounds
Mechanism	UV absorption/reflection	Antioxidant, anti-inflammatory, UV absorption, DNA protection
SPF	High	Moderate (improved with formulations)
Skin irritation	Possible	Generally lower
Environmental impact	Some filters harmful to aquatic life	More eco-friendly
Additional benefits	UV protection only	Moisturizing, wound healing, anti-aging, antioxidant
Major limitation	Allergic reactions, photoinstability	Lower stability and standardization

Herbal Sunscreen Formulations (Table 2)

Growing concerns regarding the safety, environmental impact, and long-term use of synthetic UV filters have accelerated research into herbal sunscreen formulations [70]. These formulations incorporate plant-derived bioactive compounds with intrinsic photoprotective properties, providing a natural approach to protecting the skin against UV-induced damage. In addition to absorbing or attenuating UV radiation, many herbal ingredients possess antioxidant, anti-inflammatory, moisturizing, wound-healing, and skin-rejuvenating activities, allowing them to simultaneously prevent photodamage and promote overall skin health. Herbal sunscreen formulations are generally regarded as skin-friendly because of their natural origin, lower incidence of adverse reactions, and favorable safety profile. Compared with conventional synthetic sunscreens, botanical formulations are increasingly preferred by consumers owing to their perceived biocompatibility, affordability, and multifunctional therapeutic properties. Traditionally, medicinal plants have been applied topically to prevent and manage a variety of dermatological disorders, and they continue to serve as valuable sources of bioactive ingredients for modern cosmetic and pharmaceutical formulations [71]. Depending on the intended application, herbal sunscreen products are available as creams, gels, lotions, ointments, emulsions, foams, and sprays.

Aloe vera in Herbal Sunscreen Formulations (Figure 2)

Aloe vera (*Aloe barbadensis* Miller) is one of the most extensively investigated medicinal plants for dermatological and cosmetic applications because of its moisturizing, anti-inflammatory, antioxidant, wound healing, and photoprotective properties. For centuries, it has been used in traditional medicine for the treatment of various skin disorders and is now widely incorporated into skincare and sunscreen formulations as a multifunctional natural ingredient [72].

The transparent gel extracted from the succulent leaves of *Aloe vera* is rich in biologically active constituents, including vitamins (A, C, E, B_{12}, and folic acid), minerals (calcium, magnesium, zinc, selenium, potassium, and copper), amino acids, polysaccharides, enzymes, phenolic compounds, and flavonoids. These phytochemicals collectively contribute to the plant's antioxidant and skin-protective activities by scavenging reactive oxygen species (ROS), reducing oxidative stress, and protecting cellular components from ultraviolet (UV)-induced damage [73]. Although *Aloe vera* exhibits only moderate intrinsic UV-filtering activity, it provides significant indirect photoprotection through multiple biological mechanisms. Topical application has been reported to stimulate the expression of metallothionein, an antioxidant

protein that scavenges free radicals and preserves the activity of endogenous antioxidant enzymes such as superoxide dismutase and glutathione peroxidase. Furthermore, *Aloe vera* suppresses UV-induced inflammatory responses by reducing the production of pro-inflammatory mediators and modulating immunosuppressive cytokines, thereby minimizing erythema, inflammation, and sunburn while promoting tissue repair [74].

In addition to its photoprotective effects, *Aloe vera* enhances skin hydration by virtue of its polysaccharide-rich gel, which improves moisture retention and maintains epidermal barrier integrity. It also stimulates fibroblast proliferation and collagen and elastin synthesis, thereby improving skin elasticity, accelerating wound healing, and reducing the visible signs of photoaging. These multifunctional properties make *Aloe vera* a valuable ingredient in herbal sunscreen formulations, where it serves not only as a photoprotective adjunct but also as a skin-conditioning and anti-aging agent [75]. Several formulation studies have demonstrated the potential of *Aloe vera* in sunscreen development. A polyherbal sunscreen cream containing *Aloe vera*, papaya, sandalwood, tomato, and natural oils exhibited excellent physicochemical characteristics, good spreadability, satisfactory stability, and an in vitro SPF of approximately 25, without producing skin irritation. Similarly, *Aloe vera*-based topical creams have shown desirable stability and moderate photoprotective efficacy, supporting their suitability for herbal sunscreen applications.

The incorporation of *Aloe vera* into advanced delivery systems has further improved its photoprotective performance. In one study, nanosized titanium dioxide combined with spray-dried *Aloe vera* powder produced a broad-spectrum sunscreen with enhanced SPF, improved UVA/UVB protection, and satisfactory storage stability. Another investigation developed *Aloe vera*-loaded solid lipid nanoparticle (SLN) sunscreen cream, which demonstrated improved stability, enhanced skin permeation, controlled release, good skin compatibility, and SPF values comparable to commercial sunscreen products. These findings suggest that nanotechnology-based delivery systems can significantly enhance the therapeutic efficacy and formulation performance of *Aloe vera*, supporting its application as a safe and effective natural ingredient in next-generation herbal sunscreen formulations [76].

FIGURE 2: Illustration of Aloe vera morphology

Curcumin in Herbal Sunscreen Formulations (Figure 3)

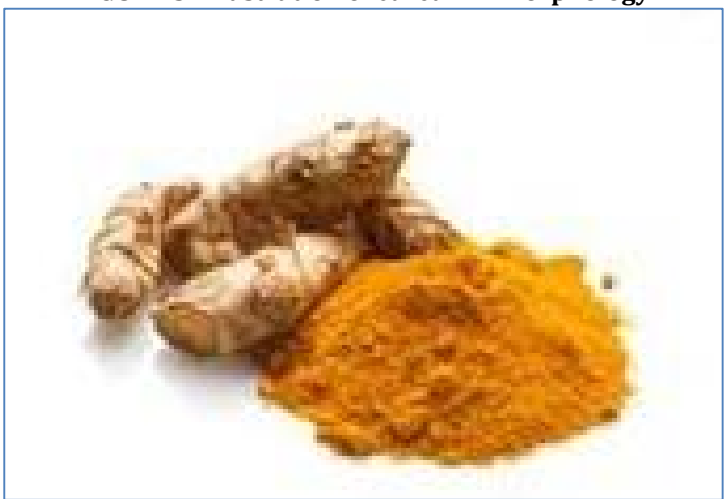
Curcumin, the principal polyphenolic constituent of *Curcuma longa* (turmeric), has attracted considerable attention as a natural photoprotective agent owing to its potent antioxidant, anti-inflammatory, antimicrobial, anti-aging, and anticancer properties. Among environmental factors responsible for premature skin aging, ultraviolet (UV) radiation is one of the most significant [77]. Curcumin protects the skin against UV-induced photodamage through multiple mechanisms, including scavenging reactive oxygen species (ROS), suppressing inflammation, inhibiting melanogenesis, and preserving collagen integrity, thereby reducing wrinkle formation and photoaging.

The photoprotective activity of curcumin is largely attributed to its ability to modulate oxidative stress-related signaling pathways. UV-induced ROS activate several molecular cascades, including the EGFR/MAPK and NF-κB pathways, which promote inflammation, extracellular matrix degradation, and cellular apoptosis. Curcumin counteracts these effects by activating the Nrf2/ARE signaling pathway. Under oxidative stress, Nrf2 dissociates from Kelch-like ECH-associated protein-1 (Keap1), translocates to the nucleus, and induces the expression of antioxidant and cytoprotective enzymes, thereby enhancing the skin's endogenous defense against UV-mediated oxidative damage [78].

Experimental studies further support the potential of curcumin as a sunscreen ingredient. In human dermal fibroblasts exposed to UVA radiation, curcumin significantly improved cell viability, reduced apoptosis, and

restored the activities of endogenous antioxidant enzymes, including glutathione, superoxide dismutase, and catalase [79]. It also attenuated endoplasmic reticulum stress and inflammatory responses while suppressing the expression of matrix metalloproteinases (MMP-1 and MMP-3), enzymes responsible for collagen degradation. Simultaneously, curcumin enhanced the transforming growth factor-β (TGF-β)/Smad2/3 signaling pathway, promoting collagen synthesis and extracellular matrix repair. Collectively, these findings highlight curcumin as a promising natural bioactive ingredient for incorporation into herbal sunscreen and advanced photoprotective skincare formulations. [79].

FIGURE 3: Illustration of curcumin morphology



Green Tea Polyphenols in Herbal Sunscreen Formulations (Table 3)

Green tea (*Camellia sinensis*) is one of the most extensively investigated botanical ingredients for photoprotection owing to its high content of polyphenolic catechins, particularly epigallocatechin-3-gallate (EGCG), epigallocatechin (EGC), epicatechin (EC), and epicatechin gallate (ECG) [80]. Among these, EGCG is the predominant and most biologically active constituent, exhibiting potent antioxidant, anti-inflammatory, and chemopreventive activities. Both topical and oral administration of green tea polyphenols have been shown to protect against UV-induced erythema, photoaging, immunosuppression, and skin carcinogenesis.

Table 3: Molecular Mechanisms of Herbal Photoprotection

Mechanism	Molecular Target	Biological Outcome	Representative Herbal Compound
ROS scavenging	ROS	Reduced oxidative stress	Curcumin, EGCG, Aloe vera
Antioxidant activation	Nrf2/ARE	Increased antioxidant enzymes	Curcumin
Anti-inflammatory	NF-κB, COX-2	Reduced cytokine production	Curcumin, EGCG
Collagen protection	MMP-1, MMP-3, MMP-9	Reduced wrinkle formation	Curcumin, Green tea
DNA protection	CPDs, DNA repair pathways	Reduced mutagenesis	EGCG
Immunomodulation	IL-10, IL-12, Langerhans cells	Prevents UV-induced immunosuppression	EGCG
Moisturizing & Repair	Fibroblasts, collagen synthesis	Skin healing and hydration	*Aloe vera*

Mechanisms of Photoprotection

Unlike conventional sunscreens that primarily absorb or reflect UV radiation, green tea polyphenols protect the skin by modulating multiple biological pathways. EGCG scavenges reactive oxygen species (ROS), suppresses oxidative stress, inhibits UV-induced inflammatory signaling, and enhances endogenous antioxidant defenses. It also reduces DNA damage, preserves epidermal keratinocyte viability, and promotes DNA repair, thereby minimizing UV-mediated cellular injury [81, 82].

Anti-photoaging and Immunoprotective Effects

Green tea polyphenols effectively prevent photoaging by inhibiting matrix metalloproteinases (MMP-2, MMP-3, MMP-7, and MMP-9), reducing collagen degradation, and limiting protein and lipid oxidation. In addition, EGCG counteracts UV-induced immunosuppression by preserving Langerhans cell function,

inhibiting $CD11b^+$ macrophage infiltration, and regulating cytokine production through suppression of IL-10 and enhancement of IL-12, thereby maintaining cutaneous immune surveillance [83].

Anticarcinogenic Activity

Numerous experimental studies have demonstrated that green tea polyphenols inhibit multiple stages of UV-induced skin carcinogenesis, including initiation, promotion, and progression. EGCG selectively induces apoptosis in transformed cells while protecting normal keratinocytes from UV-mediated damage. It also suppresses angiogenesis by reducing the expression of vascular endothelial growth factor (VEGF) and matrix metalloproteinases, thereby limiting tumor growth and progression [84].

Formulation Advances

The incorporation of green tea polyphenols into topical sunscreen formulations has significantly improved their photoprotective performance. EGCG-containing formulations exhibit enhanced antioxidant activity, reduced UV-induced oxidative damage, improved photostability, and superior protection against photoaging. These multifunctional properties make green tea polyphenols promising natural bioactive ingredients for the development of advanced herbal sunscreen formulations. Herbal bioactive compounds protect the skin against ultraviolet (UV)-induced damage through multiple molecular and cellular mechanisms rather than acting solely as UV filters. Table 5 summarizes the principal molecular targets, underlying photoprotective mechanisms, and representative herbal compounds involved in mitigating oxidative stress, inflammation, DNA damage, and photoaging.

CONCLUSION

UV radiation remains one of the major environmental factors responsible for skin damage, contributing to oxidative stress, inflammation, photoaging, immunosuppression, and skin carcinogenesis. Although conventional sunscreens containing synthetic UV filters provide effective broad-spectrum photoprotection, concerns regarding skin irritation, photoallergic reactions, environmental toxicity, and long-term safety have stimulated the search for safer and more sustainable alternatives. In this context, herbal sunscreen formulations have emerged as promising candidates owing to their multifunctional biological activities and favorable safety profiles. Medicinal plants and their bioactive phytochemicals, including polyphenols, flavonoids, carotenoids, and phenolic acids, provide photoprotection through multiple complementary mechanisms. Beyond absorbing or attenuating UV radiation, these natural compounds neutralize reactive oxygen species, suppress inflammatory signaling pathways, enhance endogenous antioxidant defenses, promote DNA repair, preserve extracellular matrix integrity, and modulate immune responses. Herbal ingredients such as *Aloe vera*, curcumin, green tea polyphenols, and other botanical extracts have demonstrated significant efficacy in reducing UV-induced skin damage in both experimental and formulation studies.

Recent advances in nanotechnology-based delivery systems, including nanoemulsions, solid lipid nanoparticles, liposomes, and polymeric nanocarriers, have further improved the stability, skin penetration, controlled release, and photoprotective performance of herbal bioactives. These innovations offer new opportunities to develop highly effective and cosmetically acceptable sunscreen formulations while reducing reliance on synthetic UV filters. Despite encouraging preclinical findings, the clinical translation of herbal sunscreens requires standardized phytochemical characterization, rigorous safety assessment, large-scale clinical validation, and regulatory approval. Future research integrating herbal bioactives with advanced nanocarrier technologies is expected to facilitate the development of safe, effective, and environmentally sustainable next-generation sunscreen formulations for comprehensive protection against UV-induced skin disorders.

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CONFLICT OF INTEREST

The authors declare that they have no conflict of interest, financial or otherwise, related to this work.

AUTHOR'S CONTRIBUTION

[Diwakar Singh]: Conceptualization, literature survey, manuscript planning, and initial draft preparation.
[Dr. Abhishek Bhardwaj]: Scientific content organization, critical revision, and manuscript editing..
[Ms. Anchal Karwal]: Literature collection, manuscript review, and editing.
[Dr. Ashutosh Badola]: Literature analysis, interpretation of published findings, and manuscript drafting.

[Dr. Amandeep Singh]: Supervision, critical review of the manuscript, and final approval.
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[Dr. Esha Vatsa]: Literature analysis, interpretation of published findings, and manuscript drafting.

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