



UNDERSTANDING THE IMPACT OF UV RADIATION ON SKIN HEALTH: MECHANISMS, RISKS, AND PHOTOPROTECTION STRATEGIES

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Abstract

This comprehensive review examines the intricate relationship between UV radiation and skin health, delving into its mechanisms, associated risks, and potential photoprotection strategies. Drawing from centuries of herbal medicine and cosmetic practices, the efficacy of antioxidant-rich herbs in shielding against UV radiation-induced damage is highlighted. Studies showcasing the protective properties of plant extracts, such as green and black tea polyphenols and aloe vera formulations, underscore the potential of botanicals in skincare. The rise of herbal sunscreens in response to growing concerns over UV exposure reflects a paradigm shift in skincare practices.

The review elucidates the diverse effects of UV radiation on the skin, encompassing sunburns, premature aging, and skin cancer development. Mechanisms underlying UVB-induced skin damage, including collagen breakdown, generation of free radicals, and interference with DNA repair mechanisms, underscore the imperative of implementing protective measures. The role of antioxidants in mitigating UV-induced damage and the development of innovative sunscreen formulations underscores ongoing efforts to enhance photoprotection strategies. Furthermore, the review underscores the multifaceted nature of UV radiation's impact on skin health, emphasizing the importance of holistic approaches to photoprotection. Future research endeavors aimed at exploring novel photoprotective agents and assessing their efficacy in preserving skin health will be instrumental in mitigating the adverse effects of UV radiation and fostering overall skin well-being.

Keywords: UV Radiation, Herbal Medicine, Antioxidant, Cosmetics

1. Introduction

Centuries of herbal use in medicine and cosmetics highlight their effectiveness in treating skin issues and improving skin health. Herbs, rich in antioxidants like vitamins C and E, flavonoids, and phenolic acids, offer protection against UV radiation, known for causing sunburns, premature

aging, and skin cancer. Studies show that extracts from plants like green and black tea, containing polyphenols, can mitigate UV-induced damage. Similarly, aloe vera formulations aid in skin repair and cell regeneration[1,2,3].

The traditional use of plants in medicine and cosmetics drives ongoing research, expanding

cosmetic science. Concerns over human exposure to UV radiation have led to the popularity of herbal sunscreens, reflecting a shift in skincare practices. Sunscreens protect against UV radiation by absorbing, reflecting, or scattering solar rays, with their effectiveness measured by the Sun Protection Factor (SPF). Higher SPF values indicate better protection against sunburn.

UV radiation, part of the Sun’s electromagnetic spectrum, affects the skin significantly. The UV spectrum includes Vacuum UV, Far UV, UVC, UVB, and UVA, with UVA further divided into UVA I and UVA II. Solar UV radiation mainly consists of UVA and UVB, as the ozone layer absorbs UVC[4,5].

The strength of solar UVB radiation at a specific location depends on factors like the solar angle,

ozone levels, and altitude. These factors affect the amount of UV exposure people experience in various[6], 37 places.

A. UVC Radiation: UVC radiation is absorbed by the atmosphere and is mainly utilized in germicidal lamps for its antibacterial properties. Accidental exposure to UVC can cause corneal burns or “welders’ flash,” resulting in sunburn-like effects on the face[7].

B. UVB Radiation: UVB radiation causes photochemical damage to DNA and is partially absorbed by the atmosphere. It leads to sunburn, cataracts, and skin cancer, with concerns about ozone depletion potentially increasing skin cancer rates[8], 42.

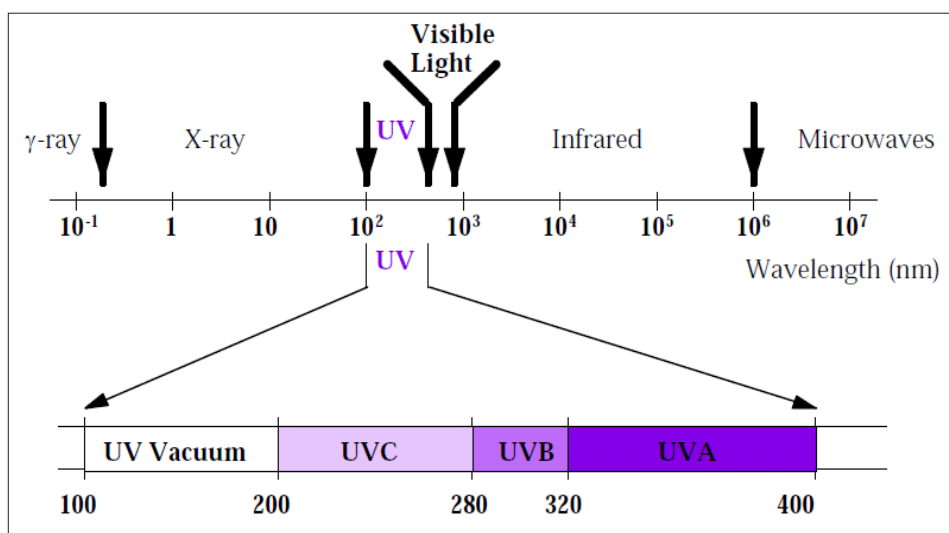


Figure 1: The ultraviolet (UV) component of the electromagnetic spectrum (A)

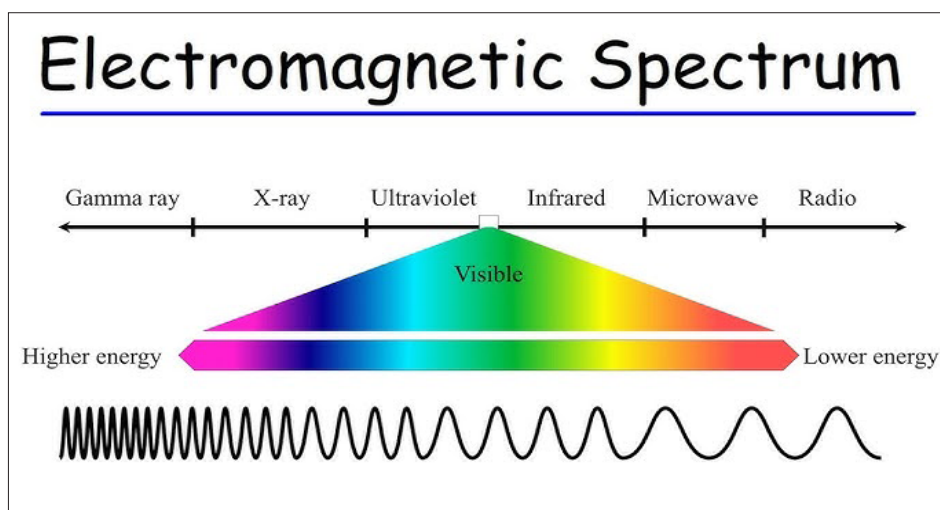


Figure 2: The ultraviolet (UV) component of the electromagnetic spectrum (B)

C. UVA Radiation: UVA radiation induces tanning and can lead to skin reddening and other health issues. It penetrates deeper into the skin and is linked to immune suppression, cataracts, and skin cancer development[9].

D. Skin Cancer: Melanoma, arising from melanocytes, poses a severe health risk due to its rapid spread. Non-melanoma skin cancer progresses slowly within the epidermis. UVB primarily affects the epidermis, causing sunburns, with peak intensity between 10:00 am and 2:00 pm[10,11,43].

2. Harmful Aspects of UVB Radiation

UVB radiation poses a significant risk of skin damage, contributing to conditions like wrinkles, aging-related skin disorders, and cancer. Mechanisms underlying UVB-induced skin damage include collagen breakdown, generation of free radicals, interference with DNA repair mechanisms, and suppression of the immune system against infections. UVB exposure stimulates the production of reactive oxygen species (ROS), which inflict harmful effects on the skin, as outlined by García et al. in 2008[12,13,14].

The intensity of Ultraviolet B (UVB) rays varies with time and season and is chiefly responsible for sunburns, a leading risk factor for both melanoma and non-melanoma skin cancers. Protective measures against UVB exposure include the use of broad-spectrum sunscreen formulations[14,15].

When UV radiation is absorbed by the skin surface, it generates harmful compounds known as free radicals or reactive oxygen species (ROS), contributing to skin cancer and premature aging. ROS derived from UVB radiation trigger oxidative decomposition, resulting in the formation of toxic components and lipid peroxidation, as elucidated[16,17].

UVB rays can also inflict protein damage, lipid peroxidation, and skin lesions. Lipid peroxidation occurs when free radicals strip electrons from lipids in cell membranes, leading to cellular damage. ROS degrade unsaturated lipids, forming malondialdehyde

(MDA), a marker enzyme of lipid peroxidation. Assessment of antioxidant enzyme levels, including superoxide dismutase (SOD), reduced glutathione (GSH), catalases (CAT), ascorbic acid (ASC), and total protein (TP), provides insights into skin tissue health[18,19,44].

Furthermore, ROS generated from UVB radiation induce structural alterations in the skin, such as erosion of the epidermis, changes in epidermal thickness and organization, fibrinoid formation, edema, and irregular collagen fibril arrangement. Deoxyribonucleic acid (DNA) readily absorbs UVB radiation, leading to molecular conformational changes, as observed[20,21].

Thus, it is essential to make skin protected from harmful effects of UVB radiation.

2.1 Sunburn (Erythema)

Sunburn, also known as erythema, occurs when the skin turns red due to increased blood flow caused by the widening of superficial blood vessels in the dermis after exposure to UV radiation. Among UV rays, UVB radiation is considered the primary culprit for sunburn, being approximately 1,000 times more effective at inducing erythema. For individuals with fair skin, it takes just 15 to 30 minutes of mid-day sun exposure to trigger erythema. Certain areas of the body, such as the face, neck, and trunk, are two to four times more prone to sunburn compared to the limbs[22,23].

2.2 Tanning

Tanning is the process of developing delayed pigmentation, or melanin pigmentation, in the skin. It typically becomes visible one to two days after sun exposure and continues to deepen over several days, lasting for weeks or even months. Tanning occurs due to heightened activity in melanocytes, the pigment-producing cells, which results in increased production of the enzyme tyrosinase. This enzymatic activity leads to the creation of new melanin and an augmentation in the number of melanin granules dispersed throughout the epidermis[24].

2.3 Premature Aging of the Skin

Repeated exposure to UVB radiation can lead to premature aging of the skin, characterized by various clinical signs reflecting structural alterations in both the epidermis and dermis. These signs encompass dryness, wrinkles, pronounced skin furrows, sagging, reduced elasticity, and uneven pigmentation, all stemming from degenerative changes in elastin and collagen. Compared to UVA, UVB radiation is significantly more potent, being 1,000 to 10,000 times more effective in inducing sunburn and nonmelanoma skin cancer, respectively. Consequently, premature aging of the skin is strongly associated with UVB exposure[25,38].

2.4 Skin Cancer

Various types of skin cancer exist, including nonmelanoma skin cancers such as basal cell carcinoma (BCC) and squamous cell carcinoma (SCC), as well as melanoma. UVB radiation exposure is considered a significant contributor to the development of each of these cancers due to its ability to cause DNA damage. However, the nature of exposure required to trigger different types of skin cancer may differ. For nonmelanoma skin cancers, cumulative sun exposure over time is believed to play a crucial role. In contrast, for melanoma, the intermittent exposure hypothesis has been proposed, suggesting that sporadic, intense exposure to UV radiation may be more closely linked to its development[26,27,28].

2.5 Damage to the Eyes

UV rays pose a threat to eye health, with over 99% of UV radiation absorbed in front of the eyes. Chronic exposure to UV radiation can result in various eye conditions including corneal damage, cataracts, and macular degeneration, potentially leading to blindness. Additionally, melanoma, a form of skin cancer, can manifest within the eye. The risk of intraocular melanomas is notably higher in individuals of Caucasian descent, with an eightfold greater risk compared to individuals of African descent[29,49].

2.6 Suppression of the Immune System

Exposure to UV radiation is known to suppress the immune system, significantly contributing to the development of nonmelanoma skin cancers. This immunosuppressive effect of UV radiation hinders the rejection of tumors by inducing a state of relative immunosuppression. UV radiation disrupts the normal function of antigen-presenting Langerhans cells in the epidermis, which are responsible for activating T-lymphocytes against foreign antigens. While these cells are altered by UV exposure, similar cells involved in the induction of suppressor lymphocyte pathways remain resistant to UV damage. This imbalance in T-cell function, shifting from helper to suppressor pathways, creates a favorable environment for tumorigenesis and cancer progression.

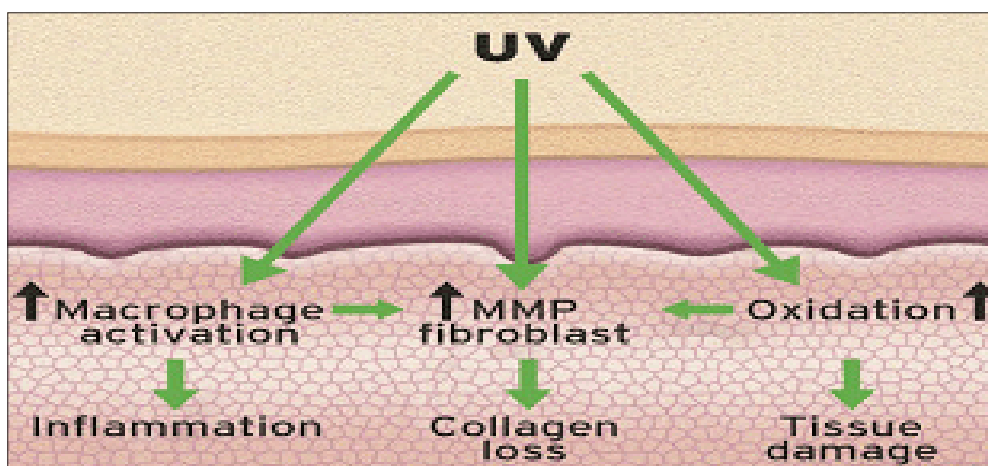


Figure 3: A schematic illustration of UV-induced inflammation, collagen loss and tissue damage

Research has shown promising results in preventing UVB-induced immunosuppression in mice through the use of green tea polyphenol (-)-epigallocatechin-3-gallate, which may alter interleukin IL-10 and interleukin IL-12 production.

Additionally, exposure to UV radiation increases the level of matrix metalloproteinases (MMP) in fibroblasts, leading to collagen loss. Terminalia catappa L. leaves have been found to enhance the production of type I procollagen by inhibiting the activity of MMP-1, -3, and -9. This property has made them valuable in anti-aging cosmetics[30,31,45].

2.7 Cell Death

The immune system's final line of defense is apoptosis, a process of programmed cell death that eliminates severely damaged cells, thus preventing them from becoming cancerous. UVB radiation triggers cell death by increasing the production of free radicals, activating apoptotic pathways, and disrupting mitochondrial membrane potential.⁴⁶

However, certain factors, including UVB exposure, can interfere with this cell death process, allowing damaged cells to persist and potentially develop into cancer. Researchers have found that the p53 gene encodes signaling molecules that play a crucial role in inducing cell cycle arrest and initiating cell death, thereby safeguarding against the formation of cancerous cells[32,33,48].

2.8 Collagen Breakdown

UVB radiation in the dermis accelerates the breakdown of collagen, surpassing the rate of breakdown observed with natural aging alone. Sunlight exposure damages collagen fibers and leads to the accumulation of abnormal elastin. This accumulated elastin triggers the production of large quantities of enzymes called metalloproteinases. Typically, metalloproteinases aid in remodeling sun-injured skin by synthesizing and restructuring collagen. However, this process isn't always efficient, and some metalloproteinases actually degrade collagen. Consequently, disorganized collagen fibers, known as solar scars, form. With

repetitive exposure to sunlight and imperfect collagen rebuilding, wrinkles gradually develop[34,39].

2.9 Free Radicals

UV radiation is a primary source of free radicals, unstable oxygen molecules commonly referred to as "free radicals." These molecules possess only one electron instead of the usual pair, rendering them highly reactive. To stabilize themselves, free radicals scavenge electrons from other molecules, initiating a chain reaction. This process, known as oxidative stress, can impair cellular function and damage genetic material, including RNA and DNA. Free radicals contribute to wrinkle formation by activating metalloproteinases, which degrade collagen, and can lead to cancer by altering the genetic material of cells[35,47].

To mitigate the generation and damage caused by reactive oxygen species (ROS), researchers advocate for the use of sunscreen to shield the skin from harmful UVB radiation. Additionally, advancements in photoprotection may involve the development of sunscreens designed to linger on the skin's surface for extended periods and incorporate antioxidants capable of neutralizing ROS. By capturing free radicals, antioxidants may enhance the effectiveness of photoprotection. As such, ongoing research aims to explore photoprotection against UVB radiation and assess its impact on biochemical parameters and histological changes in skin tissues[36,40].

3. Conclusion

In conclusion, this review underscores the multifaceted impact of UV radiation on skin health, elucidating its mechanisms, associated risks, and potential photoprotection strategies. UV radiation, comprising UVA and UVB rays, poses significant threats to skin integrity, ranging from sunburns and premature aging to skin cancer development. The deleterious effects of UVB radiation on the skin, such as collagen breakdown, generation of free radicals, and interference with DNA repair mechanisms, highlight the importance of implementing protective measures against sun exposure. Moreover, the role

of antioxidants, herbal extracts, and innovative sunscreen formulations in mitigating UV-induced damage underscores the ongoing efforts to enhance photoprotection strategies. Future research aimed at exploring novel photoprotective agents and assessing their efficacy in preserving skin health will be pivotal in combating the adverse effects of UV radiation and promoting overall skin well-being.

Conflict of Interest: None

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