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Review Article

Effects of visible lights, photodermatoses and role of antioxidants in skin health

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ABSTRACT

Visible light (VL) radiation poses potential risks to the skin, including solar urticaria, chronic actinic dermatitis (CAD), cutaneous porphyrias, and others. Photodermatoses, another skin condition, can be worsened by exposure to light. Involves the production of reactive oxygen and nitrogen species (ROS and RNS, respectively), which harms proteins, lipids, and DNA results in an inflammatory reaction and increased skin pigmentation. Studies show that plant-derived antioxidants can shield VIS-exposed skin from oxidative damage brought on by ROS. Both API (Active pharmaceutical ingredients) and BTC (Bis trichloromethyl carbonate) give protection against harm caused by VL and useful topical antioxidants that can be added to sunscreens in terms of sun protection techniques, whereas, vitamins A, C, and E are antioxidants that reduce the aging process by preventing free radicals from oxidizing sensitive biological components. With an emphasis on either VL blocking (tinted sunscreens) or production of reactive species and radical quenching (antioxidant sunscreens), availability of photoprotection products that contain VL protection is expanding. Promising advancements have been made in incorporating antioxidants and radical scavengers into sunscreen formulations to address the induction of ROS/RNS by visible light. Topical application of an antioxidant blend containing varying concentrations of a singlet oxygen quencher along with fixed concentrations of vitamin E (0.25%) and vitamin C (0.01%) has shown ability to inhibit erythema and reduce pigmentation in certain skin types. Recent advancements in understanding VL's ability to induce reactive species have paved the way for antioxidant-based formulations, which offer promising alternatives for photoprotection across all skin types.

Keywords: Photodermatoses, Antioxidants, ROS, VL

INTRODUCTION

Until recently, UV radiation remained the center of study for dermatologists as it is one of the most important modifiable risk factors for skin cancer and causes severe implications than many other environmentally-influenced skin disorders. Lately, there have been more studies on visible light as studies figured that exposure to solar radiation produce abnormal skin reaction-phototoxicity. Phototoxicity is a harmful response that occurs when certain chemicals in the skin are activated by sunlight, leading to cytotoxic effects on the skin cells. Visible light radiation has the potential to cause various biological effects on the skin, including erythema (redness), pigmentation changes, thermal damage, and production of free radicals.[1] In addition, exposure to visible light can also trigger or worsen conditions such as solar urticaria (hives), CAD, and cutaneous porphyrias, which are known as photodermatoses. Acute exposure to visible light gives birth to photodermatoses (inflammatory skin reactions induced photochemically in exposed areas) which leads to the activation of melanogenesis (pigment

production) and subsequent hyperpigmentation of the skin. VL has been observed to cause temporary or permanent darkening of human skin. Research has shown that the pigmentation caused by VL can last for several weeks, and the intensity of the pigmentation depends on the amount of light exposure. VL on skin may spark pigment changes, especially in darker tones, and worsen skin conditions related to light sensitivity and pigmentation. Some findings revealed that a solitary encounter with visible light caused minimal pigmentation, but repeated exposures led to deeper and long-lasting changes in skin coloration.[2]

VL can aggravate pigmentation disorders like melasma and post inflammatory hyperpigmentation (PIH), despite using sunblock regularly to shield against UVA/UVB rays, leading to relapses in patients' condition. VL triggers photodermatoses, including cutaneous porphyrias.[3-5] Photodermatoses, which refer to skin conditions triggered by light, can be worsened by exposure to light. When a group of photosensitive disorders manifests together, it is referred to as idiopathic photodermatoses, and the exact cause of these conditions is not yet fully understood. However, many of these disorders are believed to involve an immune-mediated response. Examples of idiopathic photodermatoses include polymorphous light eruption (PMLE), solar urticaria, and others. PMLE, known as "sun poisoning" or "sun allergy," is the most common idiopathic photodermatoses.[6,7]

UNDERSTANDING VISIBLE LIGHT COMPOSITION & ITS IMPACT

Photodermatoses are a group of skin conditions caused by light exposure. Unfortunately, many of the available sunscreens don’t offer adequate protection against visible light, leaving people vulnerable to increased risk of skin damage. Photodermatoses can be classified into five general categories-idiopathic, most likely immune-mediated; secondary to exogenous agents; secondary to endogenous agents; photo exacerbated dermatoses; and genodermatoses. Photodermatoses can have an action spectrum in the UVB, UVA, and/or visible light range, with UVA being the most common. UVB radiation, which falls within the wavelength range of 290-320 nm, is primarily responsible for causing skin redness (erythema), whereas UVA radiation, ranging from 320-400 nm, is implicated in the development of most photodermatoses.[5]

Those with action spectrum in the visible light range are solar urticaria, CAD, and cutaneous porphyrias. The damaging effects of VL on skin cells are considerably amplified by endogenous photosensitizers (ePS). Endogenous photosensitizers (ePS) are a broad set of molecules that can absorb ultraviolet radiation (UVR) ranging from 200 to 400 nm, as well as VL spanning from 400 to 750 nm. These molecules can transfer energy or electrons to nearby molecules when they absorb light and become electrically excited.[5]

Electronically excited states, such as triplet excited states, singlet oxygen, and numerous reactive oxygen and nitrogen species, such as free radicals and two-electron oxidants, are created during sunlight-induced excitation of ePS in human skin. In the presence of melanin and lipofuscin (ePS) pigments, VL induces significant oxidative damage in nucleic acids, lipids, and proteins, triggering regulated and unregulated cell death mechanisms. Light photons that interact with our skin in the visible and infrared (IR) regions have similar effects on human skin to UVA (ultraviolet A) radiation. This method involves the production of ROS and RNS, respectively, which can harm proteins, lipids, and DNA. This can result in an inflammatory reaction and increased skin pigmentation.[1]

EFFECTS OF VISIBLE LIGHT ON SKIN HEALTH

The formation of ROS, proinflammatory cytokines, and matrix metalloproteinase (MMP)-1 expression were all stimulated by visible light irradiation of human skin analogs. UVA/UVB sunscreens may not shield the skin from reactions brought on by visible light, according to research that shows they have no impact in lowering visible light-induced ROS. Visible light can trigger a surge of ROS, which sparks the release of proinflammatory cytokines and MMP (matrix metalloproteinases) expression. With our skin constantly exposed to visible light for prolonged periods, and containing multiple chromophores for visible light, the cumulative effects could lead to skin damage, potentially accelerating premature skin aging.[8]

Our bodies have built-in superpowers to fight off harmful radicals! With a team of antioxidant enzymes like SOD, catalase, and glutathione peroxidase, as well as non-enzymatic superheroes like glutathione, vitamin C, and vitamin E, human bodies can neutralize those pesky free radicals.[9] But sometimes, the harmful radicals can outnumber our defences, leading to oxidative stress. Oxidative stress seems to play a significant role in promoting melanogenesis, particularly when triggered by exposure of the skin to UVR and VL. The entire VL spectrum, including near-infrared radiations, can generate free radical species that contribute to oxidative stress.[10]The resulting oxidative damage can worsen skin pigmentation and aging, leading to changes in skin tone consistency, increased wrinkles, sagging, dryness, and roughness. Oxidative stress has the potential to induce chronic inflammation, which can disrupt the structural integrity of collagen fibers and impair normal skin cell functions, thus contributing to the development of various skin diseases, including cancer.[11]

Exogenous factors that trigger oxidative stress, such as UVR and VL, can initiate different pathways leading to pigment formation, but ultimately share a common

outcome-the possibility of undesirable hyperpigmentation, which is of cosmetic concern.[10] Despite the availability of organic and inorganic sunscreens for protecting against UVR, there is currently inadequate coverage to safeguard against the VL portion of the electromagnetic spectrum. Among VL wavelengths, shorter ones have been shown to be more biologically effective in triggering hyperpigmentation compared to UVA or UVB. Furthermore, hyperpigmentation induced by VL has been observed to be darker and more persistent. VL stimulates tyrosinase and dopachrome tautomerase to form a protein complex that leads to sustained tyrosinase activity, resulting in clinically visible pigmentation in individuals with higher melanin levels in their skin (skin phototypes III-VI).

Visible light radiation can be partially reflected from the outer surface of the skin and eyes. As this radiation penetrates through the tissue, it can scatter in different directions, including backwards, due to microscopic particles and structures such as fibers in the dermis of the skin. Additionally, the radiation can be absorbed by various molecules in the tissue. While visible radiation is generally not strongly absorbed by the bulk tissue compared to UV and long-wavelength IR radiation, it is highly absorbed by specific components such as pigments and blood. The combined effect of backscattered and absorbed visible radiation ultimately determines the color of the skin.[12]

The most noticeable and rapid alteration is noticed on skin pigmentation after exposure to natural light. There have been reports that different visible spectrum wavelengths can change skin colour. In the realm of skin physiology, visible light has been discovered to have a unique effect on the enzymes tyrosinase and dopachrome tautomerase.[10] When exposed to visible light, these enzymes come together to form a dynamic protein complex that triggers a prolonged increase in tyrosinase activity. This phenomenon ultimately results in the manifestation of visible pigmentation in skin types that are most proficient in melanin production, commonly known as melanocompetant skin types.[13]

CRUCIAL ROLE OF ANTIOXIDANTS IN PRESERVING SKIN HEALTH AND PREVENTING DAMAGE

In the field of medicine, both natural and artificial sources emit VL. Various types of devices, such as lasers, light-emitting diodes (LEDs), arc/flash lamps, halogen lamps, and fluorescent lights, are used to deliver VL as a therapeutic modality. The way light propagates and penetrates the skin depends on reflection, scattering, and absorption. Around 4% to 7% of VL is reflected by the skin surface, regardless of factors such as wavelength, pigmentation, or structure. Keratins, collagen, melanin, and hemoglobin are the main molecules in the skin responsible for VL penetration through scattering and absorption, while other substances like zinc, ion-gated channels, NADH, bilirubin, and b-carotene also absorb and scatter VL. Filamentous proteins in the skin are excited by photons, leading to scattering, with epidermal scattering potentially being greater than dermal scattering due to melanin in the epidermis.

In the skin, chromophores selectively absorb specific wavelengths of light with different affinities, as determined by their absorption coefficient. Melanin, heme, and opsin (OPN) photoreceptors are the primarily VL skin chromophores. According to the theory of selective photothermolysis, the pulse duration of the light must be shorter than the tissue's thermal relaxation time to avoid nonspecific thermal damage. Light energy parameters that do not result in photothermolysis or thermal damage, but still modify biological function, are classified as photobiomodulation.[14, 15]

In contrast to chronological aging, which is determined by an individual's inherent physiological factors, photoaging is largely influenced by the extent of sun exposure and the level of melanin in the skin. Sunlight generates ROS that can cause skin damage. While UV filters are effective in reducing ROS induced by UV radiation, they are unable to counteract oxidative stress induced by visible light (400-760 nm). As a result, the incorporation of powerful antioxidants as additives in sunscreen products is essential to mitigate the harmful effects of oxidative stress caused by visible light exposure.[16]

There is evidence that plant-derived antioxidants can shield VIS-exposed skin from oxidative damage brought on by ROS. Both API (Active pharmaceutical ingredients) and BTC (Bis trichloromethyl carbonate) give protection against harm caused by visible light and may be useful topical antioxidants that can be added to sunscreens in terms of sun protection techniques. Since the majority of damages brought on by visible light are ROS-mediated, antioxidants are essential to offer comprehensive protection.[17] Vitamins A, C, and E are antioxidant chemicals that reduce the ageing process by either preventing free radicals from oxidising sensitive biological components or by lowering free radical generation and quenching the previously produced ROS. Antioxidants that are primary or free radical scavengers prevent oxidation by chain-terminating reactions.[18] Proton transfer to the species of free radicals causes inhibition. Alpha-tocopherol, vitamin E, ascorbic acid, and vitamin C are a few examples of primary antioxidant compounds. While vitamin E is membrane-bound and able to thwart chain reactions caused by free radicals, GSH and ascorbic acid are water-soluble antioxidants. Additionally, they can replenish one another, resulting in synergistic combinations when used topically.[19,20]

Antioxidant enzymes, including GSH peroxidases, GSH reductase, glutathione S-transferases (GSTs), SODs, catalase, and quinone reductase, are part of a group of enzyme systems that help regenerate antioxidants and

directly neutralize ROS. To enhance the antioxidant protection of the skin, it is possible to boost the activities of these enzyme systems by providing them with essential metal cofactors like Cu, Mn, Zn, and Se. Topical treatments can use a range of antioxidants. To boost the antioxidant systems in the skin, topically applied enzymes that replenish these antioxidants, such as GSH peroxidases, GSH reductases, and GSTs, as well as enzymes that neutralize ROS, including SODs, catalase, and quinone reductase, are also utilized.[21, 22]

In long-term trials lasting between 3 and 6 months, the impact of antioxidant compositions on preventing chronological aging/reversing the signs of aging can be assessed. A powerful antioxidant, vitamin E prevents the creation of ROS molecules during the oxidation of lipids and the spread of the free radical process. Vitamin E can protect phospholipids and fatty acids in the phospholipids that make up the skin's membrane, and recent studies have shown that it can prevent DNA damage from oxidative stress and UV-Ae-induced cyclobutene pyrimidine dimers in keratinocytes. [23]

Since antioxidants scavenge ROS and limit the growth of damaging chain reactions, their treatment would prevent these biological effects. The ideal antioxidants should be non-toxic, demonstrate penetration in the skin, and have strong UV absorption but the adequate stability to prevent turning into the photosensitizers after the light absorption. [23]

FUTURE SCOPE IN ANTIOXIDANTS TREATING SKIN DISORDERS

With an emphasis on either VL blocking (i.e., tinted sunscreens) or the production of reactive species and radical quenching (i.e., antioxidant sunscreens), the availability of photoprotection products that contain VL protection is expanding. The currently available inorganic or physical filters are based on the ability of naturally occurring minerals (such as titanium dioxide, zinc oxide, and iron oxide) to reflect and scatter VL when the particle sizes are greater than 200 nm; however, the large-sized particles produce a lingering whitish appearance on the skin, making them unattractive from a cosmetic standpoint.

In a recent study, the use of tinted organic sunscreen following UV-A1 irradiation resulted in a significant decrease in hyperpigmentation clinical scores at day 7, when compared to an untreated control group. Promising advancements have been made in incorporating antioxidants and radical scavengers into sunscreen formulations to address the induction of ROS/RNS by visible light. Topical application of an antioxidant blend containing varying concentrations of a singlet oxygen quencher (diethylhexyl syringylidene malonate at 1% and 2%) along with fixed concentrations of vitamin E (0.25%) and vitamin C (0.01%) has shown the ability to inhibit erythema and reduce pigmentation in certain skin types.

CONCLUSION

Antioxidants used topically may enhance the skin's natural defense mechanisms. The photoprotective effect of UV filters can also be improved by topical antioxidant administration. Antioxidant-active cosmetic additives can help UV filters stay stable and prevent damage from free radicals. The natural world is one of the sources being considered for new antioxidant chemicals to integrate into cosmetic compositions. Due to their polyphenolic compositions, plant chemicals have long been recognized as powerful antioxidants. Multifunctional compounds are a requirement in the creation of new cosmetic goods due to the ongoing innovation in the cosmetic business.

The existing body of knowledge regarding the impact of VL on skin health, including erythema, photodermatoses, hyperpigmentation, and melasma, particularly in individuals with Fitzpatrick skin types IV-VI, is growing. However, current photoprotection options for both UV and VL are limited to tinted sunscreens, which are often unappealing cosmetically and underutilized. Addressing this gap in protection, the development of new filters that cover both UV and VL ranges holds potential. Furthermore, recent advancements in understanding VL's ability to induce reactive species have paved the way for antioxidant-based formulations, which offer promising alternatives for photoprotection across all skin types.

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