Jakub Graca 0009-0007-7958-8925

Silesian Medical University in Katowice, Mickiewicza 29 St., 40-001 Katowice, Poland
+48 535 802 037, kubagraca9900@op.pl

Sposób cytowania / Cite Graca J. The effect of blue light on the scalp and hair follicles. *Aesth Cosmetol Med*. 2025;14(4):167-173. https://doi.org/10.52336/acm.2025.024

The effect of blue light on the scalp and hair follicles

Wpływ światła niebieskiego na skórę głowy i mieszki włosowe

ABSTRACT

Solar radiation and blue light, which is high-energy visible light, penetrate deep into the scalp, causing oxidative stress, inflammation, hair growth cycle disorders, and also cell damage. This leads to hair loss, dry skin, microbiome dysbiosis, and photoaging.

The article aimed to analyze the potential biological mechanisms associated with the effects of blue light on the scalp and hair follicles. In particular, the study focused on oxidative stress, morphological changes in hair follicle cells, and the consequences of long-term exposure, which may affect regeneration processes and disrupt the natural hair growth cycle.

Protective strategies, including antioxidants, filters, and limiting exposure to light, are of key importance.

Keywords: blue light, HEV, scalp dysbiosis, oxidative stress, scalp, hair follicles

STRESZCZENIE

Promieniowanie słoneczne oraz światło niebieskie, czyli widzialne światło o wysokiej energii, przenikają w głąb skóry głowy, wywołując stres oksydacyjny, stany zapalne, zakłócenia w cyklu wzrostu włosów, a także uszkodzenia komórek. Prowadzi to do wypadania włosów, przesuszenia skóry, dysbiozy mikrobiomu i fotostarzenia.

Celem artykułu była analiza potencjalnych mechanizmów biologicznych związanych z oddziaływaniem światła niebieskiego na skórę głowy i mieszki włosowe. W szczególności związanych ze stresem oksydacyjnym, zmianami morfologicznymi w obrębie komórek mieszków włosowych oraz możliwymi konsekwencjami długotrwałej ekspozycji, które mogą wpływać na procesy regeneracji i zaburzać naturalny cykl wzrostu włosa.

Kluczowe znaczenie mają strategie ochronne, w tym antyoksydanty, filtry oraz ograniczenie ekspozycji na światło.

Słowa kluczowe: światło niebieskie, HEV, dysbioza skalpu, stres oksydacyjny, skóra głowy, mieszki włosowe

INTRODUCTION

The modern lifestyle involves prolonged exposure to light emitted by electronic devices, including computers, smartphones, tablets, and monitors with built-in light-emitting diodes (LEDs). A key component of this radiation is high-energy visible light (HEV), which covers a wavelength range from approximately 400 to 500 nanometers, primarily blue and violet light. Unlike ultraviolet (UV) radiation, HEV light does not cause direct damage to deoxyribonucleic acid (DNA). Still, its ability to penetrate the epidermis layers and generate reactive oxygen species (ROS) is of interest in the context of its impact on skin structures.

To date, scientific research has mainly focused on the effects of HEV radiation on facial skin, highlighting the induction of oxidative stress, epidermal barrier dysfunction, and accelerated aging processes. The impact of blue light on the circadian rhythm of skin regeneration and alterations in the expression of genes involved in cellular homeostasis have been documented in the literature. However, much less attention has been paid to the scalp.

Hair follicles, as dynamic anatomical units, are characterized by high mitotic and metabolic activity, which makes them potentially susceptible to stress-inducing environmental factors. The presence of hair follicle stem cells, keratinocytes, and fibroblasts within the scalp may constitute a potential point of interaction with HEV light, enabling its adverse effect on the structures responsible for hair regeneration and growth, leading to hair growth cycle disorders, weakened proliferative activity, and possible damage to the microcirculation around the follicles. These observations are significant in the context of increased exposure to LED light sources in indoor environments, where the distance between the skin and the screen is often minimal and contact is prolonged.

BLUE LIGHT: DEFINITION, CHARACTERISTICS AND SOURCES

Blue light, also referred to as HEV radiation, covers wavelengths ranging from 400 to 500 nm, with the highest photobiological intensity between 415 and 455 nm [1]. The main source of blue light is solar radiation. HEV constitutes a significant portion of daylight, and its intensity varies depending on the time of day and weather conditions. The presence of clouds can increase the proportion of blue light in the radiation reaching the Earth's surface – this phenomenon is known as the blue enhancement effect [2]. As a result, even on cloudy days, exposure to HEV can be high, especially in the morning and afternoon. Nowadays, a large part of the exposure to blue light comes from artificial sources, mainly LEDs, which are widely used in digital device screens (smartphones, tablets, laptops, monitors) and modern lighting. LED technology based on white light generates a relatively high proportion of blue light [3].

Studies have shown that even one hour of skin contact with a digital device screen can lead to an increase in ROS and induce apoptosis and necrosis in skin cells [4]. HEV emitted by smartphones or tablets, especially in the evening (so-called artificial light at night (ALAN)), disrupts biological rhythms and may potentially increase the risk of certain chronic diseases such as type II diabetes, breast cancer, metabolic or mental disorders [5].

Due to its high energy and short wavelength, blue light has a deeper penetration capacity than other visible light bands and can reach the retina, where it can potentially cause photochemical damage. Exposure to HEV activates oxidative stress in the retina and may contribute to DNA damage and metabolic disorders in retinal pigment epithelium (RPE) cells [1]. This mechanism results from the interaction of blue light with melanopsin-containing photoreceptors located in the retina. These cells are particularly sensitive to light with a wavelength of approximately 480 nm and send signals to the suprachiasmatic nucleus (SCN) in the hypothalamus. Under the influence of light, the SCN inhibits the secretion of melatonin by the pineal gland. Melatonin, known as the sleep hormone, is responsible for initiating and maintaining sleep, so its reduced concentration in the evening can result in difficulty falling asleep, shallow sleep, and frequent waking during the night [6]. Studies have shown that even short-term exposure to blue light in the evening, such as using a smartphone or laptop for 1-2 hours, can significantly delay the melatonin secretion phase. In one experiment, a shift in the circadian rhythm of 1.3 hours on average was observed after four hours of exposure to artificial light enriched with the blue spectrum at an intensity of 1000 lux [6]. The long-term consequences of such a rhythm include not only sleep disturbance, but also an elevated risk of depression, reduced immunity, and metabolic disorders. People exposed to increased blue light at night (e.g., shift workers, frequent travellers across time zones, children and adolescents who use electronics in the evening) have been found to have a higher incidence of sleep problems and impaired cognitive function during the day [5].

EFFECTS OF HEV ON THE SCALP AND HAIR FOLLICLES

The simplified hair growth cycle consists of three main phases: anagen (active growth phase), catagen (transitional phase), and telogen (resting phase). The correct proportions between these phases are essential for maintaining the physiological level of scalp hair. Experimental data suggest that exposure of the scalp to blue light, especially in chronic or evening conditions, leads to augmented production of ROS in skin cells and hair follicles [7]. The increase in ROS activates inflammatory pathways dependent on transcription factors such as nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB), which induces the secretion of pro-inflammatory cytokines such as tumor necrosis factor alpha (TNF-α) and interleukin 1 beta (IL-1β). Inflammation within the hair follicle disrupts its function and leads to premature termination of the anagen phase. Hair papilla cells and anagen keratinocytes under oxidative stress may undergo senescence or apoptosis, resulting in accelerated transition of the hair into the telogen phase [8]. This results in a phenomenon known as telogen effluvium – excessive hair loss associated with an abnormally prolonged telogen phase or premature termination of the anagen phase. People with existing oxidative stress or epidermal barrier disorders,

such as seborrheic dermatitis, scalp psoriasis, or atopic dermatitis, are particularly susceptible to this mechanism. Skin homeostasis is disturbed in these individuals, and excessive exposure to HEV can exacerbate pro-inflammatory processes and increase hair loss [9].

Exposure of the skin to blue light leads to the generation of ROS, which cause damage to cell membrane lipids, structural proteins, and DNA in the epidermis and dermis. *In vitro* studies have shown that just one hour of exposure of human skin cells to HEV light emitted by electronic screens causes an increase in ROS production, leading to apoptosis and cell necrosis [4]. Blue light-induced oxidative stress disrupts the functions of the epidermal barrier, leading to elevated transepidermal water loss (TEWL) and a decrease in protective lipids such as ceramides and cholesterol. This results in dryness, increased reactivity, and susceptibility to irritation and microinflammation of the scalp [4]. Disruption of the scalp microenvironment, including lipid barriers, pH, and stratum corneum structure, caused by exposure to blue light affects the composition and function of the scalp microbiome. HEV induces oxidative stress, which leads to skin dryness, epidermal cell damage, and a reduction in surface lipids, creating favorable conditions for colonization by opportunistic microorganisms [4].

One of the main opportunistic microorganisms which population can grow excessively under such conditions is *Malassezia furfur*, a lipophilic yeast-like fungus that is part of the physiological microflora of the scalp. Under dysbiosis conditions, *Malassezia* becomes pathogenic, leading to the development of seborrheic dandruff, seborrheic dermatitis, and *Malassezia folliculitis,* i.e., inflammation of the hair follicles [10].

In a retrospective clinical study of nonscarring scalp folliculitis (NSSF), as many as 96% of patients had cytologically confirmed *Malassezia* spores, and 79% responded positively to antifungal treatment, emphasising the role of this pathogen in folliculitis [10].

Inflammation caused by excessive *Malassezia* colonization involves the secretion of cytokines such as IL-1β and TNF-α, which disrupt the activity of hair follicle papilla cells, leading to their miniaturization and transition to the telogen phase. This process not only contributes to hair loss, but can also lead to permanent loss of hair follicle function, especially in individuals predisposed to androgenetic or scarring alopecia [11].

Comparative studies have also shown that the presence *of Malassezia* and its hyphae in the scalp was significantly higher in patients with androgenetic alopecia than in healthy individuals. Furthermore, antifungal treatment led to a reduction in fungal load and, at the same time, inhibition of the hair loss process [11].

DNA analysis of the scalp microbiome at different stages of seborrheic dermatitis showed a predominance of *Malassezia globosa* and *Malassezia restricta* species, which are particularly associated with the presence of scales, sebum overproduction, and the severity of inflammatory lesions [12].

POTENTIAL CLINICAL EFFECTS OF EXCESSIVE EXPOSURE TO BLUE LIGHT ON THE SCALP AND HAIR

Radiation with a wavelength in the range of 400-500 nm penetrates deep into the dermis, where it is absorbed by endogenous chromophores such as porphyrins, flavins, and nicotinamide adenine dinucleotide (NADH), which leads to the excitation of their energy state and the generation of ROS, including hydrogen peroxide (H_2O_2), superoxide anion (O_2^-) and hydroxyl radical (•OH) [13]. ROS, acting as strong oxidizing agents, lead to damage of lipids in cell membrane (lipid peroxidation), denaturation of structural proteins, and DNA fragmentation, resulting in disruption of cellular homeostasis and initiation of an inflammatory response in keratinocytes and fibroblasts [13]. Oxidative stress activates mitogen-activated protein kinase (MAPK) signalling pathways, such as extracellular signal-regulated kinases (ERK), c-Jun N-terminal kinases (JNK) and p38 mitogen-activated protein kinase, which then phosphorylate transcription factors: activator protein-1 (AP-1) and NF-κB, leading to their translocation to the cell nucleus, where they activate the expression of genes encoding extracellular matrix metalloproteinases (MMPs). In particular, it leads to overexpression of MMP-1 (collagenase 1), which degrades type I and III collagen – the basic structural proteins of the skin, MMP-3 (stromelysin 1), which breaks down other matrix components such as proteoglycans and laminin, and activates other MMPs, as well as MMP-9 (gelatinase B), which is responsible for the degradation of type IV collagen and gelatin. The resulting degradation of collagen and elastin fibers causes a loss of integrity of the extracellular matrix (ECM), weakening of the skin structure, reduction of its elasticity and flexibility, and the appearance of wrinkles [13, 14]. This process is called photoaging and leads to visible signs of skin aging, even in the absence of exposure to UV radiation, as confirmed by studies showing an increase in MMP-1 expression and ROS production under the influence of visible light alone, as well as the effects of skin damage under oxidative stress observed *in vitro* on human fibroblasts [14, 15]. In addition, it has been shown that this mechanism can be partially inhibited by antioxidants, which reduce ROS production, inhibit AP-1 activation, and decrease MMP expression, suggesting the possibility of preventing photoaging through the use of antioxidants [13, 16, 17].

HEV radiation induces the formation of ROS, which damage cell membrane lipids, structural proteins, and skin cell DNA.

This results in the activation of MMPs responsible for the degradation of collagen and elastin fibers. This process contributes to the loss of firmness, elasticity, and deterioration of the epidermal barrier function. *In vitro* studies have shown that prolonged exposure of pigmented skin to blue light results in thickening of the stratum corneum, increased dryness, and decreased hyaluronic acid levels [1]. The scalp, especially in people with thinning hair, loses its ability to effectively retain water, which increases its susceptibility to irritation, microtrauma, and chronic inflammation. *In vitro* studies on HaCaT keratinocytes cell line have shown that exposure to blue light (420-430 nm, dose 41.4 J/cm^2) leads to an immediate increase in H_2O_2 concentration. The ROS levels were elevated significantly within 30 minutes after exposure, although partially neutralized after one hour, however, their dynamic presence might disrupt the integrity of the epidermal barrier [18].

Melanocytes, the cells responsible for melanin synthesis, are located both in the epidermis of the scalp and in the hair follicle. It has been proven that blue light radiation can influence melanogenesis by activating the photoreceptor Opsin-3 (OPN3), which leads to an increase in the expression of enzymes responsible for melanin production, such as tyrosinase and tyrosinase-related protein 1 (TRP-1) and tyrosinase-related protein 2 (TRP-2) [19]. This can result in local skin hyperpigmentation, pigmentation disorders, and, in the long term, abnormal pigmentation of the hair shaft, which can clinically manifest as premature greying [19].

Blue light also affects the hair follicle matrix cells, which are crucial for maintaining the hair growth cycle. *In vitro* skin models have shown that HEV induces oxidative stress, caspase activation, and inflammatory pathways (including NF-κB), which may accelerate the transition of the hair follicle from the anagen to the telogen phase, leading to the development of telogen hair loss [8]. HEV radiation disrupts the circadian rhythm by inhibiting the synthesis of melatonin, a hormone that not only regulates sleep but also supports hair follicle regeneration and protects them from oxidative stress. Reduced melatonin levels may therefore increase the susceptibility of hair follicles to environmental factors and weaken their ability to regenerate, which may accelerate the progression of baldness [19].

In the context of anatomical exposure to HEV radiation, the presence of hair is an important factor limiting its transmission to the scalp surface. Due to its hierarchical keratin structure and melanin content, hair can absorb and scatter electromagnetic radiation in the visible range, acting as a natural optical barrier. Recent studies have shown that the elongation of the solar path length (SPL) within the hair occurs due to multiple scattering phenomena, determined by the structure of keratin fibers and the distribution of melanin in the hair cortex. These mechanisms effectively reduce the intensity of radiation reaching the scalp surface, and the absorbed energy is dissipated in the form of heat, which aids thermoregulation and protection against HEV-induced oxidative stress [20].

Studies have shown that in the 400-500 nm wavelength range, melanin contained in hair structures plays a key role in radiation absorption, while the keratin matrix of the hair intensifies the scattering phenomena, limiting the depth of light penetration into the dermis. *In vivo* studies have confirmed that the variability of absorption (μa) and scattering (μs') coefficients within the epidermis and hair shaft affects the degree of HEV attenuation [21]. The number of melanosomes contained in the hair shaft directly affects the efficiency of blue light energy conversion into heat, which is a mechanism that protects the scalp structure from photodamage. Thermal microscopy studies have shown that hair with a higher eumelanin content was more effective at converting blue light energy into heat, thereby limiting the transmission of radiation to the skin surface [22].

No direct quantitative studies are determining HEV transmission through scalp hair *in vivo*, but available data from optical hair analyses indicate that the keratin structure and melanin content limit the scalp's exposure to the harmful effects of this range of radiation. In the case of hair thinning, characteristic of androgenetic alopecia, the natural protection of the scalp becomes less effective, which argues for the need for additional forms of photoprotection.

STRATEGIES FOR PROTECTING THE SCALP FROM BLUE LIGHT

One of the most effective ways to counteract the negative effects of blue light exposure is through the topical application of antioxidants. As demonstrated in a review of scientific literature, vitamins C and E, resveratrol, niacinamide, and coenzyme Q10 effectively neutralize ROS and inhibit the expression of MMP-1 and MMP-3, which are responsible for the degradation of collagen and elastin. These substances also protect hair follicle cells from premature damage, support tissue regeneration, and improve skin hydration and elasticity. Resveratrol and niacinamide in particular can block NF-κB and AP-1-dependent inflammatory pathways, which further strengthens the scalp's defense mechanisms against oxidative stress [23]. Local therapy can be complemented with oral supplementation with astaxanthin or lycopene, which strengthen the skin's systemic resistance to oxidative stress and reduce inflammation [24].

At a later stage of the protective response to HEV radiation, topical preparations containing advanced new-generation sunscreens play a special role. Classic protective cosmetics, developed exclusively for UV radiation, do not provide complete protection against the harmful effects of visible blue light [25]. Protecting the scalp against HEV is particularly

important in patients with thinning hair or symptoms of androgenetic alopecia. In these cases, the skin's natural protection (the hair layer) is significantly reduced, resulting in increased exposure of the epidermis and hair follicles to light. Research indicates that the scalp affected by alopecia experiences increased pro-inflammatory phenomena and oxidative stress, which leads to disruption of the hair follicle microenvironment and may accelerate its miniaturization. In the context of HEV light exposure, these phenomena may be further exacerbated, making photodynamic protection a key element in the prevention of hair loss progression [26].

New formulations of protective preparations, developed for the full spectrum of light, contain physical mineral filters such as titanium dioxide (TiO_2) and iron oxides, as well as patented HEV-absorbing molecules such as TriAsorB™. These compounds have a high capacity to absorb UVA, UVB, and HEV radiation, effectively reducing ROS formation and decreasing MMP-1 and MMP-3 activity [25, 27]. *In vitro* studies have shown that formulations containing TriAsorB™ significantly reduce the formation of cyclobutane pyrimidine dimers (CPD) and 8-OHdG oxidative damage in a reconstructed human epidermis model, even in the context of blue light exposure [28]. Protection against HEV appears to be crucial in areas with reduced hair coverage, where the lack of a hair barrier promotes photodamage.

The use of mineral filters such as titanium dioxide (TiO_2) and iron oxides provides effective protection against UVA, UVB, and HEV. Studies have shown the presence of TiO_2 particles on the surface of hair shafts and in the follicular area in patients with frontal fibrosing alopecia (FFA) and in a control population. Although no penetration into the hair follicle was found, persistent retention of particles within the epidermis and at the hair root was demonstrated [29].

A practical problem associated with the use of mineral filters on the scalp is their adhesion to the skin and hair surface, which requires thorough washing of these preparations. Studies by other authors have shown that TiO_2 particles can penetrate up to 7-13 layers of the stratum corneum, while ZnO remains mainly on the skin surface and near the hair follicles. The retention of these particles, although superficial, indicates the need for their mechanical removal during evening hygiene to prevent possible accumulation and potential symptoms of cosmetic substance retention [30].

In trichological practice, it is recommended to use photoprotection in the form of light lotions, mists or sprays that allow for even coverage of the scalp, even in areas with thinning hair. These products do not weigh down the hair shafts and do not affect their volume or texture, which increases their comfort of use. These preparations are recommended in the prevention of photoaging, but also as an adjunct to the treatment of inflammatory dermatoses and androgenic and telogen alopecia [31].

Physical protection of the scalp remains equally important, especially in populations predisposed to androgenic and telogen alopecia. In people with thinning hair, the keratin barrier no longer provides effective protection against radiation, making the scalp more vulnerable to photodamage and oxidative stress, which can lead to deterioration of the hair follicles and further hair loss [32].

The use of protective clothing, such as hats, headbands, and scarves made of materials with a high ultraviolet protection factor (UPF), is an effective strategy for limiting the exposure of the scalp to the harmful effects of UV radiation and HEV. Studies show that these types of fabrics, thanks to their light-reflecting and light-absorbing micropigments, reduce the transmission of radiation to the skin surface, limiting oxidative stress and photodamage [33]. Although no direct effect of wearing high UPF protective clothing on the inhibition of hair follicle miniaturization has been documented, scientific reports are suggesting that reducing scalp exposure to radiation may indirectly support the maintenance of a healthy hair follicle microenvironment, especially in individuals with thinning hair or a predisposition to androgenetic alopecia [26].

Melatonin plays a special role in the protective response of the scalp to HEV light exposure. It has been shown to promote hair follicle regeneration by activating the Wnt/β-catenin signalling pathway, which is key to the hair growth cycle. *In vivo* studies on a depilation model in mice showed that melatonin supplementation significantly increased the expression of Wnt pathway genes and accelerated follicle regeneration, suggesting a potential therapeutic application in humans [34]. In addition, exposure to light disrupts the expression of melatonin receptors (MT1) in the skin, sebaceous glands, and hair follicles, which affects the skin's ability to respond to oxidative stress. Administration of exogenous melatonin corrected these disturbances, restoring normal MT1 receptor expression in studies on light-deprived rat models [36]. Light exposure may impair the skin's response to melatonin by disrupting the expression of its receptors (MT1), which may weaken the regenerative effects of this hormone. Melatonin supplementation restores this pathway and activates follicle regeneration processes, as confirmed by both molecular studies and animal models [35, 36].

SUMMARY

Blue light emitted by natural and artificial sources penetrates into the scalp, inducing oxidative stress, activating inflammatory pathways (NF-κB, AP-1) and MMP expression, resulting in collagen and elastin degradation and deterioration of the integrity of the epidermal barrier. Hair follicles, which exhibit high metabolic and mitotic activity, are particularly susceptible to these factors, which can lead to disruption of the hair growth cycle, miniaturization, and telogen effluvium.

In addition, HEV contributes to scalp microbiome dysbiosis by promoting the pathogenic activity of *Malassezia* yeasts, which favors the development of inflammatory dermatoses and exacerbates hair follicle damage.

In vitro and clinical studies show that HEV may be a separate factor in photoaging and scalp health deterioration, independent from UV radiation. Therefore, it is necessary to implement comprehensive photoprotection strategies, including the use of antioxidants (topical and systemic), HEV filters, and physical skin protection. Taking into account circadian rhythms and the role of melatonin in hair follicle regeneration, it opens up new therapeutic perspectives. These findings indicate the need for further in-depth translational research in the field of scalp photobiology and dermotrichology.

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otrzymano / received: 07.06.2025 | poprawiono / corrected: 11.06.2025 | zaakceptowano / accepted: 15.06.2025