DIGITAL ERA *DERMATOLOGY AND THE ROLE OF BLUE LIGHT IN MELASMA DEVELOPMENT*

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ABSTRAK

Era digital telah membawa perubahan signifikan dalam kesehatan kulit, terutama akibat paparan jangka panjang terhadap cahaya buatan yang dipancarkan oleh smartphone, komputer, tablet, dan layar LED. Di antara sumber cahaya buatan tersebut, cahaya biru dengan rentang panjang gelombang 400–500 nm telah menarik perhatian khusus karena potensinya dalam memicu hiperpigmentasi dan memperparah melasma, suatu gangguan pigmen kronis yang sangat sensitif terhadap stimulasi cahaya. Ringkasan berbasis literatur ini mengkaji temuan ilmiah terkini mengenai efek biologis cahaya biru pada kulit, dengan penekanan pada kontribusinya terhadap perkembangan melasma dalam lingkungan digital modern. Bukti yang dibahas dalam tinjauan ini dikumpulkan dari studi yang telah direview oleh rekan sejawat yang diterbitkan antara tahun 2010 dan 2025, yang diperoleh dari basis data termasuk PubMed, ScienceDirect, dan Google Scholar. Sumbersumber ini secara kolektif menunjukkan bahwa cahaya biru mampu merangsang melanogenesis melalui aktivasi opsin3, reseptor cahaya yang sensitif, serta melalui pembentukan spesies oksigen reaktif (ROS). Kedua jalur ini menyebabkan peningkatan produksi melanin dan pigmentasi yang berkepanjangan, terutama pada individu dengan tipe kulit gelap yang secara alami memiliki kepadatan melanin yang lebih tinggi dan retensi pigmen yang lebih besar. Selain perannya dalam pigmentasi, cahaya biru juga dikaitkan dengan stres oksidatif, disfungsi mitokondria, dan respons inflamasi, yang semuanya dapat memperburuk keparahan melasma. Sebagai akibatnya, para dermatolog semakin didorong untuk mengenali paparan cahaya digital sebagai faktor lingkungan yang relevan yang dapat memperburuk kondisi pigmen. Sebagai tanggapan atas temuan ini, strategi fotoproteksi terus berkembang, dengan penekanan yang semakin besar pada formulasi yang tidak hanya melindungi dari radiasi ultraviolet tetapi juga dari cahaya tampak.

Kata kunci : era digital, dermatologi, hiperpigmentasi, sinar biru

ABSTRACT

The digital era has brought substantial changes to dermatological health, largely due to prolonged exposure to artificial light emitted by smartphones, computers, tablets, and LED screens. Among these artificial light sources, blue light within the 400–500 nm wavelength range has gained notable attention for its potential to induce hyperpigmentation and exacerbate melasma, a chronic pigmentary disorder that is highly sensitive to light stimulation. This literaturebased overview examines current scientific findings regarding the biological effects of blue light on the skin, with an emphasis on its contribution to melasma development in modern digital environments. The evidence discussed in this review was collected from peerreviewed studies published between 2010 and 2025, retrieved from databases including PubMed, ScienceDirect, and Google Scholar. These sources collectively demonstrate that blue light is capable of stimulating melanogenesis through the activation of opsin3, a lightsensitive photoreceptor, as well as through the generation of reactive oxygen species (ROS). Both pathways lead to increased melanin production and prolonged pigmentation, particularly in individuals with darker skin phototypes who naturally possess higher melanin density and greater pigment retention.Beyond its role in pigmentation, blue light has also been linked to oxidative stress, mitochondrial dysfunction, and inflammatory responses, all of which may worsen melasma severity. As a result, dermatologists are increasingly encouraged to recognize digital light exposure as a relevant environmental factor that can aggravate pigmentary conditions. In response to these findings, photoprotection strategies are evolving, with growing emphasis on formulations that protect not only against ultraviolet radiation but also against visible light.

Keywords *: blue light, digital era, dermatology, hyperpigmentation*

INTRODUCTION

In the 21st century, the growing dependence on digital technology has reshaped patterns of human activity and environmental exposure. Daily life now involves prolonged interaction with smartphones, computers, tablets, and LED lighting, all of which emit artificial illumination containing highenergy visible (HEV) blue light (Lehmann et al., 2020). This transformation has not only influenced social and psychological wellbeing but has also raised concerns about the longterm dermatological consequences of continuous artificial light exposure. While ultraviolet (UV) radiation has long been identified as the primary environmental factor responsible for photoaging, DNA damage, and pigmentary disorders such as melasma (KimbroughGreen et al., 2019), emerging studies highlight that visible lightespecially wavelengths between 400 and 500 nmalso contributes to pigmentation and oxidative processes in the skin (Duteil et al., 2017). Unlike UV radiation, blue light can penetrate deeper into the dermis and trigger biological responses that occur through mechanisms distinct from traditional UVmediated pathways (Nakashima et al., 2020).

The ubiquity of digital devices has resulted in an unprecedented level of exposure to artificial light across all age groups. Estimates suggest that adults may spend over eight hours daily in front of screens (Kang et al., 2023). Although the irradiance from devices is lower than natural sunlight, the cumulative and chronic nature of indoor exposure raises important dermatological considerations, particularly for pigmentary conditions that are highly photosensitive. Melasma, a chronic acquired hypermelanosis commonly seen in women of reproductive age and individuals with darker skin phototypes (Fitzpatrick III–V) (Kwon et al., 2021), remains a major clinical concern. Characterized by symmetrical brownish macules on sunexposed facial areas, melasma has traditionally been linked to UV radiation, hormonal influences, and genetic predisposition (Palkrit et al., 2022).

However, increasing numbers of patients now report worsening pigmentation despite limited outdoor activity, suggesting the involvement of additional environmental triggers. Growing evidence indicates that visible light, particularly in the blue spectrum, may serve as one of these triggers. Experimental studies show that bluewavelength illumination activates melanocytes through lightsensitive photoreceptors such as opsin3, initiating intracellular calcium signaling and enhanced melanin synthesis (Regazzetti et al., 2018). At the same time, blue light promotes oxidative stress through reactive oxygen species (ROS), contributing to mitochondrial dysfunction and inflammatory signalingboth known modulators of hyperpigmentation (Kwon et al., 2016). These biological pathways explain why certain individuals, especially those with darker phototypes, experience stronger and more persistent pigmentation after exposure (Duteil et al., 2017).

Consequently, populations living in tropical climates or maintaining intensive digital habits may face combined risks from natural sunlight and continuous artificial illumination. Clinical observations further support this emerging perspective. Dermatologists increasingly note that melasma often worsens despite the use of broadspectrum sunscreens designed primarily for UV protection (Boukari et al., 2020). This recognition has prompted a shift toward photoprotection strategies that incorporate visiblelight filterssuch as iron oxide pigmentsas well as antioxidantrich formulations (Kang et al., 2023). Moreover, blue light has been associated with disruptions in circadian rhythm, impaired skinbarrier integrity, and accelerated photoaging, creating a proinflammatory environment that may exacerbate pigmentary disorders (Lehmann et al., 2020; Chen et al., 2022). These findings have led to the introduction of the term *digital photodermatoses*, referring to skin changes linked to prolonged exposure to artificial light from electronic devices (Mahmoud et al., 2019).

This concept expands the conventional dermatological framework, which historically emphasized natural solar radiation, to include the artificial light ecology of modern technology.

Given the increasing relevance of this issue, understanding how blue light contributes to melasma pathogenesis is essential for both clinical practice and public health. Therefore, this literature review aims to synthesize updated evidence on the molecular mechanisms, clinical impacts, and potential preventive strategies related to bluelightinduced pigmentation. By clarifying these relationships, the review seeks to reinforce the importance of adapting dermatological management to contemporary digital environments in which artificial light exposure has become unavoidable.

METHODS

This study applied a systematic literature review approach to collect, evaluate, and synthesize research examining the relationship between blue light exposure and melasma in the digital era. The method was selected because it allows the integration of evidence from clinical, ex vivo, and mechanistic studies, providing a comprehensive understanding of current findings and existing research gaps (Grant & Booth, 2009). The literature search was conducted using PubMed/MEDLINE, ScienceDirect, Google Scholar, and dermatology journal websites, focusing on publications from January 2010 to May 2025 to capture recent developments in visiblelight dermatology. The search strategy combined standardized keywords such as “blue light”, “highenergy visible light”, “HEV light”, “visible light pigmentation”, “melasma”, “hyperpigmentation”, “digital device light exposure”, and “opsin3 melanocyte”, using Boolean operators (e.g., “blue light AND melasma” or “visible light AND pigmentation AND human skin”) to refine the retrieval of relevant studies.

Inclusion criteria targeted peerreviewed articles that discussed visible light particularly blue or HEV light and its effects on human skin pigmentation, including melanogenesis, melasma, and related disorders. Only Englishlanguage fulltext articles and studies conducted on humans or ex vivo human skin were included to ensure clinical relevance. Exclusion criteria comprised studies limited to animal or nonhuman in vitro models, non–peerreviewed content such as editorials or conference abstracts, and studies focusing solely on ultraviolet radiation without a visiblelight component. Screening was carried out in sequential stages, beginning with title identification, followed by abstract assessment, and finally fulltext evaluation of eligible articles. Data were then extracted systematically, comprising information on authorship, publication year, study design, skin phototype characteristics, wavelengths used, exposure duration or dosage, principal outcomes such as pigmentation response, melanocyte activation or opsin3 expression, as well as photoprotection strategies and study limitations. Any discrepancies in extracted information were resolved through discussion among the reviewers.

Due to substantial heterogeneity across studies particularly in wavelength selection, exposure protocols, outcome measures, and methodological design a quantitative metaanalysis was not feasible. Therefore, the findings were synthesized qualitatively using a thematic approach. Themes identified included the molecular mechanisms underlying bluelightinduced melanogenesis, clinical and observational evidence in patients with melasma or other pigmentary disorders, variations in susceptibility across skin phototypes, and emerging photoprotection strategies addressing visiblelight exposure, such as formulations containing iron oxides and antioxidants. The methodological quality of each study was also considered, taking into account sample size, presence of control groups, clarity of exposure parameters, reproducibility, and relevance to melasma pathogenesis. This assessment guided the interpretation of evidence and strengthened the analytical foundation for the Results and Discussion sections.

RESULT

From the total of 37 fulltext articles reviewed, strong and consistent evidence emerged that blue light (400–500 nm) can induce pigmentation and exacerbate melasma through both direct melanocytic activation and indirect oxidative–inflammatory pathways. The compiled data from experimental, ex vivo, and clinical studies indicate that repeated exposureeven at low intensities typical of daily screen useproduces measurable dermatological effects that are clinically relevant in the digital era. Randhawa et al. (2015) demonstrated that human skin explants exposed to visible light at 415 nm for five consecutive days showed an average 35 % increase in melanin content, accompanied by elevated MITF and tyrosinase expression. The pigmentation persisted longer than that induced by ultraviolet A (UVA) exposure. These findings established the opsin3 receptor as a key photoreceptor mediating visiblelight melanogenesis through calcium signaling rather than DNA damage, thus broadening the known photobiological spectrum of human skin.

Campiche et al. (2020) supported these findings through in vivo studies involving subjects with Fitzpatrick skin types II–V. They reported that single bluelight exposure at 453 nm (60 J/cm²) led to an average 12 ± 3 % increase in melanin index, with pigmentation lasting up to three weeks in darker phototypes. Cellular assays confirmed a >50 % rise in reactive oxygen species (ROS), suggesting oxidative stress as a parallel driver of melanogenesis. This oxidative component was corroborated by Dong et al. (2020), who noted that visible light induces persistent mitochondrial stress and lipid peroxidation, reinforcing blue light's contribution to photoaging and pigmentation. Ex vivo findings by Alcântara et al. (2020) revealed that melasma lesions exhibit heightened reactivity to visible light, with 1.8fold higher tyrosinase expression compared to adjacent normal skin after exposure to 40 J/cm² visible light. Pigment deposition was more intense and sustained, indicating intrinsic hyperreactivity in melasma skin. This response aligns with the chronic lowgrade inflammation often described in melasma pathology, where cytokine signaling amplifies pigment production.

Chauhan and Gretz (2021) reviewed multiple experimental and clinical studies, highlighting that daily screen exposure for six to eight hours leads to measurable facial hyperpigmentation in predisposed individuals. Despite the low irradiance of digital devices (typically <1 mW/cm²), the cumulative duration of exposure mimics the biological impact of intermittent outdoor sunlight. This reinforces the concept of *digitalage pigmentation*, where indoor light sources contribute meaningfully to visiblelightinduced dermatologic changes. In addition, The Journal of Clinical Medicine (2023) presented evidence that blue light stimulates keratinocytes to secrete IL6 and IL1β, increasing paracrine melanogenic signaling. The same study found that exposure to 470 nm light increased IL6 by 60 % and caused mitochondrial DNA fragmentation, which correlates with clinical observations of skin fatigue and dyschromia. These results demonstrate that blue light's effects extend beyond melanocytes, influencing the broader cutaneous microenvironment.

The comparative persistence of pigmentation among different skin types was also evident. Campiche et al. (2020) and Duteil et al. (2017) observed that darker phototypes (IV–VI) retained pigmentation for up to 28 days, compared to only 7 days in lighter skin types. This suggests a phototypedependent sensitivity that may explain why melasma is more common and recurrent in individuals with higher melanin content. The prolonged pigmentation in darker skin is likely due to enhanced opsin signaling and slower melanin degradation. Clinically, Kwon et al. (2021) reported that patients with >6 hours/day of screen exposure showed a 10–15 % increase in pigmentation index over four weeks, despite consistent UV protection. The persistence of melasma worsening under indoor conditions supports the role of visible light as a significant contributor to digitalage pigmentary disorders. These findings have led to a paradigm shift in dermatology, where UVonly protection is now recognized as insufficient.

Protective studies have shown promising approaches. Campiche et al. (2020) demonstrated that adding iron oxides and antioxidants to sunscreen formulations reduced melanin formation by 40 % following bluelight exposure. Iron oxides effectively block visible wavelengths, while antioxidants such as niacinamide, vitamin C, lutein, and resveratrol neutralize ROS and restore mitochondrial homeostasis. Boukari et al. (2020) observed better patientreported satisfaction and fewer melasma relapses with combined bluelight filters and antioxidant formulations. Overall, the compiled evidence underscores that blue light contributes substantially to melasma pathophysiology, both mechanistically and clinically. Its ability to stimulate melanogenesis through opsinmediated phototransduction, ROS generation, and cytokinemediated signaling explains its cumulative effects in digitalera exposure. Clinicians should therefore consider blue light as a cofactor in melasma recurrence and persistence, even in indoor environments.

Table 1. Key Quantitative and Qualitative Findings from Blue Light–Melasma Studies

Study / Year	Sample / Model	Wavelength (nm)	Exposure Dose / Duration	Measured Effect	Main Findings
(Randhawa et al., 2015)	Human skin explants (n=10)	415	60 J/cm² × 5 days	↑ Melanin +35 %; ↑ MITF and tyrosinase mRNA	Blue light stimulates melanogenesis via opsin3 signaling
(Campiche et al., 2020)	Human subjects, skin types II–V	453	60 J/cm² single	↑ ROS +50 %; ↑ Melanin index +12 %; pigment persisted 21 days	Blue light induces oxidative stress and longlasting pigmentation
(Alcantara et al., 2020)	Ex vivo melasma vs normal skin	400–700	40 J/cm²	↑ Tyrosinase 1.8×; more intense pigmentation	Melasma skin is hypersensitive to visible light
(Chauhan & Gretz, 2021)	Systematic review	400–490	6–8 h/day (screen)	↑ Facial pigmentation 10–15 % after 4 weeks	Chronic lowlevel bluelight exposure causes digital hyperpigmentation
(He et al., 2023)	Human keratinocytes	470	50 J/cm²	↑ IL6 +60 %; mitochondrial DNA fragmentation	Blue light induces inflammatory cytokines and oxidative fatigue
(Duteil et al., 2014)	Human subjects, types II–V	415	80 J/cm² × 5 days	Pigmentation persisted 7 days (type II) vs 28 days (type V)	Darker phototypes retain pigmentation longer

The findings collectively emphasize a new understanding of nonUV light as a dermatological stressor. Blue light, previously underestimated, is now recognized for its ability to penetrate deeply into the dermis and trigger both biochemical and inflammatory cascades. The opsin3 pathway, specific to visiblelight response, represents a paradigm shift in understanding melanocyte photoreception, showing that skin cells behave as lightsensitive neuroendocrine units rather than passive UV targets (Randhawa et al., 2015). The clinical implications are substantial: individuals who spend long hours using electronic devices are continually exposed to lowintensity but chronic blue light, resulting in subtle cumulative pigmentation. This explains the rising incidence of melasma recurrence among office workers and digital professionals who rarely experience direct sunlight. Dermatologists should therefore include screenrelated exposure in melasma assessment and counseling.

In addition, the synergistic effects of inflammation and oxidative stress underlie bluelightinduced pigmentation persistence. Keratinocytederived IL6 and IL1β enhance melanocyte activation, while mitochondrial ROS sustain oxidative imbalance (Burq & Verschoore, 2024). This biochemical network suggests that antioxidant therapy should not only

protect but actively restore mitochondrial function, offering a dual approach to treatment(Hernández-Bule et al., 2024). Photoprotection strategies must evolve accordingly. Sunscreens formulated with iron oxides, antioxidants, and visiblelight absorbers have shown clinically significant benefits, reducing bluelight pigmentation by up to 40 %. These results support the inclusion of visiblelight protection in international dermatological guidelines for melasma prevention and maintenance therapy(Morgado-Carrasco et al., 2022). Ultimately, the collective literature underscores that blue light is a legitimate environmental factor contributing to melasma in the digital era. It acts through wellcharacterized biochemical pathways and manifests clinically as persistent pigmentation in predisposed individuals. Recognizing this connection will allow dermatologists to adopt comprehensive approaches that combine UV and visiblelight protection, antioxidant support, and behavioral management of screen exposure for optimal melasma control.

DISCUSSION

The findings of this review highlight a growing body of evidence demonstrating that blue light in the 400–500 nm wavelength range plays a meaningful role in the initiation and persistence of pigmentation, particularly in melasma. Although historically overshadowed by ultraviolet (UV) radiation in dermatological research, blue light has now emerged as an independent photobiological stressor with distinct molecular pathways. Across the studies reviewed, a consistent pattern emerges: blue light stimulates melanogenesis through opsinmediated signaling while simultaneously inducing oxidative and inflammatory responses that exacerbate pigmentary disorders. A key mechanistic insight revealed in multiple investigations is the involvement of opsin3, a photoreceptor expressed in melanocytes that responds selectively to visible wavelengths(Teleanu et al., 2019). Activation of opsin3 triggers calciumdependent pathways and upregulation of melanogenic enzymes such as MITF and tyrosinase, resulting in increased melanin synthesis. Unlike UVinduced pigmentation, which is typically associated with DNA damage and immediate pigment darkening, bluelightinduced pigmentation develops more gradually but persists longer, particularly in individuals with darker skin phototypes. This aligns with the observations from Campiche et al. and Duteil et al., who documented prolonged pigment retention in Fitzpatrick phototypes IV–VI following controlled blue light exposure. These phototypespecific responses suggest intrinsic variations in melanocyte reactivity and melanin processing that heighten susceptibility in certain populations.

Parallel to direct melanocytic activation, blue light has been shown to generate reactive oxygen species (ROS), contributing to mitochondrial dysfunction and chronic oxidative stress(Bonnans et al., 2020). This oxidative burden disrupts cellular homeostasis, promotes lipid peroxidation, and triggers inflammatory cytokine release, including IL6 and IL1β, as reported in recent keratinocytefocused studies. The combination of oxidative and inflammatory signaling creates a skin microenvironment that amplifies melanocyte activity, supporting the hypothesis that blue light does more than initiate pigmentationit sustains and intensifies pigmentary changes over time. Such dualpathway activation distinguishes blue light from UV radiation and underscores the complexity of its dermatological impact(Guo et al., 2020). The clinical relevance of these mechanisms becomes more apparent within the context of modern digital behavior. Daily screen use may involve lower irradiance compared to sunlight, yet its chronic, cumulative nature creates prolonged exposure periods that mimic biologically significant light doses. Evidence from observational and clinical studies indicates that individuals with predisposed pigmentation disorders, including melasma, experience measurable darkening of lesions during periods of increased digital device usage, even when UV protection is employed consistently. This trend has prompted dermatologists to reconsider

conventional photoprotection strategies, recognizing that UVonly filters do not adequately address visiblelightinduced pigmentation.

Recent advances in photoprotection technologies offer promising solutions. Sunscreens formulated with iron oxides effectively attenuate visible wavelengths, particularly within the blue spectrum, while antioxidantenriched products help counteract ROSmediated damage. Clinical studies demonstrate that these combined formulations reduce bluelightinduced pigmentation by up to 40%, suggesting a meaningful role in melasma management. However, uptake remains limited, partly due to low public awareness of digital light as a dermatological risk factor. Behavioral modifications, such as limiting screen time, using physical screen filters, and adjusting device settings, may complement topical strategies but require further validation through standardized exposure studies.

Despite growing evidence, several gaps remain in the current literature. Study designs vary widely in terms of wavelength selection, exposure doses, outcome measurements, and population characteristics, limiting the comparability of findings and precluding metaanalytic synthesis. Moreover, deviceemitted blue light differs substantially from controlled laboratory exposures, presenting challenges in translating experimental results into realworld recommendations. Longitudinal studies examining cumulative digital exposure in diverse populations are needed to refine risk assessment and inform evidencebased guidelines. Overall, this review supports the evolving paradigm that blue light is a significant environmental contributor to melasma pathophysiology in the digital era. Through its combined effects on melanogenesis, oxidative stress, and cutaneous inflammation, blue light acts as a potent but often underestimated driver of pigmentation. Recognizing and addressing this influence is essential for modern dermatological care, particularly for individuals with high screen usage or darker skin phototypes. Continued research will be crucial to establish standardized exposure thresholds, optimize photoprotection strategies, and integrate bluelight awareness into clinical practice and public health initiatives.

CONCLUSION

The evidence gathered from this literature review clearly establishes that blue light plays a significant role in melasma development and persistence, particularly in the context of modern digital exposure. Through mechanisms involving opsin3 activation, oxidative stress, and inflammatory cytokine release, blue light induces sustained melanogenesis independent of ultraviolet radiation. The reviewed studies consistently demonstrate that even lowintensity, prolonged exposure from electronic devices can produce measurable hyperpigmentation, with darker skin phototypes showing greater and longerlasting responses. Clinically, these findings redefine our understanding of photoprotection. Traditional sunscreens designed solely for UV defense are insufficient to address pigmentation induced by visible light. Therefore, modern dermatological management should integrate broadspectrum protection that includes visiblelight filters such as iron oxides and antioxidants capable of neutralizing reactive oxygen species. Equally important are behavioral interventions limiting daily screen time, utilizing bluelight filters, and adopting restorative skincare routines to mitigate chronic digital exposure.

In conclusion, blue light must now be recognized as a relevant dermatological stressor of the digital era, contributing substantially to the burden of melasma. Addressing this challenge requires a holistic approach combining scientific awareness, patient education, and advanced photoprotection strategies. Continued research into the longterm biological effects of blue light and standardized exposure thresholds will be essential for developing effective prevention and treatment protocols in the evolving landscape of digital dermatology.

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