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IMPACT OF HIGH-ENERGY VISIBLE (BLUE) LIGHT ON SKIN PIGMENTATION AND MELANOGENESIS: A COMPREHENSIVE REVIEW OF TREATMENT, PREVENTION, AND EMERGING PROTECTIVE INTERVENTIONS

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Abstract

Background *:There are ultraviolet (UV), visible light (VL), and infrared. Although UV is a recognized culprit in photoaging and pigmentation, the contribution of the high-energy visible light (HEVL; blue light 400-500 nm) spectrum is becoming more important as a critical, independent factor that causes cutaneous damage.*

Purpose: *The review summarizes existing findings on the effects of HEVL on skin pigmentation and melanogenesis, describes the biological processes involved in that effect, assesses clinically effective protection and treatment plans and highlights areas of critical gaps in the existing literature and clinical paradigms. ·*

Methods: *A systematic literature search was carried out in PubMed, Google Scholar, and Web of Science of articles published until the current month and year of year with the terms HEVL, blue light, melanogenesis, opsin-3, photoprotection, and pigmentary disorders. Findings: HEVL causes pigmentation by different mechanisms and the major one is the use of the Opsin-3 receptor in the melanocytes, resulting in long-term hyperpigmentation, particularly in those whose skin is brown pigmented (Fitzpatrick phototypes III-V). This is permanent and darker pigmentation compared to the one induced by UVA. The existing protection measures are inorganic filters (iron oxides), new organic filters (e.g., TriAsorB ™), and topical/oral antioxidants. There are new therapeutic targets under pre-clinical and clinical development including OPN3 antagonists and MITF inhibitors.*

Conclusion: *HEVL induces melanogenesis in a strong way. There is a high level of translational disparity between the biological comprehension of the HEVL-induced pigmentation and the clinical availability of specific topical treatments. This review demonstrates that it is necessary to develop standardized factors of protection and next-generation cosmeceuticals that can ultimately inhibit HEVL photoreceptors and downstream effectors.*

1.INTRODUCTION

1.1 The Evolving Situation in Photodermatology

The history of photoprotection is a story of scientific enlightenment, the development of empirical practices into evidence-based formulations. Civilisations have been trying to find the means of protecting the skin against the sun since around 4000 BC, the ancient Egyptians have been using rice, jasmine, and lupine extracts, and the Greeks have been using oil and sand mixtures(Hernández-Bule, Naharro-Rodríguez et al. 2024). It was not until the past century, however, that a scientific perception of photodamage started to form. In 1928 the first ultraviolet B (UVB) filters were invented and their effectiveness and safety was discovered in 1956(Chauhan and Gretz 2021). In 1974, a breakthrough was made when the Sun Protection Factor (SPF) rating system was introduced which, as the first time, gave a quantitative analysis of UVB protection(Ziveh, Arjmand et al. 2025). With the subsequent discovery of ultraviolet A (UVA) as a major factor in photoaging, the first UVA filters were introduced in the 1980s and the UVA star rating system was introduced in 1992(He, Jin et al. 2023). This shift of UVB to UVA/UVB photo-protection became a major breakthrough and set the tone of photoprotection where all ultraviolet radiation is covered(Furukawa, Martinez et al. 2021).

1.2 The Forgotten Spectrum

With such developments, a large part of solar radiation has been largely ignored until recently: visible light (VL). Forming about 40-50 percent of the sunlight that ever reaches the earth surface, the visible light, in sharp contrast to the ultraviolet radiation, which takes only 2-5 percent is a significant, but as yet much less noticed environment aspect in cutaneous biology(Szerej, Kot et al. 2024). VL with wavelengths of between 400-700 nm was long thought to be comparatively inert in regards to ultraviolet and infrared neighbours(Barolet 2025). There has been a mounting amount of evidence over the last twenty years that this perception is fundamentally incorrect in that VL, and especially its high-energy form, has unique and clinically relevant biological actions on human skin, specifically, the ability to induce erythema, oxidative stress, and, most famously, long-lasting pigmentation(Kaltchenko and Chien 2025).

1.3 High-Energy Visible Light (HEVL)

In the visible spectrum, high-energy visible light (HEVL), which is also known as blue light, takes up the violet/blue band between 400 and 500 nm(Bharathi, Sushma et al. 2025). The HEVL has the highest potential of photobiological activity, as it is the shortest and the most energetic wavelength in the VL spectrum(Edwar, Arofah et al. 2025). It is mostly powered by sunlight and manmade sources like light-emitting diode (LED) screens of smartphones, computers, and televisions, compact fluorescent lamps, are much smaller sources which have an irradiance that is 99 to 1000 times less than sunlight(Mun, Lee et al. 2025). HEVL penetrates the skin deeper than UVB, it reaches the dermis where it combines with certain chromophores to cause a cascade of biological reactions unlike those caused by UAVA or UAVB radiations(Passeron, Desai et al. 2025).

1.4 The Clinical Problem

The clinical significance of HEVL is greatest in that it can trigger hyperpigmentation especially in people who have skin of colour (SOC; Fitzpatrick phototypes III-VI)(Naharro-Rodriguez, Bacci et al. 2025) . In contrast to the ultraviolet-induced pigmentation, the melanogenesis caused by HEVL is maintained by a different pathway by using the Opsin-3 (OPN3) receptor on melanocytes(Mahrous, Abdel-dayem et al. 2025) . This results in darker and longer lasting pigmentation than that produced by UVA alone and it has been shown to last up to three months(Rajan 2024). Therefore, the role of HEVL today in the development of common and therapeutically difficult hyperpigmentary diseases, such as melasma and post-inflammatory hyperpigmentation (PIH) has become an important issue(Nie, Hou et al. 2025). In this patient group, even the best-quality standard

sunscreens that can offer excellent UV protection are not always enough to prevent relapse or manage pigmentation due to the lack of coverage of the HEVL factor of the radiation spectrum(Lee, Chan et al. 2025).

1.5 Identify the Objectives of the Review as well as the Critical Gap

The purpose of the review is to synthesise existing evidence on the effect of HEVL on skin pigmentation and melanogenesis, explaining the molecular mechanisms which underlie these effects, reviewing the clinically proven protection and treatment methods, and offering the gaps in the existing research and clinical paradigms. This critical gap is as follows: although the OPN3 pathway was elucidated more than five years ago, the clinical management of the disease continues to be largely dependent on the use of physical blockers like iron oxides instead of iron oxides, which is pathway-specific to the topical therapeutics(Hartmann and Valenzuela 2024). This is a serious language difference of translation between good mechanistic targets, which are OPN3, MITF and downstream melanogenic enzymes and the armamentarium that is currently in clinical practice(Purbhoo-Makan, Houreld et al. 2022). Although tinted sunscreens containing iron oxides are very effective in HEVL protection because they are capable of physically blocking and absorbing HEVL radiation, they do not target the biological cascade, once formed(Damodaran and Nair 2022). The future of treating HEVL-induced pigmentation is the creation of a new generation of specific topical agents which may prevent the melanogenesis pathway at the receptor or transcriptional step, which has the potential of providing more specific and effective interventions in the millions of patients with pigmentary disorders(Stanescu, Chiscop et al. 2025).

2. Skin Penetrance of HEVL and Physics.

2.1. Location in the Electromagnetic Spectrum

High-energy visible light (HEVL), also called blue light, exists in the violet/blue region of the electromagnetic spectrum of 400 to 500 nanometers (nm) (Gilaberte, Ederra-Galé et al. 2026). This places it between ultraviolet (UV) radiation (290400nm) and the longer wavelengths of visible light(McDaniel, Farris et al. 2018) . The energy properties of HEVL set it apart in the rest of the visible spectrum (400-700 nm): being the shortest and most energetic wavelengths of the visible spectrum, HEVL has a higher photobiological activity than longer wavelengths of the visible and green, yellow, or red light(Lee, Chan et al. 2025). In order to place UVB radiation (290320 nm) in perspective, it is highly absorbed by the epidermis, whereas UVA (320400 nm) and HEVL have the capacity to penetrate deeper into cutaneous tissue although in different ways(Hartmann and Valenzuela 2024). The amount of sunlight that falls on the Earths surface is approximately half of the light which is seen on the surface, that is HEVL makes up about 25% of all the solar radiation(Vibriani, Chen et al. 2026).

2.2. Sources of Exposure

Natural Sources: The most biologically important and most powerful source of HEVL exposure is solar radiation(Purbhoo-Makan, Houreld et al. 2022). On an average summer day, at noon, the total irradiance of the sun is about 58.5 mW/cm 2 with blue light (400-500 nm) containing about 25 per cent of the overall emission(Passeron, Brown et al. 2026). The spectrum analysis indicates that the contribution of blue light of the sun is quite constant during the strongest daytime period and its irradiance does not lessen significantly during late morning and early afternoon and has an approximate of 25-26 percent of overall irradiance(Damodaran and Nair 2022).

Artificial Sources: The HEVL exposure has presented other sources of exposure, albeit much lower, due to the increase in the use of light-emitting diode (LED) technology(Hang, Lim et al. 2026). These are smart phones, tablets, computer monitors, television screens, compact fluorescent lamps, and the lighting based on LEDs(Stanescu, Chiscop et al. 2025). During the COVID-19 pandemic, the screen time rose up to 50 percent, and the cutaneous effects of artificial HEVL exposure have been revived(Rocafort, Rivera-Díaz

et al. 2026). Nonetheless, quantitative studies prove that the contribution of artificial devices to the biologic effective irradiance is far less than that of the sun(Peñín, Losantos et al. 2026).

Table 1:Comparative Irradiance of Blue Light from Natural and Artificial Sources.

SOURCE	BLUE LIGHT IRRADIENCE ($\mu W/cm^2$)	NOTES
Sunlight(direct,noon)	3000	The dominant source
Smartphone(max brightness)	0.2-0.7	Distance-dependent (25cm vs 5cm)
Laptop Computers	0.1-0.5	Distance-dependent
LED Desk Lamp	0.5-2	Distance-dependent
LED TV (55") at 1m	0.05-0.1	Negligible compared to sun

Based on these figures, the light irradiance of the sun in blue color is roughly 200 to 1000 times more than that of the electronic gadgets in their normal conditions of operation. Nevertheless, the overall impact of long-term artificial exposure especially in photosensitive persons or persons with pigmentary defects deserves consideration as an additive and not as an alternative factor.

2.3. Depth of Penetration

The optical properties of skin, the basic ones being the reflection, absorption and scattering, and transmission of light, determine the relationship between HEVL and cutaneous tissue. When incident light hits the skin surface, there is surface and subsurface reflection then the incident light is transmitted.

Wavelength-Dependent Penetration: The wavelength of the light penetrating into the skin is directly proportional to the penetratory depth of the light in the UV-visible spectrum(Salceda 2024). The epidermis (DNA, urocanic acid and melanin) absorbs shorter wavelengths (UVB, 290320 nm) the most(Guarnieri 2024). On the contrary, HEVL (400-500 nm) penetrates much deeper up to the reticular dermis and this deep-penetration property is also similar to the UVA radiation(Ugwueke and Elbuluk 2026). This is due to the fact that the longer wavelengths are less scattered and absorbed in the epidermal structures(Wang, Tong et al. 2024).

Optical Processes in the Skin Layers: In the epidermis, melanin is the major chromophore, only absorbing certain wavelengths, especially in the UV and the visible spectrum(Samaan and Cartee 2023). When HEVL enters the dermis, hemoglobin is the primary absorber, and spectral properties of transmitted light are affected by it(de Tollenaere, Zanchetta et al. 2025). Light scattering takes place mainly in the dermis and this is mainly because of the fibrous structure of collagen which bends light in form of photon diffusion(Nie, Hou et al. 2026). There are two different scattering mechanisms, Rayleigh scattering, which is due to subcellular structures with sizes of the order of one-tenth the wavelength, and Mie scattering, which is caused by larger structures like collagen fibre and melanosomes(De La Garza, Visutjindaporn et al.). The hypodermis is primarily adipose tissue and plays a minor role in absorption but does the light scattering at significantly deeper levels .

Relative Deep Penetration at Longer Wavelengths:

Red light (620 -700 nm) and infrared radiation all penetrate even deeper than HEVL and reach to the subcutaneous tissue. They have low energy per photon, however, which leads to various biological effects, mostly thermal, as opposed to the photochemical reactions prominent of HEVL(Nasim, Khan et al. 2024). This is a clinically important difference: whereas

photochemical activation of chromophores including opsins and flavins is induced in HEVL, longer visible wavelengths are used therapeutically due to their photobiomodulatory functions(Trisnawaty, Gunadi et al. 2024).

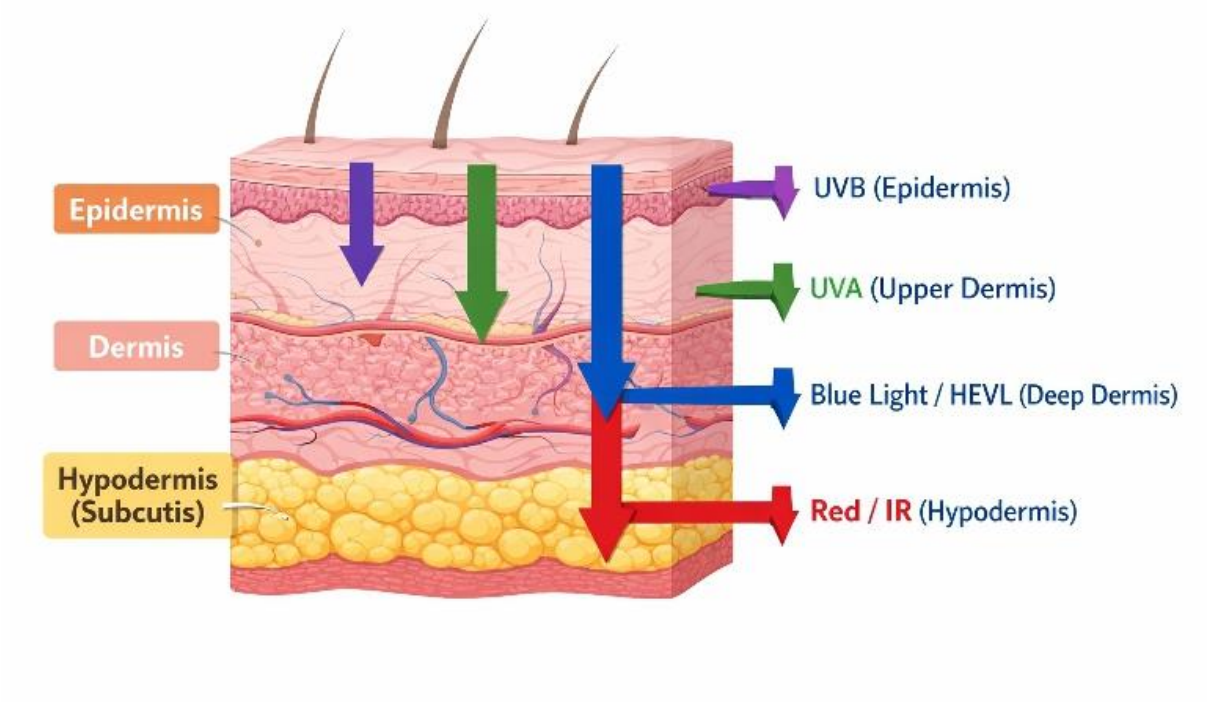

Figure 1: Depth of penetration of Electromagnetic Radiation into skin layers

3. Molecular and Cellular Pathways of Melanogenesis Caused by HEVL.

3.1. The Opin-3 (OPN3) Photoreceptor

The identification of non-visual opsins in the human skin has brought a revolution in the study of the mechanisms by which the cutaneous cells sense and react to light(Guarnieri 2024). Among these photoreceptors, the Opsin-3 (OPN3) has come out to mediate HEVL induced melanogenesis. In contrast to visual opsins which are limited to the retina, OPN3 is present on the melanocytes, the pigment-making cells of the epidermis where it is an ultra-sensitive detector of blue light .

OPN3 is activated by the HEVL wavelengths in the 415 nm range in the OPN3 pathway. When OPN3 is absorbed by photons, a conformational change process occurs to signal G-protein signalling cascades in the melanocyte(Vibriani, Chen et al. 2025). This causes calcium ion (Ca 2+) release via intracellular stores resulting in the activation of calcium/calmodulin-protein kinase II (CaMKII) and extracellular signal-regulated kinase (ERK) pathway. The phosphorylation of the downstream cyclic AMP response element-binding protein (CREB) is a key step since the phosphorylated CREB will translocate to the nucleus to trigger the melanogenic gene transcription activities. This process supports the need to suggest this OPN3 mediated mechanism and why HEVL causes pigmentation in a different pathway than the one that is activated by ultraviolet radiation(Ugwueke and Elbuluk 2026).

3.2. Downstream Signaling: the MITF Pathway

It is to this convergent point of various melanogenic signalling, comprising of those of OPN3, that the microphthalmia-associated transcription factor (MITF) universally recognised as the master of melanogenesis is converged upon. After CREB is activated, MITF is increased, and the protein itself experiences phosphorylation,

which increases its transcriptional activity(Wang, Tong et al. 2024).

Activated MITF attaches to certain DNA sequences of gene promoter regions of genes that encode the important melanogenic enzymes: tyrosinase (TYR), tyrosinase-related protein 1 (TRP-1), and dopachrome tautomerase (DCT, or TRP-2) . Tyrosinase is a rate-limiting enzyme, which catalyzes the conversion of tyrosine into the DOPAquinone, and TRP-1 and DCT participate in the further reaction that regulate the kind and amount of melanin formed(Samaan and Cartee 2023) . These enzymes are orchestrated to produce more melanin and translocate to the neighboring keratinocytes which is clinically seen as hyperpigmentation.

Comparison to UVA-induced Melanogenesis: It should be noted that HEVL-OPN3 is not comparable to UVA-induced melanogenesis. Although the pigmentation is also caused by UVA, the main mechanism of pigmentation is the transient receptor potential ankyrin 1 (TRPA1) channel in collaboration with oxidative stress, but not through the action of OPN3. Moreover, pigmentation induced by HEVL is more enduring than UVA-induced pigmentation in people with skin of colour, up to three months, which shows underlying dissimilarities in these signalling cascades

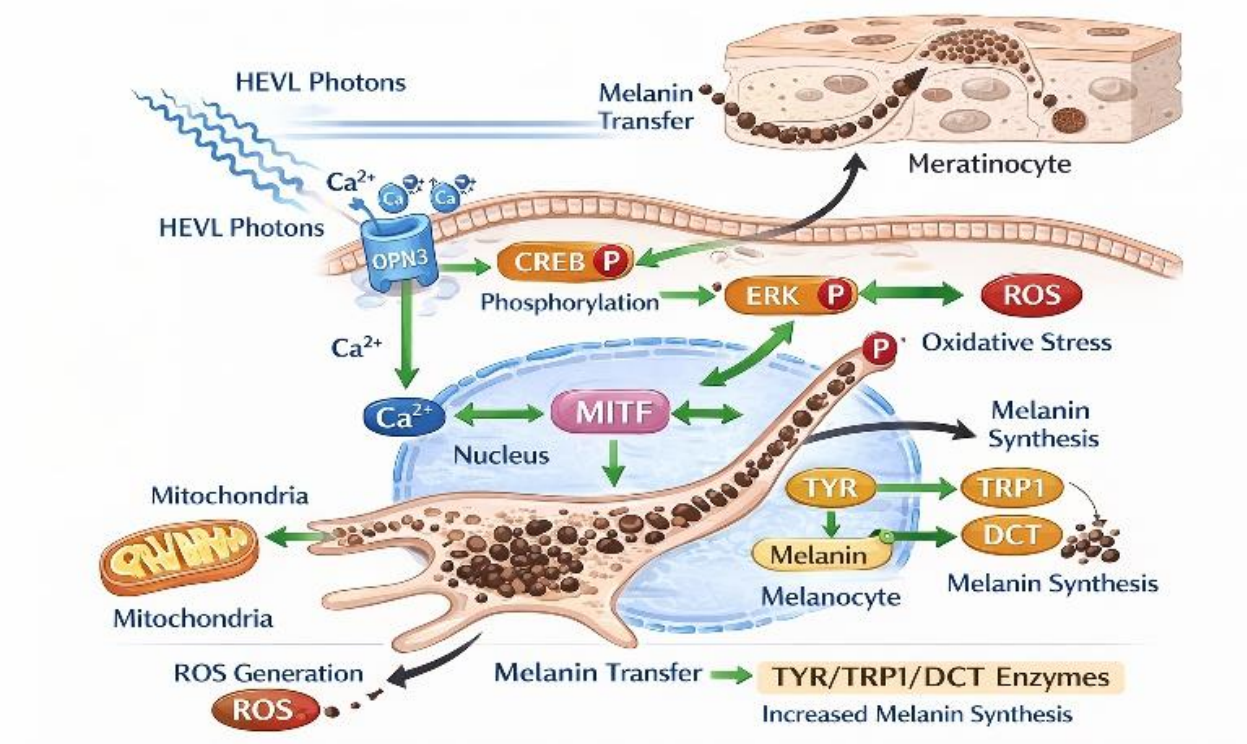

Figure 2: Molecular signaling pathways of HEVL-Induced Melanogenesis

3.3. Importance of Oxidative Stress and Synergistic Effects

In addition to receptor-mediated signalling, HEVL is a potent inducer of oxidative stress, whereby the photochemical reaction of endogenous photosensitisers with HEVL is possible. Flavin (e.g., riboflavin), porphyrins, and nicotinamide adenine dinucleotide phosphate (NADPH) oxidase are molecules with a blue light energy absorption which is excited to a higher energy state(de Tollenaere, Zanchetta et al. 2025). When they come back to baseline these photosensitivityers give energy to the molecular oxygen to create reactive oxygen species (ROS) .

Efforts by Discovering ROS Profiles: Notably, the ROS species formed by HEVL is different

compared to that formed by UVA. Mitochondrial complex inhibition and flavin excitation form superoxide anions (O2) specifically in blue light. Conversely, UVA is the primary source of singlet oxygen ($^1O2$) through reacting with other chromophores. This difference has its therapeutic implication, because antioxidant strategies should be designed to neutralise the particular ROS that is involved(Nie, Hou et al. 2026).

Synergy with UVA1: It has been convincingly shown that there are synergistic effects between HEVL and the long wavelength UVA1 (340-400 nm)(De La Garza, Visutjindaporn et al.). Put together, these wavelengths increase pigmentation and oxidative damage when compared to either separately. This synergy is clinically important since solar radiation has both spectra concurrently that is, patients are at risk of this combined insult when they are at clinics under normal sun exposure. The increased reaction perhaps represents convergent downstream responses on MITF activation and cumulative oxidative stress .

3.4. Affect on the Dermal Extracellular Matrix (HEVL)

The HEVL biological effects are not confined to the epidermis, but also affect the dermal extracellular matrix (ECM), which is a cause of photoaging. The ROS that are caused by HEVL, especially superoxide anions, trigger matrix metalloproteinases (MMPs) and especially MMP-1 and MMP-9 in particular. The proteins that cause these enzymes to break down collagen and elastin fibres are the structural proteins that make the skin stiff and firm and also make it elastic. In vitro analyses have also shown that HEVL radiation of fibroblasts results to the inhibition of proliferation, cytoskeletal reorganisation and MMP up-regulation(Nasim, Khan et al. 2024).

Contribution to Pigmentation: HEVL has indirect effects on the skin as well in which it contributes to pigmentary disorders. Melanocyte activity can be induced by ECM degradation and the resulting inflammatory response via paracrine signalling(Trisnawaty, Gunadi et al. 2024). Mediators that are released in the process of dermal remodelling like prostaglandins and endothelins are secondary triggers of melanogenesis. This forms a vicious cycle with the dermal damage caused by HEVL, where pigmentary changes are further perpetuated and increased and that is why conditions such as melasma are refractory in nature and both epidermal and dermal pathology are involved(Trisnawaty, Gunadi et al. 2024).

4.Clinical Menifestations :Pigmentation and beyond

4.1. Pigmentation of HEVL

This pigmentation appears as dark spots upon the face of the human being as compared to the body of a rat, mouse, or guinea pig.

Pigmentary reaction to HEVL is different as compared to that caused by ultraviolet radiation in both the temporal kinetics and clinical severity. These traits are important to undergo in learning of the special problems of visible light in clinical practice.

Immediate vs Persistent Pigmentation: HEVL induced two types of pigmentation, including immediate pigment darkening (IPD) and persistent pigment darkening (PPD), and delayed tanning (DT) . The darkening of pigment takes place within seconds-minutes of exposure to HEVL and is transient and appears as a temporary greyish discolouration due to the photo-oxidation of already present melanin(Vignesh Narayan and Sarkar 2024) . This instant action which is mostly seen in people with skin phototypes III-VI is an indication that there is a chemical modification of melanin polymers, as opposed to a de novo melanogenesis. Persistent pigment darkening is more clinically significant and occurs within hours and may take several days to weeks. PPD is the shift of the melanin photo-oxidation to the process of actual new melanin production, which implies the activation of the OPN3 pathway and downstream melanogenic enzymes(Eldiehy, Haraz et al. 2026). Delayed tanning, which can be compared to UV-induced tanning, happens days after sensitization and is an expression of transcriptional enhancement of melanogenic enzymes and augmented melanosome production and transfer them.

Duration and Intensity: The most notable feature of the HEVL induced pigmentation is the remarkable duration and intensity in the susceptible individuals. Earlier studies by Mahmoud and co-workers had already shown that visible light causes darker and longer-lasting pigmentation in comparison to the UVA1 radiation alone, in the pigmentation of IV- VI skin phototypes(Dimmers, Lück et al. 2025). This knowledge has been later developed to include other investigations that identified that HEVL-induced pigmentation may last as long as three months after a single exposure in people with skin of colour . This is in contrast to the pigmentation which is normally fades out within weeks due to UVA exposure. Its long duration is due to the inherent variations in the underlying processes: UVA mostly causes pigmentation by oxidative stress signaling and activating TRPA1, whereas HEVL uses the OPN3 receptor pathway, which causes a more sustained melanogenesis transcriptional programme. Quantitative studies show that HEVL is the cause of about 71 percent of visible-light pigmentation in the susceptible individuals which is the primacy of clinical significance of HEVL(Proietti, Battilotti et al. 2024).

4.2. Across Fitzpatrick Phototypes Susceptibility (FST)

The constitutive skin colour has a significant effect on the pigmentary response to HEVL and the difference between the response of the Fitzpatrick phototype scale is significant.

FST I- II: Minimal or No Pigmentation Response: Light-skinned people (FST I-II) respond qualitatively different to HEVL exposure. It used to be believed in times past that the visible light caused inconsequential pigmentation in the fair-skinned people. This, however, has been refined in more subtle research. Moreiras et al. conducted a pilot study to investigate the effect of blue (450nm) and green (530nm) light in skin histocultures of FST I-III donors(Darvin, Lademann et al. 2022). Surprisingly, although UVR only induced melanogenesis in FST II-III skin, blue and green visible light induced melanin production even in FST I skin, and no skin-specific DNA damage and apoptosis were associated with this activity. This is an indication that HEVL has the ability to induce melanogenesis in fair skin via different pathways, other than the genotoxic effects of ultraviolet radiation. Light-skinned people may have temporary erythema and slow tan with repeated exposures to HEVL shows, but the effects are less noticeable than those seen with phototypes darker in complexion. Oxidative stress and inflammation seem to be the major cutaneous manifestations of FST I-II in contrast to prolonged hyperpigmentation.

Pronounced and Sustained Pigmentation: As compared, people of colour (FST III-VI) have a strong and clinically meaningful pigmentary response to HEVL . This increased vulnerability is indicative of inherent variations in the melanocyte biology: the size of melanocytes in dark skin is higher, more active and widespread between the basal keratinocytes than in the darker skin tones(Bai, Chen et al. 2026). Besides, even melanin, which has UVB protective properties, is a photosensitiser to longer wavelengths, producing free radicals that increase the risk of photoaging and hyperpigmentation itself . The melanin-rich skin phenotype is therefore counterintuitive to become more responsive to the influence of HEVL, and pigmentation is more brown and long-term and mediated by the OPN3 pathway mentioned above. Research confirms that dark skin has a more pronounced oxidative stress response to visible light and UVA1, which has been highlighted as a reason why specific photoprotection measures are required in relation to such distinct susceptibilities(Salceda 2024).

4.3. Role in Hypermigmentary Disorders

There are direct implications of the unique features of HEVL-induced melanogenesis to pigmentary diseases that pose challenges to common and therapeutic interventions.

Melasma: Melasma is as far as the clinical example of the pathogenic role of HEVL. Melasma, which is characterised by symmetrical hyperpigmentation usually of the face, has long been considered to be induced by ultraviolet radiation but the tendency

to recur despite excellent UV protection led to the consideration of other contributing factors. It is now found that visible light especially blue light is a major driver of research(Dorf and Maciejczyk 2024). The visible light induces the dopachrome complex that causes sustained hyperpigmentation of melasma by stimulating the OPN3 pathway on melanocytes. This is the reason why the clinical picture is frustrating since high-SPF, broad-spectrum UV protection does not prevent disease recrudescence in patients adhering to it. The combined effect of UVA1 and visible light contributes to the development of pigmentary responses, and the experiments show the higher intensity of the pigments during the combined exposure than during the exposure to the visible light. Melasma is currently understood as a photodamage phenotype not limited to pigmentary alterations but also including vascular, disruption of basement membrane, and mast cell-mediated inflammation- all of which could be motivated by exposure to HEVL.

Post-Inflammatory Hyperpigmentation (PIH): HEVL is also an important contributor to the post-inflammatory hyperpigmentation. PIH is considered to be an acquired hypermelanosis after cutaneous inflammation or trauma, and it is most often severe and frequent in coloured skin. Inflammatory environment that PIH occupies, high concentration of prostaglandins, endothelins and other melanocyte-stimulating factors, provides a permissive environment in which pigment production can be enhanced by exposure to HEVL(Sharma and Malik 2025). The pathway on OPN3 can be increased during the ethos of inflammation, and melanogenic responses to even the small doses of HEVL can be exaggerated. Moreover, the inflammatory response caused by HEVL may lead to persistent low grade inflammation and extracellular degeneration of the dermis to perpetuate a chronic pigmentary state. In the patients who have survived acne, dermatological interventions or other inflammatory dermatoses, exposure to HEVL can cause PIH to last longer and become more severe when not treated, which can delay clinical recovery(Kimlin and Tenkate 2007).

4.4. Summary of Clinical Effects

Table 2:Clinical Effects of HEVL on Human Skin.

CLINICAL EFFECTS	WAVELENGHT (nm)	KEY MECHANISM	SUSCEPTIBLE POPULATION	EVIDENCE STRENGHT
Immediate Pigment Darkening	400-500	Photo-oxidation of pre-existing melanin	FST III-VI	High
Persistent Pigmentation	400-440	OPN3-mediated melanogenesis	FST III-VI	High
Erythema	400-500	Inflammatory response,thermal	AII FST (at high frequency)	Moderate
Skin Barrier Disruption	450	Increased TEWL,oxidative stress	FST I-III(studied)	Moderate
ECM Degradation	400-450	ROS'n MMP upregulation, collagen breakdown	AII FST	Emerging

5.Clinically Proven Strategies for HEVL Protection

5.1. Physical Blockers: The Best Standard

The foundation of HEVL protection is still on physical blockers, which operates through the incident radiation being reflected, scattered and absorbed at a wide spectrum. As opposed to organic UV filters, which can mainly absorb selected wavelengths, physical defense offers full coverage, which extends to the visible light range.

Iron Oxides (FeO): Iron oxides are the best type of agent in terms of visible light and HEVL protection. These coloring materials, which are usually incorporated in colored sunscreens and cosmetic foundations, are based on various mechanisms such as absorption, reflection as well as scattering of light throughout the entire visible spectrum . Iron oxides unlike ultraviolet filters have broad distribution of absorbance between 400 and 700nm, a characteristic inherently favored in HEVL protection(Wang, Wang et al. 2023). An all-time research by Kaye and others showed that, by the addition of iron oxide to scattering sunscreens, transmittance was significantly reduced compared to that determined by each individual component alone, indicating synergistic photoprotection. Quantitative studies have shown that iron oxide-containing preparations are capable of decreasing the HEVL transmission by 80 to 97 percent, which is considerably higher than the protection of untinted counterparts(Esmat, Hegazy et al. 2017). Pigmented Titanium dioxide (TiO2) and Zinc oxide (ZnO): Inorganic filters like titanium dioxide and zinc oxide have variable coverage in terms of HEVL protection that is very sensitive to particle size. Formulations larger in particle diameter, above 200 nm (non-micronized, or pigment-grade) are practical scatters and reflectors of visible light, and as such offer useful protection in HEVL(Yadav, Srivastava et al. 2025). Nevertheless, micronized and nanoparticle forms, which are created to remove the unappealing white cast of the traditional mineral sunscreens, lose its effect . The smaller the size of particles less efficient is the scattering and this forms a protection gap in the HEVL region whereby micronized zinc oxide is not as effective as a defence. It is this trade-off between cosmetic acceptability and photoprotection that has led to the investigation of alternative methods of combining the multiple agent classes(Khadamy 2024).

Clinical Evidence:

There exists strong clinical evidence that iron oxide-based formulations outperform the other iron oxide-based formulations in patients with pigmentary disorders. The two regimens of sun protection tested by Grimes, et al. on women with Fitzpatrick phototypes III-IV, III, VI were the use of SPF50 on its own and SPF50 combined with iron oxides in women with Fitzpatrick phototype III-IV over a 12 weeks period(Aurangabadkar 2020). Although, the two regimens showed an improvement in the skin, the melasma sub-group that used the iron oxide-based SPF50+FeO exhibited an early effect of overall healthy appearance in week 4(Pelletier-Aouizérate 2017). By week 12, 36 percent of melasma members of the SPF50+FeO group recorded better in skin radiance (L measurements) than none in the SPF50-alone group. Self-assessment forms verified that iron oxide formulations containing the iron oxide-enhanced the quality of life of participants with melasma, which proved the actuality of realized HEVL protection in the real world. These findings were further supported by a recent review by Zhou et al. who concluded that tinted sunscreens with iron oxides are the ones that should be recommended over non-tinted ones in patients with a tendency to develop hyperpigmentation disorders since they not only do not cause a relapse of melasma but they even augment the depigmenting effects of topical hydroquinone(Hernandez-Jimenez 2024).

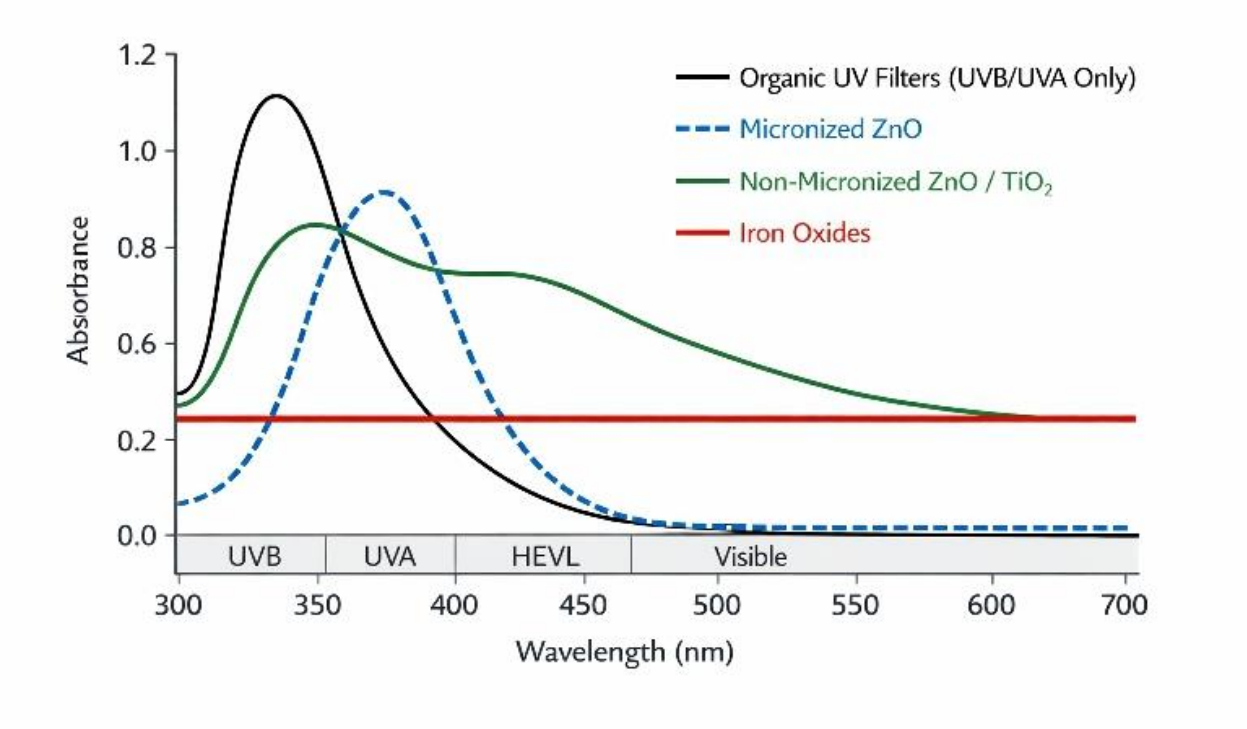

Figure 3: Spectral Absorbance of Common Sunscreen Filters

5.2. New Organic Filters: broadening the Spectrum

Innovations in organic filter technology in recent years have seen the beginning of filling the spectral gap between ultraviolet and visible protection.

TriAsorB™ (Phenylene Bis-Diphenyltriazine)**:**

TriAsorB 4 is an important addition to organic photoprotection, which is specifically formulated to expand absorbance up to the HEVL range. This new filter, also referred to as phenylene bis-diphenyltriazine (PBDT) features an absorbance up to 450 nm including the optimal 400- 450 nm range of the primary OPN3 mediated melanogenesis range(Najeeb, Suresh et al. 2021). Boyer et al. tested nine sunscreens by TriAsorB and their results showed them to be well-photostable with in vitro blue light photoprotection factors of between 30 and 50 percent BL. Representative formulations clinical analysis had shown a substantial blue light pigmentation prevention, and the colorimetric evaluation revealed a percentage photoprotective efficacy of 50.7 to 75.5 percentage whilst the visual analysis reflected a 31.2 to 72.7 percentage ($p < 0.001$) percentage(Techamontrikul, Raksasat et al.). These findings validate that TriAsorB containing preparations offer clinically significant HEVL protection to serve as an alternative to iron oxide containing preparations to patients desiring untinted preparations.

5.3. Antioxidant Defence

Since the damage caused by HEVL relies greatly on oxidative stress, antioxidants are a significant adjunctive approach .

1. **Topical Antioxidants:** L-ascorbic acid (vitamin C), 0.15 ml 1-tocopherol (vitamin E), and 0.2 ml ferulic acid have become a somewhat effective system of antioxidants(Karlsson 2015). Early studies led by Lin and others showed that ferulic acid stabilised vitamin C and vitamin E solutions and increased photoprotection of skin in vitro four-fold to about eight-fold against solar-simulated radiation of vitamins C and E respectively. Recently, Guiotto and others tested the validity of this antioxidant mixture as compared to visible light in dark skin phototypes (IV-V) using human skin explants (HUMAN 2015). Exposure to visible light augmented oxidative stress indicators (4HNE) and

pigmentation indicators and reduced collagen I expression and elevated MMP2 -adaptations that are all in line with photoaging and pigmentary vulnerability. The vitamin C, E and ferulic acid antioxidant mix pre-treatment was able to reduce these indicators substantially on days 2 and 3 and PCR analysis confirmed the antioxidant defence pathway (Nrf2-HMOX1) was activated. The results of the present studies favour the application of these antioxidant combinations to counteract the effects of HEVL especially in melanic skin typified by increased oxidative reactions(Al-Amoudi 2001).

2. **Specific Botanical Extracts:** There is some growing evidence that suggests the use of specific botanical extracts in the defence against HEVL. Extract Gardenia jasminoides has exhibited melatonin-like activity that perhaps controls circadian processes in the skin. Red rice extract and other polyphenol rich botanicals have a role in minimizing oxidative stress in numerous different ways, but clinical evidence on HEVL phenomena is scarce(McMenemy, Quinn et al. 2018).

3. **Oral Photoprotection:** Systemic photoprotection has the conceptual benefit of whole-body defence without necessarily being applied topically. Polypodium leucotomos extract (PLE) is a fern extract that is largely investigated in ultraviolet protection. Clinical evaluation and histological analysis of 17 of 22 subjects and all 22 subjects, respectively, by Kohli et al., have shown that PLE suppresses UVB-induced alterations(Sulym). Although the direct evidence to support PLE against the HEVL is unavailable, the antioxidant effect on the product implies the possibility of its utilization as an adjunction agent. The carotenoids, such as lycopene and lutein, build up in skin and could have a systemic antioxidant protection against the HEVL-induced oxidative stress but clinical evidence is required to substantiate a claim of efficacy(Pullicino 2023).

5.4.Summary of Protection

Table 3:Efficacy of Clinically Available HEVL Protection Strategies

PROTECTION STRATEGY	KEY COMPONENTS	HEVL BLOCKING EFFICACY	AESTHETIC ACCEPT-ABILITY	CLINICAL EVIDENCE (Pigmentation)
United Sunscreen	Organic UV filters	Poor (<20%)	Excellent	Ineffective alone
Mineral Sunscreen	Micronized ZnO/TiO2	Moderate (20%-40%)	Good	Limited
Pigmented Mineral	Non-Micronized TiO2/ZnO2	Good (40%-60%)	Variable (tint match)	Moderate
Tinted Sunscreen(with FeO)	Iron Oxides+UV filters	Excellent (80%-97%)	Good to Excellent	High (prevents melasma relapse)
Novel Organic Filters	TriAsorb	Good (absorption up to 450nm)	Excellent	High (in trials)
Antioxidants (Topical)	Vit C+E+Ferulic Acid	Neutralizes ROS, not blocking	Excellent	Moderate (adjunctive)

New Therapeutic Interventions and Future Cure: Attack the Pathway.

6. Emerging Treatments and Future Cures: Targeting the Pathway

6.1. The Therapeutic Gap

Although major progress has been made in comprehension of the molecular pathways of pigmentation induced by HEVL, a critical lack of translational alignment between targets of mechanistic interventions and clinical intervention tools remains. Existing photoprotection measures, although successful at

preventing or attenuating HEVL radiation are essentially broad-spectrum, as opposed to targeted. ES Formulations containing iron oxide and new organic filters like TriAsorB 7 offer outstanding physical defense by preventing the melanocytes to be affected by HEVL(de la Caridad Hernandez, Eckembrecher et al. 2024). Nevertheless, after the photons enter through inappropriate application, formulation, or inherent coverage constraints, the OPN3-induced melanogenic cascade continues despite this. The therapeutic gap is that there are no topical agents that could prevent this signalling pathway at the receptor or at the transcriptional level and provide biological intervention so that physical protection is supplemented(ZAINI 2022).

6.2. Blocking the First Signal: Opsin-3 Antagonists.

The discovery of Opsin-3 (OPN3) as the main photoreceptor involved in the melanogenesis induced by HEVL has created new possibilities of targeted intervention as never before (Opsin-3, 2010). Molecules that have the potential to inhibit the OPN3 receptor on melanocytes are actively being studied in pre-clinical studies with the aim of preventing the initial sensing of the HEVL photons(Avram 2022). These would be a new category of photo-cosmeceuticals that are not simply filters of light, but make melanocytes insensitive to light . These compounds would antagonize OPN3, block the influx of calcium, block the phosphorylation of CREB, and abrogate the stimulation of MITF at its source effectively silencing the melanogenic response at its source. Although there are certain OPN3 antagonists in pre-clinical development, the therapeutic potential of this target was validated by the studies with siRNA, which supports the therapeutic potential of this target(Motamedi and Swinnen 2024) . Selective, topically administered OPN3 inhibitors are in development, and would be a priority of future generations of anti-pigment therapy.

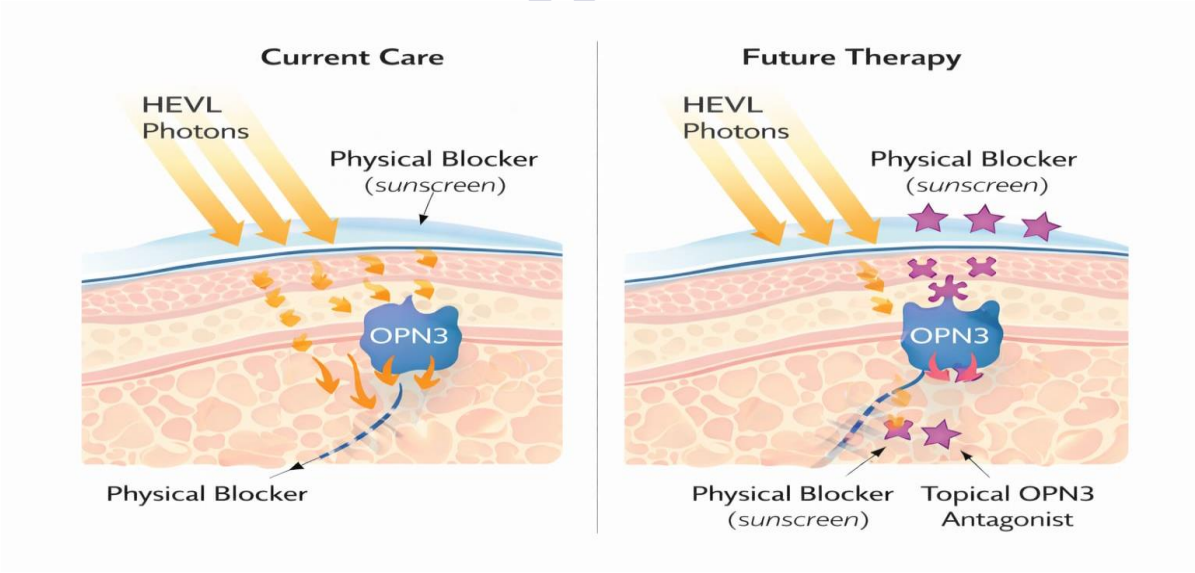

Figure 4: Current Standard of Care vs Future Targeted Therapy

6.3. MITF Inhibition: Attacking the Master Regulator

Below OPN3, there is microphthalmia-associated transcription factor (MITF) which is the master regulator that coordinates the expression of melanogenic enzymes downstream(Zhao 2026). The alternative point of intervention with the possibility of successful intervention irrespective of the activated upstream pathways is targeting MITF. Small molecules and peptides which suppress the activation of MITF, in particular when associated with HEVL exposure are being investigated(Caetano, Corrêa et al. 2022). These would decrease transcription of tyrosinase (TYR),

tyrosinase-related protein 1 (TRP-1) and dopachrome tautomerase (DCT), and thus lower the capacity to produce melanin . The difficulty is in attaining melanocyte specific action without disrupting the physiological activities of MITF in melanocyte survival and functioning. Evidence of inhibition of MITF in melanoma settings is shown by pre-clinical studies but dermatological studies would need to focus on dose-optimisation to prevent hypopigmentation or cytotoxicity(Mah, Chamyuang et al. 2025).

6.4. Specific Action Novel Antioxidants: Mitochondrial ROS

- HEVL produces reactive oxygen species (ROS) by different processes including mainly mitochondrial dysfunction and photosensitiser excitation and superoxide anions (O2) are the leading species. This knowledge has prompted the creation of new generation antioxidants tailored to address a particular subcellular location and type of ROS (Panwar and Jain 2022).
- Special mention can be made of mitochondria-targeted antioxidants. MitoQ (ubiquinone conjugated to a lipophilic triphenylphosphonium cation), SkQ1, and various other compounds are mitochondrion-selective superoxide scavengers (MitoQ). The pre-clinical investigations show that these agents inhibit mitochondrial DNA damage, maintain oxidative phosphorylation competency, and decrease down-stream MMP activation in reaction to oxidative stress(Tenenbaum, Katsambas et al. 2021) . In the case of HEVL-specific applications, mitochondrial-targeted antioxidants have the theoretical benefits of defence concentration at the main site of superoxide formation compared to non-specific antioxidants(Dong 2022).
- Another group of specific antioxidants is superoxide dismutase (SOD) mimics. These artificial molecules catalyze superoxide dismutation with kinetics comparable to native enzymes that give efficient neutralisation of O2 without the stability issues of protein-based therapeutic molecules(Arroyo 2024). These agents might be developed into topical products to specifically reverse the flux of superoxide anion that is a property of HEVL exposure.

6.5. Repairing the Damage Lipofuscin Clearance and ECM Restoration

- In addition to inhibiting pigmentation, the new approach aims at repairing the damages caused by photodamage, and lessening the susceptibility of the cutaneous to subsequent exposure of HEVL in the future .
- Lipofuscin Clearance Lipofuscin is an auto-fluorescent lipopigment which is formed in old and photodamaged skin, and is an endogenous photosensitiser which increases the oxidative stress caused by HEVL an effect amplified by lipofuscin(Nascimento, Fernandes et al. 2023). Lipofuscin clearance and lipofuscin formation inhibitory agents are another new area of lessening the baseline photosensitivity of the skin. The compounds may in theory reduce the damage caused by HEVL by eliminating the chromophores which enhance photochemical reactions(Bagalkote 2024) .
- ECM-Repairing Peptides: HEVL-induced ROS activate matrix metalloproteinases (MMPs) and MMP-1, which results in collagen and elastin degradation that causes photoaging as well as secondary pigmentary alterations(Buckett 2023) . MMP-1 inhibitors have turned out to be effective promising in counteracting such damage. The MMP-1 catalytic domain has small molecule inhibitors which target the catalytic domain, peptide-based inhibitors which mimic collagen structure, and natural compounds including flavonoids have been shown to be effective in pre-clinical studies. Topical formulas have been subjected to clinical studies demonstrating that the volume of wrinkles on the face as well as the depth of wrinkles can be greatly reduced (55.8 and 32.8 respectively) in eight weeks under the influence of MMP inhibition and the consequent collagen preservation . Outside the realm of anti-aging functionality, these types of inhibitors can be used to preserve extracellular matrix integrity, suppressing the pro-inflammatory and pro-pigmentary cues caused by degraded dermi MMP modulation is being clinically validated using

natural bioactive peptides with multi-target mechanisms(Bui, McDaniels et al. 2020).

6.6. Pipeline Summary

Table 4:Emerging Pipeline Agents for Targeted HEVL Protection/Treatment

AGENT CLASS	TARGET / MECHANISM	STAGE OF DEVELOPMENT	POTENTIAL INDICATION
OPN3 Antagonistic	Blocks HEVL sensing in melanocytes	Pre-clinical	Melasma, PIH prevention
MITF Inhibitors	Downregulates melanogenesis enzyme production	Early Clinical (for rare cancers)	Generalized hyperpigmentation
Mitochondria-Targeted Antioxidants	Scavenges superoxide at the source	Pre-clinical/Cosmeceutical	Anti-aging, anti-pigmentation adjunct
Lipfuscin-degrading agents	Reduces photosensitizer load in cells	Pre-clinical	Photoaging, pigmentation

7.The Critical Gap and Discussion.

7.1. Summary of Evidence: HEVL is Non-Negligible Factor of Skin Pigmentation

The overall evidence below this review confirms that high-energy visible light is a biologically active and clinically important part of solar radiation that cannot be overlooked in the modern photodermatology practice. In the melanocytes, specifically at 400-450nm, HEVL triggers a unique molecular pathway via Opsin-3 (OPN3) receptor (resulting in pigmentation that is more intense and longer-lived compared to that induced by the UVA radiation alone).(Regazzetti, Sormani et al. 2018) This is more exaggerated among people of skin of colour (Fitzpatrick phototypes III-VI who have an increased melanocyte responsiveness and form the largest proportion of the global population suffering hyperpigmentary syndromes like melasma and post-inflammatory hyperpigmentation. In addition to pigmentation, HEVL induces oxidative stress, matrix metalloproteinase (MMPs), depletion of dermal extra-cellular matrices, and destruction of skin barrier, which work together with UVA1 in effects on photoaging(Piffaut, De Dormael et al. 2025). All of these discoveries require a paradigm shift in photoprotection: all-inclusive sun defence should not be limited to ultraviolet radiation but should also cover the visible spectrum as well(Shahin and Samea 2010).

7.2. Rewriting the Translational Gap: Molecular Targets lack Clinical Tools.

Although the molecular mechanism of HEVL-induced pigmentation has been greatly elucidated, there has been a large translational gap between mechanistic literacy and interventions clinically available . The potent and targeted means to disrupt the HEVL response on the basis of interrupting the source of the system are the OPN3 receptor, microphthalmia-associated transcription factor (MITF), and the downstream melanogenic enzymes, namely, tyrosinase, TRP-1, and DCT. Nevertheless, pathway-specific topical therapy continues to be deprived of clinical armamentarium in favor of physical blockers and non-specific antioxidants(Visser, Amien et al. 2026). Tinted sunscreens which contain iron oxide offer excellent protection against HEVL i.e. reducing the transmission by 80-97 percent and showing clinical efficacy in preventing relapse of melasma. New organic filters like TriAsorB™ can be used to extend the absorbance into the HEVL range with great cosmetic acceptability. The combination of antioxidants (vitamin C, E and ferulic acid) helps to reduce the oxidative stress indicators in the skin exposed to HEVL. They all

work, however, by blocking the entry of photons to melanocytes or reversing downstream oxidative injury- they do not intervene in the melanogenic cascade after it has been started(Lyons, Trullas et al. 2021). There is still a severe gap in therapeutic solutions a pathway-specific, OPN3 or MITF-targeted cream is not ready yet to enter routine clinical practice. Positively, current randomised controlled studies show that a non-pigmented bioactive compound, 2-mercaptonicotinoyl glycine (2-MNG), inhibits persistent pigmentation induced by HEVL by over one-third, which is the first example of a pathway-specific intervention to be validated(Visser, Amien et al. 2026). This preclinical demonstration highlights why and how topical therapy that targets reduction of risk behaviours like physical protection should be developed urgently.

7.3. Hurdles to the Progress: Clinical Trial Design, Standardisation and Regulation

There are several obstacles to the transfer of the mechanistic findings to the clinical practice. To begin with, the design of clinical trials of HEVL is not ordinary. No standardized protocol exists in terms of inducing and measuring HEVL-induced pigmentation; that is, it is present that studies employ divergent light sources, dosimetry, exposure regimens and outcome measures hence making comparing cross-trial results and meta-analyses difficult(Francois-Newton, Kolanthan et al. 2022). Second, a unified system of :labelling the factors of Blue Light Protection (BPF) is still not standardised, which negatively affects clinical instructions and consumer education considerably. In contrast to ultraviolet protection, which has the advantage of internationally recognised SPF and PA ratings, HEVL protection is not tested or labelled (regulated) by any country. Such a regulatory gap makes clinicians powerless to make subjective comparisons of products, and patients susceptible to the false marketing premises(Diffey and Farr 1991). Third, since tinted sunscreen formulations are heterogeneous, differing in iron oxide content, particle size, and shade, not all products labeled tinted offer the same level of HEVL protection and this makes clinical recommendations even more difficult to make(Schalka, de Paula Corrêa et al. 2019). Fourth, although oral antioxidants (Polypodium leucotomos extract and carotenoids) have potential to systemically protect against photolysis, there is limited direct evidence related to their efficacy in protecting against HEVL specifically, and standardised dosing measures are still deficient in this area(Jo, Jung et al. 2020).

7.4. A Call to Standardisation: The Immediate Mover to International Testing Protocols

Managing these obstacles should involve a concerted effort on the part of scientists, clinicians, regulatory agencies and the industry actors. Internationally accepted in vivo and in vitro testing procedures are urgently required to quantitatively and qualitatively quantify and label HEVL protection, just as SPF and PA rating scales do with ultraviolet radiation(Mohammad, Kohli et al. 2019). This kind of standardisation would allow comparing the efficacy of products objectively, making clinical recommendations evidence-based, and empower consumers to make informed decisions. Encouraging progress has been made: visible light protection factor methods based on colorimetry and in vitro transmittance techniques predictive of in vivo photoprotection have been suggested and investigated in practice. A regulatory adoption and industry compliance would be achieved faster by harmonising these methodologies between international dermatology organisations in agreement with consensus statements(Zhang, Pu et al. 2024). At the same time, there is need to develop clinical trial guidelines to uphold strong and reproducible assessment of new targeted therapies, such as OPN3 antagonists, MITF suppressors, and mitochondrial-focused antioxidants. The recent approval of 2-MNG by randomised controlled trials indicates that intensive HEVL-specific clinical research is doable and must be used as a template to the forthcoming creation of drugs. It is only through this kind of standardisation and regulatory development that the translational gap can be closed, providing on the promise of pathway-specific intervention of the millions of

patients with HEVL-induced hyperpigmentation the remedy(Wang, Lu et al. 2020).

8.Conclusion

The visible light of high energy (HEVL) is a biologically active solar radiation, which can no longer be neglected in contemporary photodermatology. It activates the OPN3 receptor of melanocytes at wavelengths of 400-450 nm and leads to a darker and longer-lasting pigmentation compared to UVA alone(Regazzetti, Sormani et al. 2018). This effect is most conspicuous in Fitzpatrick skin types III-VI, who bear the greatest worldwide risk of melasma and post-inflammatory hyperpigmentation. In addition to pigmentation, HEVL induces oxidative stress, MMP activation, degradation of dermal matrix, and disrupting the skin barrier, all which cooperate with UVA1 to promote photoaging. The results require an overhaul of photoprotection approach - UV filters are not enough(Pfeifer and Besaratinia 2012). Tinted sunscreen with iron oxide is now the best line of defense against HEVL with proven clinical effectiveness in preventing melasma (reducing transmission by 80-97%). In the meantime, a fresh series of pathway-targeted biologic agents against OPN3, MITF and melanogenic enzymes is developing(Cole, FOrbes et al. 1986). The RCT-validated compound 2-MNG constitutes the first evidence that the intervention against pigmentation caused by HEVL on a receptor level can be effected at a clinical scale. Nevertheless, significant challenges still exist - specifically the fact that there are no standardised HEVL testing guidelines, that BPF labelling is not regulated, and not all clinical trials are designed. Regulations need to be changed to require HEVL protection tests similar to SPF and PA ratings. Photoprotection of the future is a multi-modal approach with broad-spectrum UV filters, formulations with iron oxide, and targeted biologic therapy that directly silences the melanogenic cascade at its point of origin(Zhang, Pu et al. 2024).

REFERENCES

Al-Amoudi, O. M. S. (2001). The effect of a long pulsed dye laser on photodamaged skin and facial telangiectasia, Boston University.

Arroyo, R. G. (2024). CERKL as a Modulator Gene of Mitochondrial Dynamics and Stress Response in the Retina, Universitat de Barcelona (Spain).

Aurangabadkar, S. (2020). Pigment specific lasers and light devices. Practical introduction to laser dermatology, Springer**:** 91-117.

Avram, M. R. (2022). "Hair Transplantation for Women and Men." Comprehensive Textbook of Cosmetic Dermatology, Laser and Energy-based Therapies: 112.

Bagalkote, P. M. (2024). "Millets Sustainable Grain for a Healthy Future (2)." ABS.

Bai, B.-X., et al. (2026). "Chinese Expert Consensus on the Clinical Application of Photobiomodulation Therapy in Dermatology (2025)." International Journal of Dermatology and Venereology **9**(1): 1-11.

Barolet, A. C. (2025). "Impact of low-intensity non-Ionizing light on human skin, pigmentation and cardiovascular health."

Bharathi, B., et al. (2025). "Photonic frontiers in melasma: Navigating layers, risks, and the future of light-based therapeutics." Cosmoderma **5**.

Buckett, L. (2023). The Human Metabolism of the Bioactive Prenylated Flavonoid Xanthohumol from Hops (Humulus Lupulus), Technische Universität München.

Bui, M. X., et al. (2020). "McNair Scholars Research Journal 2020."

Caetano, N. S., et al. (2022). Microalgae for pigments and cosmetics. Algal Biorefineries and the Circular Bioeconomy, CRC Press**:** 133-176.

Chauhan, A. and N. Gretz (2021). "Role of visible light on skin melanocytes: a systematic review." Photochemistry and photobiology **97**(5): 911-915.

Cole, C. A., et al. (1986). "An action spectrum for UV photocarcinogenesis." Photochemistry and photobiology **43**(3): 275-284.

Damodaran, A. and N. Nair (2022). Skin Pigmentation and Cosmetic Considerations for Even Skin Tone. Pigmentation Disorders-Etiology and Recent Advances in Treatments, IntechOpen.

Darvin, M. E., et al. (2022). "Carotenoids in human skin in vivo: antioxidant and photo-protectant role against external and internal stressors." Antioxidants **11**(8): 1451.

de la Caridad Hernandez, L. E., et al. (2024). Advances in aesthetic dermatology. Telemedicine and technological advances in dermatology, Springer**:** 329-365.

De La Garza, H., et al. "Pigmentation Disorders."

de Tollenaere, M., et al. (2025). "Persicaria tinctoria Extract Mitigates UV-Associated DNA Damage and Inflammation, While Boosting Vitamin D3 and Melanin in Human Skin." Cosmetics **12**(6): 237.

Diffey, B. and P. Farr (1991). "Sunscreen protection against UVB, UVA and blue light: an in vivo and in vitro comparison." British journal of dermatology **124**(3): 258-263.

Dimmers, F., et al. (2025). "SUNSCREEN EFFICACY AGAINST UVA1-AND VISIBLE LIGHT-INDUCED SKIN PIGMENTATION IS INFLUENCED BY ETHNICITY." medRxiv: 2025.2012. 2016.25342374.

Dong, Y. (2022). Engineering Injectable, Radiopaque Hydrogels For X-Ray Imaging And Therapeutic Delivery For Cancer Treatments, University of Pennsylvania.

Dorf, N. and M. Maciejczyk (2024). "Skin senescence—from basic research to clinical practice." Frontiers in Medicine **11**: 1484345.

Edwar, S. Q., et al. (2025). "Sunscreen Use in Populations of Colour: A Comprehensive Review of Barriers, Efficacy, and Clinical Recommendations." Jurnal Profesi Medika: Jurnal Kedokteran dan Kesehatan **19**(1): 69-77.

Eldiehy, K. S., et al. (2026). "Microalgal Biofactories: Sustainable Solutions for Nutrition and Cosmetics." Phycology **6**(1): 17.

Esmat, S., et al. (2017). "Phototherapy and combination therapies for vitiligo." Dermatologic clinics **35**(2): 171-192.

Francois-Newton, V., et al. (2022). "The protective effect of a novel sunscreen against blue light." International Journal of Cosmetic Science **44**(4): 464-476.

Furukawa, J. Y., et al. (2021). "Skin impacts from exposure to ultraviolet, visible, infrared, and artificial lights–a review." Journal of Cosmetic and Laser Therapy **23**(1-2): 1-7.

Gilaberte, Y., et al. (2026). "Photoprotection for Skin Cancer: What's New." Cancers **18**(4): 634.

Guarnieri, T. (2024). "Light sensing beyond vision: focusing on a possible role for the FICZ/AhR complex in skin optotransduction." Cells **13**(13): 1082.

Hang, X., et al. (2026). "Low Sunscreen Awareness and Misconceptions in Skin of Colour With Acne-Related Post-Inflammatory Hyperpigmentation: A Cross-Sectional Study Highlighting the Need for Targeted Education." Australasian Journal of Dermatology.

Hartmann, D. and F. Valenzuela (2024). "Sunproofing from within: A deep dive into oral photoprotection strategies in dermatology." Photodermatology, photoimmunology & photomedicine **40**(4): e12985.

He, X., et al. (2023). "The emerging role of visible light in melanocyte biology and skin pigmentary disorders: friend or foe?" Journal of Clinical Medicine **12**(23): 7488.

Hernández-Bule, M. L., et al. (2024). "Unlocking the power of light on the skin: a comprehensive review on photobiomodulation." International journal of molecular sciences **25**(8): 4483.

Hernandez-Jimenez, E. (2024). Lasers in Cosmetic Dermatology & Skincare Practice, Cosmetics & Medicine Publishing LLC.

HUMAN, R. I. (2015). "20% UVA Epidermis." Sunlight, Vitamin D and Skin Cancer: 430.

Jo, H. L., et al. (2020). "Clinical evaluation method for blue light (456 nm) protection of skin." Journal of Cosmetic Dermatology **19**(9): 2438-2443.

Kaltchenko, M. V. and A. L. Chien (2025). "Photoaging: Current Concepts on Molecular Mechanisms, Prevention, and Treatment: MV Kaltchenko1, AL Chien." American Journal of Clinical Dermatology **26**(3): 321-344.

Karlsson, M. (2015). Sun exposure, prevalence and localization of common melanocytic naevi in Swedish children, Karolinska Institutet (Sweden).

Khadamy, J. (2024). "Nutrients for Eye Health and Disease Management." Retrieved May.

Kimlin, M. G. and T. D. Tenkate (2007). "Occupational exposure to ultraviolet radiation: the duality dilemma." Reviews on environmental health **22**(1): 1-38.

Lee, K. W. A., et al. (2025). "Importance of Using Sunscreen After Light or Laser Facial Treatment: A Literature Review." Life **15**(9): 1484.

Lyons, A. B., et al. (2021). "Photoprotection beyond ultraviolet radiation: a review of tinted sunscreens." Journal of the American academy of dermatology **84**(5): 1393-1397.

Mah, S. H., et al. (2025). Taylor's University Graduate Research Symposium 2024: Beyond the Framework, Leaping Between Disciplines. Taylor's University Graduate Research Symposium 2024: Beyond the Framework, Leaping Between Disciplines.

Mahrous, M. H., et al. (2025). "Efficacy of Natural Products as Tyrosinase Inhibitors in Hyperpigmentation Therapy: Anti-Melanogenic or Anti-Browning Effects." Chemistry & Biodiversity **22**(8): e202403324.

McDaniel, D., et al. (2018). "Atmospheric skin aging—Contributors and inhibitors." Journal of Cosmetic Dermatology **17**(2): 124-137.

McMenemy, C., et al. (2018). "British Society for Investigative Dermatology Annual Meeting 2018 26–28 March 2018 Blizard Institute, Barts and The London School of Medicine and Dentistry."

Mohammad, T. F., et al. (2019). "Oral polypodium leucotomos extract and its impact on visible light-induced pigmentation in human subjects." J Drugs Dermatol **18**(12): 1198-1203.

Motamedi, S. and J. Swinnen (2024). "AMPK-mediated modulation of lipid droplets shapes melanoma cell vulnerability to ferroptosis."

Mun, S. J., et al. (2025). "Sunscreens in pigmentary disorders: time to revise the message." Photochemical & Photobiological Sciences **24**(2): 215-225.

Naharro-Rodriguez, J., et al. (2025). "Decoding skin aging: A review of mechanisms, markers, and modern therapies." Cosmetics **12**(4): 144.

Najeeb, S., et al. (2021). Regulation of ROS in Skin Stem Cells for Cancer Therapeutics. Handbook of oxidative stress in cancer: therapeutic aspects, Springer**:** 1-24.

Nascimento, T., et al. (2023). Microalgae proteins as a sustainable food supply. Microbiology for Cleaner Production and Environmental Sustainability, CRC Press**:** 37-71.

Nasim, M., et al. (2024). "Novel paradigm of therapeutic intervention for skin cancer: challenges and opportunities." Future Journal of Pharmaceutical Sciences **10**(1): 112.

Nie, J., et al. (2026). "Clinical efficacy and predictive factors of photodynamic therapy combined with recombinant mussel mucin repair dressing in treating post-inflammatory hyperpigmentation." Frontiers in Medicine **12**: 1654474.

Nie, X., et al. (2025). "Pigmented skin spots: Morphological characteristics, common causes, and treatment with cosmetic ingredients." Journal of Dermatologic Science and Cosmetic Technology: 100125.

Panwar, R. and A. P. Jain (2022). "RecentAdvances In PharmaceuticalSciences."

Passeron, T., et al. (2026). "An Investigator-Blinded, Randomized Trial of a Broad-Spectrum Sunscreen Containing Sclareolide and Niacinamide for the Prevention of Post-inflammatory Hyperpigmentation in Skin of Color." Dermatology and Therapy **16**(1): 405-421.

Passeron, T., et al. (2025). "Global consensus on the management of melanin hyperpigmentation disorders." Journal of the European Academy of Dermatology and Venereology.

Pelletier-Aouizérate, M. (2017). Photobiomodulation and light-emitting diodes. Cosmetic Medicine and Surgery, CRC Press**:** 409-430.

Peñín, B., et al. (2026). "Integrated Visible Light and Oxidant Protection Through Multifunctional Sunscreens." Advanced Healthcare Materials: e71125.

Pfeifer, G. P. and A. Besaratinia (2012). "UV wavelength-dependent DNA damage and human non-melanoma and melanoma skin cancer." Photochemical & Photobiological Sciences **11**(1): 90-97.

Piffaut, V., et al. (2025). "Topical prevention from high energy visible light-induced pigmentation by 2-mercaptonicotinoyl glycine, but not by ascorbic acid antioxidant: 2 randomized controlled trials." Frontiers in Pharmacology **16**: 1651068.

Proietti, I., et al. (2024). "Efficacy and tolerability of a microneedling device plus exosomes for treating melasma." Applied Sciences **14**(16): 7252.

Pullicino, J. (2023). "Analysis of Opuntia ficus-indica triggered cellular mechanisms in the protection against cellular stressor."

Purbhoo-Makan, M., et al. (2022). "The effects of blue light on human fibroblasts and diabetic wound healing." Life **12**(9): 1431.

Rajan, R. (2024). Effects of Sun Exposure in Skin of Colour. Sunscreens for Skin of Color, Springer**:** 91-110.

Regazzetti, C., et al. (2018). "Melanocytes sense blue light and regulate pigmentation through opsin-3." Journal of Investigative Dermatology **138**(1): 171-178.

Rocafort, S. L., et al. (2026). "Innovations in Refractory Melasma: Mechanism-Based Management and Emerging Therapeutic Strategies." Cureus **18**(4).

Salceda, R. (2024). "Light pollution and oxidative stress: effects on retina and human health." Antioxidants **13**(3): 362.

Salceda, R. (2024). Light Pollution and Oxidative Stress: Effects on Retina and Human Health. Antioxidants 2024, 13, 362.

Samaan, C. B. and T. V. Cartee (2023). "Treatment of periorbital vascularity, erythema, and hyperpigmentation." Clinics in Plastic Surgery **50**(3): 399-409.

Schalka, S., et al. (2019). "A novel method for evaluating sun visible light protection factor and pigmentation protection factor of sunscreens." Clinical, Cosmetic and Investigational Dermatology: 605-616.

Shahin, A. and M. Samea (2010). "Developing the models of service quality gaps: a critical discussion." Business Management and Strategy **1**(1): 1.

Sharma, M. and N. Malik (2025). "Gut-Skin Axis: Unraveling the Role." Vitamins and Human Health: 79.

Stanescu, C., et al. (2025). "Skin aging and carotenoids: A systematic review of their multifaceted protective mechanisms." Nutrients **17**(16): 2596.

Sulym, H. "DYNAMICS OF RAT SKIN RECOVERY AFTER ULTRAVIOLET IRRADIATION WITH AND WITHOUT THE USE OF HYDROQUINONE."

Szerej, K., et al. (2024). "Unveiling the hidden dangers: the impact of blue light on skin health and aging." Quality in Sport **30**: 56775-56775.

Techamontrikul, S., et al. "Efficacy and Safety of an Ultraviolet Radiation Forecasting Model (SURF-UV-NET) Assisted Heliotherapy for Psoriasis: A Split-Side Randomized Controlled Trial."

Tenenbaum, A., et al. (2021). Cosmetic Dermatology. Atlas of Dermatology, Dermatopathology and Venereology: Cutaneous Infectious and Neoplastic Conditions and Procedural Dermatology, Springer: 599-656.

Trisnawaty, S., et al. (2024). "Carotenoids in red fruit (Pandanus conoideus Lam.) have a potential role as an anti-pigmentation agent." Biomedical Reports **20**(3): 54.

Ugwueke, G. and N. Elbuluk (2026). "Beyond Conventional Paradigms: Rethinking the Evaluation and Management of Melasma."

Vibriani, A., et al. (2025). "Blue Light Exposure Disrupts Circadian Rhythms and Accelerates Skin Photoaging through Coordinated Cellular and Molecular Mechanisms."

Vibriani, A., et al. (2026). "Impact of acute blue light irradiation on the molecular clock and markers associated with photoaging in skin cell models." Journal of Molecular Medicine **104**(1): 61.

Vignesh Narayan, R. and R. Sarkar (2024). "Etiopathogenesis of Melasma." Melasma: A Monograph: 8.

Visser, W. I., et al. (2026). "Visible Light Protection Strategies for Diverse Populations." Dermatology and Therapy: 1-22.

Wang, H., et al. (2024). "Non-ionizing radiation-induced cellular senescence and age-related diseases." Radiation Medicine and Protection **5**(2): 68-74.

Wang, X., et al. (2023). "Polyphenolic natural products as photosensitizers for antimicrobial photodynamic therapy: recent advances and future prospects." Frontiers in Immunology **14**: 1275859.

Wang, Y., et al. (2020). "657 TGF-β2 upregulates OPN3 in human epidermal melanocytes independent of TGF-β2R in vitro." Journal of Investigative Dermatology **140**(7): S89.

Yadav, V. K., et al. (2025). "Melanin-Driven Delayed CPD Formation Is Independent of Melanin Biosynthesis Pathway." Journal of Investigative Dermatology.

ZAINI, D. (2022). "REGENERATIVE STEM CELLS IN AESTHETIC MEDICINE AND ITS EFFECT ON SKIN AGEING."

Zhang, R., et al. (2024). "Blue light protection factor: a method to assess the protective efficacy of cosmetics against blue light-induced skin damage in the Chinese population." Photochemical & Photobiological Sciences **23**(4): 711-718.

Zhao, C. (2026). "Artificial cell nanocapsules containing quercetin and/or nanobiotherapeutic (PolyHb tyrosinase): effect on five cancer cell lines: melanoma, lung, liver, breast, and cervix."

Ziveh, T., et al. (2025). "Biological and Therapeutic Responses of Human Skin to Different Wavelengths of Light: A Comprehensive Review." Journal of Lasers in Medical Sciences **16**: e69.