



Biological and Therapeutic Responses of Human Skin to Different Wavelengths of Light: A Comprehensive Review

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Abstract

Introduction: Light-based therapies have emerged as promising, noninvasive approaches in dermatology and regenerative medicine. Different wavelengths within the visible and near-infrared spectrum produce distinct biological effects on skin tissue, influencing pigmentation, inflammation, wound healing, collagen synthesis, and aging.

Objective: This review aims to summarize current evidence on cellular and molecular mechanisms underlying skin-specific responses to various wavelengths within the visible and near-infrared spectrum, including violet, blue, green, yellow, red, and near-infrared light.

Methods: A comprehensive literature review was conducted, including data from in vitro, in vivo, and clinical studies addressing wavelength-specific biological responses, therapeutic potential, and safety profiles.

Results: Findings suggest that violet light and blue light display antimicrobial and anti-keeloid activity but may also induce oxidative stress. Green light and yellow light mainly support wound healing, angiogenesis, and collagen preservation. Red light and near-infrared light are well established for photobiomodulation, skin rejuvenation, and wound repair, though optimal dosing and long-term safety require further study. Safety concerns such as oxidative stress, DNA damage, and pigmentation, especially in darker skin phototypes, are highlighted.

Conclusion: Light-based therapies hold significant promise for dermatological and aesthetic applications. Nonetheless, standardized treatment protocols and randomized clinical trials are essential to determine optimal wavelength parameters, dosing strategies, and long-term safety.

Keywords: Human skin; Ultraviolet (UV) radiation; Light wavelengths; Skin damage; Oxidative stress; Photobiological effects.



Introduction

The skin, as the largest organ of the body, plays a key role in protecting against environmental and stressful factors.¹ Sunlight is recognized as the most important source of ultraviolet, visible, and infrared radiation,² each of which can exert specific effects on the skin.³ This vital organ is composed of three main layers: epidermis, dermis, and hypodermis. Depending on its physical properties, optical characteristics, and light-absorbing molecules associated with each layer, light penetrates to different depths.⁴

The penetration and distribution of photons in the skin are influenced by phenomena such as reflection, scattering, and absorption. Approximately 6% of light is reflected from the skin surface, while most scattering is caused by the interaction of proteins and skin pigments.⁵

The solar radiation reaching the Earth consists of about 9% ultraviolet (UV), 50% visible light, and 40% infrared (IR). Major skin molecules such as keratin, collagen, melanin, and hemoglobin, along with vitamins, amino acids, and other macromolecules, play fundamental roles in absorbing and responding to these wavelengths.⁶

Research findings indicate that different wavelengths of sunlight have distinct and specific effects on the skin. For example, UVA radiation penetrates deeper, leading to oxidative stress and premature skin aging; blue light can induce cellular stress and pigmentation changes; and infrared radiation intensifies the negative effects of ultraviolet rays—each triggering specific skin responses.⁷

⁸ Nevertheless, ultraviolet radiation is beneficial for vitamin D synthesis and the treatment of certain diseases

such as psoriasis. Similarly, while blue light induces oxidative stress and impairs skin repair, it is also effective in reducing acne-causing bacteria.⁹ Understanding these differences is crucial for evaluating and designing effective protective agents for skin models with varying thicknesses, and it can contribute to improving methods for preventing and treating photodamage.¹⁰ Overall, these insights demonstrate that each type of light radiation has a specific role in skin damage and biochemical responses, and accurate knowledge of these mechanisms is essential for optimal skin protection.

Another group of factors includes endogenous photosensitizers (EPs), which absorb light in the skin and generate highly reactive excited states. They can directly react with surrounding molecules or induce oxidation by activating other molecules. This oxidation occurs via two pathways: Type I (contact-based reaction involving electron or hydrogen transfer) and Type II (generation of highly reactive singlet oxygen). Their function varies depending on tissue type and oxygen levels, and both processes can occur simultaneously. This mechanism underlies photosensitivity and photodamage in the skin.^{4,11,12}

This relatively new research field faces several limitations, such as the fact that the wavelength ranges and irradiation methods used in many studies do not accurately replicate real exposure conditions to sunlight or electronic devices.^{13,14} This article comprehensively investigates the effects of different wavelengths of visible light from solar radiation on the skin.

Methods

This review was conducted through a systematic literature search and critical analysis of peer-reviewed publications addressing the biological and therapeutic effects of different light wavelengths on human skin. Searches were performed in PubMed, Scopus, and Web of Science databases for articles published between January 2000 and December 2023. The search strategy combined controlled vocabulary (MeSH terms) and free-text keywords including visible light, photobiomodulation, blue light, green light, yellow light, red light, near-infrared light, wound healing, collagen synthesis, and skin rejuvenation.

All original experimental studies (in vitro and in vivo) and clinical trials investigating wavelength-specific effects on skin physiology, molecular mechanisms, or therapeutic outcomes were considered eligible. Inclusion criteria comprised (1) studies published in English, (2) clear reporting of wavelength ranges and exposure parameters, and (3) relevance to dermatological or regenerative contexts. Exclusion criteria included (1) non-English publications, (2) studies unrelated to skin tissue, (3) reviews, editorials, or conference abstracts without primary data, and (4) duplicate records. Reference lists of relevant reviews and included papers were manually

searched to capture additional sources. Extracted data covered wavelength characteristics, exposure duration and dose, cellular and molecular responses, and clinical implications. Findings were synthesized using a narrative approach, emphasizing mechanistic insights, therapeutic outcomes, and safety considerations across wavelength categories.

Light Spectra and Their Related Features

The composition of sunlight is such that approximately 44% of it consists of visible light with wavelengths ranging from 400 to 780 nm, while infrared (IR) radiation with wavelengths of 700 to 1440 nm accounts for about 53%, and ultraviolet (UV) radiation contributes to between 3 and 7% of the total energy. The ultraviolet portion itself is divided into three categories: UVA (315–400 nm), UVB (280–315 nm), and UVC (200–280 nm), each having different effects on human health. Additionally, the ozone layer plays a critical role in reducing the hazardous UVC radiation.

Overall, sunlight plays a fundamental role in supporting life on Earth, including providing the energy required for plant photosynthesis, regulating the biological cycles of living organisms, and contributing significantly to human visual processes and mental health.²

UV Radiation (100–400 nm)

Ultraviolet (UV) radiation is a part of the light spectrum with wavelengths from 100 to 400 nanometers, just below visible light, and is divided into three regions: UVA (320–400 nm), UVB (280–320 nm), and UVC (100–280 nm). The sun is the primary source of UV for humans, with approximately 95% of the radiation reaching the Earth's surface being UVA and only 5% being UVB, while UVC is almost completely absorbed by the ozone layer. Artificial sources, such as mercury lamps, tanning beds, and UVC disinfectant lamps, also contribute significantly to human exposure.

The biological effects of UV are dual. On the one hand, UVB is essential for vitamin D3 synthesis in the skin, and UV phototherapy has therapeutic applications in certain diseases¹⁵. On the other hand, excessive UV exposure leads to DNA damage through pyrimidine dimer formation, oxidative stress, and immune alterations. UVA penetrates the dermis and induces premature skin aging and collagen degradation via reactive oxygen species (ROS), while UVB, by forming thymine dimers, is the main cause of sunburn and skin cancers¹⁵. Therefore, chronic unprotected exposure carries significant health risks.

UVA (320–380 nm)

Exposure to UVA can cause structural changes in the skin, including epidermal hyperplasia and thickening of the stratum corneum.^{16,17} These effects mainly occur indirectly

through endogenous photosensitizers (EP) that generate reactive oxygen species (ROS) and damage keratinocytes, ultimately leading to increased keratin production and a thicker stratum corneum.^{18,19} These changes provide a partial protective mechanism against further UV damage, but they may also increase the risk of skin disorders such as actinic keratosis,²⁰ a precancerous lesion usually found on sun-exposed areas like the face, forehead, hands, and forearms. If untreated, it can potentially progress to squamous cell carcinoma.

UVB (280–320 nm)

Unlike UVA, which penetrates deeper layers of the skin and affects immune cells, UVB interacts primarily with the superficial cells of the skin.^{21, 22} It directly targets the DNA of skin cells and induces the formation of cyclobutane pyrimidine dimers (CPDs).^{23,24} These dimers create covalent bonds between thymine bases, disrupting the DNA double helix structure and potentially interfering with DNA transcription and replication. If unrepaired, these damages can lead to genetic mutations and skin cancers such as melanoma.²⁵ Cellular repair mechanisms, including nucleotide excision repair (NER), play a crucial role in removing these dimers and maintaining genomic integrity.²⁵ Meanwhile, melanin production in the skin increases after UVB-induced DNA damage and, by absorbing energy, protects DNA from further damage and reduces the risk of skin cancer.²⁶ This protective response helps lower the risk of genetic mutations, but severe or repeated damage may still increase the risk of UVB-related skin disorders.

Studies have shown that UVB exposure activates inflammatory pathways in the skin, accompanied by increased levels of pro-inflammatory markers such as TNF- α , IL-6, iNOS, and COX-2 in the skin.^{27, 28} These pro-inflammatory and inflammatory molecules induce various pathophysiological changes, which, along with elevated ROS production, exacerbate skin damage.^{28,29}

Molecular Mechanisms of UV (UVA/UVB) Effects on Skin

Ultraviolet radiation (UVR), as one of the most important components of sunlight, exerts complex and multifaceted effects on human skin, encompassing both beneficial and harmful outcomes. At the physiological level, UVR interacts with 7-dehydrocholesterol in the epidermis, leading to the synthesis of active vitamin D,^{29–31} a process that is essential for regulating calcium metabolism and maintaining bone strength.³² Moreover, low-dose UVR can have therapeutic effects, including antibacterial properties, acceleration of wound healing, and localized immunosuppression in conditions such as contact dermatitis.³² For this reason, narrowband UVB (311–312 nm) is clinically applied in the management of skin disorders like psoriasis and vitiligo,^{33,34} while UVA1 (340–400 nm) is used in the treatment of atopic dermatitis and

scleroderma.^{35, 36}

Alongside these beneficial effects, high doses or prolonged exposure to UVR can have detrimental consequences for skin health. The primary mechanism underlying this damage is the induction of reactive oxygen species (ROS) and oxidative stress, leading to the oxidation of lipids, proteins, and nucleic acids.³⁷ In addition to direct DNA damage,³⁸ this process causes the degradation of collagen and elastin in the dermal matrix,^{39, 40} contributing to premature aging, wrinkles, skin laxity, and an increased risk of skin cancers.⁴¹ Evidence suggests that UVA in the 330 nm range has a more pronounced effect on collagen degradation compared to UVB.⁴¹

At the molecular level, UVR activates multiple signaling pathways in epidermal cells, including the stimulation of activator protein-1 (AP-1) and the activation of kinases such as p38 MAPK and JNK.⁴² These changes modulate gene expression and disrupt cellular homeostasis, ultimately leading to inflammation, photoaging, and tissue disorders. However, this same capacity to stimulate biological responses provides a basis for the therapeutic use of UVR. Thus, ultraviolet radiation can be considered a dual-factor agent; depending on its intensity, wavelength, and duration of exposure, it can act both as a valuable therapeutic tool³⁵ and as a damaging factor in the pathophysiology of skin diseases.⁴³

Molecular Mechanisms Affected by Ultraviolet Radiation on Skin Structure Function

The skin, as the first defensive barrier of the body against environmental factors, is directly exposed to ultraviolet (UV) radiation. UVB rays are primarily absorbed in the epidermis and cause direct DNA damage in the form of cyclobutane pyrimidine dimers (CPDs) and 6-4 pyrimidine photoproducts (6-4PPs), leading to disruptions in transcription and cell proliferation.^{44, 45} These lesions activate the p53 gene and can result in cell cycle arrest or apoptosis of damaged keratinocytes.^{45, 46} In contrast, UVA penetrates deeper into the dermis and mainly acts through the production of reactive oxygen species (ROS) and reactive nitrogen species (RNS), causing DNA oxidation, strand breaks, and damage to proteins and membrane lipids.^{47, 48}

UV photons affect the skin via two mechanisms: direct absorption by chromophores and photosensitization. In the direct mechanism, molecules such as nucleic acids, certain amino acids (tyrosine and tryptophan), flavins, porphyrins, and urocanic acid (UCA) absorb UV light and induce biological responses.⁴⁹

In the indirect mechanism, endogenous or exogenous photosensitizers produce ROS and RNS. Reactive species include hydroxyl radicals, superoxide anions, singlet oxygen, hydrogen peroxide, and nitric oxide, leading to oxidative DNA changes, gene expression alterations, and cellular instability.^{2, 50, 51} ROS accumulation also stimulates

keratinocytes to secrete melanocyte-activating factors, resulting in melanogenesis and increased pigmentation.²

DNA damage and ROS activate signaling pathways such as MAPK/AP-1 and NF- κ B, leading to increased expression of matrix metalloproteinases (MMPs); these enzymes degrade extracellular matrix components such as collagen, elastin, and fibrillin-1.⁵²⁻⁵⁴ The increase in MMPs, along with reduced collagen synthesis, causes structural skin deterioration and photoaging, characterized by wrinkles, roughness, epidermal hyperplasia, and collagen fiber damage.⁵⁵

In addition to premature aging, chronic UV exposure suppresses local immunity and accumulates gene mutations, particularly in p53, paving the way for skin carcinogenesis.^{56, 57} However, sunlight at appropriate doses is beneficial for vitamin D synthesis and treatment of certain skin diseases.^{58, 59} This duality highlights the importance of preventive strategies: using broad-spectrum sunscreens, providing physical protection (clothing and sunglasses), and enhancing the skin's antioxidant system to reduce oxidative stress. Balancing the benefits of sunlight with the prevention of molecular damage is the key to skin health. Moreover, five key genes play a role in the skin's response to UVB treatment in atopic dermatitis, and this therapy has proven effective.⁶⁰ The key concepts described in this section are summarized in Figure 1.

Reactive oxygen species (ROS), generated through metabolic processes and radiation, induce oxidative stress and damage lipids, proteins, and DNA. These processes contribute to sunburn, aging, inflammation,

carcinogenesis, and pigmentation.^{61,62} UVA radiation and visible light trigger the three stages of pigmentation—immediate pigment darkening (IPD), persistent pigment darkening (PPD), and delayed tanning (DT)—the intensity and duration of which depend on skin type. Rapid pigmentation has a photochemical origin, whereas delayed pigmentation results from neomelanogenesis.⁶³ The α -MSH hormone, by reducing oxidative stress, serves as the primary stimulator of melanogenesis.¹⁶ Moreover, visible light via the opsin-3 receptor and UVA/blue light through the oxidation of pre-existing melanin further intensify the pigmentation process.⁶²

Characteristics of Visible Light

Visible light, ranging from 400 to 700 nm, accounts for nearly half of the solar radiant energy that reaches the Earth's surface.^{63,64} Each specific wavelength band within this range corresponds to a reference color.⁶⁵ The visible spectrum is commonly divided into three major regions: blue light (400–495 nm), green light (495–590 nm), and red light (590–700 nm).

Although sunlight represents the primary natural source of visible light, artificial sources include fluorescent lamps, lasers, light-emitting diodes (LEDs), mobile phones, television and computer screens, and modern smartwatches. All of these sources emit and distribute the full visible spectrum.⁶⁶

Despite the fact that the intensity of blue light emitted from digital screens is substantially lower than that of sunlight—and most daily exposure originates from

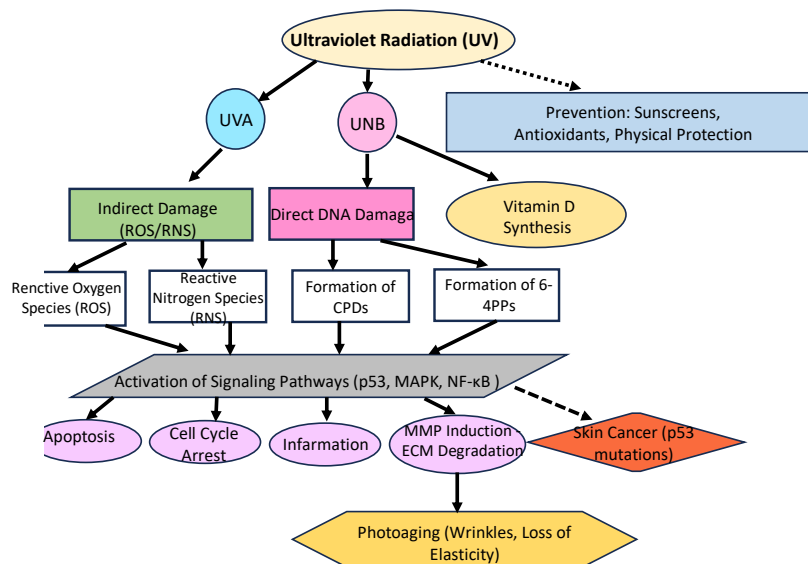


Figure 1. This diagram illustrates how ultraviolet radiation (UV) affects the skin through both direct and indirect pathways. UVB is mainly absorbed in the epidermis and causes direct DNA damage by generating cyclobutane pyrimidine dimers (CPDs) and 6-4 photoproducts (6-4PPs). UVA penetrates deeper into the dermis and primarily induces indirect damage via the production of reactive oxygen and nitrogen species (ROS/RNS). These molecular alterations activate signaling pathways such as p53, MAPK, and NF- κ B, leading to cellular responses, including cell cycle arrest, apoptosis, inflammation, and induction of matrix metalloproteinases (MMPs). The last one contributes to extracellular matrix (ECM) degradation and photoaging (wrinkles, loss of elasticity). The accumulation of unrepaired DNA mutations, particularly in p53, facilitates photocarcinogenesis. Despite these harmful effects, UVB also plays a beneficial role in vitamin D synthesis. Therefore, preventive strategies such as broad-spectrum sunscreens, antioxidants, and physical protection are essential to balance the health benefits and molecular risks of sun exposure.

natural solar radiation⁶⁷ the widespread use of digital devices, particularly following the COVID-19 pandemic, has markedly increased the duration of exposure. This phenomenon has raised growing concerns regarding the potential impacts of blue light on skin health, ocular function, and the aging process, often referred to as “digital pollution”.⁶⁸

The increasing reliance on the internet, social media, online gaming, and other digital forms of entertainment has further elevated blue light exposure, thereby amplifying concerns about skin and visual health.⁶⁹ Conversely, as the high-energy portion of the visible spectrum, blue light possesses diverse biological effects and therapeutic potential.^{70,71} Visible light is absorbed by tissue chromophores such as melanin, hemoglobin, and water; this absorption facilitates energy transfer to molecules and may induce biological effects including stimulation or localized heating.⁷²

Recent investigations (2022) have demonstrated that blue LED irradiation can induce precise and targeted cellular responses within activated tissues during the acute wound healing process. Specifically, blue light stimulates mast cells, triggers the initial inflammatory response, enhances angiogenesis through the secretion of signaling factors such as TNF- α ,⁷³ and promotes the differentiation of fibroblasts into myofibroblasts. Collectively, these processes play a pivotal role in wound closure and tissue remodeling.⁷⁴ Animal studies further confirm that blue LED light, in addition to accelerating collagen deposition and epithelial regeneration, expedites the entire wound healing process, making it faster and more effective.⁷⁵

Red light, with a wavelength of 600–700 nm, is characterized as low-energy radiation with a high skin penetration capacity, exerting notable stimulatory effects on skin cells. Due to its lower absorption by endogenous photosensitizers, red light generates fewer reactive oxygen species and primarily functions through the activation of intracellular signaling pathways.⁴ Consequently, red light promotes metabolic responses, regulates gene expression, modulates nitric oxide reservoirs, and activates key transcription factors, ultimately leading to enhanced epithelial regeneration, improved tissue repair, and reduced inflammation. These effects highlight its potential clinical applications in the treatment of certain dermatological conditions.¹²

In addition, studies have shown that individuals with darker skin exhibit greater sensitivity to therapeutic wavelengths within the 400–700 nm spectrum, with a higher likelihood of adverse effects. Visible light within this range may induce persistent pigmentation in individuals with skin of color.⁷⁶ Clinical investigations have further reported that the sensitivity of this group to red LED light is up to 50% higher compared with individuals with lighter skin tones, a difference observed in the tolerance threshold for irradiation dosage.⁷⁷ Figure 2 summarizes

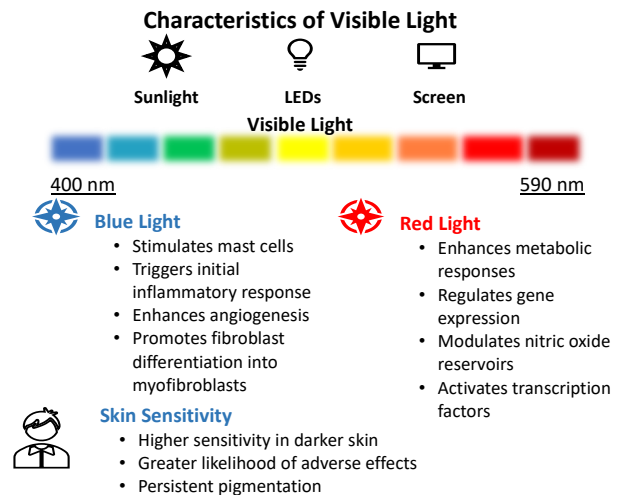


Figure 2. Overview of the visible light spectrum (400–700 nm), its primary sources, and the documented biological effects of blue and red wavelengths, including wound-healing activity, cellular responses, and variations in skin sensitivity.

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Effects of Different Visible Light Spectra on the Skin

Violet Light

Irradiation with UV–violet light (410 nm, 10–50 J/cm²) significantly alters the expression of cellular differentiation factors.⁷⁸ In vitro studies have demonstrated that exposure to violet–blue light (e.g., 410 nm) can downregulate the expression of differentiation markers and antimicrobial peptides, induce ROS production, trigger inflammation, and cause DNA damage in keratinocytes and fibroblasts, while also suppressing the initial formation of keloids.⁷⁹

Reduction of keratinocyte differentiation markers

Exposure to 410-nm light increases rhodopsin (an opsin) expression in epidermal keratinocytes, while simultaneously decreasing mRNA expression of epidermal differentiation markers (e.g., K1, K10, transglutaminase-3, and filaggrin). The same study also revealed that rhodopsin overexpression reduces phosphorylated CREB (p-CREB)—the active form of a transcription factor associated with differentiation pathways—suggesting that violet light can impair intracellular signaling related to keratinocyte differentiation via opsins.⁷⁸

Suppression of innate immune responses/effects on AMPs

Several reviews and reports have indicated that short-wavelength visible light (410 nm, 10–50 J/cm²) may suppress innate immune responses. Some reviews specifically noted reduced mRNA levels of antimicrobial peptides (AMPs). However, direct and quantitative evidence for decreases in LL-37 or β -defensin at a precise dose of 30 J/cm² remains limited and heterogeneous. Thus, while a potential “AMP reduction” effect has been suggested, definitive and quantitative confirmation

requires more targeted studies.⁹

ROS generation, inflammatory response, and DNA damage

Irradiation at 410 nm and nearby wavelengths has been shown to enhance ROS production in cells. In some models, this ROS increase was accompanied by proinflammatory cytokine release (e.g., elevated TNF- α or altered IL-6/IL-8) and markers of oxidative and DNA damage. These effects were reported to be dose- and wavelength-dependent, with some studies even describing protective or anti-inflammatory outcomes depending on the experimental model.⁸⁰

Effects on fibroblasts and keloid formation

In human keloid fibroblasts, in vitro irradiation with 410-nm LEDs reduced type I collagen mRNA and protein expression.⁷⁹ The authors concluded that this observation may indicate a preventive effect against early keloid formation; however, as these results were derived from in vitro data, clinical translation requires further human studies and long-term follow-up.^{79, 81}

Blue Light

Blue light (450–490 nm) constitutes a major portion of the visible spectrum and exerts both beneficial and potentially harmful effects on the skin. At low intensities, it reduces fibroblast metabolic activity, whereas at higher intensities it induces cytotoxicity.⁸² Blue light also generates mitochondrial oxidative stress in keratinocytes, leading to delayed recovery of the epidermal barrier.⁸³

Effects on skin fibroblasts

Cellular studies have shown that human skin fibroblasts exposed to low-dose blue light (450 nm, <30 J/cm²) display decreased metabolic activity, likely due to mitochondrial dysfunction and impaired cellular energy metabolism. Conversely, higher doses (>30 J/cm²) result in cytotoxicity, cell cycle arrest, and cell death. These findings were first reported by Mignon et al and subsequently confirmed in later studies.⁸²

Oxidative stress and effects on keratinocytes

Human keratinocytes exposed to blue light show increased mitochondrial ROS production.⁸⁴ This surplus ROS induces oxidative stress, DNA damage, lipid peroxidation, and activation of inflammatory pathways (e.g., IL-6, IL-8, and TNF- α release). At the same time, low-dose exposure may exert antimicrobial effects and even improve acne outcomes, as blue light can activate porphyrins in *Cutibacterium acnes*.^{50, 85}

Impact on the epidermal barrier and wound repair

Animal and human studies have demonstrated that blue light irradiation in the 430–510 nm range can significantly delay epidermal barrier recovery after injury (e.g., tape

stripping or stratum corneum removal). The proposed mechanisms include altered keratinocyte proliferation, induction of apoptosis, and disruption of ceramide synthesis, along with other lipid components of the skin barrier.^{86, 87}

Green Light

Green light at approximately 520 nm with an irradiation dose of 240 J/cm² has shown beneficial effects on the healing of third-degree burns in animal models. These effects are primarily mediated through the stimulation of angiogenesis and myofibroblast differentiation. In a study by Simões et al, the effects of red light (630 nm) and green light (520 nm) on the healing of third-degree wounds in Wistar rats were compared. Results demonstrated that green light significantly enhanced angiogenesis during the early healing phase and increased the number of myofibroblasts in later stages, highlighting its critical role in promoting wound repair.⁸⁸ Another study by Szymański et al reported that low-dose green light (520 nm) increased proliferation and cell cycle changes in keratinocytes and fibroblasts, thereby facilitating cellular processes essential for tissue repair.⁸⁹

Molecular mechanisms of green light

Green light may contribute to tissue repair by activating various signaling pathways. For instance, irradiation can activate TGF- β 1, a protein crucial for tissue healing and myofibroblast differentiation. Moreover, green light has been shown to reduce inflammation and promote collagen synthesis at the injury site.⁹⁰

Yellow Light

Yellow light at approximately 590 nm is recognized as an effective modality for improving skin condition by enhancing oxygenation and cellular energy. Irradiation reduces skin inflammation and erythema, improves texture, and diminishes wrinkles. It also regulates fibroblast activity, thereby facilitating repair of damaged skin tissue.

Molecular mechanisms of yellow light

At the molecular level, yellow light modulates the balance of free radical production and inhibits the expression of matrix metalloproteinase-1 (MMP-1), thereby preventing collagen degradation.^{91–93} Since MMP-1 is a key enzyme involved in extracellular matrix breakdown, its suppression helps preserve the skin structure. Furthermore, yellow light activates autophagy in adipocytes, facilitating triglyceride degradation through lysosomes. This process is marked by increased LC3-I to LC3-II conversion and decreased p62/SQSTM1 protein levels, indicating efficient cellular clearance. The inhibition of lysosomal function reduces these effects, underscoring the pivotal role of lysosomes in this mechanism.⁹⁴ In

addition, enhanced oxygenation and improved cellular metabolism induced by yellow light stimulate the cycles of tissue repair and skin rejuvenation.⁹³ Collectively, these molecular activities highlight the potential of yellow light as an effective therapeutic tool for skin disorders and fat metabolism, contributing to improved tissue repair and overall skin health.

Red Light

Red light (620–700 nm) penetrates deeply into the skin layers and, as a noninvasive and safe method, stimulates skin cell activity. It can enhance blood circulation and promote vasodilation, leading to improved nutrient delivery and oxygenation of tissues. This improved cellular support, combined with stimulated cell growth and regeneration, accelerates wound healing and repair of skin damage.

Molecular mechanisms of red light

Red light protects fibroblasts against UVB-induced DNA damage while increasing type I collagen expression and reducing MMP-1 levels, thereby improving skin elasticity and slowing the aging process. Unlike harmful UV radiation, red light therapy does not cause heat damage or tissue injury,^{95–97} making it a safe and widely adopted approach in photomedicine and dermatological care.

At the cellular level, red light primarily acts through its molecular mechanisms. Photon absorption by cytochrome c oxidase in mitochondria enhances ATP production,^{98,99} providing greater cellular energy to promote growth, regeneration, and repair. Additionally, red light reduces oxidative stress by limiting ROS production and modulating inflammatory pathways such as NF- κ B, TNF- α , and IL-6, thereby lowering skin inflammation.¹²

Moreover, red light increases the production of growth factors such as TGF- β and FGF, which activate fibroblasts and enhance collagen and elastin synthesis.¹⁰⁰ Together, these molecular effects improve the skin structure and quality, increase elasticity, and accelerate the repair of damaged tissue. Overall, the combined clinical and molecular impacts of red light establish it as a highly effective modality for skin rejuvenation,¹⁰² wound healing, and mitigating signs of aging.

Near-Infrared Light

Near-infrared (NIR) light, spanning 700–3000 nm, has attracted significant interest in photomedicine due to its deeper tissue penetration. In vitro and in vivo studies have shown that NIR irradiation, particularly at wavelengths of 830 and 640 nm, upregulates the expression of collagen-related genes and elastin in human fibroblasts and skin samples, thereby enhancing procollagen and elastic fiber production. Clinical evidence has also reported that LED treatments at 830 and 633 nm improve skin appearance

by reducing roughness and increasing epidermal uniformity. Furthermore, several animal and clinical studies demonstrated that low-intensity NIR irradiation (810 nm) can accelerate cell proliferation, collagen deposition, and re-epithelialization, thereby promoting wound healing through photobiomodulation.¹⁰³

On the other hand, spectroscopic data indicate that NIR exposure may increase reactive oxygen species (ROS) production and, under certain conditions, induce collagen degradation and molecular alterations resembling those observed in ultraviolet radiation (UVR)-induced damage.¹ These findings raise concerns regarding the potential adverse effects of high-dose NIR exposure, although at lower doses, its reparative and regenerative effects appear to predominate. Thus, while current evidence supports the beneficial role of NIR in enhancing skin quality, stimulating collagen synthesis, and accelerating wound healing, further randomized controlled clinical trials with larger sample sizes are still required to establish optimal dosing, treatment duration, and long-term safety (Table 1).

Discussion

The present review integrates a large body of experimental, translational, and clinical evidence to elucidate how human skin differentially responds to distinct wavelengths within the visible and near-infrared (NIR) spectrum. While ultraviolet radiation has long dominated photobiology research, recent advances have revealed that visible and NIR wavelengths also modulate cellular physiology, inflammatory pathways, pigmentation dynamics, and wound-repair mechanisms through chromophore-specific interactions and redox-dependent signaling. The findings of the current review highlight both therapeutic promise and wavelength-specific risks that must be considered when developing light-based strategies for dermatologic and regenerative applications.

Violet and Blue Light: Dualistic Biological Effects

Violet ($\approx$ 400–420 nm) and blue light ($\approx$ 420–470 nm) display complex dose-dependent effects on skin cells. Multiple studies demonstrate that these wavelengths can impair keratinocyte viability, reduce fibroblast proliferation, and activate oxidative stress pathways through TRPV1-mediated calcium influx and photoexcitation of endogenous chromophores such as flavins and porphyrins.^{1, 14, 80} Blue-violet light also reduces carotenoid levels in human skin, indicating rapid free radical formation.⁸³ These mechanisms help explain barrier-recovery delay and inflammation reported after acute exposure.⁸⁶

Despite these risks, controlled low-fluence blue-light regimens exhibit therapeutic potential. In vitro and in vivo studies show antimicrobial activity, suppression of keloid fibroblast collagen synthesis, and accelerated

Table 1. Effects of Visible and Near-Infrared Light on Skin

Spectrum	Wavelength (nm)	Key Effects
Blue Light	450–495	• Low dose (<30 J/cm²): ↓ Fibroblast metabolism, mitochondrial modulation ⁷⁵ • High dose (>30 J/cm²): Cytotoxicity, apoptosis, cell cycle arrest ^{83, 14} • Keratinocytes: ↑ ROS → oxidative stress, DNA damage, inflammation ^{1, 86} • Antimicrobial: Activates porphyrins in <i>Cutibacterium acnes</i> → acne improvement ^{93, 86}
Green Light	490–560	• Promotes angiogenesis and myofibroblast activity • Potential for burn wound healing ⁸⁸
Yellow/Orange Light	560–630	• 590 nm: ↓ UVA-induced ROS, ↓ MMP-1 expression ⁹¹ • 595 nm: ↑ Collagen I, modulation of MMP-1 ⁷⁹ • 590 nm: ↓ Triglycerides via autophagy–lysosomal pathway (fat reduction) ⁹³
Red Light	620–700	• Mitochondria: absorbed by Cytochrome c oxidase → ↑ ATP ^{99, 12} • ↓ ROS, anti-inflammatory (NF-κB, TNF-α, IL-6) ^{79, 77} • ↑ TGF-β, ↑ FGF → fibroblast activation, ↑ collagen/elastin ^{97, 95} • Clinical: wound healing, anti-aging, UVB protection ^{103, 76, 96}
Near-Infrared Light	700–3000	• 810–830 nm: ↑ Collagen, elastin, proliferation, epithelialization ^{97, 99} • Clinical: smoother skin, reduced roughness, rejuvenation ^{103, 76} • Risks: ↑ ROS, potential collagen degradation at high doses ⁷⁷ • Consideration: Optimal dose/safety require further RCTs ⁶³

wound healing through modulation of inflammatory cytokines and fibroblast metabolism.^{79, 81, 75} These dualistic effects underscore the importance of defining safe therapeutic windows and considering phototype-specific pigmentation responses.^{64, 65}

Green and Yellow Light: Wound Healing and Collagen Preservation

Green (≈ 520–560 nm) and yellow light (≈ 570–600 nm) demonstrate distinct biological actions associated with tissue regeneration. Green light enhances fibroblast activity and stimulates angiogenic responses, improving third-degree burn repair when used alongside red light.⁸⁸ Yellow light, particularly around 590 nm, appears to exert antioxidant and anti-inflammatory effects. Studies confirm that yellow LED exposure reduces UVA-induced MMP-1 expression, elevates catalase activity, and mitigates oxidative stress in fibroblasts.^{91, 93} These findings suggest that green and yellow wavelengths may help preserve extracellular matrix integrity and attenuate photoaging in clinical settings.

Additionally, yellow-light modulation of immune-related pathways, including S-nitrosylation signaling, reveals its potential for fine-tuning epidermal inflammatory responses.⁹² However, clinical translation remains limited, with insufficient randomized studies investigating dose thresholds and long-term outcomes.

Red and Near-Infrared Light: Established Photobiomodulation Pathways

Red (≈ 630–660 nm) and near-infrared (≈ 780–850 nm) wavelengths remain the most extensively studied in photobiomodulation research. Their effects largely stem from interactions with cytochrome-c oxidase, modulation of mitochondrial membrane potential, and enhanced ATP generation, collectively promoting cell survival, collagen synthesis, and wound repair.^{12, 99} Transcriptomic studies confirm that red light upregulates genes related to DNA-repair responses (GADD45A) and antioxidant

defense, thereby counteracting UV-induced damage.^{95, 96}

In vivo and clinical studies further support its benefits in full-thickness wound healing, graft survival, and dermal rejuvenation.^{97, 103, 76} Despite its generally favorable safety profile, emerging data suggest that beam characteristics and irradiance distribution significantly influence therapeutic consistency.⁹⁹ Additionally, variation in patient age, phototype, and tissue hydration can modulate the magnitude of photobiomodulation, warranting systematic clinical validation.

Comparative Evaluation and Mechanistic Integration

A comparative analysis of wavelengths reveals that shorter wavelengths within the violet-blue range impose higher oxidative burdens due to potent excitation of endogenous photosensitizers such as porphyrins, melanin precursors, and flavins.^{4, 11} Conversely, longer wavelengths, such as red and NIR, penetrate more deeply and modulate cellular bioenergetics rather than generating reactive oxygen species. Green and yellow wavelengths occupy an intermediate space, influencing matrix remodeling, angiogenesis, and inflammatory regulation through chromophores that remain incompletely characterized.

Cross-talk among light-induced reactive oxygen species (ROS) signaling, opsin-mediated phototransduction, and mitochondrial pathways reveals a complex multisensory network in human skin capable of integrating wavelength-specific signals. This intricate interaction underscores the importance of delineating beneficial hormetic ROS signaling from detrimental oxidative stress overload through detailed mechanistic studies.

Safety Considerations and Phototype-Specific Vulnerability

Short-wavelength visible light generates substantial oxidative stress, induces matrix-degrading enzymes, and accelerates photoaging markers, particularly in darker phototypes where melanin acts as a strong visible-light chromophore.^{65, 69, 2} Blue light emitted by digital devices

has been implicated not only in cutaneous oxidative stress but also in circadian disruption and neurocutaneous pathways.^{1,70,68} Although melanin provides protection against UV, its photoexcited states can prolong cellular damage through chemiexcitation and delayed CPD formation even after exposure ceases.⁵¹

Long-term safety data for therapeutic red/NIR exposures remain incomplete. Although these wavelengths are generally considered safe, variations in irradiance, pulse structure, and device type may impact mitochondrial homeostasis and cellular redox balance over time.⁷⁷ Standardized reporting of dose parameters is essential to reduce heterogeneity in study outcomes.

Future Directions and Clinical Translation

Despite rapid advances, several critical gaps persist. First, a universal dosing framework for visible-light therapies is lacking. Variability in irradiation parameters obstructs reproducibility and comparative evaluation across studies. Second, skin-phototype diversity is poorly represented in clinical trials; given the higher visible-light sensitivity of melanin-rich skin, phototype-specific protocols are urgently needed.⁶³

Conclusion

Different wavelengths of light elicit specific biological responses in human skin, ranging from the modulation of keratinocyte differentiation and antimicrobial peptide expression to the stimulation of angiogenesis, fibroblast activation, and collagen synthesis. Violet and blue light show dual roles, providing antimicrobial and anti-keratoid benefits at therapeutic doses but posing risks of cytotoxicity and delayed barrier recovery at higher exposures. Green and yellow light can support tissue repair and collagen homeostasis, while red light and near-infrared light have the strongest evidence for promoting wound healing, anti-aging, and skin rejuvenation through photobiomodulation. Exposure to ultraviolet (UVA/UVB), blue, and visible light can also induce oxidative stress, DNA damage, and cellular dysfunction. Clinically, integrating protective and therapeutic strategies—including broad-spectrum sunscreens, blue-light filters, antioxidant-enriched topicals, and controlled red/near-infrared photobiomodulation—can help prevent photoaging, minimize light-induced skin damage, and enhance regenerative interventions. Careful dose optimization, consideration of skin phototype-specific responses, and standardized protocols are essential for safe and effective translation of these findings. Future research should focus on defining treatment parameters and exploring combination light therapies to maximize therapeutic benefits while minimizing adverse effects.

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Competing Interests

The authors declare that they have no competing interests

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