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Visible Light Part I. Properties and Cutaneous Effects of Visible Light

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Abstract

Approximately fifty percent of sunlight reaching the Earth's surface is visible light (400–700 nm). Other sources of visible light include lasers, light-emitting diodes (LEDs), and flash lamps. Photons from visible light are absorbed by photoreceptive chromophores (e.g., melanin, heme, and opsins), altering skin function by activating and imparting energy to chromophores. Additionally, visible light can penetrate the full thickness of the skin and induce pigmentation and erythema. Clinically, lasers and light devices are used to treat skin conditions by utilizing specific wavelengths and treatment parameters. Red and blue light from LEDs and intense pulsed light (IPL) have been studied as anti-microbial and anti-inflammatory treatments for acne. Pulsed dye lasers are used to treat vascular lesions in adults and infants. Further research is necessary to determine the functional significance of visible light on skin health and wellness without confounding the influence of ultraviolet and infrared wavelengths.

Keywords

visible light; optical radiation; photobiomodulation; chromophores; lasers; photodermatitis; porphyria; phototherapy

1. Electromagnetic radiation

- Optical radiation includes ultraviolet, visible, infrared radiation.
- Most sunlight reaching the Earth's surface is visible or infrared.

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Electromagnetic radiation (EMR) includes gamma rays, X-rays, ultraviolet radiation (UVR), visible light (VL), infrared (IR), microwaves, and radio waves (Figure 1). UVR, VL, and IR are considered optical radiation (10 nm-1 mm) and defined as *Light* for clarity and convenience. The cutaneous effects of UVR and IR have been well studied; recently, advances in VL understanding on the skin have been made.[1–10] VL is the narrow spectrum (400–700 nm) of EMR that the human eye can detect.[3] VL accounts for ~50% of solar radiation reaching Earth's surface and can be further divided by color and wavelength.[3,11,12] UVR (10–400 nm) comprises ~5% of solar radiation reaching Earth's surface.[11,13,14] UVC and Extreme UV (EUV) are filtered by the atmosphere.[11,13,14] IR (700 nm-1 mm) comprises the remaining 45% of solar radiation that reaches Earth's surface.

2. Parameters, devices, and safety

- Natural and artificial sources emit VL.
- Devices produce different forms of light based on operating principles.

Parameters.

Natural and artificial sources emit VL. Light exposure in a medical context can be described by specific terms and parameters (Table 1).[15–20]

Devices.

Multiple types of devices deliver VL as a therapeutic modality, including lasers, light-emitting diodes (LEDs), arc/flash lamps, halogen lamps, and fluorescent lights (Figure 2).[3]

Safety.

Solar irradiance at the Earth's atmosphere is referred to as the solar constant (~136 mW/cm^2).[21,22] VL comprises ~53 mW/cm^2 of the solar constant.[21,22] The solar irradiance at the Earth's surface (~100 mW/cm^2) will vary depending on the latitude, time, and weather/atmospheric conditions.[21,22] Commercially available VL sources used in dermatology practices, such as lasers and LEDs, may have power densities exceeding 100 mW/cm^2.[17,23] According to the United States (US) Occupational Safety and Health Administration (OSHA) regulatory standards, lasers in the VL domain can damage the retina. Therefore, wavelength-specific safety goggles are recommended for VL procedures. Concurrent US Federal Drug Administration (FDA) regulations for skin photoprotection are based on minimal erythema dose (MED) and critical wavelengths; these are discussed in Part 2.

3. Depth of penetration

- Red light (625–700 nm) penetrates the skin deeper than blue light (400–500 nm)
- Red light can penetrate the full skin thickness

Light propagation and penetration through the skin are dependent on reflection, scattering, and absorption.[24] Approximately 4–7% of VL is reflected by the skin surface, regardless of incident wavelength, pigmentation, or structure.[24,25] Keratins, collagen, melanin, and hemoglobin are the primary skin molecules responsible for the VL light penetration

via scattering and absorption.[24,25] Zinc, ion gated channels, NADH, bilirubin, and β-carotene also absorb and scatter VL. Most skin scattering occurs when photons are absorbed by filamentous proteins and re-emitted. Epidermal scattering may be greater than dermal scattering due to melanin in the epidermis.[24–27] In the dermis, scattering occurs mainly in the direction of the incident beam as the diameters of collagen fibers are similar to VL wavelengths (i.e., Mie scattering), resulting in an enhanced depth of penetration.[24–27] Chromophores, such as cytochrome c oxidase (COX), absorb specific light wavelengths, which excite electrons into a higher energy state. Subsequent activation of second messengers including, reactive oxygen species (ROS), ATP, nitric oxide, and cAMP, elicit modulation of inflammation, proliferation, and tissue repair.[28] This process is called photobiomodulation or low-level light therapy. VL is not known to induce cyclobutane pyrimidine dimers (CPDs) and 6–4 photoproducts directly, as DNA is a primary UVR chromophore.[4–6,29,30] However, VL generates ROS, which can cause CPDs through a photochemical response.[31–34] Water, comprising ~15–20% of the stratum cornea and ~70–75% of the remaining epidermis and dermis, has low VL absorption.[3,35–39] However, water is the primary chromophore for IR (e.g., CO_2) laser therapy.

Wavelength is directly proportional to the depth of penetration but inversely related to energy, according to Beer-Lambert and Planck's laws, respectively.[40,41] Consequently, blue light (BL) is higher energy than red light (RL) but has less penetration. Pathologic skin changes, including edema, erythema, pigmentation, and fibrosis, can alter light penetration by changing chromophore concentrations and tissue density.[3] Along with demonstrating increased penetration depth with increasing wavelength, a custom Monte Carlo simulation of a multi-layered skin model demonstrated that increasing beamwidth for IPL enhances VL skin penetration.[42] Monte Carlo computer simulations use skin and light parameters to model depth of penetration.[43,44] Penetration depth increased markedly as beamwidth increased from 1–5 mm.[42,45] Maximum penetration was achieved with a 10 mm beamwidth, and larger beamwidths did not further increase penetration depth.[42,45]

As depth of penetrations can vary based on beamwidth, wavelength, and power density, published measurements, and simulations of light penetration can vary substantially.[25,27,46–49] RL penetrates 6–50 mm deep.[25,27,47–50] BL likely has a maximum skin penetration of 0.5–1 mm.[42,51] Epidermal thickness is ~30–100 μm, while epidermal plus dermal skin thickness is ~0.5–6 mm.[48,52–54] VL when administered at the correct settings, can likely penetrate entirely through the epidermis. RL can likely reach most, if not all, dermal structures.

4. Skin chromophores

- Melanin, heme, and opsins are chromophores receptive to VL.
- Chromophores have specific absorption spectra.
- The biological effects of VL are mediated through skin chromophores.

The primary VL skin chromophores are melanin, heme, and opsin (OPN) photoreceptors.[3,11,28] Chromophores in the skin absorb discrete wavelengths at varying affinities (i.e., the absorption coefficient). It is possible to target chromophores using

appropriate wavelengths and pulse widths selectively.[55,56] According to the theory of selective photothermolysis, the pulse width must be shorter than the tissue's thermal relaxation time, or non-specific thermal tissue damage occurs.[56] Light energy parameters that do not cause photothermolysis or thermal damage but still alter biological function are considered photobiomodulatory.[28,57] Photobiomodulation affects skin according to hormetic paradigms. Hormesis is the principle that an agent, substance, stimuli, or condition can induce a biphasic dose-response. VL's lower fluences can have stimulatory effects, while higher fluences are inhibitory, cytotoxic, or destructive.[58–61] The skin has an optical wavelength window (600–1300 nm) in which melanin, hemoglobin, and water absorption coefficients are the smallest.[3,25] Within this optical window, RL and IRA light is absorbed by COX to trigger non-thermal photobiomodulatory effects.[58–61]

Melanin.

Human skin melanocytes produce two main types of melanin from dihydroxyphenylalanine (DOPA) precursors: yellow-red pheomelanin and black-brown eumelanin.[62,63] BL, also called high energy visible light (HEVL), stimulates the expression and activity of tyrosinase, the rate-limiting enzyme in melanin production, via the OPN3 receptor present on keratinocytes and melanocytes.[64,65] In melanocytes from skin of color (but not lighter skin types), BL activation of OPN3 leads to tyrosinase/tyrosinase-related protein complex formation.[65] Therefore, BL may increase melanogenesis.[64,65]

Melanin has a poorly characterized structure and an absorption spectrum of 200–900 nm.[3,62,63,66–69] Peak absorption varies depending on the melanin moiety, but light absorption is greatest in the UV spectrum and decreases into the VL and IR spectra.[3,66–69] However, due to melanin's broad absorption spectrum in the VL range, melanosomes may absorb light intended for other chromophores during phototherapy.[70] Additionally, melanin has a thermal relaxation period of 70–250 ns.[3,66–69] Patients with skin of color benefit from laser treatment with picosecond lasers, longer wavelengths, and lower power densities to limit thermal tissue damage.[71–73] As a result, near-IR lasers (e.g., 1064 nm Nd:Yag) are considered safer than VL lasers for skin of color and pigmentation.[74]

In melanin-containing cells, ROS and reactive nitrogen species (RNS) generated by VL exposure can cause radiation-independent CPDs.[34,67,75–77] Radiation-independent CPDs develop 3 hours after UVA exposure due to melanin interactions with ROS and RNS.[34] In contrast, most CPDs are formed by direct absorption of UVB by DNA. However, potential VL-induced CPDs from ROS and RNS generation has been poorly studied.

Hemoglobin and heme derivatives.

Hemoglobin has a peak absorption in the blue (418 nm) and yellow/orange (542/577 nm) waveband.[3,24] Erythrocyte concentration is the primary determinant of light absorption by hemoglobin.[3,24] Porphyrins are tetrapyrrole macromolecules that serve as the intermediate for heme.[78–80] Accumulation of porphyrins due to enzyme dysfunction causes cutaneous porphyrias. Porphyrins have an absorption spectra at 400–405 nm, known as the Soret band, and weaker absorption spectra of 500–750 nm.[81,82] During photodynamic therapy (PDT), exogenous aminolevulinic acid (ALA), a non-photosensitive porphyrin precursor,

is applied to the skin. ALA accumulates in cells and is converted to protoporphyrin IX (PP-IX).[83] Subsequent irradiation with BL or RL from LEDs, fluorescent lights, or halogen lamps induces ROS generation and cell apoptosis.[84–87] PDT, commonly used to treat actinic keratosis and skin aging, is considered a non-photothermolytic process.

COX is a heme and copper-containing protein in the mitochondrial electron transport chain that absorbs RL and IRA.[28,57] RL and IRA absorption by COX is one of the primary drivers of photobiomodulation.[28,57] RL and IRA activate the electron transport chain, which increases mitochondrial membrane potential and leads to intracellular accumulation of second messengers: ATP, ROS, cAMP, Ca^{2+}, and nitric oxide.[28,57] This can alter gene expression, protein activity, redox reactions, inflammation, and metabolism.[28,30,31,57]

Opsins.

OPN photoreceptors absorb specific wavelengths of light and are responsible for VL phototransduction (Table 2).[88–90] Photoactivation of OPN1 and OPN2 in the retina's cone and rod cells provides visual image signals.[89,90] However, non-imaging forming OPNs have been identified in the skin.[64,88,91–96] Skin OPNs can regulate circadian rhythms, epidermal barrier function, and melanogenesis.[64,88,91–96] For example, BL activation of OPN3 may increase keratinocyte differentiation and tyrosinase activity in melanocytes.[65,91]

Skin reactions and pathologies

- VL induces greater hyperpigmentation in skin of color than light skin.
- VL triggers photodermatoses, including cutaneous porphyrias.

Hyperpigmentation.

Recent studies have examined the effects of pure VL and UVA1 on pigmentation and erythema, as the skin may not be protected from these spectra by standard sunscreen. Broad-spectrum and discrete VL wavelengths induce pigmentation with synergistic effects when combined with UVA exposure.[97–104] Pigmentation, lasting at least 2 weeks, occurred following broad-spectrum VL irradiation (>97.5% VL, <0.2% UVA, 0.8–2.4% IRA) at 40–80 J/cm^2 in skin types III-VI.[98,105,106] 40–80 J/cm^2 is the equivalent of ~15–30 minutes of VL solar irradiation.[21] BL at a fluence of 58±20 J/cm^2 induced pigmentation in skin types III and IV, persisting at least 21 days.[107] In another study, RL at 320 J/cm^2 from LEDs induced hyperpigmentation in skin of color subjects, resolving 1 week to 3 months post-treatment.[77] Pigmentation from VL is more intense and sustained than UVA1-induced pigmentation in dark-skinned subjects.[77,97,98] VL causes a redistribution of melanin from the basal layer to the upper epidermis and activation of OPN3 in dark-skinned subjects, leading to Ca^{2+} and MITF dependent melanogenesis.[64,65,98,102] Yellow light (YL) at a fluence of 5–20 J/cm^2 inhibits melanogenesis in vitro.[108]

Photodermatoses.

Photodermatoses are diseases caused or exacerbated by light. Photodermatoses are classified as immunologically-mediated, photoaggrevated, or secondary to exogenous/endogenous agents and DNA-repair deficiencies.[109–113] UVR is known to be the action spectra of most

photodermatoses. However, VL is the action spectrum for solar urticaria and cutaneous porphyrias, and less commonly, in polymorphous (polymorphic) light eruption (PLE) and chronic actinic dermatitis.

Solar urticaria: This is an uncommon mast cell-mediated photodermatosis.[97,114,115] Symptoms of solar urticaria include erythema, pruritus, and whealing. Phototesting studies indicate that 14–90% of patients with solar urticaria reacted to VL alone or in combination with UVR.[115–118] Fluorescent PDT light sources can induce urticaria and whealing.[119,120] Phototesting for solar urticaria must be evaluated minutes post-exposure as urticaria resolves within a few hours. Management of solar urticaria includes photoprotection with broad-spectrum sunscreen, anti-histamine, UVA/UVA1 hardening, methotrexate, cyclosporine, and omalizumab.[121–123] For those with action spectrum in the VL range, tinted sunscreen is necessary.

Porphyrias: These are mostly autosomal dominant diseases caused by accumulations of porphyrins, known phototoxic agents. They are classified as cutaneous (i.e., skin blistering) or neurovisceral (i.e., abdominal pain, vomiting, and tachycardia).[78,113] 10 mW/cm^2 of BL (405 nm) for 1000 seconds activates PP-IX.[124,125] Management of porphyria cutanea tarda, the most common cutaneous porphyria, includes sun protection, avoidance of exacerbating factors (i.e., alcohol), phlebotomy, and low dose chloroquine.[86,87] The FDA approved afamelanotide (Scenesse®), an α-melanocyte stimulating hormone (α-MSH) analog, in October 2019 as a new treatment for the second most common porphyria in the US, erythropoietic protoporphyria (EPP).[126] Afamelanotide increases melanin production and serves as an antioxidant, hence down-regulates porphyrin-induced phototoxicity.

Polymorphous (polymorphic) light eruption (PLE): This is the most common immunologically-mediated photodermatosis with multiple clinical presentations, including vesicular, papular, hemorrhagic, and eczematous; in dark-skinned individuals, it presents as a distinctive pin-head papular eruption.[127] PLE is the most common photodermatosis worldwide and can affect all races and skin types.[127] The majority of reactions occur due to UVA alone, but UVB and, uncommonly, VL may also responsible.[127,128] In one study, 100 J/cm^2 of VL (~40 minutes of VL sun exposure) induced PLE compared to 20–35 J/cm^2 for UVA (~40–75 minutes of UVR sun exposure).[21,129] Management of PLE include photoprotection, anti-histamines, and induction of tolerance (i.e., UVB hardening).[115–118,127]

Chronic actinic dermatitis: This presents with eczematous and frequently lichenified skin lesions. UVB, UVA, and rarely VL have been identified as the action spectra, but the most severe reactions are associated with UVB exposure.[130–132]

Secondary photodermatoses: 141 to 313 J/cm^2 of VL may induce skin reactions in some secondary photodermatoses, including systemic lupus erythematosus.[133]

Erythema and inflammation.

Exposure of subjects with skin phototypes I-III to VL with ≤2% UVA1 resulted in immediate erythema.[134] In dark-skinned individuals (phototypes IV-VI), 480 J/cm^2 of VL with 0.5% UVA, but not pure VL, induced immediate erythema assessed by investigator's clinical inspection.[106] In another study, VL and UVA1 light elicited immediate erythema lasting less than 24 hours in skin types II-IV patients.[103] In two randomized controlled trials on the safety of LED RL (633 nm) on human skin, 320 J/cm^2 and above induced prolonged erythema in some light-skinned patients, while RL at 480 J/cm^2 and above caused blistering in some patients of various skin types.[77] Erythema from visible light may be due to a combination of inefficient photoenergy transfer in the skin and inflammation; for some light sources, the device's thermal effects might also contribute.[77,102,135,136] Many laser and light protocols employ cooling devices to prevent thermal damage and minimize adverse events such as pain, erythema, blistering, scarring, and edema.[77,137]

5. Photomedical phototherapy

- For photobiomodulation, light is delivered at parameters that do not induce thermal destructive processes
- Treatment efficacy of VL is wavelength specific for each condition.
- Lasers, IPL, LEDs, and fluorescent bulbs are commonly used in VL treatment protocols.

Acne.

In clinical trials, BL and RL from devices at 400–445 nm and 625–700 nm, respectively, treated mild to moderate acne by decreasing *Cutibacterium acnes* colonization, pore size, and inflammation (Table 3).[17,138–140] Inflammatory lesions may respond better than non-inflammatory lesions.[17,138–141] *C. acnes* directly produce porphyrins, which can absorb VL to produce ROS.[17,140,142] Long-incubation PDT (>3-hours) with high-intensity RL activation may induce long-term acne remission but is associated with pain, inflammation, and photosensitivity.[143]

Psoriasis.

Narrow-band UVB is commonly used to treat psoriasis, but researchers have also explored using VL.[17,144] Blue and red LEDs may reduce erythema and improve the local psoriatic severity index (PASI).[17,145,146] VL phototherapy inhibits keratinocyte and endothelial proliferation by increasing cellular differentiation.[147,148] LED phototherapy may be better suited for reducing psoriatic erythema rather than improving desquamation.[17,145,146] However, psoriatic recurrence may occur following cessation of treatment.

Wound healing.

Significant clinical research has examined the use of lasers, IPL, and LEDs for acute and chronic wound healing. Treatment parameters and patient selection vary considerably among publications.[17,149–151] RL reduces wound healing time, lesion size, and erythema in diabetic foot ulcers.[17,149–153] Venous ulcers may not respond to photobiomodulation.[154,155]

VL improves wound healing by reducing the expression of proinflammatory proteins and cytokines, increasing OPN3 mediated keratinocyte differentiation, and preventing bacterial growth.[91,156–158]

Hair growth.

Multiple devices, including caps, combs, and handheld units, are currently available to stimulate hair growth using LEDs or laser diodes (i.e., lasers with a semiconductor gain media). RL increases hair density, growth, and tensile strength in androgenic alopecia by stimulating the anagen growth phase.[159] Clinical studies commonly use RL and near-IR photobiomodulation with fluences of 0.1–150 J/cm^2 for hair regrowth.[55,150]

Rejuvenation and photodamage.

RL, YL, and BL have been studied as anti-aging therapeutics.[17,140] The proposed mechanism of light-mediated anti-aging is photobiomodulatory activation of Ca^{2+}, nitric oxide, matrix metalloproteinase, and collagen-stimulating pathways.[17,140] PDT may improve photodamaged facial skin texture, wrinkles, and mottled pigmentation.[160,161]

6. Photothermolytic phototherapy

- Lasers and IPL target chromophores and induce local tissue destruction via photothermolysis

Vascular lesions.

Pulsed dye laser (PDL; 585–600 nm) targets oxyhemoglobin and is the treatment of choice for vascular lesions, including port-wine stains (PWS), spider angiomas, and hemangiomas.[3,162–164] In plaque and nail psoriasis, PDL induces photothermolysis of capillaries.[147,165–169] PDL has been shown to have better efficacy than UVB excimer laser in treating nail bed and matrix lesions.[147,165–169] Frequency-doubled Nd:Yag (532 nm) are also used for vascular lesions but do not penetrate as deeply as PDL.[162]

Special care is recommended when using PDL in skin of color and infants. The high density of melanin in individuals with skin of color results in absorption of VL, which produces heat generation and blistering. Asian patients have been safely treated with PDL and IPL for vascular lesions.[74,170] Infants have thinner and less pigmentated skin, and long-pulsed treatment settings are recommended.[171–174]

Tattoo removal.

Q-switched frequency-doubled Nd:YAG (532 nm) and ruby (694 nm) lasers are used to remove tattoos.[3] Ruby lasers target black and blue pigments, while 532 nm Nd:YAG lasers target yellow, orange, and red pigments. Typically, black tattoo pigments are easier to remove than colored pigments.[175] 532 nm picosecond pulsed lasers have enhanced safety and efficacy for tattoo removal compared to nanosecond lasers.[176,177] Common adverse events include immediate erythema, hyper-/hypopigmentation, and hypersensitivity.

7. Future directions and conclusion

- VL has the potential to treat a variety of skin diseases and affect skin health
- Additional research is needed to determine the optimal treatment parameters for VL phototherapy.

VL is currently used for the treatment of skin conditions, and use may expand to new applications.[98,178] Skin of color is more susceptible to high fluences of VL and requires conservative treatment parameters. Further research is necessary to determine VL's functional significance on skin health without confounding UVR and IR wavelengths. A better understanding of VL therapeutic effects would improve treatment protocols and patient care. With the availability of light sources emitting broad but pure VL, these types of studies can now be performed.[98,178] Comparative clinical trials can elucidate the optimal efficacy of specific wavelengths, fluences, and power densities.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Henry W. Lim is an investigator for Incyte, L'Oreal, Pfizer, and PCORI, has served as a consultant for Pierre Fabre, ISDIN, Ferndale, Beiersdorf, and La Roche-Posay, and has participated as a speaker in general educational session for La Roche-Posay, and Cantabria labs. Indermeet Kohli is an investigator (grant funding received by the institution) for Ferndale, Estee Lauder, L'Oreal, Unigen, Johnson and Johnson, Allergan, and Bayer and is a consultant (fee and equipment received by the institution) for Pfizer, Johnson and Johnson, and Bayer. Jared Jagdeo is a member of the GlobalMed Scientific advisory board and a consultant for UV Biotek. Iltefat Hamzavi is an investigator for Estee Lauder, Ferndale Laboratories, Galderma, Bayer, Loreal, Lenicura, and Unigen.

ABBREVIATIONS

ALA	Aminolevulinic acid
BL	Blue light
CME	Continuing medical education
CPD	Cyclobutane pyrimidine dimer
COX	Cytochrome c oxidase
DOPA	Dihydroxyphenylalanine
EMR	Electromagnetic radiation
EPP	Erythropoietic protoporphyria
EUV	Extreme UV

FDA	Food and Drug Administration
HEVL	High energy visible light
IPL	Intense pulsed light
IR	Infrared
LED	Light-emitting diode
MED	Minimal erythemal dose
MITF	Microphthalmia-associated transcription factor
OPN	Opsin
OSHA	Occupation Safety and Health Administration
PASI	Psoriasis severity index
PDL	Pulsed dye laser
PDT	Photodynamic therapy
PLE	Polymorphic light eruptions
PP-IX	Protoporphyrin IX
PWS	Port Wine Stains
RL	Red light
RNS	Reactive nitrogen species
ROS	Reactive oxygen species
US	United States
UVR	Ultraviolet radiation
UV	Ultraviolet
VL	Visible light
YL	Yellow light

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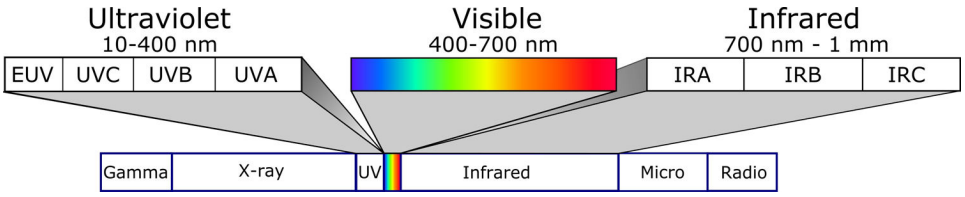

Figure 1: Electromagnetic radiation spectrum.

UVR, VL, and IR are optical radiation. VL can be divided by color: blue/violet (400–500 nm), green (500–565 nm), yellow (565–590 nm), orange (590–625 nm), or red (625–700 nm). Similarly, UVR is separated into separate spectra: UVA (320–400 nm), UVB (290–320 nm), UVC (200–290 nm), and extreme (EUV; 10–120 nm). IR can be subdivided into IRA (near-IR; 700–1440 nm), IRB (mid-IR; 1440–3000 nm), and IRC (far-IR; 3000 nm-1 mm) wavelengths. Spectral boundaries are not discrete, and there is an overlap in the biological effects between adjacent forms of EMR.

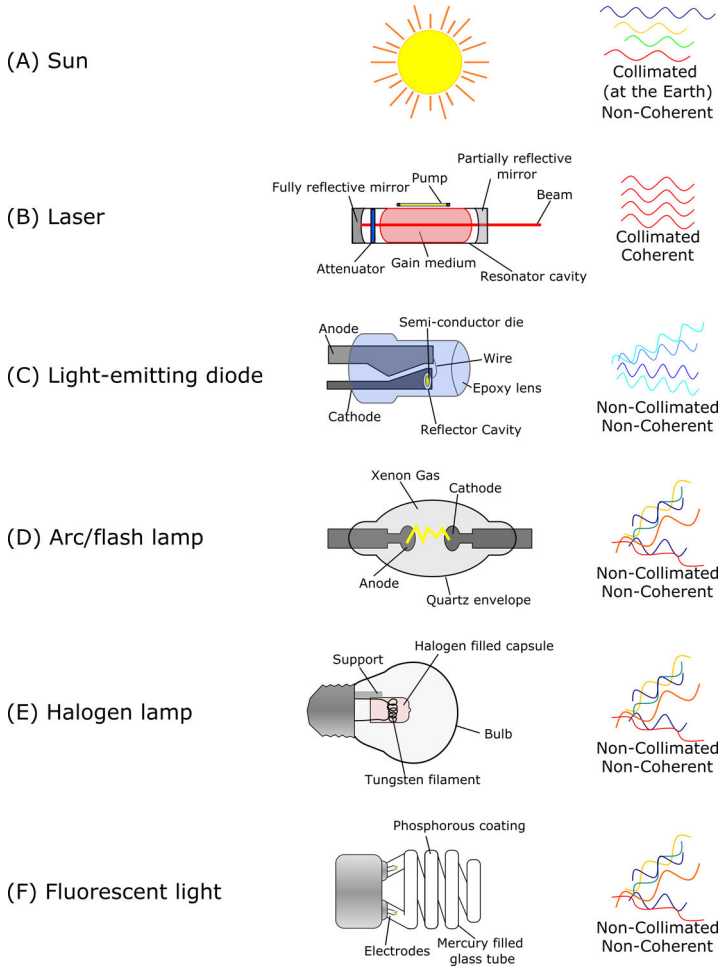

Figure 2: Diagram of natural and artificial visible light sources.

Devices are labeled with significant parts. Collimation (i.e., parallel), coherence (i.e., in-phase), and chromaticity are provided for each light source. **(A)** Sunlight is relatively collimated at the surface of the Earth. **(B)** Lasers pump energy through a gain medium (e.g., crystal or gas) to generate or amplify light between mirrors in the resonator cavity. The variable attenuator in the resonator cavity of Q-switched lasers allows for beam pulsing. Lasers are highly monochromatic, coherent, and collimated upon emission. **(C)** Light-emitting diodes (LEDs) pass an electrical current through a semiconductor. LEDs produce light in a narrow range to appear as a single color. Multiples LEDs can be placed in an array to generate white light or higher power densities. **(D)** Arc and flash lamps (e.g., intense pulsed light [IPL]) arc electricity through a mercury or xenon gas chamber and are optimized for continuous and pulsed operating conditions, respectively. Filters are applied to achieve specific wavelengths. **(E)** Halogen lamps heat a tungsten filament in a sealed chamber with small amounts of halogen gas. **(F)** Fluorescent lights excite electricity through mercury gas to produce UVR, which is converted to VL via the phosphorescent coating on the lamp's inner surface.

Table 1:

Definition of light parameters.

Terminology	Definition	Standard unit
Wavelength	Distance between two peaks of a wavefunction.	Meters
Irradiance (power density)	The power (energy/second) of light delivered to a unit of surface area.	Watts/meter^2
Fluence (dose)	The amount of energy delivered to a unit of surface area over a given time. Fluence = irradiance x time.	Joules/meter^2
Duration of exposure	The amount of time that the skin is exposed to light.	Seconds
Pulse width	The length of time that a pulsed device delivers light (i.e., “flashes” in a repetitive mode).	Seconds
Beamwidth	The diameter of the beam. The edge of the beam is defined by having an irradiance of at least $1/e^2$ of maximum irradiance.	Meters
Duty cycle	Percentage of time that the signal is active during one cycle.	Percentage of time
Luminance	The intensity of light at the source weighed by human perception of VL.	Lux
Photon flux	The number of photons delivered per unit of surface area at a given time.	Photons/meters^2/ seconds
Coherence	Light waves have an identical wavelength and constant phase.	N/A
Collimation	Light waves are parallel.	N/A

Table 2:

Identified skin opsins.

Listed opsins, activation wavelengths, and identified expressed skin locations and cell types.

Opsin	Activation Wavelength (nm)	Skin Expression
OPN1-SW	420–425	Epidermis, melanocytes, keratinocytes, and fibroblasts
OPN1-MW	527–530	Epidermis
OPN1-LW	557–560	Epidermis
OPN2	500–505	Melanocytes, keratinocytes, fibroblasts, and hair follicle stem cells
OPN3	420–527	Melanocytes, keratinocytes, fibroblasts, and hair follicle stem cells
OPN4	480	Fibroblasts
OPN5	380	Melanocytes, keratinocytes, and fibroblasts
Peropsin	380–400	Keratinocytes

Abbreviations: OPN – opsin, nm – nanometer.

Table 3:

Therapeutic applications of VL.

VL therapy efficacy depends on patient selection, disease severity, modality, and device settings.

Condition	Spectrum	Wavelength (nm)	Device/Modality
Acne	Broad	400–700+	IPL
	Blue	405–420	LED
	Blue	405–420	Fluorescent bulb
	Yellow	585 or 595	PDL
	Red	630–670	LED
Hair Regrowth	Red	633 or 660	Laser Diode
	Red	630–660	LED
Neonatal Jaundice	Blue	450–470	LED
	Blue	400–550	Halogen
	Blue	400–550	Fluorescent
Tattoo Removal	Green	532	Frequency-doubled Nd:Yag
	Red	694.3	Ruby
Psoriasis	Blue	405–420	LED
	Yellow	585 or 595	PDL
	Red	625–670	LED
Skin Rejuvenation	Yellow	570–590	LED
	Red	625–670	LED
	Red	633 or 660	Laser Diode
Vascular Lesions	Broad	400–700+	IPL
	Green	532	Frequency-doubled Nd:Yag
	Yellow	578	Copper Bromide
	Yellow	585 or 595	PDL
Wound Healing	Blue	405–420	LED
	Green	515 or 520	Laser Diode
	Yellow	570–590	LED
	Red	625–660	LED
	Red	633 or 660	Laser Diode

Abbreviations: IPL – Intense pulsed light; LED – light-emitting diode, PDL – pulsed dye laser, Nd:Yag – neodymium-doped yttrium aluminum garnet, nm – nanometer.