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Actinic Damage: The Major Cause of Skin Aging

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Abstract

The authors have previously reported on the deleterious effects of sunlight on human skin tissue. Despite ongoing evidence that actinic damage and skin ageing are caused not only by ultraviolet radiation, but also visible light and near-infrared energy, few studies have focused on the additive impact of the sun's total energy. In the past, ultraviolet radiation was assumed to be the only biologically significant factor in skin actinic damage, but in recent decades this view has been considerably revised to include the impact of the sun's complete energy reaching the earth affecting terrestrial life, including humans. In this paper, we summarize the evidence supporting actinic damage and the cumulative negative effects of ultraviolet, visible light and near-infrared radiation as the major cause of photoaging, pigmentation, vascular irregularity, muscle thinning, skin ptosis, photoimmunosuppression and photocarcinogenesis. Visible skin aging associated with actinic damage is differentiated from chronologic aging, which is less defined by the stigmata of photoaging associated with changes to skin tone, texture and laxity. Awareness of the biological effects of total solar energy output and understanding actinic damage may assist physicians and patients to seek comprehensive solar protection and repair strategies including sun avoidance and topical preparations that protect from ultraviolet, visible light and near-infrared as well as skin repair mechanisms including retinoids and DNA repair enzymes.

Keywords

Actinic Damage, Near-Infrared, Photoaging, Ultraviolet, Visible Light

1. Introduction

Actinic damage, also called sun damage, represents skin changes due to excessive sun exposure. Actinic damage of the skin manifests itself as extrinsic skin aging (photoaging) and photocarcinogenesis [1]. Facial aging is influenced by intrinsic

factors such as genetics and cellular metabolism, as well as by extrinsic environmental exposure and lifestyle choices [2]-[5]. Understanding these mechanisms is crucial for developing strategies that encourage optimal aging and mitigate the negative impacts of sunlight on skin appearance and function [5].

The deleterious changes in the structure and function of human skin, particularly due to the amount of exposure to ultraviolet (UV) are well documented in the dermatologic literature [2] [3], and studies have recently begun to explore the effects of non-UV on skin physiology [6]. The main extrinsic factors such as acute and chronic sun exposure may accelerate the signs of facial actinic damage and are a major cause of facial aging caused by UV, visible light (VL) and near-infrared (NIR) radiation [7]-[14]. Despite the wide prevalence of a variety of UV blocking materials, including sunscreens, sunglasses, treated glass, film, umbrellas, and fibers, motivated by the desire to prevent actinic damage and unwanted facial aging, solar radiation continues to pose a health threat worldwide as these protective measures only consider UV and not VL or NIR damage [7]-[10]. In reality, over 90% of solar radiation affecting the Earth consists of VL and NIR, and intensive or ongoing exposure to these electromagnetic wavelengths is the major cause of actinic damage and photoaging [8] [12]-[16]. It is estimated that in excess of 80% of the signs of facial ageing (wrinkles, sagging, hyperpigmentation etc.) are directly caused by the biological effects of solar energy reaching the earth and interreacting with human skin tissue [17]. It must be noted once again that standard sunscreens and UV blocking materials filter only UV, and will not prevent facial aging induced by VL and NIR [7]-[16].

The authors previously reported that NIR can penetrate skin and sclera and affect the deeper tissues, such as muscles, lens, and retina, heralding profound biological effects throughout the body [8] [15] [16]. Continual long-term exposure to NIR can induce various kinds of actinic damage and diseases, such as undesirable photoaging [18]-[22], long-lasting vasodilation [19], muscle thinning [18], sagging and skin ptosis [18], photoimmunosuppression, and photocarcinogenesis, when innate biological protection against the relevant electromagnetic spectral radiation is inadequate [21].

Due to the facts that human tissue is subjected to significant doses of biologically active solar spectral radiation daily, and that most protective measures including sunscreens and sunglasses are only capable of blocking UV (and not harmful VL and NIR), due consideration should also be given to developing and deploying photoprotective materials and strategies that offer comprehensive protection from the combined effects of UV, VL and NIR [7]-[16].

2. Discussion

2.1. Sunlight

Sunlight is the vital source of light and heat essential to almost all forms of terrestrial life. Plants need sunlight for photosynthesis, and mammals need plants for many vital purposes [11]. In Ancient Egyptian friezes from around 4000 BC, the

sun is depicted as delivering rays to every form of life, with each ray ending in a little hand "patting" the target [11]. In front of the face of the Pharaoh, the sun's ray ends in the ankh, the symbol for life, which illustrates the sun as the source of light and life [11].

Incident solar energy comprises approximately 6.8% UV, 38.9% VL, and 54.3% NIR [23] (**Figure 1**). Despite widespread UV blocking material use such as sunscreens, sunshades and UV filtering glass being deployed to prevent actinic damage, pigmentation, skin cancer, and facial aging continue to pose a health threat worldwide [15] [16].

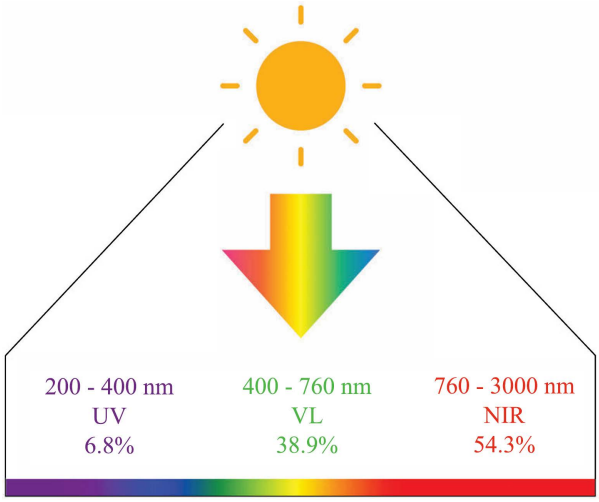

Figure 1. Incident solar energy comprises approximately 6.8% UV, 38.9% VL, and 54.3% NIR.

It has been established that longer wavelength VL and NIR have the potential to penetrate beyond the dermal structures to deeper biological tissue and exposure to this solar energy form can improve ATP production and mitochondrial function. This phenomenon could translate to improvements in physiologic performance, particularly in the central nervous system, which includes the visual system [24].

The medical literature is rich in research and documentation of the effects of sunlight on human skin tissue. Beginning at birth, the face is exposed to solar wavelengths, resulting in the cumulative visible signs of photodamage including hyper and hypopigmentation, degradation of connective tissue elasticity and integrity, erosion of skin tone and texture (sallowness, elastosis, hyperkeratosis) and vascular irregularity. Convincing and consistent evidence also links progressive sun exposure throughout life with various forms of skin cancer [17].

On the other hand, we must consider the potentially positive benefits of solar exposure on other bodily systems once the risk of actinic skin damage has been mitigated [24].

The recurrent interaction of skin with sunlight is an intrinsic constituent of human life, exhibiting both beneficial and detrimental effects [25]. The apparent ro-

bust architectural framework of skin conceals remarkable mechanisms that operate at the interface between the skin's surface and the environment [25].

2.2. Facial Ageing

It is generally agreed that between 80% - 90% of facial aging is caused by the sun [3] [17] [26] (**Figure 2**), yet what remains largely unexplored is the complex relationship between intrinsic and extrinsic aging. The passage of time is directly attributable to the combination of chronological and actinic damage [27]. Intrinsic structural changes occur as a natural consequence of ageing and are genetically determined [3]. The skin ageing rate in any individual can also be dramatically influenced by personal and environmental factors, particularly the amount of exposure to UV [3].

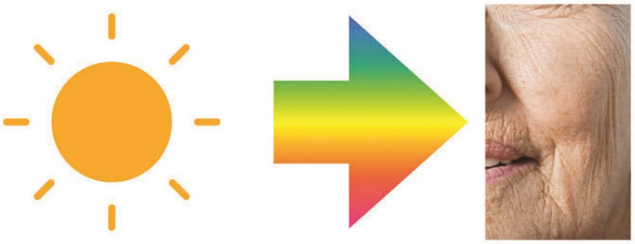

Figure 2. It is generally agreed that between 80% - 90% of facial aging is caused by the sun.

Photoaging has clinical, histologic, and functional characteristics of older skin that can be observed in habitually sun-exposed areas, consisting of chronic sun damage superimposed on so-called intrinsic or programmed aging [4]. Photoaging accounts for most of the unwanted changes in skin appearance over time and also exaggerates or accelerates the loss of physiologic reserve and various protective capacities [4].

The ageing process is unique to the individual and depends on the interplay between an individual's genetics and external environmental factors. Through understanding the molecular and cellular mechanisms, an appreciation of the consequent structural and functional changes can be achieved [28].

As the population ages, dermatological focus must shift from ameliorating the cosmetic consequences of skin ageing to decreasing the genuine morbidity associated with problems of the ageing skin [3]. A better understanding of both the intrinsic and extrinsic influences on the ageing of the skin, as well as distinguishing the retractable aspects of cutaneous ageing (primarily hormonal and lifestyle influences) from the irretractable (primarily intrinsic ageing), is crucial to this endeavour [3].

A comprehensive understanding of both extrinsic and intrinsic factors contributing to skin aging is essential for developing effective prevention and intervention strategies. Multifaceted approaches, incorporating lifestyle modifications, advanced skincare routines, and emerging therapeutic technologies are important to mitigate the effects of aging and promote healthier, more resilient skin [5].

The fact that the longer we live, the more our face is exposed to the sun's total energy renders the concept of a control population impossible [17].

2.3. UV

UV-radiation is the invisible part of light spectra having a wavelength between visible rays and X-rays. Based on wavelength, UV rays are subdivided into UV-A (320 - 400 nm), UV-B (280 - 320 nm) and UV-C (200 - 280 nm). UV has both beneficial and harmful effects [29] [30].

Most of the positive effects of UV are mediated via UVB induced production of vitamin D in skin [30]. UVB-induced, delayed tanning acts as a sunscreen [30] [31]. Several human skin diseases, like psoriasis, vitiligo, atopic dermatitis localized scleroderma and multiple sclerosis, can be treated with solar radiation (heliotherapy) or artificial UV radiation (phototherapy) [30]. Furthermore, UV generates nitric oxide (NO), which may reduce blood pressure and generally improve cardiovascular health [32] [33]. UVA-induced NO may also have antimicrobial effects and furthermore, act as a neurotransmitter [30]. Finally, UV exposure may improve mood through the release of endorphins [34]-[36].

UV-C has the property of ionization thus acting as a strong mutagen, which can cause immune-mediated disease and cancer in adverse cases [29]. However, UVC does not currently constitute a health hazard as it is almost completely absorbed by the ozone layer of the earth. Incident UVA and UVB cause observable, measurable damage to skin structures [37]-[39].

Human skin has various molecular mechanisms to protect itself from UV damage. The Stratum Corneum, epidermis, and immune cells such as Langerhans cells and T lymphocytes located within the skin are protective defense systems against harmful external agents [39]. UV activates the cutaneous immune system, which leads to an inflammatory response by various mechanisms [29]. The unique stratified epithelial architecture of human skin along with its endogenous antioxidant-response pathways constitutes the important defense mechanisms against UV [25]. Another line of protection for the skin is the melanocytes. Melanin, a photoactive pigment synthesized by these cells, impedes the penetration of UV into the epidermis by absorption [39]. The intricate pigmentary system and its intersection with the immune-system cytokine axis work synergistically to balance tissue homeostasis [25].

The elaborate adaptive mechanisms, elegant multilayered architecture and evolutionary selection pressures involved in skin and sunlight interaction makes this a compelling model to understand biological complexity [25]. Furthermore, to maintain homeostasis, UV-induced DNA damage can be repaired at the molecular level by nucleotide repair and base excision mechanisms or apoptotic mechanisms as well as activated cell cycle checkpoints [25] [29] [39].

Photoaging and photocarcinogenesis are primarily due to solar UV, which alters DNA, cellular antioxidant balance, signal transduction pathways, immunology, and the extracellular matrix (ECM) [38]. The DNA alterations include UV-

induced thymine-thymine dimers and loss of tumor suppressor gene p53 [38]. UV reduces cellular antioxidant status by generating reactive oxygen species (ROS), and the resultant oxidative stress alters signal transduction pathways such as the mitogen-activated protein kinase (MAPK), the nuclear factor-kappa beta (NF-κB)/p65, the janus kinase (JAK), signal transduction and activation of transcription (STAT) and the nuclear factor erythroid 2-related factor 2 (Nrf2) [38]. In addition to the formation of photo-dimers in the genome, UV can also induce mutation by generating ROS and nucleotides are highly susceptible to these free radical injuries [29]. UV induces pro-inflammatory genes and causes immunosuppression by depleting the number and activity of the epidermal Langerhans cells [38] [39]. Further, UV remodels the ECM by increasing matrixmetalloproteinases (MMPs) and reducing structural collagen and elastin [38].

Photoaging is the premature aging of the skin structures over time due to repeated exposure to UV which is evidenced by dyspigmentation, telangiectasias, roughness, rhytides, elastosis, and precancerous changes [40]. Exosomes are associated with aging-related processes including, oxidative stress, inflammation, and senescence. Stem cell-derived exosomes can restore skin physiological function and regenerate or rejuvenate damaged skin tissue through various mechanisms such as decreased expression of MMPs, increased collagen and elastin production, and modulation of intracellular signaling pathways, which is promising for the therapeutic potential of exosomes in skin photoaging [40].

UV radiation increases the risk of long-term tissue damage such as photoaging, photoimmunosuppression, and photocarcinogenesis [37]-[39]. UVA radiation has its negative effect on the epidermal keratinocytes and dermal fibroblasts and induces long-term changes [39]. Changes arising as a result of UVB radiation are visible mainly within the epidermis but also penetrate the upper part of dermis [41]. The harmful effects of ultraviolet exposure principally include sunburn, photodermatoses, hyperpigmentation, photoaging of the skin and precancerous lesions and cancers [39]. Despite the ability of the stratum corneum to absorb considerable UV, attenuated UV energy reaches viable epidermal cells, causing direct DNA damage as evidenced by the formation of cyclobutene-pyrimidine dimers following UV exposure [42]. In addition to direct DNA corruption, UV causes indirect damage through the generation of ROS which impair the photoprotective role of endogenous antioxidant defenses, resulting in the numerous pathologies associated with photoaging, and immunosuppression, a major factor in melanoma formation [6] [43]-[45]. It has also been established that UV irradiation leads to sustained elevations of MMPs that degrade skin collagen and contribute to photoaging [46].

In summary, extrinsic skin damage from UV exposure is far more harmful but more capable of significant modification than intrinsic aging factors [3].

2.4. VL

VL (400 - 700 nm) accounts for approximately 40% of the sun's Electromagnetic

Radiation energy reaching the Earth's surface (**Figure 3**). The skin's photoreceptive chromophores (including melanin, opsins and heme) absorb VL with biological consequences including erythema and hyperpigmentation.

VL encompasses the electromagnetic radiation visible to the human eye, spanning from 400 to 700 nm of the electromagnetic radiation spectrum [6] [13], and photons from VL are absorbed by photoreceptive chromophores (melanin, heme, and opsins), altering skin function by activating and imparting energy to chromophores [47].

VL can induce more intense and longer lasting pigmentation compared to UVA1 (340 - 400 nm) and UVA1 has been shown to potentiate these effects of VL [48] [49], with VL emerging as a key player in photodermatology [49].

Clinically, lasers and light devices are used to treat skin conditions by utilizing specific wavelengths and treatment parameters [47]. Red and blue light from light-emitting diodes and *intense pulsed light* have been studied as antimicrobial and anti-inflammatory treatments for *acne*, and pulsed dye lasers are used to treat vascular lesions in adults and infants [47].

However, similar to UV, the effects of blue light, yellow light, red light, and broad VL have been implicated in photoaging by way of interaction with specific photoreceptors, ROS production, pigmentation, cytokine formation, MMPs, and other photon-mediated reactions [6] [12] [45] [50]-[53].

VL can penetrate the full thickness of the skin, resulting in sequelae throughout the entire epidermis, dermis and hypodermis, and induce pigmentation, erythema, which contribute to photoaging [6] [47] [54]. The induction of free radicals, the generation of reactive species, is one driving mechanism of skin pathologies induced by VL, leading to the induction of melanogenesis and hyperpigmentation [13]. The synergistic effects of VL + UVA1 on inducing pigmentation and erythema in skin have been demonstrated and linked to exacerbation of dermatologic conditions including melasma and post-inflammatory hyperpigmentation [55] [56].

Photoprotection against VL includes avoiding the sun, seeking shade, and using photoprotective clothing [57]. The organic and inorganic UV filters used in sunscreens and commercially available sunscreens do not protect the skin from VL [6] [56] [57].

Inorganic filters (zinc oxide and titanium dioxide) are used in the form of nanoparticles in sunscreens to minimize the chalky and white appearance on the skin but do not protect against VL [56]. Tinted sunscreens use different formulations and concentrations of iron oxides and pigmentary titanium dioxide to provide protection against VL [56] [58]-[69]. Many shades of tinted sunscreens are available by combining different amounts of iron oxides and pigmentary titanium dioxide to cater to all skin phototypes [56]. Tinted sunscreens are beneficial for patients with VL-induced photodermatoses and those with hyperpigmentation disorders such as melasma and postinflammatory hyperpigmentation [56].

Sunscreen containing an antioxidant combination significantly reduced the

production of ROS, cytokines, and MMP expression *in vitro*, and decreased oxidative stress in human subjects after VL irradiation [6]. The effectiveness of topical sunscreen with antioxidant combinations in inhibiting VL + UV-A1-induced erythema in Fitzpatrick skin types (FSTs) I to III and reducing pigmentation in FSTs IV to VI [13] [48] [56] [57].

It is essential that dermatologists and the public are aware of the impact of VL on skin and understand the available options for VL protection [6] [13] [65]-[69].

2.5. NIR

Infrared (IR) radiation ranging from 760 nm to 1 mm is non-ionizing radiation located "below the red", *i.e.* adjacent to the red part of the visible radiation range and extending up to the microwave range [20]. The IR spectral region is arbitrarily divided according to wavelength into sub-regions of NIR (760 - 3000 nm), middle IR (3000 - 30,000 nm), and far IR (30,000 nm^{-1}·mm). Incident solar NIR is selectively filtered by atmospheric water [70] [71] (**Figure 3**); thus, most NIR radiation that reaches the Earth's surface readily penetrates the superficial layers of the skin. In addition to natural NIR, human skin is increasingly exposed to artificial NIR from medical devices and electrical appliances [8] [72] [73]. Therefore, we are exposed to tremendous amounts of NIR every day.

Incident solar NIR wavelengths above 2500 nm are completely filtered by atmospheric water and cannot be delivered to the Earth at sea level. The peak wavelengths of solar NIR are 500 - 600 nm, which is different from NIR of incandescent filament lamps whose wavelengths include NIR and mid-wavelength IR, with peak wavelengths of 800 - 1500 nm.

Notably, NIR devices using an incandescent filament lamp emitting wide wavelengths of NIR without a water filter or contact cooling were used in previous studies [74]-[78]. With these methods, NIR immediately increases the temperature of the superficial layer of culture fluid in a laboratory dish or skin, as NIR is predominantly absorbed by hydrogen bond-containing molecules, such as water and hemoglobin. The energy of NIR then decreases as it penetrates deeper and will not reach enough target cells in the basal layer or deeper tissues. Therefore, previous reports were only able to describe the superficial and thermal effects of NIR, and the experimental designs were not appropriate to examine the biological effects of NIR [21].

Wavelengths below 1100 nm will be absorbed by melanin in the skin, and wavelengths between 1400 and 1500 nm and those above 1850 nm will be absorbed by water in the skin, which results in heating and possible induction of painful sensations and burns [79]. To imitate the incident solar NIR and deliver NIR energy to the deeper tissues, filtering out the wavelengths below 1100 nm, around 1450 nm, and above 1850 nm is essential [80]. NIR increases the surface temperature and induces thermal effects, thus, to reduce the skin surface temperature, perspiration, and blood vessel dilation, contact cooling is recommended.

To accurately investigate the biological effects of solar NIR, a water-filter that

excludes wavelengths between 1400 and 1500 nm and the cooling system is indispensable.

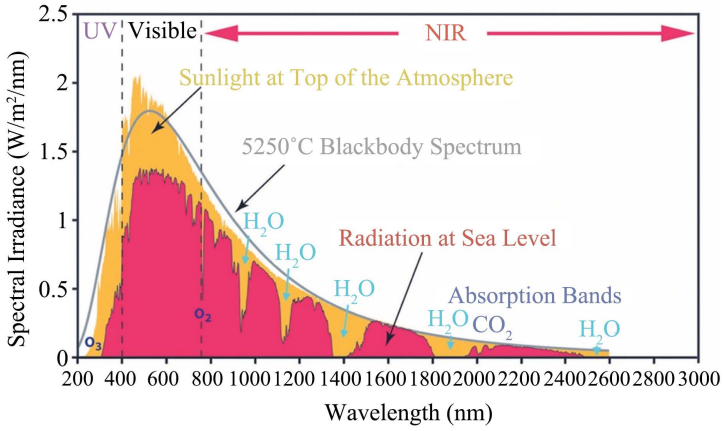

Figure 3. Solar radiation. This graph shows the radiation spectrum for direct light both at the top of the Earth's atmosphere (yellow) and at sea level (red). The sun produces light with a distribution similar to that expected from a 5250˚C blackbody (gray), which is approximately the temperature of the Sun's surface. As light passes through the atmosphere, it is partially absorbed by gases with specific absorption bands (blue). These curves are based on the American Society for Testing and Materials Terrestrial Reference Spectra, which are standards adopted by the photovoltaic industry to ensure consistent test conditions and are similar to the light levels expected in North America. Cited and revised from **Figure 2**. Tanaka *et al.* (2010) [18].

The authors previously elucidated that NIR (1100 - 1800 nm together with a water-filter that excludes wavelengths 1400 - 1500 nm) non-thermally affects the skin into the deeper tissues [18]-[22] [75] [81]-[83].

NIR exhibits both wave and particle properties and is strongly absorbed by water in the skin, hemoglobin in dilated vessels, myoglobin in the superficial muscle, and bone cortical mass, and is scattered by adipose cells [8] [20]. NIR induces photochemical changes and affects a large volume and depth of tissue [70] due to its high permeability.

NIR reaches deeper layers of the skin and is also related to the generation of ROS, photoaging and erythema while VL is responsible for generating ROS, pigmentation, cytokine formation, and matrix metallopeptidases (MMPs) [6] [12] [45] [51]-[53].

Where biological NIR protection is not complete NIR performs as a facial aging factor [8] [21] and NIR significantly contributes to extrinsic skin aging [84]. NIR induces facial aging similar to that observed in solar elastosis and enhances UV-induced actinic damage [74]. NIR has been shown to induce ROS formation and lead to the subsequent increased expression of MMPs [73]. In addition to UV, VL and NIR within natural sunlight increase MMP-1 and MMP-9 expression *in vivo* [85].

The biological effects of NIR have both beneficial applications and deleterious effects [8] [86] [87]. NIR stimulates wound healing [76] [88]-[90] and can treat malignant tumors [20] [79] [91]-[94]. Actively proliferating cells show increased

sensitivity to NIR [95] [96], and NIR induces DNA strand breaks and apoptosis [97] as well as the death of cancer and bone marrow cells [18] [20] [94]. NIR also stimulates skin rejuvenation and skin tightening [82]-[84], induces long-lasting vasodilation that is beneficial for ischemic disorders [8] [83], and relaxes and weakens dystonic and hypertrophic muscles to reduce wrinkles and myalgia [8] [83]. In addition to its usefulness in cancer detection and imaging, NIR induces DNA damage in mitotic cells should be investigated further as an effective anti-cancer treatment [20] [94]. NIR can also activate stem cells, which may be beneficial in regenerative medicine [8] [83].

Long-term exposure to NIR from various heat sources, such as fires and stoves, results in histopathological changes similar to those seen in actinically damaged skin [98]. Various kinds of tissue damage and diseases, such as undesirable facial aging, long-lasting vasodilation, muscle thinning, skin ptosis, sagging, cataracts, and potentially photocarcinogenesis may be induced by long-term NIR exposure [21] [83]. In addition, skin tumors appeared faster after irradiation with exposure to solar simulations containing UV, VL, and NIR compared to irradiation with UV alone [99]. Despite the wide prevalence of a variety of UV blocking materials, such as sunblock, sunglasses, films, and fibers, effective methods for blocking NIR are not currently deployed.

A wide range of protective mechanisms has been evolutionarily maintained in organisms to protect against actinic damage [8] [83]. Humans have acquired protective mechanisms against actinic damages especially NIR on multiple levels, including perspiration, blisters, vasodilation, hair, skin, adipose tissue, and cotton or wool clothing [8] [83] [100].

Chronic NIR exposure can induce rosacea, which is more common in Caucasians and fair-skinned populations [101]. Erythema ab igne can be induced by long-term exposure to sources of heat and NIR, such as fires and stoves [102], and exhibits histopathological changes similar to those seen in actinically damaged skin [98]. The occurrence of telangiectasia appears to increase with age, increased sunbathing, and poor pigmentation ability [101]. These lesions may develop into thermal keratosis, such as hyperkeratosis, keratinocyte dysplasia, and dermal elastosis, which are similar to the changes that occur in actinically damaged skin [103]. Apoptosis of vascular smooth muscle cells and degeneration of myoglobin are non-thermally induced by NIR, resulting in long-lasting vasodilation [8]. Muscles appear to be easily damaged by NIR, as they contain hemoglobin and myoglobin [104], which are oxygen-carrying proteins with many hydrogen bonds and alpha helices [8].

Previous studies have shown that hydrogen bonds and helical structures are resonated by NIR [8] [105]. Thus, it is possible that NIR induces resonance of helical structures in the oxygen-carrying proteins and degenerates proteins containing hydrogen bonds and helical structures, which results in damage to the storage and transport of oxygen [8] [21].

Compared to darker skin with dense melanin and a thicker dermis, fair skin with lower concentrations of melanin and a thin dermis might allow NIR to pen-

etrate deeper into human tissue, resulting in damage to superficial muscles, resulting in muscle thinning and skin ptosis. Fair skin tends to wrinkle and sag earlier in life [106] [107], and characteristics of age-related changes occur at a more accelerated rate in Caucasians [108].

Carcinomas often arise from heat-induced erythema ab igne [74] [109] [110], and NIR induces photoaging and potentially photocarcinogenesis [72]. In addition, skin tumors appeared faster after irradiation with the full lamp spectrum containing UV, VL, and NIR compared to irradiation with UV alone [99].

Subcutaneous adipocytes may be induced by NIR to protect the underlying tissues, including the panniculus carnosus in animals and superficial muscles in humans, against NIR damage. Subcutaneous adipocytes located above superficial muscles are effective and desirable for temperature retention and protection from NIR, as fatty tissue can scatter NIR optically [111] and fatty acids are the major NIR absorbing materials in soft tissues [112]. Although various kinds of UV blocking materials are often used to prevent tissue damage from UV exposure, these materials do not block visible light or NIR [8] [100]. Therefore, we should protect ourselves with clothing, sunscreen and glasses that not only block UV, but also VL and NIR, in order to prevent sun damage. Additional studies are required to investigate the generally quantified dose limit for the body and the necessity of sun protection.

Over 90% of solar radiation affecting the Earth consists of VL and NIR, and intensive or ongoing exposure to VL and NIR contributes to facial aging and skin cancer [7]-[14] (**Figure 4**). It must be noted that the global sunscreen industries have not significantly embraced effective formulation technologies designed to filter UVA, VL and NIR [7]-[14]. As the biological effects of combined solar energy (referred to as the Solar Constant, comprised of UV, VL and NIR) are significant, solar protection from UV, VL and NIR is desirable and effective in preventing facial aging [7]-[14].

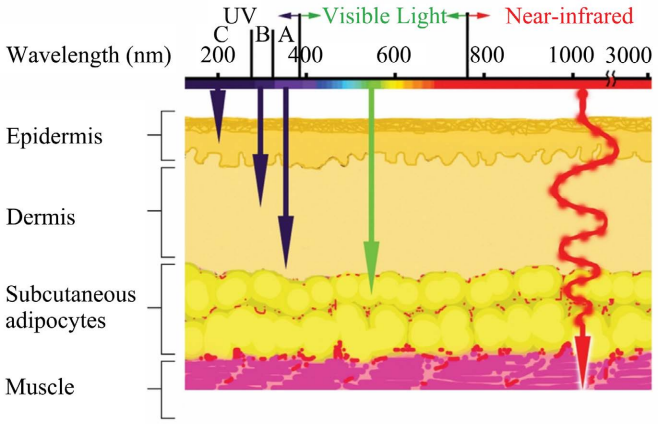

Figure 4. A schematic of penetration depth at each wavelength.

3. Conclusions

This review has provided a comprehensive overview of actinic damage in facial

aging and the biological effects of UV, VL and NIR as extrinsic facial aging factors. Understanding the mechanisms underlying facial aging has significant implications for healthcare and skincare practices.

Actinic damage and facial ageing have various physical and mental health ramifications, increasing the imperative for daily home-based, non-invasive skin maintenance. Comprehensive solar protection from UV through to NIR radiation and solar repair should be standard dermatologic practice in preventing actinic damage and facial ageing. By embracing interdisciplinary approaches and leveraging cutting-edge technologies, we can pave the way for transformative advancements in anti-photoaging medicine and skincare, ultimately enhancing the quality of life for individuals worldwide.

Disclosure

Authors are paid employees of RATIONALE.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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