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REVIEW

Assessing the Biological Impacts and Health Risks of UV Radiation in the Context of Sustainable Development Goals: Review

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ABSTRACT

Ultraviolet (UV) radiation, while essential for certain biological processes, presents significant risks to human health and ecosystems. Increased UV exposure, driven by ozone depletion and climate change, heightens threats such as DNA damage, skin cancer, immune suppression, and eye disorders, directly aligning with Sustainable Development Goal (SDG) 3: Good Health and Well-being. Additionally, UV radiation disrupts marine and terrestrial ecosystems, impacting biodiversity and productivity, relevant to SDGs 14 and 15. This review evaluates the health and environmental impacts of UV radiation and highlights recent advancements in UV monitoring technology. Key findings reveal substantial evidence linking UV exposure to accelerated skin aging, immune system impairment, and ecological imbalances, with specific emphasis on the long-term health effects, genetic variability in UV sensitivity, and interactions with the skin microbiome. This review proposes strategies to mitigate UV radiation impacts, emphasizing the integration of SDG-oriented approaches to foster resilience in public health and ecosystems.

Keywords: UV protection, Skin cancer, Ozone depletion, Climate change, Wearable UV sensors

1. Introduction

Ultraviolet (UV) radiations are 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) [1]. Ultraviolet rays can have both harmful and beneficial effects. UV-C has the property of ionization thus acting as a strong mutagen, which can cause immune-mediated disease and cancer in adverse cases. UV radiation is a form of electromagnetic energy emitted by the sun, essential for several biological processes, but excessive exposure to it poses serious risks to human health and ecosystems [2]. The UV Index, designed as a public health tool, measures the intensity of UV radiation, and provides guidance on how to minimize harmful exposure [3]. This index has gained relevance due to climate change and ozone layer depletion, which increases exposure to UV radiation [4]. Fig. 1 illustrates UV Index levels and key factors affecting exposure, emphasizing the associated health risks. At the molecular level, UVB radiation is especially damaging, causing direct damage to DNA, such as pyrimidine dimers, which can trigger mutations and cancer [5]. UVA radiation, although less energetic, generates reactive oxygen species (ROS) that damage cellular components [6]. Repair mechanisms, such as nucleotide excision repair (NER), help mitigate this damage, but excessive exposure can overload these processes and lead to mutagenesis [7]. The biological implications of exposure to UV radiation are profound, affecting organisms at the molecular, cellular, and ecological levels [8]. In humans, UV

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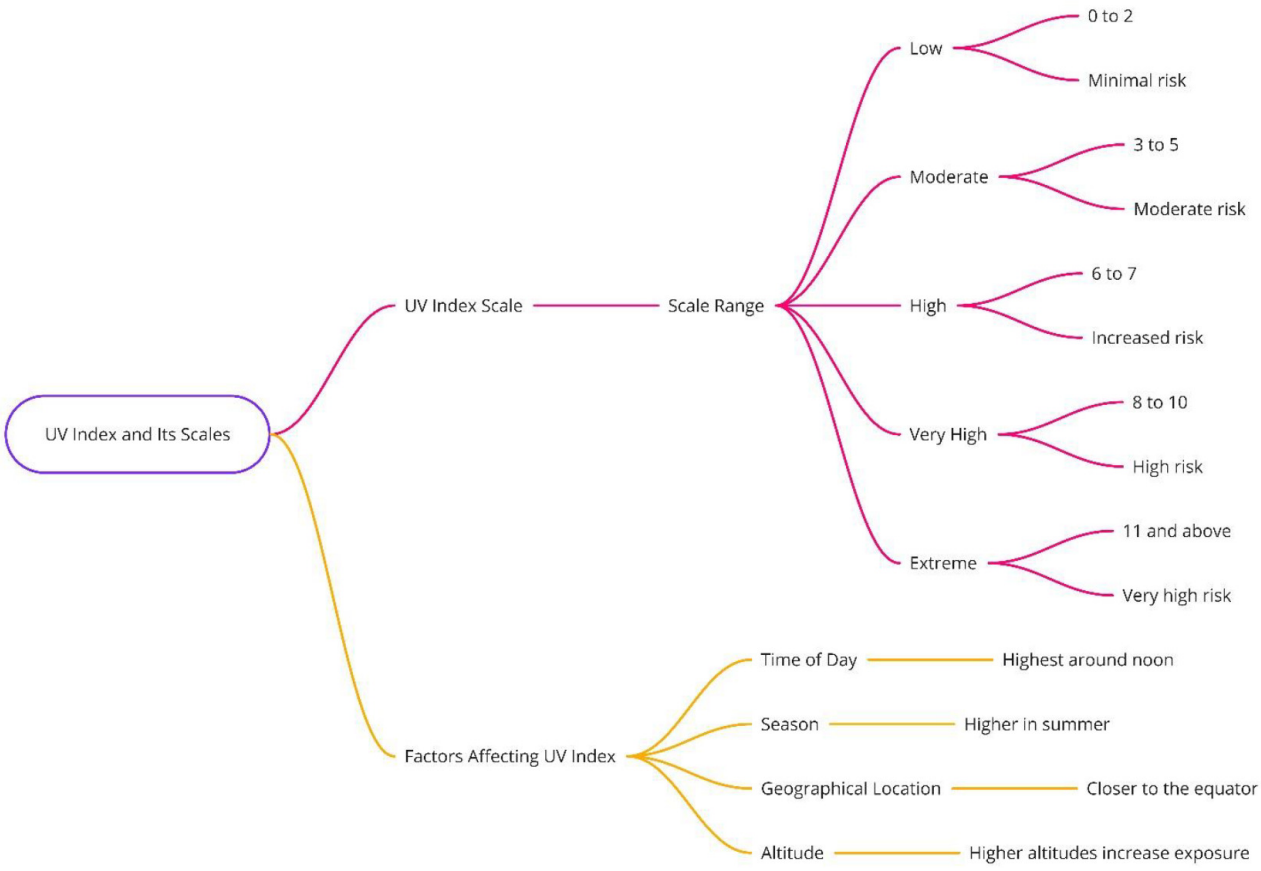

Fig. 1. UV Index scale and the environmental factors that influence UV radiation exposure.

radiation causes immediate effects such as sunburn, but also long-term consequences such as premature skin aging, DNA damage, and increased risk of cancer [9]. In addition, several solutions like protective glasses and contact lenses are studied in the subject of eye disorders UV exposer such as cataracts [10], and recent studies indicate that it can weaken the immune system, increasing susceptibility to infections and reducing the effectiveness of vaccines.

UV radiation has profound biological and environmental impacts that extend far beyond human health [11]. On one hand, increased UV exposure, exacerbated by ozone depletion and climate change, harms both terrestrial and marine ecosystems, disrupting biodiversity and ecosystem functions [12]. For example, A 2019 study highlights the increased health risks from rising UV exposure due to ozone depletion and climate change, including skin cancer, DNA damage, and immunosuppression [13]. It emphasizes the need for effective public health strategies, climate resilience, and UV protection, aligning with SDG 3, SDG 10, and SDG 13 to protect human health. Phytoplankton, a key organism in marine ecosystems, and terrestrial plants are particularly vulnerable to UV radiation, leading to reduced productivity and cascading effects on food chains and carbon cycling. These disruptions align with concerns addressed in SDGs 14 (Life Below Water) and 15 (Life on Land), emphasizing the need to protect ecosystems from environmental stressors like UV radiation. On the other hand, UV radiation also impacts human health through its immunosuppressive effects, increasing susceptibility to infections and potentially reducing vaccine efficacy [14]. Tahmasebi et al. in their study on oxygen-ozone therapy's effects on regulatory T-cell (Treg) responses in multiple sclerosis patients, provide insights into how therapies can modulate immune function [15]. This is highly relevant to UV-induced immunosuppression, where regulatory T-cells play a key role in maintaining immune balance. The mechanisms explored in their research may offer potential strategies for mitigating the adverse immune effects of UV radiation, particularly in regions where UV exposure is intensified by climate change. Phytoplankton, which are foundational to the marine food web, are particularly vulnerable to UV radiation [16]. As primary producers, they play a crucial role in global carbon cycling and oxygen production.

Even minor reductions in phytoplankton populations due to increased UV radiation can have cascading effects on oceanic ecosystems and biodiversity [11]. Terrestrial plants are also affected, with UV radiation inhibiting photosynthesis and damaging plant tissues, potentially reducing crop yields, and affecting food security [17].

This review aims to comprehensively examine and address critical research gaps related to UV radiation and its biological impacts, aligning closely with the Sustainable Development Goals (SDGs). The primary focus of this review is to explore the long-term health effects of chronic, low-level UV exposure, particularly regarding its cumulative impact on skin aging, immune suppression, and DNA damage. We also delve into the mechanisms of UV-induced immunosuppression and its implications for disease susceptibility and vaccine efficacy, as these remain insufficiently understood. Another major area of interest is the interaction between climate change, ozone depletion, and UV radiation patterns, focusing on how these environmental factors may alter regional UV exposure and impact ecosystem resilience. Additionally, the review highlights the emerging area of UV radiation's effects on the human skin microbiome, emphasizing the need for further investigation into how UV exposure alters microbial communities and influences dermatological health. We also address the poorly understood genetic factors influencing individual susceptibility to UV-induced damage, including DNA repair and melanin production variability. Finally, this review covers the ecological effects of increased UV exposure on non-human organisms in extreme environments and assesses the long-term efficacy of current UV protection strategies and public health campaigns. By addressing these gaps, this review seeks to provide a holistic understanding of UV radiation's biological impacts and propose future research directions to support public health and environmental resilience.

2. UV radiation and human health

2.1. Short-term and long-term effects on skin

UV radiation is a powerful environmental factor that significantly impacts the skin, the body's largest organ and its primary barrier against external threats [18]. Directly exposure of the skin to UV radiation, which can have both short-term and long-term biological consequences [19]. These effects range from temporary changes like sunburn to more serious outcomes, such as DNA damage and skin cancers, including melanoma [20]. Both types of radiation contribute to skin damage through distinct mechanisms, and understanding their roles in skin pathology is crucial for developing effective preventive measures [21]. Far-UVC, especially at 222 nm, effectively inactivates pathogens while being safer for human skin and eyes compared to traditional UVC (254 nm) sources [22]. A dose of 100 mJ/cm^2 significantly reduces pathogens without harming humans, making it a promising method for preventing surgical site infections [23]. It also holds potential in combating viruses, though current limitations hinder immediate large-scale use. Safety concerns and limited testing over long durations suggest caution. Furthermore, far-UVC sources, such as excimer lamps and far-UVC LEDs, are scarce and have limited lifespans. Thus, while promising, far-UVC is not a short-term solution but may benefit future decontamination applications.

2.1.1. Short-term effects: Sunburn and acute cellular damage

One of the most immediate effects of UV radiation on the skin is sunburn, a painful and inflammatory response to excessive UV exposure. Sunburn, or erythema, is primarily caused by UVB radiation, which directly penetrates the epidermis, the outermost layer of the skin. When the skin is overexposed to UVB rays, it absorbs the energy, leading to the formation of cyclobutane pyrimidine dimers (CPDs) in the DNA of epidermal cells [24]. According to Schuch et al. treefrog tadpoles (Hypsiboas pulchellus) exhibit both molecular and sensory mechanisms to mitigate UV-induced DNA damage [25]. While they are sensitive to UVB radiation due to slow DNA repair rates, their ability to activate photolyases under UVA exposure and detect and escape harmful UV wavelengths helps protect their genomic integrity. Additionally, UVB exposure stimulates the production of pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), which contribute to the acute inflammatory response [26]. Sunburn is not only an immediate reaction but also a marker of underlying DNA damage, setting the stage for more serious long-term effects if such damage is not properly repaired [27]. In 2022, Chiou W in his study stated that approximately 120,000 health workers in USA identified a strong linear correlation between severe sunburn (SS) and skin cancer incidence, suggesting a likely causal relationship [28]. Even short-term UV exposure, particularly UVB rays, can lead to immediate effects like sunburn, where DNA damage triggers an inflammatory response. While these effects may seem temporary, repeated exposure leads to cumulative DNA damage, elevating long-term risks such as skin cancer and premature aging. The authors suggest that severe UV exposure

overwhelms the skin's repair capacity, potentially resulting in cancerous tumors if repair mechanisms fail, aligning with the wound-to-tumor hypothesis. Furthermore, Human Papillomavirus can lower the threshold for cancer initiation by increasing vulnerability to DNA lesions [29]. UVA rays, which penetrate deeper, contribute to oxidative stress, indirectly affecting cancer risk. The study also addresses skin cancer risks across different professions, critiques common experimental practices using unphysiological UV doses, and draws parallels between skin aging kinetics in exposed versus unexposed skin. The findings emphasize the significance of UV protection, even with brief exposure, and encourage further research on these mechanisms.

2.1.2. Long-term effects: DNA damage and skin aging

The biological effects of UV radiation extend far beyond the acute responses seen in sunburn. Over time, repeated exposure to UV radiation, particularly UVB, can lead to cumulative DNA damage in skin cells. According to Chouinard et al. repeated exposure to low doses of UVB radiation induces considerable damage in human skin cells, including the accumulation of CPDs, structural destabilization, and cellular dysfunction [30]. These findings suggest that even suberythemal UVB exposure may contribute to long-term skin damage and increase the risk of skin cancer. The most significant form of this damage is the mutation of key genes responsible for regulating cell growth and repair, including tumor suppressor genes such as p53 [31]. Mutations in the p53 gene impair the cell's ability to halt the cell cycle and repair DNA, increasing the likelihood that mutations will persist and accumulate over time [32]. This can lead to the initiation of carcinogenesis, the process by which normal cells transform into cancer cells.

According to Jagoda and Dixon, 1,25-dihydroxyvitamin D3 (1,25D) and its analogs play a protective role in mitigating UV radiation-induced oxidative stress by reducing both direct and indirect DNA damage in skin cells [33]. This is achieved, as shown in Fig. 2a, through a reduction in reactive oxygen species (ROS) and the enhancement of DNA repair mechanisms like p53 activation. This directly relates to the exacerbation of UV-induced DNA damage by oxidative stress from UVA radiation, as 1,25D helps to combat this oxidative stress and protect against further cellular damage as shown in Fig. 2b. Unlike UVB, which directly interacts with DNA, UVA radiation penetrates deeper into the skin, reaching the dermis [1].

Here, it generates ROS, highly reactive molecules that damage cellular components, including lipids, proteins, and nucleic acids. ROS initiate oxidative damage that weakens the skin's structural integrity and promotes premature skin aging, or photoaging. This process is characterized by the breakdown of collagen and elastin fibers, leading to wrinkles, loss of skin elasticity, and changes in skin texture. Long-term oxidative damage can also impair the skin's immune function, reducing its ability to defend against infections and repair damage.

2.1.3. Skin cancers: The role of UVB and UVA in carcinogenesis

One of the most concerning long-term effects of UV radiation is its role in the development of skin cancers, including melanoma, basal cell carcinoma (BCC), and squamous cell carcinoma (SCC). These cancers arise from different types of skin cells but share a common risk factor in UV-induced DNA damage [34]. Fakhrioliaei et al. contributed to a broader understanding of cancer mechanisms, which indirectly supports research on UV-induced skin cancers by elucidating the roles of Nrf2 (nuclear factor erythroid 2-related factor 2), HER2 (human epidermal growth factor receptor 2), and ALDH (aldehyde dehydrogenase) in cancer development and resistance [35]. Their findings are aligned with the goals of SDG 3 in improving cancer treatment and prevention, particularly for cancers driven by environmental factors like UV radiation.

- Melanoma is the most dangerous form of skin cancer and arises from melanocytes, the cells responsible for producing melanin, the pigment that helps protect the skin from UV damage [36]. Melanoma is strongly linked to intermittent, intense UV exposure, such as severe sunburns, particularly during childhood and adolescence [37]. The DNA damage caused by UVB radiation in melanocytes can lead to mutations in genes such as BRAF and NRAS, which drive the uncontrolled growth of these cells [38]. The delayed repair of CPDs in melanocytes also contributes to the accumulation of mutations, increasing the risk of cancer development [39]. According to Cherrie et al. outdoor construction workers in Britain are at significant risk of non-melanoma skin cancer (NMSC) due to prolonged exposure to solar UV radiation [40]. The study found that workers accumulate enough UV exposure over 30-40 years to more than double their risk of developing NMSC.
- BCC and SCC are more common than melanoma but less aggressive. Both types of cancer are strongly associated with chronic UV exposure, particularly in individuals who spend prolonged

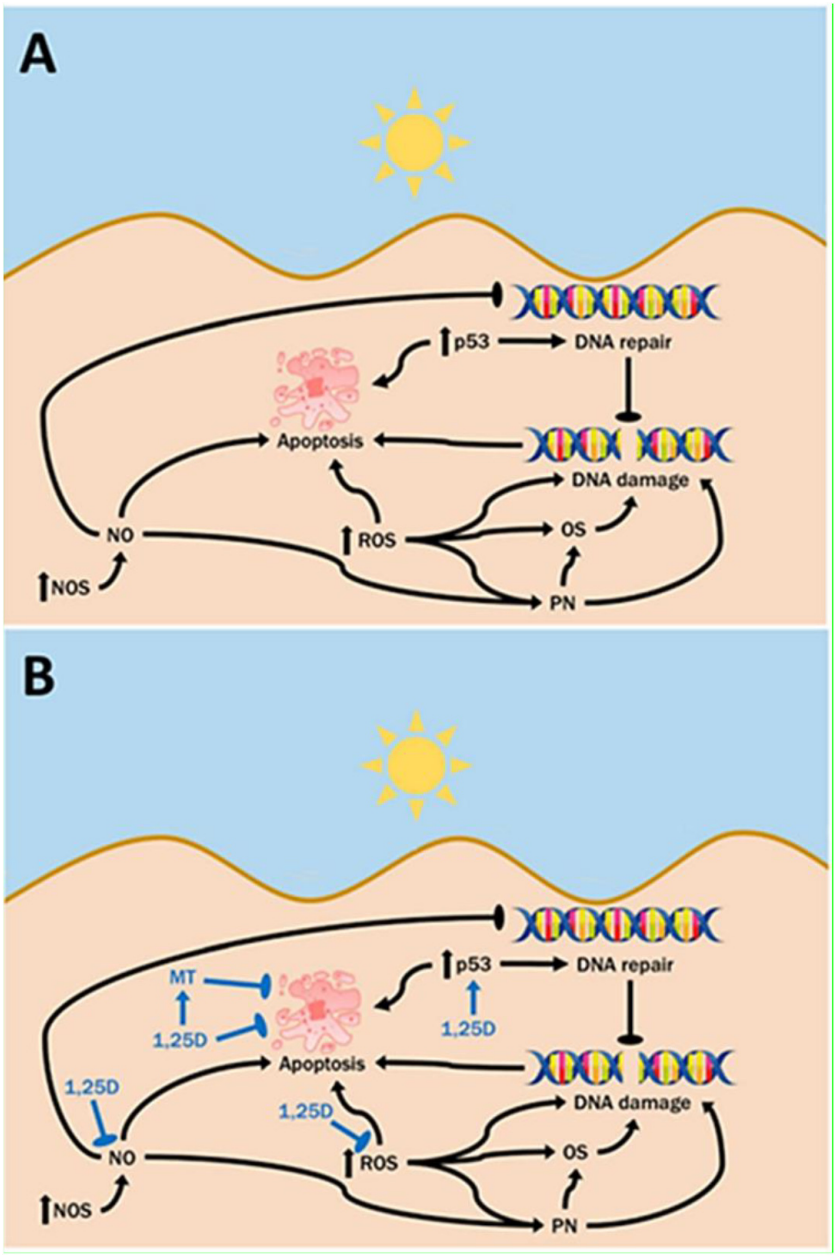

Fig. 2. UV exposure causes a. direct and b. indirect DNA damage, while 1,25D reduces damage and promotes repair.

periods outdoors [41]. BCC originates from basal cells in the epidermis [42], while SCC arises from keratinocytes, the most abundant cell type in the epidermis [43]. UV-induced mutations in the hedgehog signaling pathway are a hallmark of BCC, while SCC is often linked to UVB-induced mutations in p53 [44]. These cancers often develop in sun-exposed areas of the skin, such as the face, neck, and arms, and are more likely to occur in older individuals with a history of cumulative sun exposure.

2.1.4. Protective mechanisms and skin adaptations

In response to UV exposure, the skin has evolved several protective mechanisms. The most notable is the production of melanin, a pigment synthesized by melanocytes that absorbs and dissipates UV radiation, reducing its penetration into deeper skin layers. According to Marcovici et al. melanin and melanin-functionalized nanoparticles have shown great promise in cancer research due to their unique physicochemical properties, such as high biocompatibility, antioxidant, and photoprotective effects

Fig. 3. a. Before and b. after applying Er^{3+}/TiO_2 doped contact lenses without affecting the retinal image quality.

[45]. These nanoparticles enhance targeted cancer therapies, providing more efficient treatment options with reduced systemic toxicity, which aligns with advancements in UV-related skin cancer research. Tanning is the skin's adaptive response to UV exposure, where melanocytes increase melanin production to shield the underlying tissues from further damage [46]. However, this protection is not foolproof, and even tanned skin remains vulnerable to UV-induced damage. Additionally, the skin has DNA repair mechanisms, such as NER, which helps fix UV-induced lesions like CPDs [47]. However, these repair processes are not always efficient, and mutations can persist if damage is too extensive, or repair is incomplete.

2.2. Ocular effects

UV radiation, while essential for certain biological processes, poses a significant threat to eye health. Chronic or acute exposure to UV radiation can lead to several ocular conditions, including cataracts, pterygium, photokeratitis, and macular degeneration [48]. Among these, cataracts are the most common cause of blindness worldwide, and UV radiation plays a well-documented role in their development [49]. These lenses are used in sunglasses, contact lenses, and prescription glasses. High-quality lenses, such as those labeled "UV400," block 99-100% of UV rays [50]. Some modern contact lenses also offer UV protection, but they should be paired with sunglasses for full coverage of the eye area. [51] In addition to UV protection, advancements in UV-blocking lenses include the use of materials like Er^{3+}/TiO_2 nanoparticles, which block both UV and blue light. As noted by Shaker et al., these nanoparticles enhance optical properties without compromising image quality as shown in Fig. 3 [52]. Blue light, emitted by digital devices and the sun, is linked to eye strain and potential retinal damage, and some UV-blocking lenses now provide protection against it [53].

By preventing long-term eye conditions and promoting eye health, UV-blocking lenses contribute to SDG 3: Good Health and Well-being. They represent an important tool in mitigating UV exposure and ensuring healthy vision in regions with high sunlight intensity. According to Rahmani et al., Acuvue Oasys soft contact lenses met the ANSI Z80.20 criteria for UV transmission, providing effective protection against both UVA and UVB rays [54]. This makes them a suitable choice for individuals seeking enhanced UV protection for their eyes, aligning with public health goals under SDG 3: Good Health and Well-being.

2.2.1. UV radiation and cataracts

Cataracts occur when the lens of the eye becomes cloudy, impairing vision [55]. This condition is closely linked to UVB radiation, which is absorbed by the lens and cornea [56]. According to Taylor et al. high cumulative exposure to UVB radiation significantly increases the risk of cortical cataract formation [57]. Their epidemiologic survey of 838 watermen revealed that those with the highest UVB exposure had a 3.3 times greater risk of developing cortical cataracts, underscoring the importance of UVB protection for eye health. Over time, exposure to UVB rays causes oxidative damage to lens proteins, leading to protein aggregation and the formation of opacities in the lens [58]. This damage disrupts the normal transmission of light, resulting in the characteristic cloudiness of cataracts. UVB-induced oxidative stress also leads to lipid peroxidation, which further exacerbates protein aggregation and damage to the lens fibers [59]. The accumulation of this damage over the

years is particularly evident in individuals exposed to high levels of UV radiation, such as outdoor workers or people living in regions with strong sunlight [60]. Research suggests that UVB radiation accelerates the aging process in the lens, increasing the risk of cataracts at an earlier age than would otherwise occur.

2.2.2. Photokeratitis and corneal damage

The cornea, the eye's outermost protective layer, absorbs all UVB radiation and a portion of UVA radiation [61]. While the cornea plays a key role in filtering UV radiation to protect the inner eye, it is also vulnerable to UV damage. Photokeratitis, often referred to as "sunburn of the eye," is an acute condition that results from intense UV exposure, typically after activities like skiing or spending extended periods in bright sunlight without protection. Excessive exposure to UV radiation, according to Yustheresani et al., significantly increases the risk of photokeratitis, particularly among informal welding workers in Depok, West Java, Indonesia [62]. Their study revealed that 84% of workers experienced photokeratitis, with UV exposure levels exceeding the Threshold Limit Values (TLV) in 76% of cases. Factors such as inadequate eye protection, low safety knowledge, and close welding distance further amplified the risk, emphasizing the need for improved safety measures to prevent UV-induced eye damage.

Symptoms include pain, redness, tearing, and blurred vision, caused by UV-induced damage to the corneal epithelial cells. The integration of TiO_2 nanoparticles into contact lenses, as demonstrated by Shaker et al. represented a significant advancement in optical health technology, enhancing image correction while offering potential for energy-harvesting capabilities [63]. This innovation is particularly relevant in light of the cornea's role in filtering UV radiation, as the cornea itself is susceptible to UV-induced damage, such as photokeratitis, a condition resulting from acute UV exposure. Photokeratitis, often referred to as "sunburn of the eye," can lead to symptoms like pain, blurred vision, and redness, with repeated exposure potentially causing permanent damage such as pterygium [64]. The development of advanced contact lenses, such as PHEMA-TiO_2 lenses, not only improves visual acuity by reducing aberrations but also holds promise for protecting the cornea from UV damage [65]. Both the enhancement of optical systems and the protective role of these lenses align with the goals of SDG 3: Good Health and Well-being, focusing on innovative solutions to safeguard eye health against the adverse effects of UV radiation. In most cases, photokeratitis is reversible, but repeated episodes can lead to more permanent damage, including pterygium, an abnormal growth of tissue on the cornea.

2.3. Immunosuppression

UV radiation has well-documented effects on the immune system, leading to immunosuppression [66]. As shown in Fig. 4, UV-induced immunosuppression primarily occurs when UV radiation interferes with the function of specific immune cells, such as Langerhans cells in the skin, which are critical for recognizing and responding to harmful pathogens [67]. When these cells are damaged, the body's ability to detect and fight infections diminishes, making individuals more vulnerable to a range of diseases [68]. One of the most well-known examples of UV-induced immunosuppression is its role in increasing the risk of skin infections [69]. Studies have shown that excessive UV exposure can impair the skin's defense mechanisms, allowing pathogens like herpes simplex virus (HSV) to become reactivated, causing cold sores [70].

Similarly, research has highlighted that individuals exposed to prominent levels of UV radiation are more susceptible to fungal infections like tinea and bacterial infections such as Staphylococcus aureus. The inflammatory processes described by Lv et al. can be extrapolated to conditions beyond IBD, such as UV-induced skin inflammation [71]. Chronic UV exposure can trigger inflammatory responses in the skin, and Th22 cells may be implicated in the repair processes following UV-induced tissue damage. Further research into how UV radiation affects Th22 activity could lead to improved therapeutic approaches for managing inflammation caused by excessive UV exposure, thus contributing to better public health outcomes under SDG 3.

In addition to infections, UV radiation can also reduce the efficacy of the immune system's response to vaccines [72]. Some studies suggest that excessive UV exposure can lead to a reduced production of antibodies following vaccination, limiting the body's ability to protect against diseases [73]. This phenomenon has been observed in conditions such as influenza vaccination, where people exposed to higher levels of UV radiation showed a weaker immune response compared to those with lower UV exposure [74]. UV radiation-induced immunosuppression can be particularly concerning in regions with a high UV Index, where populations may face greater risks of recurrent infections and less effective vaccine responses [75]. Understanding these effects is crucial for developing strategies to protect the immune system in individuals exposed to higher UV levels [76].

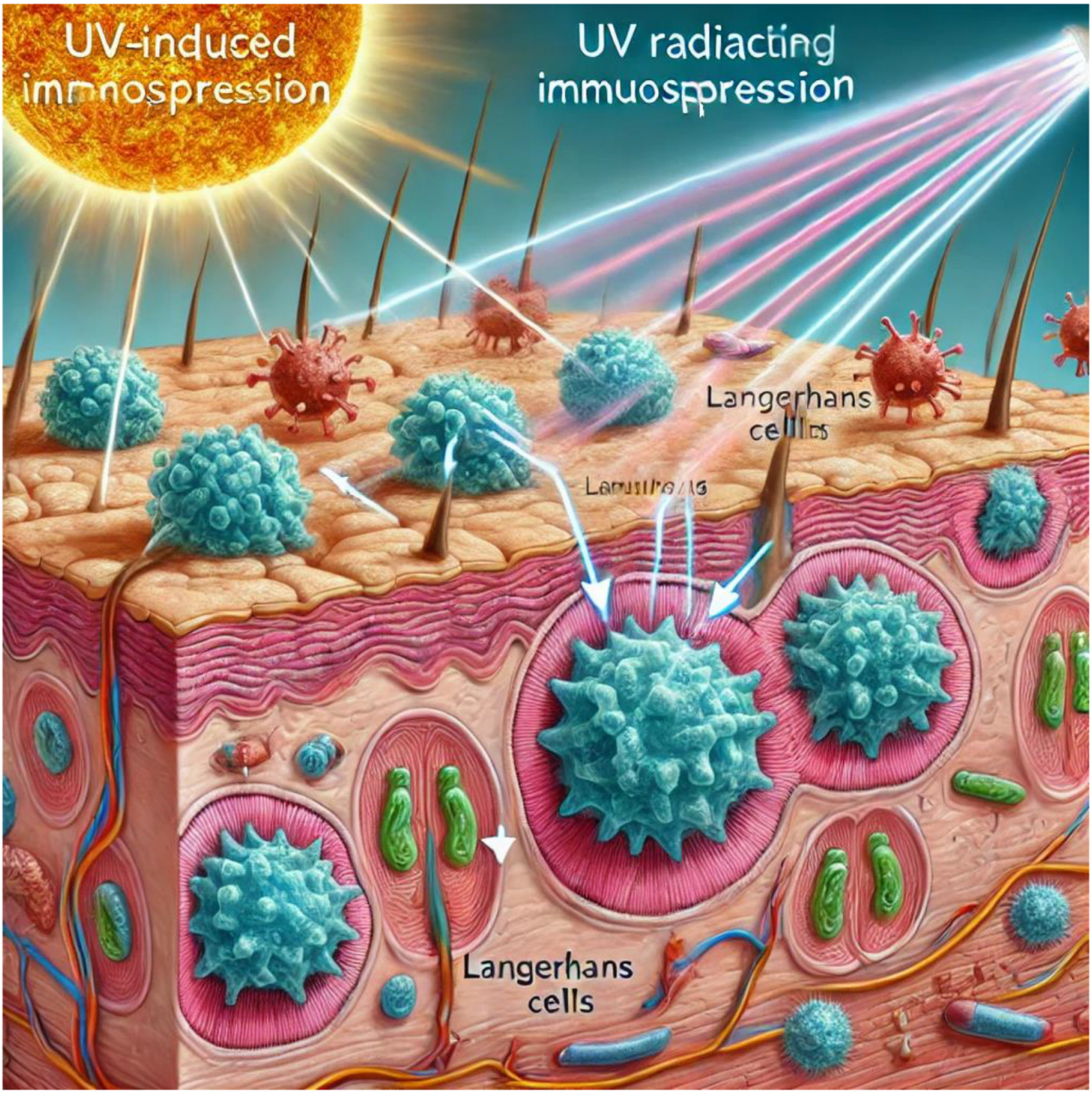

Fig. 4. Visual representation of UV-induced immunosuppression, illustrating how UV radiation affects Langerhans cells in the skin, impairing their immune function.

3. UV-induced DNA damage and repair mechanisms

3.1. DNA damage by UV radiation

UV radiation is a potent environmental factor that can cause considerable damage to the genetic material in living cells [77]. DNA is particularly vulnerable to UV radiation, especially in the UVB range, which can penetrate cells and directly interact with the DNA molecules [47]. According to Berens and Molinier, UV radiation acts as a genotoxic agent, causing photolesions that threaten genomic integrity and organism survival [78]. To counter this, specific DNA repair mechanisms are activated to maintain genome stability at UV-damaged sites. However, a key research gap exists in understanding how UV-induced DNA damage, DNA repair mechanisms, and epigenetics intersect shown in Fig. 5. Addressing this gap is crucial for comprehending how genome complexity influences DNA repair processes [79]. The primary forms of DNA lesions induced by UV radiation are

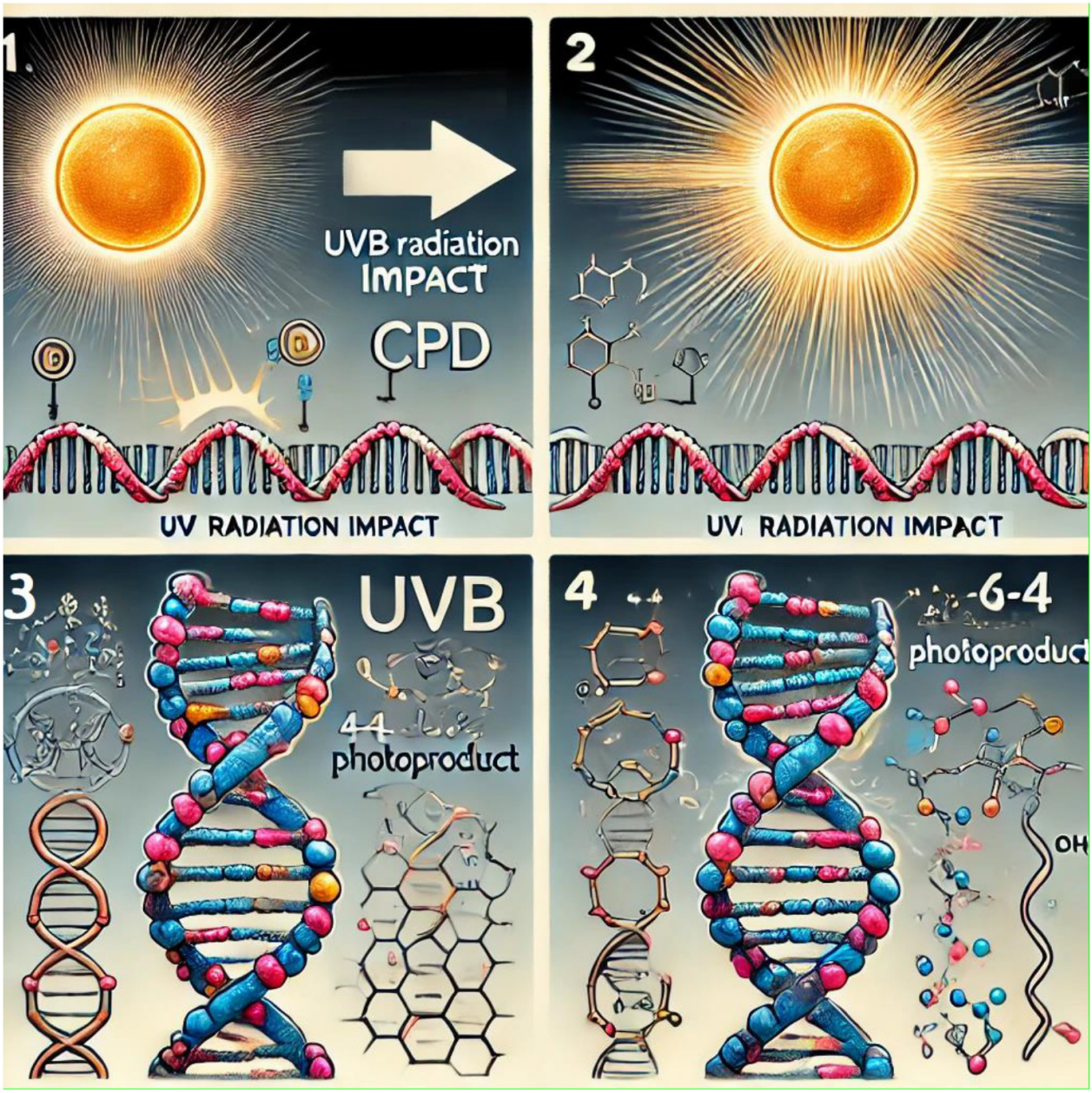

Fig. 5. Clear illustration of DNA damage by UV radiation.

CPDs and 6-4 photoproducts, both of which distort the DNA structure, disrupt normal cellular processes, and, if left unrepaired, lead to mutagenesis and cancer development.

3.1.1. Cyclobutane pyrimidine dimers (CPDs)

CPDs are the most common type of DNA lesion caused by UVB radiation [80]. These lesions occur when two adjacent pyrimidine bases, most often thymine (T) or cytosine (C), on the same DNA strand become covalently bonded. According to Mitchell and Cleaver, CPD formation also occurs with UVC irradiation, but the yield of photoproducts is influenced by distinct factors compared to UVB [81]. UVC light induces CPDs based on the sequence context, with purines at the 5′ position inhibiting dimer formation, particularly when guanine is present. Their study highlights that the wavelength of ultraviolet light plays a crucial role in determining the specificity and extent of DNA damage caused by UV exposure [82]. CPDs interfere with the ability of the DNA to properly replicate and transcribe. The resulting kink in the

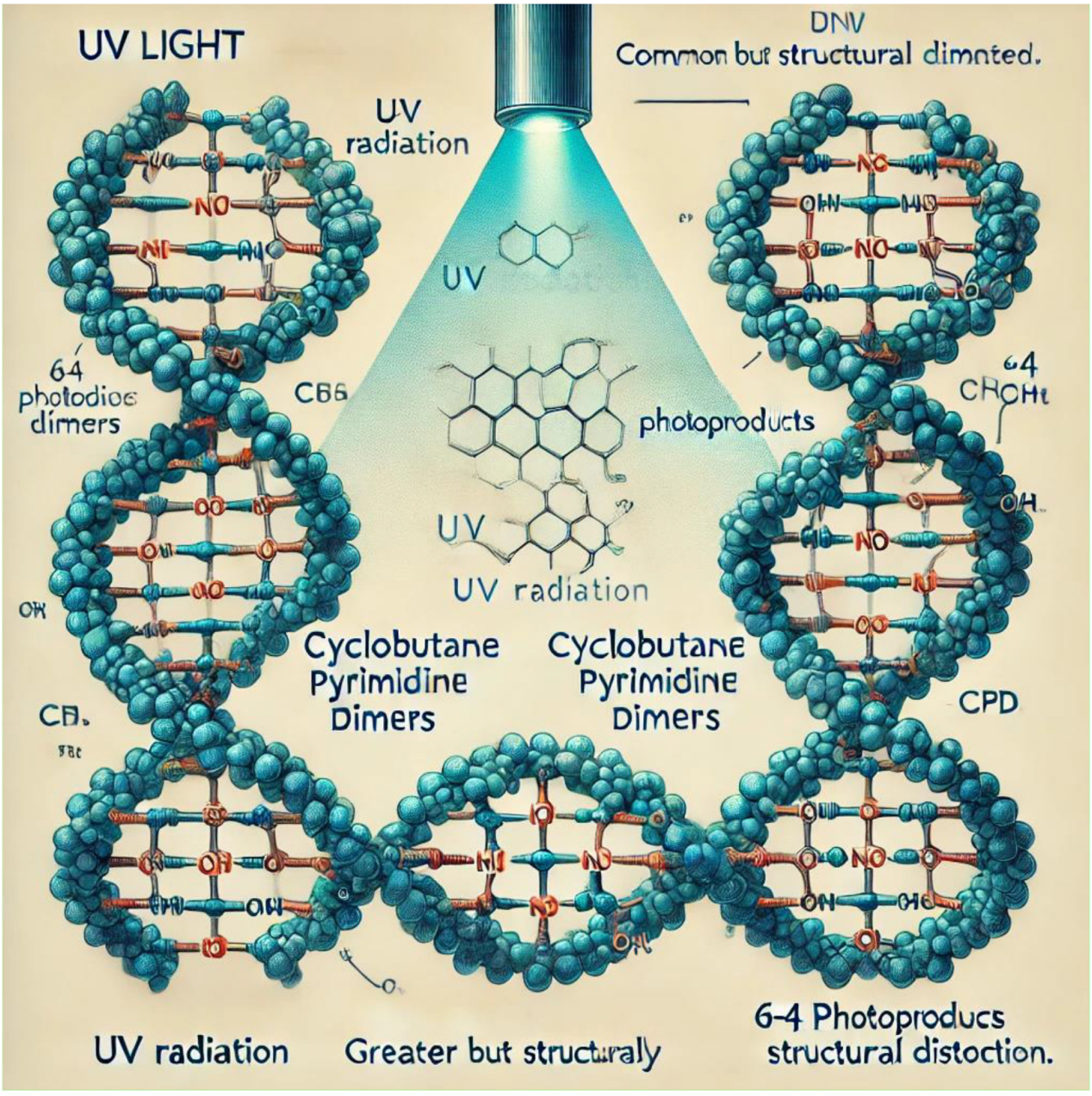

Fig. 6. UV radiation causing DNA lesions to the formation of CPDs and 6-4PPs, their structural impact on DNA, and their recognition by repair machinery.

DNA helix blocks the action of DNA polymerase during replication, potentially causing the polymerase to insert incorrect nucleotides at the site of the lesion [83]. If these errors are not corrected, they can lead to permanent mutations, which increase the risk of cellular malfunction or the development of cancer. The formation of CPDs is a key mechanism by which UV exposure leads to the accumulation of mutations, particularly in genes that control cell growth and division, such as p53, a tumor suppressor gene often mutated in skin cancers [84].

3.1.2. 6-4 photoproducts

6-4 photoproducts (6-4PPs) are another form of UV-induced DNA damage [85]. Understanding the photochemistry of DNA lesions such as cyclobutane pyrimidine dimers (CPDs), pyrimidine (6-4) photoproducts (6-4PPs), and spore photoproducts (SPs) is essential for studying UV radiation's effects on DNA as illustrated in Fig. 6 [86]. By using organic synthesis and chemical analysis, Li selectively modified pyrimidine residues to reveal key insights into the mechanisms of these photoreactions, contributing to

our knowledge of UV-induced DNA damage and its biological implications, particularly in skin cancer development [87]. In 6-4PPs, a bond forms between the C6 atom of one pyrimidine and the C4 atom of an adjacent pyrimidine, creating a more pronounced structural distortion in the DNA than CPDs. The 6-4 photoproducts are less common than CPDs but are more disruptive to the DNA structure because they produce a greater distortion in the double helix [88]. This distortion makes 6-4PPs more easily recognized by the cell's DNA repair machinery. However, if left unrepaired, 6-4PPs also interfere with DNA replication and transcription, leading to errors and potential mutations [89]. Both CPDs and 6-4PPs are significant contributors to mutagenesis. If these lesions are not quickly and accurately repaired, they can result in permanent mutations, increasing the risk of developing skin cancer, such as melanoma and BCC [90]. Cells rely primarily on the NER pathway to recognize and repair UV-induced DNA damage. This repair process removes the damaged section of DNA and fills in the gap with the correct nucleotides using the undamaged strand as a template.

3.2. DNA repair mechanisms

Cells are constantly exposed to various environmental factors that can damage their DNA, with UV radiation being one of the most common and harmful sources [91]. UV radiation can induce a variety of DNA lesions, particularly CPDs and 6-4 photoproducts, which distort the DNA double helix [92]. If left unrepaired, these lesions can lead to mutations that increase the risk of cancer, particularly skin cancer. Fortunately, cells have evolved sophisticated repair mechanisms to counteract such damage, ensuring genomic stability and preventing malignant transformations. One of the primary DNA repair pathways that addresses UV-induced damage is NER [93]. NER is a versatile repair mechanism that can recognize and remove a wide variety of helix-distorting lesions, including those caused by UV radiation [94]. This process, shown in Fig. 7, occurs in several steps: damage recognition, excision of the damaged strand, synthesis of a new strand to replace the excised section, and ligation to seal the DNA backbone. NER operates through two sub-pathways:

1. Global Genome NER (GG-NER): This sub-pathway scans the entire genome for DNA lesions, regardless of whether the DNA is being actively transcribed [95]. It recognizes bulky distortions, like CPDs and 6-4 photoproducts, which affect the integrity of the double helix. Once detected, the damage is excised by specialized enzymes, such as endonucleases, and the gap is filled in by DNA polymerases [96].
2. Transcription-Coupled NER (TC-NER): This sub-pathway focuses on repairing damage in actively transcribed genes [97]. When RNA polymerase II encounters a DNA lesion during transcription, it stalls, signaling the need for repair. TC-NER ensures that transcription can resume by quickly removing the block caused by the lesion.

Defects in this pathway are linked to disorders like xeroderma pigmentosum (XP), a condition characterized by extreme sensitivity to sunlight and a dramatically increased risk of skin cancer due to the inability to repair UV-induced DNA damage. Individuals with XP lack functional NER enzymes, leading to the accumulation of mutations, some of which may result in oncogenic changes. In addition to NER, other DNA repair mechanisms also play crucial roles. For example, base excision repair (BER) deals with smaller, non-helix-distorting lesions, while mismatch repair (MMR) corrects errors that arise during DNA replication. In some cases, homologous recombination (HR) and non-homologous end joining (NHEJ) can repair DNA double-strand breaks that may arise indirectly from UV-induced oxidative stress.

4. Biological adaptations to UV radiation

4.1. Human adaptations

The human body has evolved various mechanisms to protect itself from the harmful effects of UV radiation, a natural component of sunlight that can damage cellular DNA, proteins, and lipids. Human adaptation to UV radiation is highly variable and has been shaped by geographical, environmental [98], and genetic factors over thousands of years [99]. The variations in skin color and UV sensitivity among different populations are examples of evolutionary responses to varying levels of sun exposure in various parts of the world [100].

4.1.1. Melanin production: A natural defense against UV radiation

Melanin is the primary pigment responsible for skin color in humans, produced by cells called melanocytes located in the basal layer of the epidermis [101]. It plays a critical role in protecting the skin from the harmful effects of UV radiation by absorbing up to 99% of UV rays [102]. This process prevents the radiation from penetrating deeper into the skin, where it can damage DNA and lead to mutations that may result in skin cancers, such as melanoma [90]. Additionally, melanin helps to

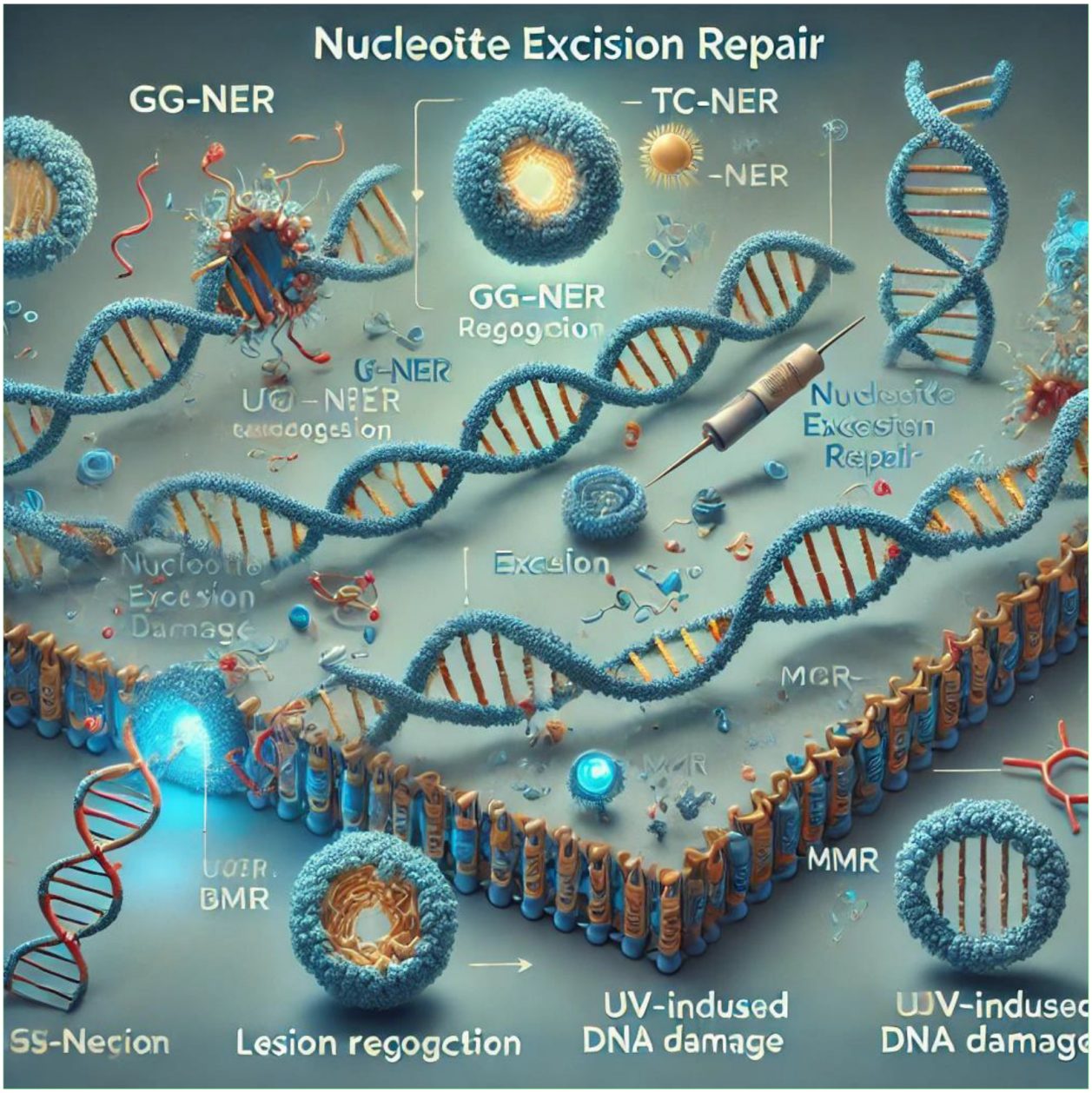

Fig. 7. DNA repair mechanisms, particularly focusing on the NER pathway with sub-pathways GG-NER and TC-NER, along with additional repair mechanisms.

neutralize ROS generated by UV exposure, which can cause oxidative stress and damage cellular components. Melanin acts by scattering UV rays and dissipating their energy as harmless heat, preventing the direct interaction of UV radiation with DNA molecules [1]. According to Ye et al., melanin's ability to absorb and transform sunlight into heat has been harnessed for a novel cancer immunotherapy [103]. Using a melanin-mediated transdermal microneedle patch, the therapy promotes antitumor immune responses by enhancing T-cell infiltration and cytokine release. This innovation aligns with efforts to reduce skin cancer risks, potentially integrating into future UV protection strategies. By protecting the skin from excessive UV exposure, higher melanin levels help prevent skin cancers and other UV-related health issues [104]. Conversely, populations that evolved in regions with lower UV radiation, particularly at higher latitudes, developed lighter skin over time. The reason for this adaptation is linked to the balance between UV protection and the need for UVB radiation to synthesize vitamin D [105]. UVB rays are essential for converting 7-dehydrocholesterol in the skin into vitamin D, a critical nutrient for bone health,

Table 1. Varying levels of vulnerability to UV-induced skin damage and skin cancers across fair, medium, and dark skin tones, as well as recommended protective measures for each group.

Skin Type	Melanin Levels	UV Vulnerability	Sunburn Risk	Skin Cancer Risk	Protection Required
Type I and II (Fair Skin)	Low	High	High	High	Sunscreen, clothing, shade
Type III and IV (Medium Skin)	Medium	Moderate	Moderate	Moderate	Sunscreen, moderate protection
Type V and VI (Dark Skin)	High	Low	Low	Low	Less protection needed

immune function, and other physiological processes. In areas with lower UV levels, having lighter skin allows for more efficient absorption of UVB radiation, facilitating sufficient vitamin D production. This explains why populations in Northern Europe, for instance, tend to have lighter skin compared to populations in equatorial Africa.

4.1.2. Variability in UV sensitivity among populations and skin types

Human populations exhibit a wide range of skin types, classified by the Fitzpatrick skin phototype scale, which measures how the skin reacts to UV exposure. As shown in Table 1, the scale ranges from Type I, characterized by very fair skin that burns easily and rarely tans, to Type VI, which describes very dark skin that rarely burns and tans easily [100]. These skin types reflect the varying degrees of melanin production and, consequently, the level of natural protection against UV radiation. According to Miyamura et al., UVA tanning provides minimal photoprotection, despite similar visual tanning to UVB-induced tans [46]. UVB exposure stimulates melanin production, offering greater protection against DNA damage, whereas UVA tanning results from photooxidation with negligible protective effects. Both forms of tanning, however, contribute to DNA damage and potential photocarcinogenesis [106]. However, despite the lower risk, dark-skinned individuals are not immune to UV damage and can still develop skin cancers, albeit at lower rates [107]. Furthermore, darker skin has been linked to a higher risk of vitamin D deficiency in areas with low UVB exposure, as the high melanin content reduces the skin's ability to synthesize vitamin D efficiently [108].

4.1.3. Genetic variability in UV sensitivity

Apart from melanin production, genetic factors also play a role in how different individuals and populations respond to UV radiation [101]. Variations in genes involved in melanin synthesis, such as MC1R (melanocortin 1 receptor), contribute to the diversity of skin colors and UV sensitivity among humans [109]. For example, certain variants of the MC1R gene are associated with red hair, fair skin, and a higher susceptibility to UV damage. Moreover, some individuals have genetic mutations that impair their DNA repair mechanisms, making them more susceptible to UV-induced skin damage and cancers. XP, for instance, is a rare genetic disorder that results in extreme sensitivity to UV radiation due to defects in NER pathways [110].

5. Public health initiatives and preventive measures

Exposure to UV radiation poses significant health risks, including skin cancer, premature aging, and eye damage, underscoring the need for effective public health strategies. Governments, health organizations, and advocacy groups have implemented various preventive measures to educate the public on UV exposure risks [111], and promote protective actions like applying UV-blocking windows thin films to the buildings' windows [112]. One effective method is UV Index awareness campaigns, which encourage individuals to monitor daily UV levels and adjust their activities accordingly, especially during high UV Index days [3]. These campaigns highlight the importance of limiting outdoor exposure during peak sun hours, using sunscreen, and wearing protective clothing [113]. Sunscreen usage is another critical protective measure [114]. Sunscreens with a high Sun Protection Factor (SPF) shield the skin by absorbing or reflecting UV radiation [115]. Broad-spectrum sunscreens protect against both UVA and UVB rays, reducing the risks of sunburn, premature aging, and skin cancer, although reapplication every two hours remains essential for sustained protection.

In addition to sunscreen, protective clothing with a high Ultraviolet Protection Factor (UPF) [116], wide-brimmed hats, and UV-blocking sunglasses are recommended to further reduce exposure [117]. Technological advancements have also played a crucial role in UV protection, with wearable UV sensors and smartphone apps providing real-time UV exposure data. These devices alert users when they reach

Table 2. Impact of various UV protection strategies along with specific recommendations for effective use.

Strategy	Impact (%)	Recommendations
UV Index Awareness Campaigns	85%	Monitor daily UV Index, adjust outdoor activities
Proper Sunscreen Usage	90%	Use SPF 30+, reapply every 2 hours
Protective Clothing and Accessories	80%	Wear UPF-rated clothing and dark fabrics
Wearable UV Sensors	75%	Use daily to track cumulative UV exposure
Smartphone UV Monitoring Apps	70%	Check UV levels before going outside
Reapplication of Sunscreen	88%	Reapply after sweating or swimming
Seeking Shade during Peak Hours	78%	Avoid outdoor activities from 10 a.m. to 4 p.m.
UV-Protective Sunglasses	82%	Wear sunglasses with 100% UV protection
Broad-Spectrum Sunscreen Use	92%	Use broad-spectrum sunscreens to protect from UVA/UVB
Wide-brimmed Hats	84%	Use wide-brimmed hats to protect face, neck, and ears

unsafe UV levels, allowing them to take timely action, such as applying sunscreen or seeking shade. For instance, smartphone apps like SunSmart Global UV [118], and QSun provide location-specific UV Index readings and personalized advice on sun protection, enhancing users' ability to manage their exposure [119]. Overall, the combination of public awareness campaigns, protective measures, and real-time UV monitoring technologies empowers individuals to reduce their UV exposure and minimize the associated health risks. Basing on the general effectiveness and prominence of each strategy discussed in the literature, Table 2 highlights the relative impact of different public health strategies and technological advancements in reducing UV exposure.

6. Discussion: Aligning the research gaps of the current study with sustainable development goals (SDGs)

In addressing the complex biological impacts of UV radiation, it is essential to frame the identified research gaps within the broader context of the United Nations' Sustainable Development Goals (SDGs). By aligning these research priorities with the SDGs, we ensure that future studies not only advance scientific understanding but also contribute to global health, environmental sustainability, and equitable development. UV radiation impacts various aspects of human health, ecosystems, and climate, all of which align with specific SDGs as illustrated in Fig. 8.

The following discussion explores how the critical research gaps identified in this study intersect with specific SDGs, highlighting their broader significance and potential impact.

Long-Term UV Exposure Effects (90%) – SDG 3: Good Health and Well-being: This research gap addresses the cumulative impact of chronic, low-level UV radiation exposure on health, which is directly aligned with SDG 3, promoting healthy lives and well-being for all. Understanding how sustained UV exposure contributes to skin aging, DNA damage, and immune suppression is critical for mitigating skin cancer risks, which continue to rise globally. As this gap directly impacts human health outcomes, the prioritization of this area is crucial for developing interventions that protect populations at risk, particularly in regions with high UV indices.

1. UV-Induced Immunosuppression Mechanisms (85%) – SDG 3: Good Health and Well-being

The immune system's response to UV radiation is a growing concern, particularly regarding its role in suppressing immune function and increasing susceptibility to infections and reduced vaccine efficacy. This is highly relevant to SDG 3, as it affects public health outcomes, including the fight against infectious diseases and maintaining robust immune defenses. Deeper investigation into the mechanisms of UV-induced immunosuppression is essential for regions exposed to intense sunlight, where populations may experience compromised immune responses, exacerbating vulnerabilities to diseases.

2. Climate Change, Ozone Depletion, and UV Radiation Patterns (75%) – SDG 13: Climate Action

The interplay between climate change, ozone layer depletion, and shifting UV radiation patterns presents a critical research area linked to SDG 13, which focuses on combating climate change and its impacts. Changes in atmospheric conditions directly affect regional UV exposure levels, influencing human health and ecosystem resilience. Research in this area is vital for understanding future trends in UV radiation and developing adaptive strategies to reduce environmental and health risks, particularly as climate change progresses.

3. Impact of UV Radiation on the Skin Microbiome (60%) – SDG 3: Good Health and Well-being

Emerging studies on the human skin microbiome and its interaction with UV radiation are still in their infancy, yet they hold significant potential for understanding dermatological health. As the skin microbiome plays a pivotal role in immune defense and maintaining skin integrity, disruptions caused by UV

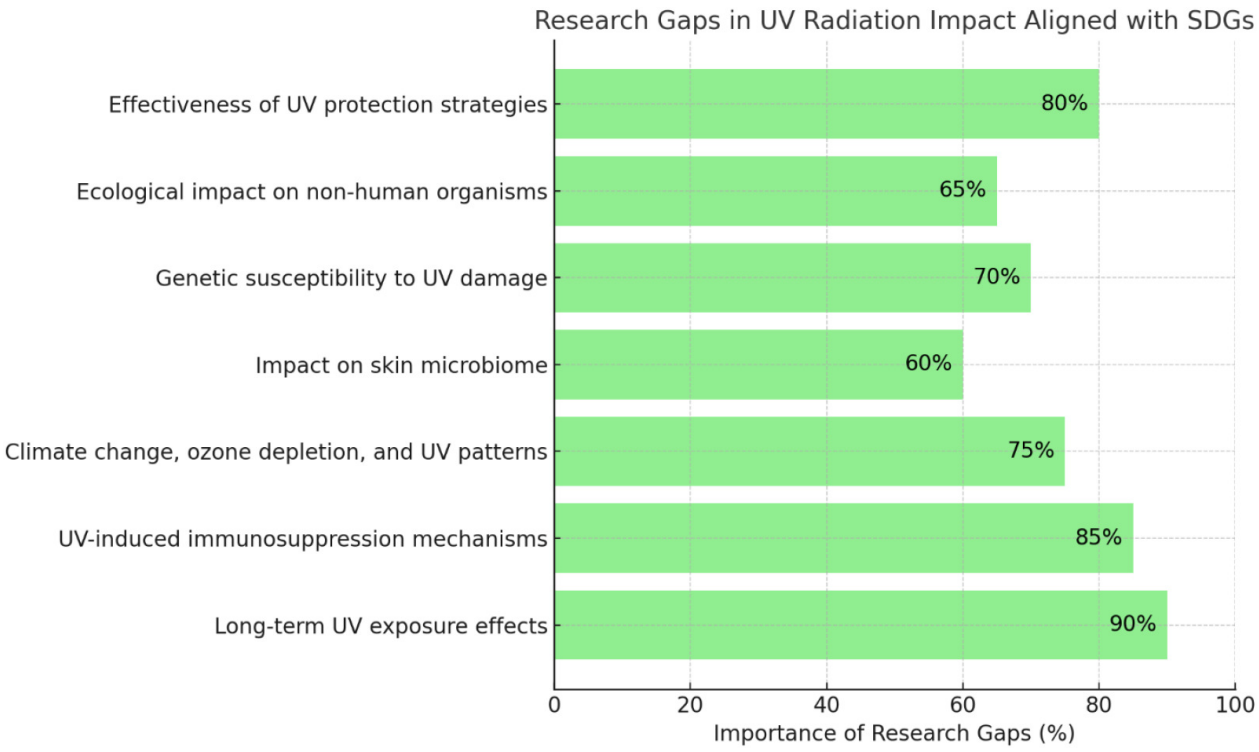

Fig. 8. Relative importance of each research area in percentage, highlighting its alignment with specific SDGs. This chart illustrates the prioritization of this work research gaps in in UV radiation based on their potential contributions.

exposure could have broader implications for human health. Further research in this area will contribute to SDG 3 by advancing knowledge on how UV radiation affects microbial communities and influencing the development of protective skincare measures.

4. Genetic Susceptibility to UV Damage (70%) – SDG 3: Good Health and Well-being

This gap, which involves understanding individual genetic variability in response to UV radiation, particularly regarding DNA repair and melanin production, is closely related to SDG 3. Populations with different genetic profiles exhibit varying levels of UV sensitivity, with some individuals at greater risk of skin cancers and other UV-induced damage. Researching these genetic differences will enable the development of personalized sun protection strategies, which could reduce the global burden of UV-related health conditions, particularly skin cancer.

5. Ecological Impact on Non-Human Organisms (65%) – SDG 14: Life Below Water and SDG 15: Life on Land

Increased UV radiation has profound implications for both marine and terrestrial ecosystems, affecting biodiversity and ecosystem functions. Phytoplankton, a foundational element of the marine food web, is particularly vulnerable to UV-induced damage, impacting global carbon cycles (SDG 14). Likewise, UV radiation poses threats to terrestrial plants and animals, affecting crop yields and biodiversity (SDG 15). These ecological disruptions warrant further research to develop conservation strategies and resilience frameworks to protect life on land and below water.

6. Effectiveness of UV Protection Strategies (80%) – SDG 12: Responsible Consumption and Production

The long-term effectiveness of UV protection strategies, including sunscreens, clothing, and wearable UV monitors, aligns with SDG 12, which promotes sustainable consumption and production practices. Evaluating the efficacy of these measures over time and across different populations is essential for reducing UV-induced health risks while ensuring that protective products are produced in an environmentally responsible manner. Sustainable development requires not only effective health measures but also practices that minimize ecological harm, making this area of research highly relevant.

7. Future recommendations

- **Mechanisms of UV-Induced DNA Damage and Repair:** this topic focuses on the investigation

the molecular mechanisms underlying the body's repair processes in response to UV-induced DNA damage, especially the efficiency of NER and BER in different cell types. This includes exploring the threshold of UV exposure that overwhelms DNA repair pathways and the role of genetic polymorphisms in susceptibility to UV-induced mutations. This research could contribute to cancer prevention strategies by identifying biomarkers of impaired DNA repair and lead to personalized UV protection approaches for high-risk individuals.

- **The Role of UV Radiation in Immune System Modulation and Disease Susceptibility:** this topic focuses on studying the effects of UV radiation on the immune system, particularly its role in immunosuppression and how it increases susceptibility to infectious diseases, autoimmune conditions, and reduces vaccine efficacy. Explore the underlying molecular pathways linking UV radiation and immune system dysregulation. Findings could enhance public health guidelines on UV exposure, improve vaccination strategies, and identify populations at higher risk for immune-related complications from UV exposure.
- **Genetic Susceptibility and Personalized UV Protection Strategies:** this topic focuses on the conduction of genomic studies to investigate how genetic variations in genes related to melanin production, DNA repair, and oxidative stress response affect an individual's sensitivity to UV radiation. This includes studying how specific populations may be more vulnerable to UV-induced skin cancer and other diseases. This research can pave the way for precision medicine in dermatology, where UV protection guidelines are tailored based on individual genetic risk profiles, improving skin cancer prevention.
- **Impact of Artificial UV Sources on Public Health:** this topic focuses on the investigation of the health effects of artificial UV exposure from sources such as tanning beds, UV lamps, and certain industrial processes. Focus on the risks of artificial UV radiation in contributing to skin cancers, premature aging, and eye diseases. This research could lead to improved regulations and public health warnings around the use of artificial UV sources, reducing the incidence of preventable diseases related to UV exposure.
- **Smart Technologies for Personalized UV Exposure Management:** this topic focuses on studying the development and integration of smart technologies, such as wearable UV sensors and smartphone apps, for personalized UV exposure tracking and tailored protection strategies. Analyze the potential of these technologies in reducing UV-related health risks over time. Findings could accelerate the adoption of real-time UV monitoring technologies and improve user engagement, leading to more effective sun protection practices and a decrease in UV-related diseases.

8. Conclusion

The findings presented underscore the profound short- and long-term effects of UV radiation on skin, eyes, and broader ecological systems. Our analysis reveals that while the skin's natural defenses, such as melanin production and DNA repair mechanisms, provide some protection, excessive exposure leads to critical health concerns, including sunburn, premature aging, and increased risks of skin cancer. UVB radiation primarily causes DNA damage, while UVA contributes to oxidative stress, with both types of radiation weakening the immune system, raising susceptibility to infections and potentially diminishing vaccine efficacy in high UV regions. Our study also highlights the impact of UV radiation on marine and terrestrial ecosystems, affecting organisms like phytoplankton and plants, with implications for biodiversity and food security.

The best results from this review emphasize the urgent need for public health interventions, including UV index awareness, proper sunscreen use, and protective clothing, to mitigate these risks. Advanced UV monitoring technologies, such as wearable sensors and smartphone apps providing real-time data, are promising in enhancing public awareness and individual action against UV exposure. Aligning research with the SDGs provides a structured framework to address UV-related health challenges comprehensively, promoting both climate resilience and sustainable development. Future research focused on genetic variability, long-term exposure effects, and protective strategies for ecosystems is essential to minimize UV radiation's adverse effects, safeguarding human and environmental health.

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Conflict of interest

The authors have no competing interests to declare that are relevant to the content of this article.

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