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Review Article

The Impact of Ultraviolet Radiation on Human Health

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Abstract

This comprehensive review examines the significant health effects of ultraviolet (UV) radiation, highlighting its impact on skin and eye health, immune function, and reproductive health. Prolonged exposure to UV rays is a well-established risk factor for various forms of skin cancer, including melanoma and non-melanoma types, as well as conditions such as cataracts and photokeratitis. The mechanisms through which UV radiation exerts these harmful effects include DNA damage, oxidative stress, and immune suppression, underscoring the need for protective measures. The UV Index is introduced as a vital tool for assessing UV intensity and informing individuals about the risks associated with sun exposure. By implementing effective sun safety strategies, such as using sunscreen, wearing protective clothing, and seeking shade, individuals can significantly mitigate their risk of UV-related health issues. As the understanding of UV radiation's health effects continues to evolve, public awareness and education remain crucial in promoting long-term skin and eye health, as well as overall well-being. This review emphasizes the importance of proactive measures in protecting against the growing risks associated with increased UV exposure in today's environment.

Keywords: Ultraviolet Radiation, Skin Cancer, Immune Suppression, UV Index, Sun Protection

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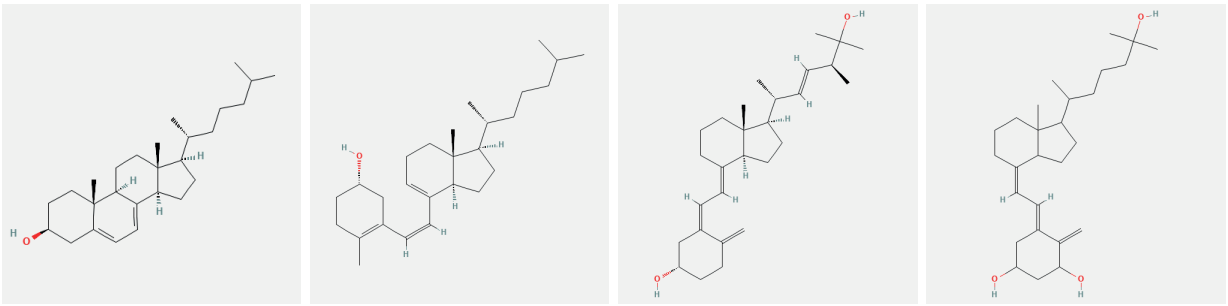
1. Introduction

The world has been experiencing hot days in recent years due to climate change and global warming. People are concerned about the effects of sunlight on their health. UV radiation is electromagnetic radiation with wavelengths shorter than visible light and longer than X-rays (10 nm to 400 nm) [1]. Based on its wavelength, UV radiation is divided into three categories: A, B, and C. UV-A radiation has the least energy and mostly reaches the Earth's surface [2]. Although this part of UV radiation can cause premature skin aging and some types of skin cancer, it also plays a key role in the production of vitamin D in the human body [3, 4]. Therefore, adequate exposure to this radiation is essential for maintaining appropriate vitamin D levels in the body. Vitamin D production in the skin begins when it is exposed to sunlight, specifically UV-B radiation [5]. When the skin absorbs this UV-B radiation, a chemical reaction is triggered that is essential for vitamin D synthesis. In the skin, there is a substance known as 7-dehydrocholesterol ($C_{27}H_{44}O$), which is a derivative of cholesterol [6]. 7-dehydrocholesterol has a complex sterol structure (see Figure **1**a). When UV-B rays penetrate the skin, 7-dehydrocholesterol is exposed to UV light and can absorb enough energy, which causes the breaking of the carbon-hydrogen bond at the C9 position [7]. Then the photochemical reaction causes a rearrangement of the molecule, like breaking the double bonds and forming new bonds in the 7-dehydrocholesterol molecule structure, leading to the cyclization of the structure (see Figure **1**b). The reaction results in the rearrangement of the molecular structure, leading to the formation of previtamin D3 ($C_{27}H_{44}O$) which is also a storied family [8]. This reaction can be represented as shown in Eq. 1.



(a) 7-dehydrocholesterol (b) Previtamin D3 (c) 25-hydroxyvitamin D (d) 1,25-Dihydroxyvitamin D3

Figure 1: Chemical structures of chemicals involved in Vitamin D formation.

After the formation of previtamin D3, it undergoes a heat-dependent transformation to become vitamin D3, also known as cholecalciferol (see Eq. 2) [9]. During this reaction, the opening of the B-ring occurs, allowing the molecule to rearrange. These reactions happen naturally in the skin and occur relatively quickly after UV exposure. Once vitamin D3 is produced, it enters the bloodstream and is transported to the liver. In the liver, vitamin D3 undergoes further processing. Here, it is converted into 25-hydroxyvitamin D ($C_{27}H_{44}O_2$), also referred to as calcidiol (see Figure **1**c) [10]. This form is the primary circulating version of vitamin D in the body and serves as an important marker for assessing vitamin D status in individuals.

The final activation of vitamin D occurs in the kidneys [11]. In this organ, 25-hydroxyvitamin D is converted into 1,25-dihydroxyvitamin D ($C_{27}H_{44}O_3$), also known as calcitriol (see Figure **1**d). Calcitriol is the active form of vitamin D and plays a vital role in regulating calcium and phosphorus levels in the body [12]. It is essential for maintaining bone health and also influences various bodily functions, including immune response. The following pathways highlight the intricate process through which UV radiation contributes to the production of vitamin D in the human body, emphasizing the importance of sunlight exposure for maintaining adequate vitamin D levels (Eq. 1 to 4).

$$7-dehydrocholesterol\ \left(C_{27}H_{44}O\right)\overset{UV\ B\ Radiation}{\longrightarrow} previtamin\ D3\ \left(C_{27}H_{44}O\right) \quad (1)$$

$$Previtamin\ D3\ \left(C_{27}H_{44}O\right)\overset{Heat}{\longrightarrow} Vitamin\ D3\ \left(C_{27}H_{44}O\right) \quad (2)$$

$$Vitamin\ D3\ (C_{27}H_{44}O)\overset{In\ liver}{\longrightarrow} 25-hydroxyvitamin\ D\ \left(C_{27}H_{44}O_2\right) \quad (3)$$

$$25-hydroxyvitamin\ D\ (C_{27}H_{44}O_2)\overset{In\ kidneys}{\longrightarrow} 1,25-dihydroxyvitamin\ D\ \left(C_{27}H_{44}O_3\right) \quad (4)$$

The energy of a UV-A photon (at 400 nm) is approximately 4.97×10^{-19} Joules while the energy of a UV-B photon (at 280 nm) is about 7.10×10^{-19} Joules [13]. This means that UV-B photons carry about 1.43 times more energy than UV-A photons**.** This higher energy is why UV-B radiation has more potential to cause skin damage and induce processes like vitamin D synthesis in the skin. The ozone layer absorbs most of the UV-B radiation emitted from the sun [14]. UV-C radiation has the highest energy and can be very dangerous, but it is usually absorbed by the ozone layer and the Earth's atmosphere and does not reach the Earth's surface [15]. The energy of a UV-C photon (at 100 nm) is approximately 1.99×10^{-18} Joules [16]. The energy of a UV-C photon (at 100 nm) is approximately 2.8 times greater than the energy of a UV-B photon (at 280 nm). This significant increase in energy contributes to UV-C's higher potential for causing molecular and cellular damage. UV radiation has various applications, including disinfection, vitamin D production in the body, and some industrial and scientific uses [17].

Scientists introduced the UV index to measure how much UV radiation could be harmful to human health [18]. The UV Index is a globally recognized scale designed to assess and communicate the intensity of UV radiation from the sun at a particular time and place [19]. This index serves as a crucial tool for helping individuals protect themselves from harmful UV exposure by providing specific guidance on appropriate sun protection measures [20]. The UV Index ranges from 0 to 11+, with higher values indicating increased UV intensity and a greater skin and eye health risk [21]. Each level of the index corresponds to a specific risk category, allowing people to easily understand the necessary precautions they should take. For instance, a UV Index of 0 to 2 represents a low risk for the general population [21]. In this category, minimal protection is needed, and basic measures like wearing sunglasses are typically sufficient. As the index rises to a range of 3 to 5, indicating moderate risk, individuals are advised to take additional precautions,

such as wearing a hat and sunglasses and applying sunscreen [21]. When the UV Index reaches levels of 6 to 7, it is classified as high risk, which calls for stronger protective measures, including wearing long clothing and using high-SPF sunscreen to shield the skin from harmful rays. The risk becomes even more serious as the UV Index moves into the "Very High" category, defined as 8 to 10. During these levels, it is recommended to seek shade during peak sun hours, typically from 10 AM to 4 PM, when UV radiation is most intense. At an "Extreme" level of 11+, all protective measures are crucial, and it is best to avoid direct sun exposure during these times altogether [21]. Meteorological organizations play an essential role in reporting the UV Index daily, and this information is readily accessible through weather websites and news outlets worldwide. This widespread availability empowers individuals to make informed decisions about sun exposure and to take necessary precautions to protect themselves.

Overall, UV radiation is generally strongest during the summer months and peaks around midday, when levels can reach potentially hazardous heights [22]. To effectively reduce the risk associated with excessive UV exposure, it is advisable to limit outdoor activities during peak hours and to wear protective clothing that covers most of the body [23]. Additionally, using a high-SPF sunscreen is recommended to further minimize the harmful effects of UV radiation [24]. These proactive measures are essential for promoting long-term skin and eye health in people around the world.

The significance of this study lies in its comprehensive examination of the multifaceted health effects of UV radiation, addressing a critical area of public health that impacts individuals globally. By consolidating current research findings on the detrimental impacts of UV exposure—including skin cancers, ocular conditions, and immune dysfunction—this study aims to enhance awareness and understanding of the risks associated with UV radiation. This is particularly crucial in the context of increasing UV exposure due to environmental changes and lifestyle factors. The novelty of this study is reflected in its integrative approach, which not only highlights established health concerns but also explores emerging insights into the effects of UV radiation on reproductive health and overall well-being. Furthermore, by emphasizing the importance of the UV Index as a tool for risk assessment, this study provides practical recommendations for effective sun protection strategies tailored to different risk levels. Ultimately, the findings aim to contribute to public health initiatives and educational programs that promote proactive measures against UV-related health issues, fostering a more informed and health-conscious society.

2. Effects of UV Radiation on Health

2.1. Skin Damage

Sunburn is a type of skin damage that happens when the skin is exposed to too much UV radiation [25]. UV radiation mainly comes from the sun but can also come from artificial sources like tanning beds [26]. Tanning beds, also known as sunbeds, are devices that people use to get an artificial tan. They are designed to emit UV radiation, which darkens the skin, mimicking the effect of natural sunlight. A tanning

bed typically has a series of fluorescent lamps that produce UV-A and sometimes UV-B rays. When a person lies down or stands in bed, the UV light stimulates the production of melanin in the skin, which creates a tanned appearance. When the skin absorbs more UV radiation than it can handle, the result is sunburn [25]. This can cause redness, pain, swelling, and even blisters in severe cases. The skin's reaction to UV overexposure is a sign that it has been damaged.

Sunburn occurs primarily due to two types of UV rays: UV-A and UV-B. UV-B rays are usually the main cause of sunburn [27]. They have shorter wavelengths and deliver more energy to the skin's outer layer. UV-A rays penetrate deeper into the skin and are linked to skin aging. Both types of UV rays damage the DNA in skin cells [28]. When the DNA in skin cells gets damaged, it can lead to problems such as skin cancer [29]. To repair itself, the skin releases chemicals, such as cytokines and prostaglandins. These chemicals trigger inflammation, which is why sunburned skin turns red and feels sore [30]. The body's immune system plays a key role in the sunburn reaction. Blood vessels in the sunburned area expand, causing redness and warmth [31]. White blood cells then move to the area to help repair damaged cells. However, this immune activity also makes the skin more irritated, which leads to pain [32]. This response acts as a warning sign to prevent further UV damage.The pain of sunburn often peaks between 6 to 48 hours after exposure [33]. In the days following, the skin may begin to peel. Peeling is a natural way for the body to remove damaged cells [33]. These damaged cells might contain harmful DNA mutations, so shedding them is protective. However, repeated sunburns or long-term UV exposure can lead to more serious issues, like skin cancer or premature aging [34]. Preventing sunburn is important and can be achieved with protective measures. Sunscreens with high sun protection factor (SPF) ratings provide defense against both UV-A and UV-B rays [35]. Sunscreens protect the skin by using ingredients that absorb, reflect, or scatter UV radiation before they can reach deeper skin layers and cause harm. There are two main types of active ingredients in sunscreens, each with a unique way of providing protection. The first type is chemical filters, such as avobenzone or oxybenzone [36]. These ingredients work by absorbing UV radiation, transforming it into heat, and releasing it harmlessly away from the skin [37]. This prevents the UV rays from penetrating and damaging skin cells. The second type is physical or mineral filters, like zinc oxide and titanium dioxide. These ingredients sit on the skin's surface and act as a physical barrier, reflecting or scattering UV rays to prevent them from entering the skin [38]. By combining these mechanisms, sunscreens effectively reduce the risks of sunburn, DNA damage, premature skin aging, and skin cancer, making them essential for daily skin protection. Wearing protective clothing, seeking shade during peak sunlight hours, and avoiding tanning beds are also other effective ways to protect ourselves against UV radiation [38].

2.2. Premature Aging

Premature aging of the skin caused by UV radiation, commonly referred to as photoaging, is a significant concern in dermatology [39]. This phenomenon occurs when the skin is subjected to UV rays from the sun or artificial sources, leading to accelerated changes that mimic the natural aging process [40]. The primary culprits behind photoaging are UV-A and UV-B rays, with UV-A being the major contributor to long-term skin damage [41]. Understanding the mechanisms by which UV radiation causes premature aging is crucial for developing effective prevention strategies. One of the key mechanisms of skin aging due to UV exposure is the damage to collagen and elastin fibers, which are vital components of the skin's structure [42]. UV radiation, particularly UV-A rays, penetrates deeply into the skin, reaching the dermis where collagen and elastin are located. This deep penetration leads to the generation of reactive oxygen species, which are unstable molecules that can cause oxidative stress. The oxidative stress damages collagen and elastin fibers, leading to a breakdown in their structural integrity [43]. As a result, the skin loses its firmness and elasticity, contributing to the formation of wrinkles and sagging.

The oxidative stress caused by UV exposure also stimulates the production of matrix metalloproteinases [43]. These enzymes play a crucial role in the degradation of collagen and other components of the extracellular matrix [44]. When matrix metalloproteinases activity increases in response to UV radiation, it leads to the breakdown of collagen, further accelerating the aging process [45]. This enzymatic activity is a critical mechanism through which UV radiation promotes structural degradation of the skin, enhancing the appearance of aging signs. Additionally, UV radiation triggers an inflammatory response in the skin [46]. This inflammatory response involves the release of pro-inflammatory cytokines and other mediators that can lead to inflammation and skin damage. Chronic inflammation can exacerbate the breakdown of collagen and elastin, contributing to a loss of skin barrier function and promoting further signs of aging [47]. Over time, the accumulation of inflammation can result in a more damaged and aged appearance.

Prolonged exposure to UV radiation can also disrupt the normal functioning of skin cells, including keratinocytes, fibroblasts, and melanocytes [48]. Fibroblasts, responsible for producing collagen and elastin, may become less effective in their roles due to UV-induced damage [49]. Keratinocytes, the primary cells in the outer layer of skin, can exhibit abnormal differentiation and proliferation, leading to an uneven skin texture and increased pigmentation [50]. Melanocytes, which produce melanin, can become hyperactive in response to UV exposure, resulting in the development of dark spots known as solar lentigines or age spots [51]. Moreover, UV radiation can directly damage the DNA in skin cells, leading to mutations that disrupt normal cellular function [52]. If this DNA damage is not properly repaired, it can result in cellular senescence, a state where cells no longer divide or function effectively. The accumulation of senescent cells in the skin contributes to the overall decline in skin health and accelerates the appearance of aging signs. This process underscores the long-term effects of UV exposure on the skin's cellular landscape. Lastly, the effects of UV exposure are cumulative, meaning that repeated sun exposure over the years compounds the damage to the skin. This cumulative effect can lead to significant changes in the

skin's appearance, including deep wrinkles, leathery texture, and uneven pigmentation [53]. The onset of these changes can occur much earlier in individuals who do not consistently practice sun protection compared to those who take measures to shield their skin from UV radiation.

2.3. Skin Cancer

2.3.1. Melanoma

Melanoma is the most serious form of skin cancer due to its aggressive nature and ability to spread, or metastasize, to other parts of the body if not detected early [54]. It originates in melanocytes, the skin cells responsible for producing melanin, the pigment that colors our skin and protects it from harmful UV rays [55]. However, excessive exposure to UV radiation—especially from the sun or tanning beds—can cause mutations in the DNA of melanocytes, disrupting their normal growth and triggering uncontrolled cell division that can lead to tumor formation [56]. Key genetic mutations, such as those affecting the BRAF and NRAS genes, are frequently involved [57]. Mutations in the BRAF gene, found in about 50% of melanoma cases, keep cell growth signals turned on, causing cells to multiply uncontrollably [58]. Pyrimidine dimers are abnormal covalent bonds that form between adjacent thymine or cytosine bases in the DNA molecule under UV radiation [59]. These dimers disrupt normal base pairing and can lead to errors during DNA replication. If not properly repaired, this damage can result in mutations that affect the genes controlling cell growth and division. Key genes that are often affected include important tumor suppressor genes, with p53 being one of the most well-known [60]. Tumor suppressor genes are crucial for maintaining normal cell function and play a significant role in preventing cancer [61]. Specifically, p53 helps control the cell cycle, ensuring that cells with damaged DNA do not continue to divide and proliferate [62]. These genes are crucial for regulating the cell cycle and stopping the growth of damaged cells. Mutations in p53 can allow damaged melanocytes to escape normal regulatory mechanisms, increasing the risk of tumor formation [52].

UV radiation also generates reactive oxygen species, which are highly reactive molecules that can damage cellular components, including lipids, proteins, and DNA [63]. The body has natural antioxidant defenses to counteract reactive oxygen species, but excessive UV exposure can overwhelm these defenses, leading to oxidative stress. This condition not only causes direct DNA damage but can also activate signaling pathways that promote inflammation and further cell proliferation, creating an environment conducive to tumor growth. UV radiation can weaken the immune system in the skin, making it harder for the body to find and destroy abnormal cells [63]. This immune evasion happens because UV rays can increase the number of regulatory T cells and release substances that suppress the immune response [64] As a result, the immune system might not recognize and get rid of melanoma cells, allowing them to grow uncontrollably. The risk of developing melanoma rises with the total amount of UV exposure a person gets over their lifetime. Short periods of intense sun exposure that result in sunburn

are dangerous [25]. Additionally, some factors can increase the damaging effects of UV radiation and the chances of developing melanoma. These factors include having fair skin, a history of severe sunburns, and certain genetic traits [65].

Detecting melanoma early is essential for better results because treatments work best before cancer has a chance to spread [66]. Melanoma often appears as an unusual mole or skin lesion [67]. Dermatologists use the "ABCDE" rule to identify it: A is for asymmetry, B for irregular borders, C for varied color, D for diameter larger than 6 mm, and E for evolving or changing characteristics [68]. Melanomas often differ from other benign moles due to their rapid changes in appearance [69]. Early detection and surgical removal offer the best chance of a cure, whereas advanced stages require more intensive therapies [66]. A major concern with melanoma is its high potential for metastasis, which involves complex mechanisms that enable cancer cells to spread throughout the body [70]. Recent studies show that melanoma cells can escape detection by the immune system by producing proteins that block immune responses [71]. This helps them avoid being destroyed by the body's defenses. They also create matrix metalloproteinases, which are enzymes that break down the extracellular matrix [72]. The extracellular matrix is the structure that usually helps keep cells in their proper place [73]. This breakdown of the extracellular matrix enables melanoma cells to move into surrounding tissues. Furthermore, through a process called epithelial-to-mesenchymal transition (EMT), melanoma cells change their shape and acquire mobility, allowing them to detach from the primary tumor, invade nearby structures, and enter the bloodstream or lymphatic system [74].

Treating advanced melanoma is challenging, but recent advances have significantly improved outcomes [75]. Targeted therapy uses drugs that are designed to specifically block mutations that drive cancer, like BRAF inhibitors for melanomas with BRAF mutations [76]. These drugs can shrink tumors and help patients live longer, but over time, the cancer may become resistant to the treatment. Immunotherapy has been a significant breakthrough, particularly for advanced melanoma cases [77]. It works by boosting the immune system so that it can recognize and attack melanoma cells [71]. Immune checkpoint inhibitors, such as pembrolizumab and nivolumab, prevent melanoma cells from hiding from immune cells, effectively slowing disease progression and extending survival [78]. Preventing melanoma largely depends on minimizing UV exposure by using sunscreen, wearing protective clothing, and avoiding tanning beds [65]. Genetic predispositions also play a role; individuals with a family history of melanoma or specific inherited mutations are at higher risk [79].

2.3.2. Non-Melanoma

Non-melanoma skin cancers primarily include basal cell carcinoma and squamous cell carcinoma [80]. While these types of skin cancer are less likely to spread to other parts of the body compared to melanoma, they can still cause significant local damage and lead to serious health complications if left

untreated. UV exposure is a significant risk factor for the development of non-melanoma skin cancers, which primarily include basal cell carcinoma and squamous cell carcinoma [81]. Studies have shown that both chronic and intermittent UV exposure can lead to DNA damage in skin cells, triggering mutations that promote tumor formation [82]. Specifically, UV radiation induces the formation of cyclobutane pyrimidine dimers, which disrupt normal cellular processes and can lead to uncontrolled cell growth [83]. The risk of developing these cancers increases with cumulative sun exposure, particularly in individuals with lighter skin types who are more susceptible to the harmful effects of UV rays [84]. Additionally, UV radiation can suppress local immune responses in the skin, reducing the body's ability to detect and eliminate abnormal cells [85]. This combination of direct DNA damage and impaired immune surveillance contributes to the higher incidence of basal cell carcinoma and squamous cell carcinoma in populations with significant sun exposure, highlighting the importance of sun protection measures to reduce these cancers' prevalence.

Basal cell carcinoma is the most common form of skin cancer, accounting for approximately 80% of non-melanoma skin cancer cases [86]. Basal cell carcinoma arises from the basal cells in the lower layer of the epidermis [87]. The primary cause of basal cell carcinoma is prolonged exposure to UV radiation from the sun, although other factors, such as exposure to artificial UV light (like tanning beds) and certain chemicals, can also contribute [88]. Recent research has revealed that basal cell carcinoma tumors exhibit mutations in several key pathways. The most frequently mutated gene in basal cell carcinoma is the PTCH1 gene, which is part of the Hedgehog signaling pathway. This pathway plays a crucial role in cell growth and differentiation. Mutations in PTCH1 disrupt normal signaling, leading to uncontrolled growth of basal cells. Other genetic alterations may involve TP53, SMO, and various oncogenes, which further promote tumorigenesis [89]. Clinically, basal cell carcinoma typically presents as a small, shiny bump or a sore that does not heal, often appearing on sun-exposed areas of the skin, such as the face and neck [90]. While basal cell carcinoma is rarely life-threatening, it can cause significant local damage by invading surrounding tissues, including cartilage and bone [91]. Aggressive forms of basal cell carcinoma, such as infiltrative or morpheaform variants, can be particularly destructive, making early detection and treatment essential [92].

Squamous cell carcinoma is the second most common type of non-melanoma skin cancer, accounting for about 20% of cases [93]. Squamous cell carcinoma originates in the squamous cells, which are flat cells found in the outer layer of the skin [94]. Like basal cell carcinoma, squamous cell carcinoma is primarily caused by UV exposure, but other risk factors include a history of skin burns, chronic skin inflammation, and exposure to certain chemicals, such as arsenic [95]. Squamous cell carcinoma often arises from precancerous lesions known as actinic keratosis, which appear as rough, scaly patches on sun-damaged skin [96]. Genetic studies have shown that mutations in the TP53 gene are also common in squamous cell carcinoma, leading to impaired DNA repair and promoting the survival of abnormal cells [97]. Additionally, alterations in the Ras signaling pathway are frequently observed, which can further contribute to the uncontrolled growth of squamous cells [97]. Squamous cell carcinoma typically presents

as a firm, red nodule or a scaly patch that may bleed or crust over [98]. While squamous cell carcinoma is also less likely to metastasize, it can invade deeper layers of the skin and spread to lymph nodes or other organs if left untreated [99]. The risk of metastasis is higher in high-risk patients, such as those with immunosuppression or significant sun damage.

Treatment for both basal cell carcinoma and squamous cell carcinoma usually involves surgical excision to remove the tumor completely [100]. Other treatment options include cryotherapy, topical chemotherapy, photodynamic therapy, and Mohs micrographic surgery for larger or more aggressive tumors [101]. Preventive measures are crucial in reducing the incidence of non-melanoma skin cancers [81]. Regular use of broad-spectrum sunscreen, wearing protective clothing, and avoiding excessive sun exposure, especially during peak UV hours, are essential steps. Additionally, routine skin examinations by a dermatologist can help detect early changes in the skin that may indicate the development of these cancers.

2.4. Eye Damage

Cataracts are a common eye condition where the normally clear lens becomes cloudy, leading to blurred or impaired vision [102]. One of the primary risk factors for cataract formation is prolonged exposure to UV radiation, particularly UV-B rays [103]. When UV radiation reaches the lens, it directly damages the proteins and fibers within the lens, causing them to clump together and form opaque areas that block or scatter light [104]. This process disrupts the normal pathway of light through the lens, resulting in clouded vision. As UV exposure increases, so does the likelihood of cataract formation, especially among people who spend a significant amount of time outdoors without adequate eye protection [105].

The mechanisms of UV-induced cataract formation are complex, involving several damaging processes. First, UV radiation generates reactive oxygen species which is known as ROS within the lens, which are highly reactive molecules that can harm cellular structures [106]. These reactive oxygen species target the proteins in the lens, leading to oxidative changes that degrade their structure. Over time, this oxidative stress accumulates, causing the lens proteins to lose their transparency and flexibility. Oxidative damage also affects lipid membranes within lens cells, weakening the cell structure and further contributing to the cloudy appearance of cataracts [107]. UV radiation also influences protein aggregation within the lens [108]. The lens contains unique proteins called crystallins, which are essential for maintaining its clarity [109]. In a healthy eye, these proteins are organized to allow light to pass through easily. However, UV exposure causes crystallins to unfold or form aggregates, disrupting this organization [110]. When these proteins clump together, they scatter light, contributing to the cloudiness that characterizes cataracts. This protein aggregation is particularly common in lenses exposed to prolonged UV radiation and contributes significantly to the visual impairment caused by cataracts. Additionally, UV radiation can cause DNA damage in the epithelial cells on the lens's surface [106]. This damage accelerates cellular aging, limiting

the cells' ability to repair and maintain lens clarity. As these cells age and become less functional, damaged or dying cells accumulate, which further promotes cataract formation. This cellular aging process, driven by DNA damage, is a critical factor in the development of cataracts, as the lens's ability to stay clear is gradually compromised over time.

UV exposure contributes to two main types of cataracts [103]. Nuclear cataracts form in the center of the lens and are commonly associated with both aging and UV exposure [111]. These cataracts cause the lens to be yellow or brown, which not only reduces visual clarity but also affects color perception. The second type, cortical cataracts, develop along the edges of the lens, often progressing in a spoke-like pattern [112]. UV radiation plays a significant role in the formation of cortical cataracts by damaging the lens fibers that radiate outward [108]. This type of cataract can lead to increased sensitivity to glare and may affect peripheral vision, further limiting the quality of sight. The impact of UV-induced cataracts on vision and quality of life can be substantial. Early symptoms may include blurry vision, sensitivity to bright light, and difficulty seeing at night [113]. As the cataracts progress, these symptoms can worsen, severely restricting daily activities like reading, driving, and recognizing faces. If left untreated, cataracts can lead to significant visual impairment or even blindness, particularly in older adults or those with high cumulative UV exposure.

Preventing cataracts through UV protection is crucial to preserving eye health. Wearing UV-blocking sunglasses and wide-brimmed hats can help reduce the amount of UV radiation reaching the eyes, significantly lowering the risk of cataract formation. Research suggests that regular use of UV-protective eyewear can delay cataracts' onset, especially for people who spend a lot of time outdoors [114]. Additionally, a diet rich in antioxidants—such as vitamins C and E—may help counteract oxidative stress in the lens, providing further protection from UV-induced damage.

Photokeratitis is a painful eye condition often described as a "sunburn" of the eye, caused by intense exposure to UV-B radiation [115]. This condition results from high levels of UV-B rays directly damaging the surface of the eye, particularly affecting the cornea and the conjunctiva, which is the thin membrane covering the eye [116]. Although photokeratitis is usually temporary, the symptoms can be very uncomfortable and disruptive, underscoring the importance of awareness and prevention to protect eye health [117]. The cornea, the transparent front surface of the eye, is especially sensitive to UV radiation. When exposed to excessive UV-B rays, the epithelial cells on the corneal surface absorb the radiation, leading to cellular damage and inflammation [118]. This process is similar to sunburn in the skin, which is why photokeratitis is often called "eye sunburn." UV radiation exposure triggers inflammation in the cornea and conjunctiva, leading to symptoms such as redness, swelling, tearing, a gritty sensation, and intense pain. In severe cases, photokeratitis can cause temporary vision loss, extreme light sensitivity, and involuntary eyelid spasms known as blepharospasm [119]. These symptoms can be particularly distressing and may make daily activities challenging.

Photokeratitis is commonly associated with high-altitude environments, snow-covered areas, and water surfaces, where UV-B exposure is intensified due to reflection [120]. For instance, “snow blindness” is a specific type of photokeratitis that occurs when UV rays are reflected off snow, effectively doubling the intensity of radiation that reaches the eyes. Similarly, environments like beaches or coastal areas pose a risk because water and sand also reflect UV rays, increasing the amount of radiation exposure to the eyes. Artificial sources of UV light, such as welding torches, tanning beds, and certain types of lamps, can also cause photokeratitis [121]. In the context of welding, photokeratitis is often referred to as “arc eye” due to the intense UV light produced during the welding process.

At the cellular level, photokeratitis is driven by the formation of reactive oxygen species when UV radiation interacts with eye cells [106]. These reactive oxygen species molecules lead to oxidative stress, which can damage cell membranes, proteins, and DNA within the corneal cells. The body responds to this cellular injury with inflammation, causing blood vessels in the eye to dilate, which results in redness and swelling. This inflammatory response is responsible for much of the pain and discomfort associated with photokeratitis. Fortunately, the corneal epithelium, which serves as the eye’s first line of defense, has a remarkable ability to regenerate [122]. As a result, photokeratitis typically resolves on its own within 24-48 hours if further UV exposure is avoided, although the symptoms can be intense during this recovery period.

While photokeratitis generally resolves without long-term damage, repeated or severe episodes of UV-induced eye damage can lead to lasting consequences [123]. For instance, cumulative UV damage is associated with a higher risk of developing cataracts and pterygium, a growth on the conjunctiva that can potentially encroach upon the cornea and impair vision. Taking preventative measures is crucial to avoid these risks. Wearing UV-protective eyewear, especially in high-risk settings like snowfields, beaches, or during activities like skiing and welding, is essential for shielding the eyes. Sunglasses with 100% UV-blocking lenses or wraparound goggles are effective in protecting the eyes from both direct and reflected UV rays. In addition, wide-brimmed hats can further reduce exposure by blocking sunlight from directly reaching the eyes.

2.5. Immune System Suppression

Excessive exposure to UV radiation can significantly suppress the immune system, compromising the body’s ability to detect and fight off infections, diseases, and even cancerous cells [85]. This immunosuppressive effect of UV radiation occurs primarily through its impact on skin cells and immune signaling pathways [124]. While UV radiation is necessary in small amounts for vitamin D production, excessive exposure triggers complex processes that reduce immune effectiveness, particularly in skin-exposed areas. The immune-suppressing effects of UV radiation begin at the cellular level [125]. When skin cells absorb UV radiation, they release a variety of signaling molecules, including cytokines and

immunosuppressive factors like prostaglandin E2 [45]. These molecules can interfere with the normal immune response by sending signals that dampen inflammation and reduce the activity of immune cells. For example, UV radiation can stimulate the production of regulatory T cells (Tregs), a type of immune cell that suppresses other immune responses [126]. While Tregs play a role in preventing autoimmune diseases by moderating the immune system, an increase in their activity due to UV exposure can weaken the body's ability to detect and attack abnormal cells, including pathogens and precancerous cells [127].

One key pathway affected by UV exposure involves the production of reactive oxygen species in skin cells. These reactive oxygen species cause oxidative stress and damage DNA, which can lead to mutations if left unrepaired. The immune system usually identifies and destroys damaged or mutated cells; however, under conditions of excessive UV exposure, immune suppression allows these damaged cells to evade detection. This process is one of the ways UV exposure is linked to an increased risk of skin cancers like squamous cell carcinoma and basal cell carcinoma, as immune surveillance is compromised.

UV-induced immunosuppression primarily affects the skin, where it can lead to localized immune tolerance. This means that skin exposed to high levels of UV radiation is less able to mount an effective immune response to infections or abnormal cells. For instance, the skin's response to viral infections like herpes simplex can be weakened by excessive UV exposure, leading to more frequent and severe outbreaks in some individuals. Over time, this localized immune suppression may contribute to an increased risk of skin cancers, as the body's ability to identify and destroy cancerous cells is impaired. Interestingly, the effects of UV exposure on immunity are not limited to the skin. Prolonged or high-intensity UV exposure can also have systemic effects, reducing immune function throughout the body. Studies have shown that excessive UV exposure can decrease the effectiveness of vaccines by altering how immune cells recognize and respond to antigens, the substances that vaccines use to provoke an immune response. These systemic effects underscore the importance of understanding UV exposure's full impact on immune health.

Reduced immunity due to excessive UV exposure has wide-ranging health implications. The increased susceptibility to infections, especially those affecting the skin, can lead to more frequent and severe episodes of conditions like cold sores or bacterial skin infections. Additionally, the immune system's compromised ability to recognize and eliminate abnormal cells can increase the risk of skin cancers. Cancers such as basal cell carcinoma and squamous cell carcinoma are more likely to develop in areas frequently exposed to the sun, where immune surveillance has been reduced over time. Preventing immune suppression from UV exposure involves limiting UV exposure, especially during peak sunlight hours. Protective measures like wearing sunscreen, UV-blocking clothing, and hats are essential to minimize the harmful effects of UV on both local and systemic immunity. By taking these precautions, individuals can reduce the risk of immune-related health issues and help maintain the immune system's ability to respond to infections and prevent the growth of abnormal cells.

2.6. Reduction of Vitamin B9 (Folate)

Excessive exposure to UV radiation can lead to a reduction in vitamin B9, or folate, which is an essential nutrient involved in DNA synthesis, repair, and cell division [128]. Folate plays a crucial role in many bodily functions, especially in rapidly dividing cells, making it particularly important during pregnancy for fetal development [129]. However, high levels of UV radiation can degrade folate, resulting in decreased levels in the bloodstream [130]. This depletion has serious health implications, as folate deficiency is associated with an increased risk of birth defects, certain cancers, and other health conditions [131]. UV radiation, particularly UV-A and UV-B rays, can penetrate the skin and lead to biochemical reactions that break down folate in the blood. Folate is photosensitive, meaning it can be degraded by exposure to light, especially the wavelengths present in UV radiation. When skin is exposed to high amounts of sunlight, UV radiation induces the formation of reactive oxygen species, which are unstable molecules that can cause oxidative damage [63]. These reactive oxygen species interact with folate molecules in the bloodstream, leading to chemical changes that break down the vitamin and reduce its bioavailability in the body.

Folate degradation primarily affects individuals who are exposed to high levels of UV radiation without adequate protection. For instance, people living in regions with strong sunlight year-round or those who spend extended periods outdoors without sun protection are at a higher risk of folate depletion. The protective measures for preserving folate levels include wearing sunscreen, clothing that shields the skin, and seeking shade during peak sunlight hours. The reduction in folate levels due to UV exposure can have significant health consequences, particularly for pregnant women and individuals with higher folate requirements. Folate is crucial for DNA synthesis and repair, which are vital processes during fetal development. Low folate levels in pregnant women increase the risk of neural tube defects in the fetus, such as spina bifida and anencephaly. These birth defects can lead to severe developmental issues, lifelong disability, or even infant mortality. This is why maintaining adequate folate levels is a priority during pregnancy, and why prenatal supplements containing folic acid (a synthetic form of folate) are often recommended.

Beyond pregnancy, folate is also essential for general cellular health. Cells with rapid turnover, such as those in the bone marrow, skin, and digestive tract, rely heavily on folate for replication and repair. Low folate levels can impair the function of these cells, leading to issues like megaloblastic anemia—a condition in which red blood cells are abnormally large and unable to function effectively. This form of anemia can cause fatigue, weakness, and other symptoms related to poor oxygen transport throughout the body. Reduced folate levels due to UV exposure may also play a role in cancer risk. Since folate is involved in DNA synthesis and repair, a deficiency in this nutrient can lead to DNA damage and genomic instability, which are risk factors for cancer development. Studies have shown that low folate levels are associated with an increased risk of certain cancers, including skin cancers, due to the accumulation of DNA damage in cells. As folate is essential for maintaining DNA integrity, a deficiency can make cells more susceptible to mutations and potentially cancerous changes.

To prevent folate depletion caused by UV radiation, protective measures against UV exposure are crucial, especially for those at higher risk of folate deficiency. Using broad-spectrum sunscreen, wearing protective clothing, and limiting time in direct sunlight can all help reduce the risk of folate degradation. In addition to sun protection, a diet rich in folate can support adequate levels of this essential nutrient. Foods such as leafy greens, citrus fruits, beans, and fortified cereals are excellent sources of folate and can help offset any UV-related folate loss. For those with increased folate needs, like pregnant women or individuals with certain health conditions, folic acid supplements may be beneficial. These supplements can help maintain folate levels even with some degree of UV exposure, ensuring the body has enough of the nutrients for vital functions.

2.7. Reproductive Health

Environmental and external factors significantly influence fertility rates, impacting both individuals and populations [132, 133]. Excessive UV radiation exposure has been shown to impact fertility in both males and females, with recent studies highlighting its effects on male sperm quality, as well as potential effects on female reproductive health [134]. UV radiation can induce oxidative stress and DNA damage, mechanisms that play a significant role in altering reproductive functions and health. While the effects are often more pronounced in males due to the sensitivity of sperm to oxidative damage, there is also evidence that UV-related stress can impact ovarian health in females, potentially affecting overall fertility [135, 136].

UV radiation primarily impacts male fertility through oxidative stress, which can alter sperm motility and morphology [137]. When the skin absorbs high levels of UV rays, it triggers the production of reactive oxygen species, which are unstable molecules that can damage cell membranes, proteins, and DNA [137]. Sperm cells are particularly vulnerable to oxidative stress because they have limited antioxidant defenses to counteract the effects of reactive oxygen species [138]. This oxidative damage can lead to altered sperm motility, impairing their ability to reach and fertilize an egg. Furthermore, abnormal morphology can result in sperm that are misshapen or have structural defects, further reducing fertility potential [139]. Research has shown that men with high exposure to UV radiation often have decreased sperm quality compared to those with lower UV exposure levels [140]. In particular, studies have found that excessive sun exposure or exposure to artificial sources of UV light can decrease sperm count and result in an increased proportion of sperm with damaged DNA [141]. Sperm DNA fragmentation-induced oxidative stress can impair the genetic integrity of the sperm, which may lead to complications in conception or result in an increased risk of miscarriage if fertilization does occur[142] .

Although research on UV radiation's effects on female fertility is less extensive, there is emerging evidence that UV-induced oxidative stress may also impact ovarian health [143]. The ovaries, which are responsible for producing and releasing eggs, are sensitive to changes in the oxidative environment

within the body [144]. High levels of reactive oxygen species can disrupt the ovarian follicles where eggs mature, leading to poorer egg quality and potentially impacting ovulation [145]. Animal studies suggest that prolonged exposure to UV radiation may reduce the ovarian reserve (the number of viable eggs in the ovaries), though further research is needed to confirm this effect in humans [146, 147]. In addition, UV-induced oxidative stress may affect hormonal balance, which is essential for normal ovulation and menstrual cycles [148]. Disruptions in hormone levels due to oxidative stress could interfere with the menstrual cycle's regularity, impacting a woman's fertility over time [149].

Additionally, since UV radiation can cause DNA damage, there is a concern that prolonged UV exposure could contribute to genetic instability in germ cells, which might have implications for offspring health [150, 151].

To mitigate the effects of UV radiation on fertility, adopting protective measures is essential. For men, reducing direct sun exposure, especially during peak hours, and wearing protective clothing can help minimize UV-induced damage to sperm [136]. Consuming a diet rich in antioxidants, such as vitamins C and E, may also help counteract some of the oxidative stress associated with UV exposure [152]. Antioxidants play a crucial role in protecting male reproductive health by countering the damaging effects of oxidative stress on sperm [153, 154]. For women, wearing protective sunscreen and clothing, and maintaining a healthy antioxidant-rich diet, can support overall reproductive health.

Recent studies on the interactions between ozone (O_3) and UV radiation have underscored their potential combined effects on reproductive health, implicating mechanisms at the cellular, molecular, and physiological levels [155]. Ozone itself is a potent oxidant, and UV exposure induces a range of photobiological effects; together, they can amplify oxidative stress, disrupt endocrine functions, and influence signaling pathways critical for reproductive health. Here's a detailed exploration of how these interactions occur and the mechanisms involved. Ozone and UV radiation each cause oxidative stress by creating reactive oxygen species [156]. When combined, they increase reactive oxygen species levels even more, leading to greater oxidative damage in reproductive tissues, like the testes, ovaries, and uterine lining. High reactive oxygen species levels can harm cells by damaging lipids, DNA, and proteins, which impacts their function and survival [157]. For example, in men, high reactive oxygen species levels can interfere with sperm production by damaging spermatogonial stem cells, which are especially sensitive to oxidative stress [158]. When reactive oxygen species levels rise, cells usually respond by activating the Nrf2 pathway, which controls antioxidant defenses [159].

The endocrine system is especially sensetive to environmental pollutants like ozone, which, when combined with UV radiation, can disrupt hormone balance even more. The endocrine system is especially sensetive to environmental pollutants like ozone, which, when combined with UV radiation, can disrupt hormone balance even more [123]. UV exposure increases the production of signaling molecules and cytokines, which can boost ozone's effects on hormone regulation [160]. Recent studies suggest that

UV-related damage to the adrenergic system in reproductive tissues affects the release of gonadotropin-releasing hormone (GnRH). This change, in turn, disrupts the secretion of FSH and LH, leading to hormonal imbalances that can interfere with reproductive cycles [161]. This disruption is especially significant for women, as it may impact ovulation, implantation, and fetal development when there is prenatal exposure [162]. Both ozone and UV radiation can induce direct and indirect DNA damage, but when combined, their effects may lead to more complex genetic and epigenetic alterations. UV radiation primarily causes DNA strand breaks and thymine dimers, which, if left unrepaired, can trigger mutations and contribute to carcinogenesis [52]. Ozone, by increasing ROS levels, exacerbates DNA damage by hindering DNA repair processes.

Exposure to both ozone and UV has been shown to activate the NF-κB pathway, which is crucial for regulating inflammation [163]. When NF-κB is activated, it increases the production of inflammatory cytokines like IL-6 and TNF-α [164]. These inflammatory molecules can disrupt key reproductive processes, such as implantation and placental development in females and sperm production in males [165]. Ongoing inflammation is associated with reproductive health issues, including endometriosis and decreased sperm quality, both of which can significantly impact fertility [166]. Mitochondria are essential for energy production in cells and play a crucial role in reproductive health, particularly in spermatogenesis and oogenesis [167]. Both ozone and UV exposure can lead to mitochondrial dysfunction through direct damage to mitochondrial DNA and the disruption of energy-producing pathways, which are highly dependent on redox balance [168].

Research indicates that exposure to both UV and ozone can cause epigenetic changes, such as DNA methylation and histone modification, which alter gene expression in germ cells (sperm and eggs) [169]. These changes can influence fertility, embryo quality, and possibly lead to reproductive health effects in future generations [170]. For example, recent animal studies show that offspring of parents exposed to UV and ozone display altered DNA methylation patterns in genes tied to reproductive function and stress response, suggesting that these environmental exposures may impact the health of subsequent generations [171]. The complex interactions between ozone and UV exposure highlight a relationship that scientists are still working to understand. Recent studies stress the importance of exploring how these combined environmental factors impact reproductive health, as effects may differ based on species, exposure levels, and individual sensitivity.

Table **1** provides a comprehensive overview of recent studies investigating the health effects of UV exposure across a range of biological models, environmental contexts, and health outcomes. The studies span various UV wavelength ranges (UV-A, UV-B, and in some cases UV-C) and employ methodologies including in vivo animal models, in vitro cellular assays, epidemiological analyses, and literature reviews. The findings highlight diverse health implications, from reproductive and respiratory health impacts to skin cancer, vitamin D synthesis, photoaging, and ocular damage. Key insights include the importance of proper UV protection strategies, especially for outdoor workers and vulnerable populations, to minimize cancer

risk and prevent other UV-related health issues. The studies underscore the protective role of antioxidants and the Nrf2 pathway in mitigating oxidative damage induced by UV, though prolonged activation of these defenses may carry its risks. Additionally, findings stress the value of targeted policies—like the Montreal Protocol—which have had a global impact in reducing harmful UV exposure. The table concludes with evidence that UV exposure affects not only humans but also animals, influencing their welfare and productivity. The collected information from recently published papers emphasizes the complex interplay between UV exposure and health, pointing to the need for balanced sun exposure, effective protective measures, and continued research into UV's role in human and animal health.

Table 1: A summary of recent investigations on health effects of UV exposure.

Primary Focus	Key Findings	UV Wavelength Range	Methodology	Health Implications	Sample or Model Used	UV Exposure Duration/Intensity	Impact/Insight	Ref.
UV and pollution impacts on infant health	UV and pollutants correlated with lower birth size and respiratory issues	Not specified	Epidemiological study	Infant growth, respiratory health	Human infants in Jakarta, Indonesia	Chronic exposure during prenatal and postnatal periods	Highlights need for targeted policies in low-income settings to reduce UV-related health risks for infants	[172]
Phthalate exposure and health risk from UV-reactive chemicals	Minimal health risk from phthalate exposure via UV-related products like sunscreen	UV-A and UV-B	Urine sample analysis and exposure modeling	Phthalate exposure from personal care products	Human urine samples	Estimated low-level daily exposure	Confirms low systemic risk for UV-induced phthalate exposure in daily use scenarios	[173]
Efficacy of sunscreen in UV protection	Effective in blocking UVB rays, with recommendations for proper reapplication	UV-A and UV-B	Literature review	Skin protection and cancer prevention	Sunscreen products and UV exposure	Daily exposure scenarios under varying SPF conditions	Reinforces need for correct application to minimize skin cancer risk from UV exposure	[174]
UV's role in tanning and skin cancer risk	Genetic predisposition influences susceptibility; UV linked to various skin cancers	UV-A and UV-B	Molecular and epidemiological analysis	Skin cancer risk and pigmentation changes	Human skin cells and epidemiological data	Chronic or high-level exposure	Provides insight into genetic factors and UV exposure balance for skin health management	[175]
UV-driven vitamin D synthesis and health	Balances UV exposure benefits for vitamin D with cancer risk	UV-B	Literature review	Vitamin D synthesis vs. skin cancer risk	Human health perspective	Routine sun exposure	Advocates for balanced sun exposure to achieve vitamin D levels without excessive cancer risk	[176]
Global health impacts of UV exposure	Montreal Protocol significantly reduced UV-related skin cancer cases	UV-B and UV-C	Global environmental impact assessment	Skin cancer prevention and ocular health	Population-level data, U.S. estimates	Decreased exposure due to Montreal Protocol	Demonstrates global policy impact on UV exposure reduction and associated health benefits	[20]

Table 1: Continued.

Primary Focus	Key Findings	UV Wavelength Range	Methodology	Health Implications	Sample or Model Used	UV Exposure Duration/Intensity	Impact/Insight	Ref.
Evolutionary impact of UV exposure	Examines adaptation of skin pigmentation and vitamin D synthesis across regions	UV-B	Evolutionary biology and anthropology analysis	Vitamin D synthesis, skin adaptation	Human populations across regions	Chronic exposure over generations	Explores UV's historical significance in human physiological adaptations	[177]
UVA1's specific health risks	UVA1 penetrates deeper, causing oxidative stress and skin aging	UVA1 (340-400 nm)	Review of molecular and cellular studies	Photoaging, skin carcinogenesis	Human skin cells and theoretical modeling	Chronic UVA1 exposure	Highlights the need for UVA1-specific photoprotection strategies	[34]
UV exposure risk among outdoor workers	Direct correlation between UV intensity, skin cancer risk, and protection practices	UV-A and UV-B	Field study with environmental data	Skin cancer risk for outdoor workers	Outdoor workers in Baghdad	Varying exposure based on job conditions	Reinforces need for sun protection strategies for high-UV occupations	[178]
Oxidative stress from UV-exposed cosmetics	UV exposure of cosmetics can increase skin phototoxicity; antioxidants may help mitigate risks	UV-A and UV-B	Cellular assays on cosmetic compounds	Phototoxicity and oxidative damage	Cosmetics applied to skin cells	Daily exposure with potential high-intensity sunlight	Emphasizes safe cosmetic use in UV-rich environments to minimize skin damage	[179]
UV-induced eye damage	Highlights UV's potential to damage the cornea, retina, and lens	UV-B and UV-A	Histopathological animal model study	Cataract, photokeratitis, and retinal health	Corneal, lens, and retinal tissue	Prolonged exposure in high UV index areas	Underscores UV protection for ocular health due to increased cataract and eye disease risk	[118]
Nrf2 pathway's protective role in UV exposure	UV activates Nrf2-driven antioxidant defenses, though prolonged activation can affect skin health	UV-B and UV-A	Cellular study with skin cells	Skin protection and cellular stress response	Human skin fibroblasts and keratinocytes	Repeated exposure to UV-A and UV-B	Demonstrates Nrf2's dual protective and potentially disruptive roles under chronic UV exposure	[48]
Occupational UV exposure and cancer risk	Inadequate sun protection observed, with increased skin cancer risk	UV-A and UV-B	Epidemiological study on occupational exposure	Skin cancer risk for outdoor workers	Outdoor workers in Italy	Chronic occupational exposure	Calls for improved UV prevention measures for outdoor workers in high-exposure areas	[180]
UV-induced health issues in animals	UV exposure increases oxidative stress, skin lesions, and optical tumors in animals	UV-B and UV-A	Review of veterinary studies and field observations	Animal welfare and productivity	Livestock and domestic animals	Prolonged outdoor UV exposure	Highlights UV exposure's impact on animal welfare and productivity, with economic implications	[181]

Table 1: Continued.

Primary Focus	Key Findings	UV Wavelength Range	Methodology	Health Implications	Sample or Model Used	UV Exposure Duration/Intensity	Impact/Insight	Ref.
Cellular antioxidant response to UV	UV triggers Nrf2 pathway activation in human skin cells, with protective antioxidant roles	UV-B and UV-A	In vitro cellular study	Oxidative stress and cellular protection	Human skin fibroblasts and keratinocytes	Moderate to chronic exposure	Reaffirms Nrf2's role in defending skin cells from oxidative damage due to UV exposure	[48]

3. Conclusion

In conclusion, the effects of ultraviolet (UV) radiation on health are profound and multifaceted, impacting various aspects of well-being. Prolonged exposure to UV rays has been linked to serious conditions such as skin cancers, including melanoma and non-melanoma types, as well as cataracts and photokeratitis. The mechanisms behind these effects often involve DNA damage, immune suppression, and oxidative stress, which can lead to significant health risks if protective measures are not taken. The UV Index serves as a critical tool for raising awareness about UV radiation intensity and guiding individuals in taking appropriate precautions. By understanding the levels of UV risk and implementing protective strategies—such as wearing sunscreen, protective clothing, and seeking shade during peak hours—individuals can significantly reduce their risk of adverse health effects. Additionally, it is important to recognize the broader implications of UV exposure, including its potential impact on reproductive health and overall immune function. As our understanding of UV radiation continues to evolve through ongoing research, public education and awareness remain key in promoting skin and eye health. By staying informed and vigilant about UV exposure, individuals can take proactive steps to protect themselves and maintain their overall health in the face of increasing UV radiation levels due to environmental changes.

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