Investigating the Impact of UV-B Radiation on Corneal Epithelial Cells

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Abstract

Ultraviolet (UV) radiation is an environmental exposure that has significant effects on human health, including damage to the eyes [17]. The eyes have several layers of protection. This paper focuses on the outermost surface of the eye: the cornea. Due to its absorption properties, UV-B radiation is transmitted to the eyes and therefore increases the risk rate [17]. UV-B exposure systematically inflicts harm to the corneal cells through several mechanisms: DNA damage, an increase in oxidative stress, disruption of normal cell function, and triggering programmed cell death in corneal epithelial cells [1,11,12,16]. This review synthesizes findings from previously published studies to provide a detailed but accessible explanation of how UV-B radiation affects corneal epithelial cells. It focuses on several key processes: direct DNA damage [1], activation of DNA damage receptors such as γH2AX [19], cell regulation pathways like p53 [13,14], and the role of reactive oxygen species (ROS) in amplifying injury [12,15]. The review also discusses protective strategies.

Protective strategies include antioxidant eye drops [16], dietary measures [2,3,4], circadian regulation of DNA repair [20], and retinoids [9]. Finally, UV-related eye diseases such as photokeratitis [5,6] and pterygium [7] are discussed within a public health framework, emphasizing that the majority of UV-related vision loss is oftentimes preventable suffering [21,22]. Response mechanisms, if implemented, can mitigate the risk of UV vision loss [17,21,22]. By connecting cellular mechanisms to real-world outcomes, this review highlights how understanding biological mechanisms can guide simple, affordable strategies to protect long-term vision and prevent optic diseases [17,21,22].

1. Introduction

Sunlight plays a major role in biological functions and maintaining human health. For example, moderate exposure to UV radiation helps the body generate vitamin D. However, exposure to excessive UV radiation can cause greater implications such as ocular melanoma, tissue aging, and ocular disease [17]. There are many public health campaigns that focus on protecting the skin from UV radiation through applications like sunscreen. Despite this, exposure to the eyes persists as they are continuously exposed to sunlight [17]. The outer layer of the eye, the cornea, plays a role in maintaining vision. The cornea also serves as the eye's first layer of protection from any environmental hazards. The cornea absorbs UV-B radiation; its epithelial cells experience increased exposure compared to other regions like the retina [17]. To protect the eyes, all types of sunglasses reflect sunlight. Many believe that UV-blocking lenses are the most essential feature of sunglasses. The corneal epithelial cells are exposed to oxidative stress, which can lead to DNA damage or even apoptosis. [12,13,14]. Then, due to the accumulation of this damage, this would in turn lead to disorders such as photokeratitis. Repeated UV injuries can lead to long-term issues for vision. [5,6,7] The pathways associated with UV-B injury are important to take into consideration when it comes to treatments and

medication. It is important to identify protective therapies for DNA damage and oxidative stress damage to the cells in the cornea [21,22]. This paper aims to provide a comprehensive yet clear explanation of the cellular pathways involved in UV-B–induced corneal damage.

1.1 Preventing UV-B Damage

The eye is continuously exposed to UV radiation, which makes it prone to environmental damage. Radiation is absorbed through the cornea, but this imposes high levels of oxidative stress onto the corneal cells [17]. The amount of radiation that has the ability to reach the eye is dependent on specific environmental factors. Spending prolonged periods of time outside or surrounded by surfaces like snow and ice can significantly increase exposure to UV radiation [23]. Without the correct eye protection, these surfaces create a high risk for ocular vulnerability [17].

Despite these widespread challenges, eye protection is consistently overlooked. Many people recognize the importance of protecting their skin but do not actually go to the lengths to address ocular concerns [24]. Limited awareness and inconsistent use of eye protection like UV-blocking sunglasses can increase the risk of preventable UV-related damage [17,24]. UV-B radiation damages the corneal cells through DNA damage and oxidative stress [1,14]. As discussed before, the damage leads to long-term cellular injury and ocular diseases [8].

1.2 Reactive Oxygen Species

UV-B radiation disrupts the function of corneal epithelial cells by inflicting both direct and indirect damage to the DNA. Direct exposure to UV-B can produce DNA lesions, while indirect damage takes place through reactive oxygen species (ROS). Both of which place stress on the cell [1,14]. The cornea does absorb the majority of the UV radiation. One of the primary ways that UV-B radiation can damage the corneal epithelial cells is through producing reactive oxygen species (ROS) that enter the eye, but the epithelial cells are the ones that succumb to the harmful effects. ROS are unstable molecules generated during normal cellular processes. However, excessive UV exposure can cause these levels to increase rapidly. When the amount of ROS surpasses the cell's natural defenses, oxidative stress and cellular dysfunction occur [1,14]. Elevated ROS levels can damage parts of cells including lipids, proteins, and DNA [15].

To limit the amount of genetic damage, the cells activate a DNA damage response which detects the injured DNA and coordinates repairs. These pathways help decide whether a cell which is damaged can recover or needs to undergo apoptosis [8]. To achieve this balance between recovery and removal is important to maintain the health of the corneal cells. Protection of the corneal epithelial tissue is extremely crucial for maintaining clear vision. When the cell has quick cellular responses and defense mechanisms to help reduce the effects of UV radiation, it can mitigate any long-term development of ocular diseases [12,14].

2.1 γH2AX Biomarker

Maintaining stability is essential for the function of the corneal epithelial cells. One of the earliest responses to DNA damage is the formation of γH2AX. γH2AX is a phosphorylated form of the histone protein H2AX. This protein serves as one of the earliest indicators of DNA damage. Preceding the DNA injury, H2AX is phosphorylated near the site of the damaged region. The protein γH2AX forms visible foci that act as a signal for the recruitment of DNA repair proteins and responding to DNA damage [1].

γH2AX has become one of the most widespread biomarkers for combating DNA damage. γH2AX does not repair DNA directly; however, it alerts the cell to DNA injury. γH2AX marks this injured region and summons the proteins that repair the cells. The necessity of γH2AX becomes more pressing after extreme UV-B radiation. The accumulation of γH2AX following UV irradiation is associated with stress and DNA breakage [19]. This means that with this protein, the injury can be determined even before they have fully developed.

Because of this role, γH2AX is not only a biomarker. γH2AX helps organize cellular responses to DNA damage by directing the repair proteins [1]. If this process works as intended, the DNA is repaired to prevent any future complications and mutations. In the corneal epithelial cells, which are exposed to UV-B radiation, this mechanism helps reduce any long-term cellular damage.

2.2 p53 Cell Cycle Protein

The damage to DNA does not only determine what can happen to a cell. After UV-B radiation damages the corneal epithelial cells, the cell must decide what part of the damage to repair or whether to let it die. This decision is determined by the tumor suppressor protein p53, which ensures genetic stability [1]. Under regular conditions, p53 remains inactive. When UV-B radiation disrupts DNA, a series of pathways activates p53 to coordinate a response to the damage [13]. p53 acts as a control center since it receives information from other sites regarding damage and directs the cell to the next steps. p53 does not act by itself; rather, it connects different parts of the cell [13].

One of the protective actions of p53 is to pause the cell cycle. This stops cell division during this time period and gives time for the cell to repair the damaged DNA. This is to avoid the replication of the damaged DNA during cell division, which would prolong its harmful effects. This is essential because if damaged DNA gets copied, then this could introduce mutations into the cell. If the p53 facilitates a safe repair process, this would allow the cell to return to its normal functions.

Not all damage is reparable. When UV-B exposure causes extensive DNA damage, the p53 is not only a survival factor but also a protective factor. This can be expected through cell death, otherwise known as apoptosis [13]. Although deliberately killing these cells may seem harmful, this process actually provides long-term benefits. The cornea remains protected by preventing the damaged cells from proliferating in the tissue.

The ability of p53 to control multiple different outcomes is one of the most important defense mechanisms against UV exposure. It has the ability to decide whether the cell should repair,

stop cell division, or undergo cell death. p53 maintains the integrity of the corneal epithelium tissue and helps reduce UV exposure.

2.3 Caspase-Mediated Apoptosis

Apoptosis is a regulated process of cell death that helps remove cells that contain severe DNA damage. After cells experience substantial UV radiation, corneal epithelial cells activate the apoptotic pathways, which prevent the damaged cells from surviving and limit the passage of mutation [10,13]. An important part of this process includes the activation of caspases. Caspases are a family of enzymes that carry out the breakdown of damaged cells. Research shows that UV-induced apoptosis involves the activation of caspases. This is a crucial defence mechanism against DNA-damaging cells [10,11]. Although apoptosis results in cell loss, it helps with the maintenance of the cornea. The elimination of irreparable cells facilitates stability and reduces the risk of future and long-term cellular dysfunction [11,13]

3.1 Antioxidant Ocular Treatments

UV radiation increases the production of reactive oxygen species. This contributes to cell damage and oxidative stress. Antioxidants can help mitigate the effects of the stress by neutralizing ROS [1]. The antioxidant treatments can be shown to be effective as they act on the surface of the corneal cells. A study found that a formulation containing riboflavin and vitamin E TPGS improved the density of corneal cells and tissues after UV exposure [16]. These results showed that the antioxidant therapy helps reduce the injury following ultraviolet radiation.

Another study investigated the effects of hyaluronic acid and found that it had the potential to reduce the oxidative damage from stress and inflammation in the corneal epithelial cells. Not only did it reduce the stress, but the hyaluronic acid supprepairs of the damaged tissue. However, greater research is still needed to measure the efficiency and effectiveness of the antioxidants. The current evidence on these topics shows an improvement in cell survival and helps protect the cornea from the UV-B oxidative damage [1,16,18].

3.2 Dietary Protection Mechanisms

Outdoor activities often exacerbate the risk of sun damage for individuals, with a common implication of skin cancer. Both squamous cell carcinoma and basal cell cancers impact millions of individuals. Still, recent research studies have shown that changes in dietary habits have a strong tendency to mitigate cellular damage from the sun. Recommended treatment strategies surround eating foods rich in certain vitamins, including Vitamin E, C, and A. However, what is not fully grasped about the consumption of these diverse foods is that they cannot be taken individually to be effective. Supplements are often taken in various quantities and types. Similarly, foods need to be consumed meticulously, noting nutritional value as well as the proportions of vitamins found in each food source. Although eating vitamin-rich foods is generally beneficial for the body, certain foods contain more nutrients stopping skin cancer than others. Beta-carotene is a primary nutrient that helps prevent skin cancer and protects the immune system from being compromised. Orange colored fruits and vegetables are excellent

sources of the nutrient, including oranges, carrots, sweet potatoes, and mangoes. Another beneficial nutrient is Omega-3 Fatty Acids. They inhibit COX-2, a chemical that promotes the rise of various types of skin cancer. But foods are not the only moderators for protecting against the sun and the potential rise of cancers. Polyphenols in tea are a great example. Studies exhibit a correlation between drinking green and black tea and tumor-inhibiting characteristics. In addition, the drink has components that prevent DNA damage, repair DNA, and reduce cellular damage [4]. Aside from vitamins, acids, and tea polyphenols, dietary compounds have also shown significant ability to protect the skin from sun damage. A clinical study explored the impact of a collagen-containing yogurt on 70 Japanese women, tracking UV-induced skin erythema and minimal erythema dose (MED) levels. MED indicates the minimum UV radiation to produce skin irritation/redness, with higher MED levels indicating greater resistance to UV radiation and more sun exposure necessary for redness expression.

For the study, active and placebo groups were established, with baseline MED quantifications of 26.0 ± 5.6 mJ/cm² for the active group and 26.1 ± 5.3 mJ/cm² for the placebo group. Results after 35 days indicated that participants with skin phototype II (MED 14.5–25.2 mJ/cm²) showed a significantly greater increase of approximately 6.5 mJ/cm² versus 2.8 mJ/cm² in the placebo group ($P = 0.028$). The study's findings indicated that the dietary intervention increased resistance to UV-based sun damage [3]. A separate study explored the chemoprotective impact of dietary grape powder on UVB radiation in female SKH-1 mice (a hairless mouse strain used in cancer research). Results showed grape consumption in the UVB-exposed mice reduced tumor growth. Tumor volume decreased substantially from 104.52 mm³ in control mice to 14.26 mm³ and 45.51 mm³ in mice fed diets containing 3% and 5% grape powder, respectively. Another indicator of protection was the percentage of mice with malignant lesions. While 73% of control mice developed malignant lesions, only 50% of grape-fed mice had lesions [2].

3.3 Retinoid Protection

Retinol is crucial for protecting the ocular surface and preventing various ocular surface diseases. Vitamin A (retinol) promotes greater mucin expression, augmenting epithelial health and healing different injuries in the conjunctiva and cornea. In addition, it protects from corneal keratalization, a condition in which the corneal membrane turns from a smooth surface to a rough, opaque layer. Transcribing Vitamin A into a usable form for the ocular surface is a complex pathway that requires several steps. First, Retinol-binding protein 4 (RBP4) transports the retinol, circulating it through the bloodstream to peripheral tissues. Peripheral tissues receiving the retinol receive the neural impulse, and once secreted via tears, it gets activated by retinoic acid 6 (STRA6). After being turned into different forms, including retinoic acid (RA) via retinal dehydrogenase (RALDH) oxidation, the body's homeostasis is maintained. The cornea and conjunctiva are the two major structures that detect irritation or sensitivity of the eyes from environmental stimuli. When sensory signals are activated, a reflex pathway is triggered, stimulating the lacrimal gland via the sympathetic and parasympathetic nerves. The lacrimal gland releases tears and Vitamin A, helping maintain healthy epithelial cells. However, when tear production or secretion is impaired, conditions such as dry eye disease and ocular inflammation can persist [26].

3.4 Eyewear Protection

Sunglasses employ mechanisms to respond to the three main ways light interacts with different objects: transmission, absorption, and reflection. The primary objective of sunglasses is to absorb incoming light before it reaches the cornea, preventing sunlight from reaching the cornea and causing ocular damage on the cellular level. They also allow minimal visible light transmission, enabling minimal exposure to sunlight. Reflection, although a great protective strategy for UV sunglasses, is not widely adopted. It is an additional optical technology, further protecting the eyes. The two main technologies are reflective (mirrored) coatings and polarization filters. With the refractive technology controlled and the transmission and absorption properties of the glasses, around 80-90% of total UV radiation from the sun is blocked, leaving only 10-20% of the radiation reaching the eyes. Typically, sunlight reaching the eyes depends on sunglass quality. If bought from low-quality manufacturers, the UV coating will typically peel off, making the sunglasses less effective. Additional coatings come in several variants and enable different properties for the glasses. One in particular includes a hydrophobic coating, making it water-repellent. Another coating is an oleophobic coating, which serves as a repellent for oily substances [24]. Choosing a pair of sunglasses is often a different part of the purchasing process, but focusing on UV-blocking capability is the most important factor to consider. A UV400 rating indicates lenses with a high level of UV protection. People unsure about the rating of their sunglasses often assess them utilizing a photometer, focusing on identifying whether UV-blocking technology is present in the glasses. In addition to the functional use of UV-blocking, there are two other vital factors to consider. One in particular is fit. Ensuring that the glasses properly cover the face and eye regions minimizes light exposure. It is crucial not to prioritize style. Fit is comparatively a greater factor to consider. Lastly, sunglasses are made depending on the activity being engaged in. For reflective surfaces like snow and sand, polarized sunglasses are the ideal option. For yardwork or other intensive activities involving airborne debris, protective goggles preventing ocular damage are ideal. Regular sunglasses, even UV-blocking ones, cannot effectively protect against debris particles [17].

4. Circadian Rhythm

The molecular circadian time-keeping center relies on feedback loops known as transcription-translation feedback loops (TTFLs) that identify the cycles of day and night. Several genes and transcription factors are involved in the timing of the circadian rhythm, including the clock-controlled genes (ex. Three Period (*PER1–3*) and two Cryptochrome (*CRY1–2*)) and transcription factors like Circadian Locomotor Output Cycles Kaput (CLOCK) and Brain-and-Muscle-ARNT-like 1 (BMAL1). For testing of circadian rhythm cycles, albino rats were first domesticated to undergo scientific research. Similarities in anatomy and physiology make them ideal to test on. Having ~95% genomic concordance with humans, they are also small and have high fertility rates [25]. A study shows the impact of changes in eating times on mice's biological clocks. The mice were designated to receive food at variable times during the day, rather than the night. Mice are innately nocturnal, making the discrepancy in normal feeding times critical for analysis. Results showed that abnormalities in eating in treated mice led to greater UVB damage during the daytime than the night. For the control mice fed during the normal evening time, there were no changes in the xeroderma pigmentosum group A (XPA) cycle. The XPA cycle contains enzymes to repair UV-damaged skin, so alterations to the XPA cycle occur with abnormalities in the feeding cycle. In addition to the apparent disruption of the XPA cycle, studies have shown several other implications. One in particular is the impact on skin: evidence shows ~10% of skin genes have been altered by the changes in feeding times.

Secondary impacts show that feeding times impact the quantity of weight loss, incentivizing more research to educate groups for maintaining longevity [9].

5.1 Photokeratitis

Ultraviolet (UV) radiation is notable for the inflammatory impacts it can cause for various ocular structures, including the cornea. Various sources of radiation, whether natural or artificial, and a range of wavelengths are significant risk factors for UV exposure. Photokeratitis is an ocular disease from several natural and artificial sources. The sun is the primary source, but other artificial sources causing the disease include tanning lamps and other therapeutic UV treatments. Common symptoms that typically emerge between 6-12 hrs following exposure are pain and redness; yet, other more severe issues include blepharospasm and lacrimation [6]. In blepharospasm, the eyes shut involuntarily, caused by uncontrollable eye twitches and spasms from excessive sunlight [27]. A second ocular issue under photokeratitis is lacrimation. Tear production from the lacrimal glands happens in response to light stimulus and is a key characteristic of lacrimation [28]. Photokeratitis side effects typically persist for up to 48 hrs. Solar radiation activates various inflammatory processes, including upregulation of nerve signals. These include TRPA1 and TRPV1 channels. In addition, UVR damage to the corneal epithelium disrupts cellular functions, including inhibiting mitosis and fragmenting the nucleus. The same study examined rats, exposing them to 300 nm UVR for a duration of 15 minutes. Results proved detrimental to corneal health, causing apoptosis in all layers; first, it was detected after 5 hrs, and TUNEL staining showed peak apoptotic levels at 24 hrs [5].

5.2 Pterygium

Pterygium arises from limbal stem cell alterations due to lengthy durations of UV exposure. It is labeled as degenerative, with several symptoms and physical feature changes. Pterygia can often have tumor-like features, with infected ocular tissue, leading to abnormal growth paired with premalignant lesions. In addition, the disease is often found in a wing-like shape and encroaches upon the bulbar conjunctiva and ultimately the cornea. Typically, the pterygia are benign lesions that grow slowly; however, if they become larger, treatments are often based on the cosmetic ideals a patient desires. The same study collected information from 100 patients with pterygia, recruited from the Prince of Wales Hospital. The results supported the origin of the condition pterygium. The origin was from limbal stem cells (LSCs), and about 18% of specimens with these markers had certain cell clusters known as Fuchs' flecks, demonstrating this condition. In addition, the study showed premalignant lesions in a high proportion of cases, with 12% of cases containing abnormalities like melanocytic and dysplastic abnormalities [12]. Pterygium often can come with recurrent complications. Corneal scarring and epithelial defects that do not heal on their own are common complications, despite efforts to repair. Pterygium prevalence varies from region to region worldwide and has a great range. It can be as low as 1.7% in South Florida, but can be as high as 9.8% in Australia. A common trend of the percentage of individuals with pterygium is the UV radiation index; areas with higher UV radiation have higher rates of pterygium, and vice versa [7].

6. Public Health Significance

UV-related eye diseases are deadly and a major public health problem [21,22]. However, it is widely preventable if proper precautions are taken [17]. Populations with limited access to

protective eyewear and eye care experience higher rates of UV-induced vision impairment compared to countries with more accessible ocular care [21,22]. Public health initiatives need to be implemented in order to alleviate the situation [21,22].

Social media campaigns help spread ideas and can focus on the benefits of eyecare. Additionally, education seminars in impoverished areas with topics like UV protection, access to basic eye care, diet, and even the body's circadian clock could substantially reduce preventable vision loss worldwide [17,20,21,22].

7. Limitations and Future Directions

Although laboratory studies provide valuable insight into UV-induced corneal damage, more long-term human studies are needed [11,15,16]. Future research should utilize longitudinal studies to explore benefits of various antioxidant diets [2,3,4]. Additionally, translating the research studies into active literacy for underserved communities is a necessity to increase the effectiveness of sustained protective strategies [17,21,22].

8. Conclusion

Learning how UV-B affects corneal cells is key for vision protection, especially in sunny places with little eye protection [17,21,22]. This study shows UV-B triggers apoptosis and ROS stress [11,12,15], but antioxidants like riboflavin/TPGS, vitamin E, and hyaluronic acid help cells survive strong UV-B [16,18]. Additionally, signals such as γH2AX and p53 help cells decide between repair and apoptosis [13,14,19]. Affordable solutions could prevent cell-level eye damage worldwide [17,21,22]. Preventable suffering can only be solved through education on simple protective measures, including UV-blocking eyewear, antioxidant support, and public health responses [16,17,21,22,24]. A clear understanding of these mechanisms supports efforts to preserve vision and reduce preventable eye disease [17,21,22].

Bibliography

[1] Kciuk, M., Marciniak, B., Mojzych, M., & Kontek, R. (2020). Focus on UV-induced DNA damage and repair—disease relevance and protective strategies. *International Journal of Molecular Sciences*, *21*(19), 7264. https://doi.org/10.3390/ijms21197264

[2] Mintie, C. A., Musarra, A. K., Singh, C. K., Ndiaye, M. A., Sullivan, R., Eickhoff, J. C., & Ahmad, N. (2020). Protective Effects of Dietary Grape on UVB-Mediated Cutaneous Damages and Skin Tumorigenesis in SKH-1 Mice. *Cancers*, *12*(7), 1751. https://doi.org/10.3390/cancers12071751

[3] Morifuji, M. (2019). The beneficial role of functional food components in mitigating ultraviolet-induced skin damage. *Experimental Dermatology*, *28*, 28–31. https://doi.org/10.1111/exd.13825

[4] Skin Cancer Foundation. (2017, June 8). *Can Your Diet Help Prevent Skin Cancer?* The Skin Cancer Foundation. https://www.skincancer.org/blog/can-your-diet-help-prevent-skin-cancer/

[5] Alemzadeh-Ansari, M., Izadi, M., Jonaidi-Jafari, N., Pourazizi, M., & Hoseinpourfard, M. (2018). Photokeratitis induced by ultraviolet radiation in travelers: A major health problem. *Journal of Postgraduate Medicine*, *64*(1), 40. https://pmc.ncbi.nlm.nih.gov/articles/PMC5820813/

[6] Photokeratitis (Ultraviolet [UV] burn, Arc eye, Snow Blindness). (2024, January 23). Www.college-Optometrists.org. https://www.college-optometrists.org/clinical-guidance/clinical-management-guidelines/photokeratitis_ultraviolet_uv_burn_arceye_snowblin

[7] Caldwell, M. (2022, October 31). *Pterygium - EyeWiki*. Eyewiki.org. https://eyewiki.org/Pterygium

[8] Volatier, T., Schumacher, B., Meshko, B., Hadrian, K., Cursiefen, C., & Notara, M. (2023). Short-Term UVB Irradiation Leads to Persistent DNA Damage in Limbal Epithelial Stem Cells, Partially Reversed by DNA Repairing Enzymes. *Biology*, *12*(2), 265. https://doi.org/10.3390/biology12020265

[9] Berbis, P. (2010). [Retinoids: mechanisms of action]. *Annales de Dermatologie et de Venereologie*, *137 Suppl 3*, S97-103. https://doi.org/10.1016/S0151-9638(10)70036-3

[10] Sitailo, L. A., Tibudan, S. S., & Denning, M. F. (2002). Activation of Caspase-9 is required for UV-induced apoptosis of human keratinocytes. *Journal of Biological Chemistry*, *277*(22), 19346–19352. https://doi.org/10.1074/jbc.m200401200

[11] Glupker, C. D., Boersma, P. M., Schotanus, M. P., Haarsma, L. D., & Ubels, J. L. (2016). Apoptosis of Corneal Epithelial Cells Caused by Ultraviolet B-induced Loss of K+ is Inhibited by Ba2+. *The Ocular Surface*, *14*(3), 401–409. https://doi.org/10.1016/j.jtos.2016.05.001

[12] Chui, J., Coroneo, M. T., Tat, L. T., Crouch, R., Wakefield, D., & Di Girolamo, N. (2011). Ophthalmic Pterygium. *The American Journal of Pathology*, *178*(2), 817–827. https://doi.org/10.1016/j.ajpath.2010.10.037

[13] Kapoor, M., & Lozano, G. (1998). Functional activation of p53 via phosphorylation following DNA damage by UV but not γ radiation. *Proceedings of the National Academy of Sciences*, *95*(6), 2834–2837. https://doi.org/10.1073/pnas.95.6.2834

[14] Jiang, Guo-Jian, et al. “Ultraviolet B Irradiation Induces Senescence of Human Corneal Endothelial Cells *in Vitro* by DNA Damage Response and Oxidative Stress.” *Journal of Photochemistry and Photobiology B: Biology*, vol. 235, Oct. 2022, p. 112568. *ScienceDirect*, https://doi.org/10.1016/j.jphotobiol.2022.112568.

[15] Reduced UVB-induced corneal damage caused by reactive oxygen and nitrogen species and decreased changes in corneal optics after trehalose treatment. (2010). *PubMed*. https://doi.org/10.14670/HH-25.1403

[16] Palazzo, M., Vizzarri, F., Ondruška, L., Rinaldi, M., Pacente, L., Guerra, G., Merolla, F., Caruso, C., & Costagliola, C. (2020). Corneal UV protective effects of a topical antioxidant formulation: a pilot study on in vivo rabbits. *International Journal of Molecular Sciences*, *21*(15), 5426. https://doi.org/10.3390/ijms21155426

[17] Light, P. Y. E. F. T. S. U. (2025, January 14). Protecting your eyes from the sun’s UV light. *National Eye Institute*. https://www.nei.nih.gov/about/news-and-events/news/protecting-your-eyes-suns-uv-light

[18] Wu, W., Jiang, H., Guo, X., Wang, Y., Ying, S., Feng, L., Li, T., Xia, H., Zhang, Y., Chen, R., Chen, T., & Lou, J. (2017). The protective role of hyaluronic acid in CR(VI)-Induced oxidative

damage in corneal epithelial cells. *Journal of Ophthalmology*, *2017*, 1–6. https://doi.org/10.1155/2017/3678586

[19] Dhuppar, S., Roy, S., & Mazumder, A. (2020). γ H2AX in the S Phase after UV Irradiation Corresponds to DNA Replication and Does Not Report on the Extent of DNA Damage. *Molecular and Cellular Biology*, *40*(20). https://pmc.ncbi.nlm.nih.gov/articles/PMC7523655/

[20] Eating habits affect skin's protection against sun. (2017, August 17). ScienceDaily. https://www.sciencedaily.com/releases/2017/08/170815141712.htm

[21] World Health Organization: WHO. (2023, August 10). *Blindness and vision impairment*. https://www.who.int/news-room/fact-sheets/detail/blindness-and-visual-impairment

[22] International Agency for the Prevention of Blindness. (2021). *Vision atlas: Magnitude of vision loss.* https://www.iapb.org/wp-content/uploads/2021/02/Vision-Atlas_Evidence-Series_Magnitude-of-VIsion-Loss_24022021.pdf

[23] Pavitt, D., & Pavitt, D. (2025, September 28). The Albedo effect – How snow and ice cools the world - The average scientist. *The Average Scientist -*. https://theaveragescientist.co.uk/2023/08/17/the-albedo-effect-how-snow-and-ice-cools-the-world/

[24] Science, S. (2024, November 18). How do sunglasses actually work? *Sunglass Science*. https://www.sunglassscience.com/post/how-do-sunglasses-actually-work

[25] Goodenow, D., Greer, A. J., Cone, S. J., & Gaddameedhi, S. (2022, February 17). Circadian effects on UV-induced damage and mutations. *Mutation Research/Reviews in Mutation Research, 789*, 108413. https://pmc.ncbi.nlm.nih.gov/articles/PMC9188652/

[26] Ge, H., Di, G., Song, P., Han, W., Chen, P., & Wang, Y. (2024). Role of Vitamin A on the Ocular Surface. *Experimental Eye Research*, 110179. https://doi.org/10.1016/j.exer.2024.110179

[27] Blepharospasm: What It Is, Causes & Treatment. (n.d.). Cleveland Clinic. https://my.clevelandclinic.org/health/diseases/21546-blepharospasm

[28] Petersen-Jones, S., & Stanley, R. (2009). Ocular discharge. *Elsevier EBooks*, 253–293. https://doi.org/10.1016/b978-070202861-8.50012-9

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