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The Role of UV Radiation in Chromosomal Mutations: Mechanisms, Impacts, and Implications for Genomic Stability

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Abstract

Through both direct and indirect genotoxic pathways, ultraviolet (UV) radiation is a ubiquitous and powerful environmental mutagen that causes a broad range of chromosomal abnormalities. UV-B and UV-C exposure cause primary lesions like cyclobutane pyrimidine dimers (CPDs) and 6-4 photoproducts, but UV-A exposure causes oxidative damage through reactive oxygen species (ROS). These lesions cause structural abnormalities (deletions, duplications, and translocations) and numerical anomalies (aneuploidy) by interfering with DNA replication and chromosome segregation. In people with compromised DNA repair mechanisms, especially those with Cockayne Syndrome (CS) or Xeroderma Pigmentosum (XP), continuous DNA damage leads to chronic genomic instability and an increased risk of skin malignancies, such as melanoma, squamous cell carcinoma, and basal cell carcinoma.

Oncogenes is made worse by mutational inactivation of tumour suppressor genes such as TP53 and PTEN. Public health hazards have increased due to increased UV-B exposure caused by environmental stresses, particularly ozone layer loss. An integrated strategy is needed to address this issue, integrating policy-driven public education and early screening initiatives with molecular diagnostics, gene-targeted medicines, and advancements in UV protection including wearable sensors and sophisticated sunscreens. In the face of continuous environmental change, such tactics are crucial to lowering the burden of mutations and maintaining genomic integrity.

Keywords: Chromosomal Mutations, Mechanisms, Impacts, Implications, Genomic Stability, Role of UV Radiation

I. Introduction

With its well-established mutagenic and carcinogenic qualities, ultraviolet (UV) radiation is a common environmental agent that has a substantial effect on human health and genetic integrity. Based on wavelength, it is separated into three primary groups: UV-A (320–400 nm), UV-B (280–320 nm), and UV-C (100–280 nm). UV-A and UV-B reach the surface and have significant biological effects, especially on the skin, which is the main interface between environmental UV exposure and internal physiology, whereas UV-C is mostly absorbed by the Earth's ozone layer (Cadet & Douki, 2018). Because it can directly interact with DNA molecules to form cyclobutane pyrimidine dimers (CPDs) and 6-4 photoproducts (6-4PPs), which distort the DNA helix, obstruct transcription, and interfere with DNA replication fidelity, UV-B is thought to be the most genotoxic of these (Brash, 2015; Wang et al., 2024). By producing reactive oxygen species (ROS), which cause oxidative base lesions, single- and double-strand breaks, and crosslinking events that collectively jeopardise DNA and chromosomal stability, UV-A, despite being less energetic, penetrates deeper into the dermis and aids in mutagenesis (Buchanan et al., 2021).The accumulation of somatic mutations and chromosomal abnormalities, such as deletions, duplications, translocations, and aneuploidy, which are indicative of genomic instability and disease development, occurs when UV-induced DNA lesions are not sufficiently repaired. This is especially noticeable in skin tumours with distinctive UV mutational signatures, such as C-T transitions at pyrimidine sites, such malignant melanoma, squamous cell carcinoma, and basal cell carcinoma (BCC,

SCC, and BCC) (Shao et al., 2021). Notably, UV radiation causes oncogenesis by causing mutations in important genes such as TP53, CDKN2A, and BRAF, which interfere with cellular processes that are involved in DNA damage sensing, cell cycle arrest, and apoptosis. In addition to carcinogenesis, long-term exposure to UV light causes photo aging, or premature skin ageing, which is marked by telomere shortening, collagen degradation, decreased dermal flexibility, and increased cellular senescence (Zouboulis et al., 2021). Particularly susceptible to the genotoxic effects of UV radiation are people who have abnormalities in DNA repair pathways, specifically nucleotide excision repair (NER), base excision repair (BER), and mismatch repair (MMR). For example, NER pathway gene mutations cause xeroderma pigmentosum (XP), a rare autosomal recessive condition that causes gradual neurological degeneration, early-onset skin malignancies, and hypersensitivity to UV light (Cleaver et al., 2023). Defective DNA repair exacerbates cellular damage and speeds up systemic ageing in other UV-related hereditary illnesses, including trichothiodystrophy and Cockayne syndrome. These disorders demonstrate how important healthy DNA repair systems are for preserving chromosomal stability and halting UV-induced mutagenesis.

In addition to genetic sensitivity, anthropogenic environmental changes are making the biological consequences of UV light worse. Increased UV-B radiation has reached the Earth's surface as a result of ozone layer depletion brought on by climate change and chlorofluorocarbon (CFC) emissions, especially in areas nearer the poles (Guerreiro et al., 2022). With epidemiological research showing a global rise in UV-associated illnesses such skin malignancies, ocular cataracts, and immunosuppressive disorders, this increase in ambient UV exposure presents a major public health concern. Artificial UV exposure sources, like tanning beds, phototherapy units, and germicidal UV-C lamps, exacerbate this problem by disproportionately affecting younger populations and occupational workers (Narayanan et al., 2010). Developing integrated preventative and intervention techniques is crucial from a public health and policy standpoint. These include routine genetic screening to identify high-risk individuals, the development of next-generation UV-blocking technologies (e.g., nanoparticle-based sunscreens, UV-sensitive wearable sensors, and smart textiles), and educational campaigns encouraging behavioural changes (e.g., sun avoidance during peak

hours, consistent use of sunscreen, and wearing protective clothing) (Zouboulis et al., 2021). Recent developments in precision medicine, including CRISPR-Cas gene editing and small-molecule DNA repair enhancers, present encouraging therapeutic options for reducing UV-induced genomic instability and enhancing the prognosis of patients with impaired DNA repair ability (Wang et al., 2024).Furthermore, our knowledge of UV-induced mutation landscapes is being transformed at a never-before-seen resolution by high-throughput technologies including single-cell transcriptomics, next-generation sequencing (NGS), and systems biology techniques. These methods facilitate the creation of predictive models for UV-associated disorders, the discovery of tissue-specific mutational signatures, and the clarification of DNA repair dynamics. Ultimately, a comprehensive strategy that crosses the boundaries of molecular science, environmental policy, public health infrastructure, and therapeutic innovation is needed to address the complex threat posed by UV radiation. With an emphasis on the function of DNA repair mechanisms in reducing these consequences, this article examines the many molecular processes via which UV radiation causes chromosomal mutations and genomic instability. As we emphasise current preventive measures, public health consequences, and new therapeutic methods, we look at the pathophysiological effects of UV-induced damage, specifically skin malignancies and aging-related illnesses. Understanding and managing the effects of UV-induced chromosomal alterations is still crucial for protecting both individual and global health in an era of rising UV exposure and environmental unpredictability.

II. Mechanisms of UV-Induced Chromosomal Mutations

Chromosome mutations can result from a variety of DNA lesions caused by UV radiation, especially UV-A and UV-B. These lesions include pyrimidine dimers, DNA cross-linking, and oxidative damage. When neighbouring pyrimidine bases in the DNA strand are exposed to UV-B radiation, they produce pyrimidine dimers, specifically thymine-thymine (T-T) dimers. This is the most prevalent type of UV-induced DNA damage (Setlow & Carrier, 1962). These dimers seriously deform the DNA helix, obstructing normal base pairing and disrupting transcription and DNA replication. When the DNA polymerase comes into contact with the dimer during replication, this results in the insertion of point mutations or deletions. Apart from pyrimidine dimers, UV radiation can also cause DNA cross-links, which obstruct

transcription and replication by covalently binding two neighbouring nucleotides across both strands of the DNA helix (Michaels & Miller, 2021). Furthermore, reactive oxygen species (ROS) such superoxide anions, singlet oxygen, and hydroxyl radicals are produced by UV radiation, which also indirectly damages DNA. These extremely reactive substances can interact with DNA to cause oxidative damage, which can include single-strand breaks, base alterations (such 8-oxo-guanine), and, in extreme situations, double-strand breaks. If untreated, ROS-induced lesions may result in alterations such base substitutions or deletions, which could weaken genomic stability and encourage the growth of tumours (Buchanan et al., 2021). Cells use a variety of repair processes, mostly involving the nucleotide excision repair (NER) and base excision repair (BER) pathways, to lessen the genetic effects of UV-induced DNA damage. In order to repair pyrimidine dimers and other large DNA damages brought on by UV light, NER is essential. In this repair route, the damaged area is identified, a brief DNA segment containing the lesion is removed, and the excised strand is resynthesised using the complementary strand as a template (Cleaver et al., 2023). Because NER is necessary to preserve genomic stability, its malfunction results in diseases like xeroderma pigmentosum (XP), which makes people more vulnerable to skin malignancies because they are unable to repair UV-induced DNA damage (Cunningham et al., 2022). Apart from NER, BER is in charge of fixing oxidative DNA lesions brought on by ROS-induced damage, including 8-oxo-guanine. This process entails the identification of the damaged base by certain glycosylases, its removal, and DNA polymerase's subsequent filling of the gap (Lopez et al., 2023). BER plays a key role in maintaining genomic integrity and avoiding mutations that can result from oxidative stress.

In addition to these repair pathways, homologous recombination (HR) or non-homologous end joining (NHEJ) are the main methods utilised to repair double-strand breaks (DSBs) brought on by UV-induced DNA damage. HR is a high-fidelity repair method that precisely fixes DSBs by using a homologous sequence, usually from a sister chromatid (Sung, 2021). When a sister chromatid is available during the S and G2 phases of the cell cycle, HR is especially crucial. However, NHEJ, a repair process that ligates damaged DNA ends directly without the use of a homologous template, is more prone to errors. Chromosome rearrangements may result from NHEJ's propensity to introduce mutations, like as insertions

or deletions, despite its speed and efficiency (Sung, 2021). Significant chromosomal mutations can result from malfunctioning DNA repair systems, which can have a major impact on an organism's health and cellular function. For example, XP, a hereditary condition marked by excessive sensitivity to UV radiation, accelerated ageing, and an increased risk of skin malignancies, is caused by mutations in genes involved in NER, such as XPA, XPC, or POLH (Cleaver et al., 2023). Similarly, because oxidative DNA lesions build up in genes connected to BER, including OGG1 or APE1, mutations in these genes have been linked to an increased risk of cancer (Lopez et al., 2023). Chromosome translocations, deletions, and other abnormalities can result from impaired DSB repair brought on by faulty HR or NHEJ, which can aid in the development of tumours (Sung, 2021). Wide-ranging effects on cellular function may result from the accumulation of UV-induced chromosomal alterations. The buildup of mutations in healthy cells can result in senescence, or cell death; in malignant cells, it can cause unchecked growth and malignancy. It is commonly known that UV radiation contributes to the development of cancer, particularly skin cancer. UV-associated skin malignancies frequently exhibit UV-induced mutations in tumour suppressor genes like p53 and oncogenes like Ras (Buchanan et al., 2021). Moreover, UV-induced mutations in additional genes that control DNA repair, apoptosis, and cell cycle progression could intensify UV radiation's carcinogenic effects.

In conclusion, direct and indirect DNA damage caused by UV radiation can result in mutations that impair cellular functions, hence posing a serious danger to genomic stability. For the genome to remain stable and damaging mutations to not accumulate, DNA repair processes must remain intact. The need of further research into the molecular mechanisms underpinning UV-induced DNA damage and repair is highlighted by the fact that failure in these repair pathways not only raises the chance of chromosomal abnormalities but also predisposes people to a number of genetic illnesses and malignancies.

III. Chromosomal Mutations Induced by UV Radiation

Numerous chromosomal changes are brought on by UV radiation, and these mutations have a big impact on the stability of the genome and the integrity of cells. These mutations fall into two groups: chromosomal abnormalities that are structural and those that are numerical. Direct damage to the DNA structure results in structural mutations, which include deletions,

duplications, inversions, and translocations. For instance, deletions cause chromosomal portions to be lost, which frequently leads to the loss of vital tumour suppressor genes like p53 and encourages the growth of cancer (Setlow et al., 1962). Gene dosage imbalances and the overexpression of oncogenes, which can promote cancer, can result from duplications, which occur when a chromosomal fragment is duplicated (Michaels & Miller, 2021).

Chromosome rearrangements, such as inversions and translocations, can cause important genes to be misplaced or express themselves under the wrong promoters, which might worsen cancer (Buchanan et al., 2021). Conversely, numerical mutations such as aneuploidy and polyploidy involve variations in the number of chromosomes. Since UV radiation damages the mitotic spindle, resulting in chromosomal missegregation, aneuploidy which arises from abnormalities in chromosome segregation during cell division is a characteristic of many malignancies (Cunningham et al., 2022). Cancer cells frequently exhibit polyploidy, which allows them to survive under harsh circumstances. This phenomenon, in which entire sets of chromosomes are duplicated, can result from improper cell division (Sung, 2021). The creation of pyrimidine dimers and replication mistakes are two important processes that are part of the molecular underpinning of chromosomal instability brought on by UV exposure. DNA distortion brought on by UV-induced pyrimidine dimers, particularly thymine-thymine dimers, results in mistakes during DNA synthesis and replication stalling. These mistakes can build up and cause structural mutations like deletions and translocations or even numerical mutations like aneuploidy if they are not appropriately fixed (Setlow et al., 1962). Moreover, aneuploidy can develop from mitotic mistakes brought on by UV exposure, which produce uneven chromosomal segregation. Defects in the spindle assembly checkpoint, which guarantees correct chromosome alignment before to mitosis, worsen UV-induced chromosomal missegregation.

Chromosome instability results from UV radiation that compromises this checkpoint, which promotes carcinogenesis (Buchanan et al., 2021). Furthermore, the persistence of these mutations is significantly influenced by flaws in DNA repair processes. UV-induced pyrimidine dimers are mostly repaired by the nucleotide excision repair (NER) pathway. However, because of the build-ups of unrepaired mutations, people with NER-deficient disorders like Xeroderma Pigmentosum (XP) have a far greater prevalence of UV-induced

skin malignancies (Cleaver et al., 2023). Similar to this, the base excision repair (BER) process helps repair UV-induced oxidative DNA damage, but when it malfunctions, it can also lead to long-lasting mutations.

Furthermore, the failure of double-strand break repair mechanisms such as homologous recombination (HR) and non-homologous end joining (NHEJ) can worsen chromosomal instability and raise the risk of oncogenesis. These mechanisms are essential for repairing breaks that occur during replication stress caused by UV (Michaels & Miller, 2021). The mechanisms driving UV-induced chromosomal alterations have been better understood thanks to experimental models, which include both in vitro and in vivo systems. Pyrimidine dimer production, mistakes in DNA replication, and mitotic missegregation in response to UV radiation have all been studied in vitro using cultivated cell lines. These findings have improved our understanding of how repair inadequacies lead to chromosomal instability by demonstrating that abnormalities in DNA repair pathways can worsen UV-induced mutations (Buchanan et al., 2021). Studying the long-term effects of UV radiation on genomic stability and cancer development has required the use of in vivo models, especially mice models with faulty DNA repair genes like those implicated in NER. These models give researchers insights that are not achievable from in vitro studies alone by enabling them to investigate how UV-induced chromosomal changes appear in entire organisms and lead to disorders like skin cancer (Cunningham et al., 2022). Additionally, since various tissues may show varying sensitivity to UV-induced mutations, these animal models also aid in clarifying the tissue-specific reactions to UV radiation.

In conclusion, pyrimidine dimer formation, replication errors, and mitotic errors are some of the ways by which UV radiation causes both structural and numerical chromosomal alterations.

The development of cancer and chromosomal instability are greatly aided by the accumulation of these mutations, particularly when DNA repair systems malfunction. The intricate relationships between UV radiation, DNA damage, and cellular response mechanisms are clarified by using experimental models to understand these processes. This helps to shed light on how UV exposure can have long-term genomic effects. Better methods

to prevent and treat UV-induced malignancies and other genetic illnesses linked to chromosomal instability depend on the continued investigation of these pathways.

IV. Impacts of UV-Induced Chromosomal Mutations on Genomic Stability

UV-induced chromosome mutations have a major impact on genomic stability and can result in a variety of health issues, including cancer, early ageing, and various genetic disorders. The connection between UV radiation and genetic instability is evident in the development of skin malignancies, such as melanoma, basal cell carcinoma (BCC), and squamous cell carcinoma (SCC). UV-B light in particular forms pyrimidine dimers, which have the ability to bend the DNA helix and cause mutations during DNA replication (Setlow et al., 1962).

These changes may have an impact on tumour suppressor genes like p53, which are crucial for regulating the cell cycle, apoptosis, and DNA repair. The loss of p53's tumor-suppressive activity encourages carcinogenesis by boosting cellular proliferation and survival, making it one of the most common genetic alterations observed in UV-related skin cancers (Buchanan et al., 2021). Additionally, mutations in other tumour suppressors, such as PTEN, which regulate cell growth and survival, speed up the development of cancer by interfering with the PI3K/AKT signalling pathway (Cunningham et al., 2022).

A common mechanism of UV-induced mutations is the formation of pyrimidine dimers, primarily thymine and cytosine dimers, which inhibit DNA replication and cause replicative errors. If these dimers are not sufficiently rectified, they can result in chromosomal instability by generating mitotic mistakes like chromosomal missegregation, in which chromosomes fail to split effectively during cell division. These errors can lead to numerical chromosomal anomalies such aneuploidy or polyploidy, which can promote tumorigenesis and genomic chaos (Michaels & Miller, 2021). According to research, UV-induced DNA damage also promotes structural chromosomal alterations including deletions, inversions, and translocations, which further jeopardise the integrity of the genome and hasten the onset of cancer.In addition to its function in skin cancer, UV-induced genomic instability has been connected to accelerated ageing and a number of hereditary issues. The enzymes required for nucleotide excision repair (NER), the primary DNA repair process that corrects pyrimidine dimers brought on by UV light, are absent in people with conditions like Xeroderma Pigmentosum (XP). Failure to repair UV-induced damage leads to premature onset of skin

cancer and accelerated ageing (Cleaver et al., 2023). Growth retardation, increased sensitivity to UV light, and severe brain degradation are symptoms of Cockayne Syndrome (CS), another hereditary disease brought on by transcription-coupled repair mistakes.

The fact that the DNA repair mechanisms of both XP and CS are impaired highlights how important efficient DNA repair is to maintaining genomic integrity and preventing disease. The genomic instability associated with UV-induced mutations worsens over time due to the cumulative impact of UV exposure. Long-term exposure to UV radiation causes DNA damage, particularly in the skin, the organ most frequently exposed to UV radiation. The long-term effects of this accumulated damage include actinic keratosis, BCC, and melanoma (Michaels & Miller, 2021). Additionally, the eyes are another vital organ that is damaged by UV radiation. Over time, UV rays harm the lens of the eye, which can result in cataracts and ocular melanoma. Furthermore, long-term UV exposure and immune cell abnormalities caused by UV light diminish local immune responses, making the skin more vulnerable to infection and cancer development (Setlow et al., 1962).

In conclusion, genomic instability which has a profound impact on ageing, cancer, and genetic disorders is largely caused by UV radiation. Chromosomal damage accumulation, chronic mutation of tumour suppressor genes, and a failure of DNA repair systems are the causes of skin cancers and aging-related illnesses. Long-term UV exposure also accelerates these processes, which eventually increases the load of mutations. Understanding the processes via which UV radiation alters chromosomes is necessary to develop preventative measures and treatment approaches aimed at decreasing UV-induced damage. Research is uncovering crucial new details about the molecular mechanisms underlying UV-induced genomic instability, highlighting the need for better DNA repair, early detection of UV-induced damage, and effective sun protection strategies to reduce the incidence of UV-related diseases.

V. Implications for Public Health and Environmental Concerns

The consequences of UV-induced chromosomal abnormalities go well beyond personal health; they also involve environmental and public health issues that have grown more pressing in recent years. The earth's natural defence against damaging ultraviolet (UV) radiation, the ozone layer, is being destroyed, and this has had a significant impact on UV

exposure levels worldwide. Human-induced pollutants, especially chlorofluorocarbons (CFCs), have caused a dramatic reduction in the ozone layer, which is made up of ozone molecules that absorb and filter the majority of the Sun's harmful UV rays (Solomon et al., 2022). Higher quantities of UV-B radiation, which is especially damaging to DNA, are currently received in locations that are undergoing considerable ozone thinning, such as the regions over Antarctica and parts of the Arctic. Thus, ozone depletion has caused UV radiation levels to rise, greatly increasing the risk of UV-induced DNA damage like chromosomal mutations worldwide (Solomon et al., 2022). A major contributing factor to the increased prevalence of skin malignancies, including melanoma, basal cell carcinoma (BCC), and squamous cell carcinoma (SCC), which are all directly related to UV-induced DNA alterations, is the rise in UV exposure. Skin cancer rates have increased dramatically in nations like Australia, where ozone depletion is most severe, putting a significant strain on healthcare systems (Barton et al., 2023).

The threats to public health are growing increasingly obvious as environmental UV radiation levels keep rising. Global trends in UV exposure show that the number of skin cancer diagnoses is rising globally. Certain groups, especially those with lighter skin phototypes and fair skin, are more vulnerable to UV-induced chromosomal alterations and subsequent carcinogenesis (Barton et al., 2023). These mutations can result from direct DNA damage caused by UV radiation, such as the creation of pyrimidine dimers, which can cause chromosomal abnormalities and improper DNA replication. Furthermore, indirect damage caused by reactive oxygen species (ROS) production aggravates genomic instability even more, which promotes the development and spread of cancer. Chromosome instability, a characteristic of many cancers, especially those pertaining to the skin, is caused by the cumulative effect of UV radiation over time (Buchanan et al., 2021). Preventive steps to reduce UV exposure have been implemented in response to the growing threats to public health. Sunscreens, which include active chemicals like avobenzone and zinc oxide, function as chemical or physical barriers that reflect or absorb UV rays, shielding the skin's cellular DNA from harm. To avoid excessive exposure to UV radiation, it is now essential to wear protective apparel and accessories, such as wide-brimmed hats, UV-blocking textiles, and sunglasses, in addition to sunscreens.

Additionally, public awareness programs are essential for teaching people the value of sun protection and the dangers of prolonged UV exposure. Particularly in high-risk areas, these programs, which are led by governmental and health organisations, frequently emphasise the use of protective apparel and high-SPF sunscreens as well as safe sun habits. Government rules are also essential for reducing the health concerns associated with UV radiation. Standards for sunscreen products have been set by numerous nations, guaranteeing their effectiveness in preventing dangerous UV radiation. To guarantee that customers have access to correct information regarding SPF levels and broad-spectrum protection, regulatory agencies including the European Medicines Agency (EMA) and the U.S. Food and Drug Administration (FDA) have established criteria for sunscreen labelling. Additionally, by delaying the ozone layer's depletion, environmental protection initiatives that attempt to reduce CFC emissions have played a crucial role in controlling the rise in UV exposure (Solomon et al., 2022). Future developments in UV protection technologies could significantly lower the likelihood of chromosomal alterations brought on by UV radiation. Since UVB rays cause sunburns and more immediate skin damage, while UVA rays penetrate deeper into the skin and cause long-term damage, new sunscreen formulations are being developed to give greater protection against both UVA and UVB radiation. Nano-encapsulated sunscreens, which provide more effective and long-lasting protection, are the result of research into nanotechnology. There are also more wearable UV-monitoring gadgets on the market, like smartwatches and UV sensors built into clothes. By using these gadgets, people can keep an eye on their UV exposure in real time and take prompt action to avoid overexposure (Buchanan et al., 2021). Additionally, genetic screening is becoming more widely acknowledged as a useful method for identifying people who are more vulnerable to UV-induced mutations. UV-induced chromosomal damage is especially dangerous for those with abnormalities in DNA repair processes, such as those who have Xeroderma Pigmentosum (XP), a rare genetic condition that affects nucleotide excision repair (NER). According to Cleaver et al. (2023), early identification of these people and customised preventative measures can greatly lower their risk of 8UV-related genetic diseases and malignancies. Gene therapy and DNA repair enhancement are being investigated as potential future treatment options in addition to genetic screening. Research on improving NER

pathways to repair UV-induced DNA damage, for instance, may help avoid the long-term effects of UV exposure, such as the emergence of skin cancer (Cleaver et al., 2023). Finally, lowering the worldwide burden of UV-induced chromosomal alterations will depend heavily on environmental regulations targeted at minimising UV exposure, as well as developments in protective technology and genetic strategies. Ensuring the longevity of ozone protection initiatives and continuing to promote public health education on UV safety will be vital in reducing the incidence of UV-induced malignancies and genetic diseases in the next decades.

VI. Recommendations

A concerted effort is necessary to lessen the effects of UV-induced chromosomal alterations. Consistent application of broad-spectrum sunscreens, UV-blocking apparel, and avoiding the sun during peak hours should be the focus of public awareness campaigns, especially for high-risk populations including outdoor workers, those with fair skin, and people with DNA repair problems. To find and safeguard susceptible people, healthcare systems should implement early detection initiatives including genetic screening. Prevention and therapy can be improved by expanding research into DNA repair processes and creating tailored medicines, such as pharmacological and gene-based interventions. Priority should be given to innovations in UV protection, such as wearable UV sensors, eco-friendly sunscreens, and materials that block UV rays. Furthermore, international initiatives to stop ozone depletion are still essential for lowering UV-B exposure and preserving genetic integrity.

VII. Conclusion

UV-induced chromosomal aberrations are associated with skin cancers, early ageing, and genetic illnesses, and they pose a serious risk to genomic stability, especially in individuals with impaired DNA repair processes, as those with Xeroderma Pigmentosum. These mutations, caused by pyrimidine dimers and oxidative damage, are exacerbated by ozone depletion, which increases UV exposure globally, and other environmental factors. Preventive measures, such sunscreen, protective clothing, and public education, are crucial in addition to developments in wearable sensors, UV-blocking technologies, and genetic screening. In the future, minimising the negative health effects of UV radiation and

preserving genetic integrity would necessitate a coordinated effort involving environmental protection, public policy, and scientific research.

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