Sources and Types of Ultraviolet Radiation

Subjects: Dermatology

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Ultraviolet (UV) radiation plays a crucial role in the development of melanoma and non-melanoma skin cancers. The types of UV radiation are differentiated by wavelength: UVA (315 to 400 nm), UVB (280 to 320 nm), and UVC (100 to 280 nm). UV radiation can cause direct DNA damage in the forms of cyclobutane pyrimidine dimers (CPDs) and 6-4 photoproducts (6-4PPs). In addition, UV radiation can also cause DNA damage indirectly through photosensitization reactions caused by reactive oxygen species (ROS), which manifest as 8-hydroxy-2′-deoxyguanine (8-OHdG). Both direct and indirect DNA damage can lead to mutations in genes that promote the development of skin cancers.

Keywords: ultraviolet radiation ; UVA radiation ; UVB radiation ; melanoma ; squamous cell carcinoma ; basal cell carcinoma ; cyclobutane dimers

1. Introduction

Ultraviolet (UV) radiation is an environmental stress factor that extensively affects the skin. Persistent UV exposure over extended periods contributes to various adverse events that weaken our immune suppression, accelerate photoaging, and increase our susceptibility to developing skin cancer.

2. Sources of Ultraviolet Radiation

Ultraviolet radiation (UVR) is in a distinct portion of the electromagnetic spectrum, ranging from 200 to 400 nanometers (nm), placing it just beneath the visible light range of 400 to 700 nm. UVR is primarily sourced from the sun and is differentiated into three categories based on wavelength: UVA (315 to 400 nm), UVB (280 to 320 nm), and UVC (100 to 280 nm) [1]. Alongside natural sources, artificial sources of UV radiation are becoming more common in dermatological conditions. Such sources include tanning beds and various therapeutic medical devices capable of emitting UVA, UVB, or UVC radiation. Industrial equipment, such as welding torches, also serves as a considerable artificial source of potent UV radiation [2]. The assortment of natural and artificial UV sources enriches the spectrum of UV exposure encountered in different environments, which potentially results in an elevated risk of skin cancer.

2.1. Ozone Layer

The stratosphere’s ozone layer serves as a critical barrier, absorbing much of the sun’s UV radiation and thereby shielding the Earth’s surface from its harmful effects [3]. In regions near the equator, the ozone layer is thinner; these areas experience higher levels of UV radiation, which correlates with increased rates of skin cancer at decreasing latitude [4]. Conversely, the ozone layer is thicker near higher latitudes, providing greater protection against UV radiation. In the last century, human activities, notably the emission of chlorofluorocarbons (CFCs) and other ozone-depleting substances (ODSs), have significantly weakened the ozone layer, increasing the risk of UV radiation reaching the Earth. The Montreal Protocol, established in the 1980s, has played a pivotal role in halting the depletion of the ozone layer by eliminating 99% of ODS emissions through bans on various applications, such as freon refrigerators. As a result of the Montreal Protocol, the ozone layer is anticipated to return to its baseline state by the mid-21st century [3]. Projections indicate a decrease in UV radiation between 30 and 60 degrees latitude by 2 to 5% in the north and 4 to 6% in the south, reflecting the positive impact of the protocol [5]. However, challenges remain due to rising greenhouse gas emissions, contributing to variability in the ozone layer and influencing UV radiation levels. Factors such as cloud cover, aerosols, and surface reflectivity are becoming more significant in determining UV exposure outside polar regions. Climate change, marked by increasing temperatures, further modifies UV radiation exposure, with research suggesting that the carcinogenic potential of UV radiation could increase by 5% with each degree Celsius rise in temperature [6]. This complex interplay of environmental changes highlights the ongoing need to monitor and adapt our strategies for protecting public health against the effects of UV radiation.

2.1.1. Melanoma

The incidence of melanoma is on the rise in many developed countries, though the rates differ across various populations and age groups. Specifically, in countries like Canada, Italy, Norway, France, and Lithuania, there has been a notable increase in melanoma cases among older age groups. Conversely, younger demographics have seen a decline or stabilization in melanoma incidence [3]. This difference in trend between older and younger groups can largely be explained by the cumulative effect of UV radiation exposure over the years in older populations, including UV exposure prior to the implementation of the Montreal Protocol [7]. For younger individuals, the reduced incidence of melanoma is likely due to heightened awareness and use of sun protection measures [3]. Additionally, shifts in occupational trends toward more indoor jobs and preferences for indoor leisure activities have contributed to lower UV exposure among this group. These trends highlight the importance of continued public health efforts to promote sun safety and the impact of global environmental policies on public health outcomes.

2.1.2. Squamous Cell Carcinoma and Basal Cell Carcinoma

Squamous cell carcinoma and basal cell carcinoma, collectively known as keratinocyte skin cancers (KCs), have seen varying trends in incidence across different regions. From 2005 to 2019, Australia, New Zealand, Iceland, and the United Kingdom reported an increased incidence of KCs, while the United States experienced relatively stable rates [3]. This disparity in KC incidence rates can be linked to a variety of factors, including differences in health policies, public awareness, accessibility to healthcare services, practices in reporting, and, potentially, genetic predispositions affecting skin cancer susceptibility. A significant aspect influencing the risk of developing KCs is exposure to ambient UV radiation, with populations in regions with high UV exposure facing a substantially increased risk. For instance, the lifetime risk of developing KCs in Australia is 3.5 times higher than in the United States [3]. This data implies that geographic areas subjected to more intense and consistent UV radiation exhibit higher incidences of KCs, underlining the critical impact of environmental factors on the prevalence of skin cancers.

3. Types of Ultraviolet Radiation

3.1. UVA Radiation

UVA rays have the longest wavelength within the ultraviolet spectrum, extending from 320 to 400 nanometers (nm), and constitute the majority—between 90% and 95%—of UV radiation that reaches the Earth's surface. These rays are known for their ability to penetrate the skin deeply, reaching beyond the epidermis to the lower layers of the dermis. UVA radiation adversely impacts both epidermal keratinocytes and dermal fibroblasts, leading to long-term skin structure and function changes. The penetration of UVA radiation is significant, about 100-fold more than UVB radiation that can infiltrate the skin [1]. Despite their ability to reach deeper into the skin, UVA rays are generally less directly involved in skin carcinogenesis than UVB rays due to their relatively lower absorption by DNA [8]. The mechanism in which UVA rays cause DNA damage is primarily through indirect means, such as photosensitization reactions, which lead to the formation of 8-hydroxy-2′-deoxyguanine (8-OHdG) [9]. When skin chromophores absorb UVA rays, this interaction prompts the formation of reactive oxygen species (ROS), which can cause oxidative stress within the epidermal and dermal layers of the skin [10]. This oxidative stress is a key player in the pathogenesis of photoaging. It activates pathways like mitogen-activated protein kinases (MAPKs) and nuclear factor-kappa B (NF-κB), which elevate the levels of matrix metalloproteinases (MMPs) in the skin. MMPs are enzymes that break down structural proteins such as collagen and elastin. These proteins are crucial for maintaining skin elasticity and integrity [11]. Consequently, the breakdown of structural proteins by MMP contributes to the development of wrinkles and other signs of aging skin. Moreover, the use of tanning beds, which predominantly emit UVA radiation, has been linked to increased risks of erythema (skin reddening) and melanoma. Studies have shown that UVA radiation is responsible for 50% to 80% of the erythema associated with tanning beds, underscoring the potential risks associated with artificial UVA exposure [12] rather than the potential risks associated with UVB radiation exposure.

3.2. UVB Radiation

UVB rays have a wavelength ranging from 280 to 320 nm and are the primary cause of carcinogenic damage found in the skin. Despite comprising only 1% to 10% of sunlight that reaches our planet, UVB rays are the primary cause of overt skin damage, such as sunburns [13]. These rays primarily target the outermost skin layer, the epidermis, but have the capacity to penetrate the upper dermis [1]. For UVB rays to initiate a biological response, they must be absorbed by cellular molecules capable of transforming light energy into chemical signals. Genomic DNA is the primary absorber of UVB, and its interaction with UVB often leads to the formation of thymine dimers, which are distinctive indicators of UVB damage [1]. This results in the formation of cyclobutane pyrimidine dimers (CPDs) and 6-4 photoproducts (6-4PPs), which can accumulate and, if not properly repaired by the nucleotide excision repair (NER) system, significantly heighten the risk of skin cancer [14]. UVB exposure also has the potential to generate reactive oxygen species (ROS) via nicotinamide adenine dinucleotide phosphate hydrogen (NADPH) oxidase and cyclooxygenase (COX) enzymes in keratinocytes.

3.3. UVC Radiation

UVC has a wavelength between 100 and 280 nm, representing the most deleterious form of UV radiation due to its high energy [1]. Fortunately, UVC rays are completely filtered by the Earth's ozone layer and never reach the ground.

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