Skin Cancer: Pathophysiology and Epidemiology

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Introduction

Definition and significance

Skin cancer is defined as the uncontrolled or abnormal proliferation of skin cells due to various reasons. One of the most well-known and referred reasons is DNA damage and mutations from UV radiation that trigger growth. The mutations are caused by ultraviolet (UV) radiation, ionizing radiation, immunosuppression, or viral infections, and are commonly seen in elderly patients (Kaur and Yusuf, 2021). Ever since the 19th century, skin cancer has been associated with sun exposure (Al-Matouq, 2025).

Skin is the largest organ of the body, and it performs multiple vital functions. It protects the body from environmental factors like pathogens and UV rays, acts as the first line of immunological defense against infections, keeps the organ and tissue integrity, and helps conserve homeostasis. The three primary layers of skin are: epidermis, dermis, and hypodermis. The epidermis is the most superficial layer, and it is mostly made up of keratinocytes and melanocytes at the basal layer. Langerhans cells and Merkel cells are also seen in the epidermal layer. The dermis lies beneath the epidermis and constitutes the thickest part of the skin. It contains fibroblasts, vessels, nerves, and appendages. The hypodermis is the deepest layer and contains fat tissue.

Skin cancer is stated as the most frequent malignant condition among light-skinned individuals. Both malignant melanoma and non-melanoma skin cancer are reportedly on the rise. The incidence among light-skinned people is now as high as 1.2 million cases a year (Al-Matouq, 2025; Ferlay et al., 2021). It is speculated that there is a skin cancer epidemic with the increased incidence of melanoma cases, yet the mortality rates do not comply with it. This raises the question of whether the increased number of biopsies and overdiagnosis is causing the growing incidence data (Apalla et al., 2017).

Because of its high frequency, related morbidity, and mortality, skin cancer (SC) is

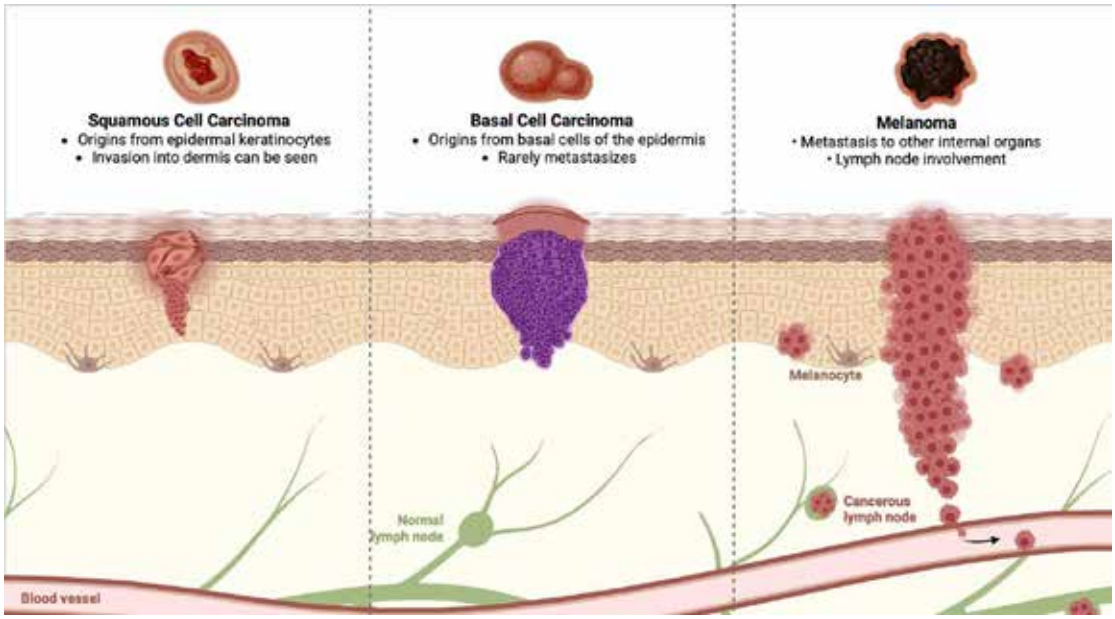
referred to as a serious global public health concern. Healthcare institutions and the overall economy are severely strained by the number of skin cancer patients. The Australian health system spent AU$1.8 billion on the diagnosis and treatment of skin cancer in 2020–2021 (Collins et al., 2024). The increasing global prevalence, high financial cost, and the fact that malignant melanoma is still the most aggressive type of cancer, skin cancer continues to be a significant concern that requires immediate attention for care and prevention.

Types of skin cancer

Skin cancer types differ in their clinical presentation, place of origin, and extent of spread. They are mainly separated into melanoma and non-melanoma skin cancer (NMSC). Melanoma skin cancer is also known as malignant melanoma or cutaneous melanoma. It arises from melanin-producing cells: melanocytes. It constitutes a smaller percentage of skin cancer cases but holds the majority of skin cancer-related deaths. The four major subtypes of melanoma include: Superficial Spreading Melanoma (SSM), Nodular Melanoma (NM), Lentigo Maligna Melanoma (LMM), and Acral Lentiginous Melanoma (ALM). NMSCs originate from keratinized epithelial cells. The two main divisions of NMSC are basal cell carcinoma (BCC) and squamous cell carcinoma (SCC) (Figure 1).

Figure 1

Types of Skin Cancer



BCCs originate from the basal cells at the deepest layer of epidermis. They rarely metastasize but are locally damaging (Roky et al., 2025). SCCs arise from epidermal keratinocytes. It tends to be more aggressive compared to BCCs, and if left untreated,

it may develop deeper into the epidermis and metastasize to other tissues (Zeng et al., 2023). Actinic keratoses and Bowen's disease are accepted as precursors of BCC lesions (Das et al., 2021; Al-Matouq, 2025). Other less common NMSCs are: Merkel cell carcinoma, which develops quickly and is aggressive, kaposi sarcoma, related to viral infections HHV-8, and Cutaneous Lymphoma. Since NMSCs have a lower mortality rate and are usually detected in their early stages, they are not reported to the skin cancer registries. Although they don't usually metastasize out of the tumor site, in rare cases, if they are left untreated, they may cause fatalities (Kivisaari & Kähäri, 2013).

Epidemiology of Skin Cancer

Physicians have described specific subtypes of skin cancer in the early 19th and early 20th centuries (Zeng et al. 2023). The relation between excessive sun exposure and skin cancer was first noted in the early 19th century, and in the early 20th century, for the treatment of skin cancer, radiation was utilised (Leiter et al., 2020; Natelauri and Jghamadze, 2025). Epidemiological observations started to firmly establish our knowledge of environmental risk factors and disease frequency by the middle of the 20th century.

Since its incidence, morbidity, and mortality constitute a public health issue, skin cancer is one of the biggest medical problems of today. Melanoma skin cancer is the 17th most common type worldwide. Europe has the highest frequency and fatalities. Nonetheless, the highest rates of incidence and mortality are found in Australia and New Zealand (Roky et al. 2025). Non-melanoma skin cancers (NMSCs) are most common in North America, and the majority of deaths from NMSCs occur in Asia. Australia and New Zealand have the greatest incidence rates of basal cell carcinoma (BCC).

The global burden and risk of skin cancer are significantly influenced by demographic factors. Fair-skinned people of European ancestry have the highest prevalence of skin cancer. Globally, melanoma is more commonly seen in men than women in regions other than the United States (Arnold et al., 2022). Generally speaking, incidence rises with age. Over-50-year-olds account for over 80% of newly diagnosed melanoma cases. But among young individuals, melanoma is also one of the most prevalent malignancies (Bhattacharya et al., 2021).

Changes in incidence and prevalence over the years

An estimated 325,000 new cases of melanoma were reported worldwide in 2020. The International Agency for Research on Cancer (IARC) estimated that the newly diagnosed melanoma cases to increase by more than 50% and melanoma deaths to increase by more than 68% by 2040. These rates were calculated in accordance with the population growth and changes in age composition. Age-adjusted change assumptions on the incidence and

mortality rates were not considered. But the increasing trend is not the same worldwide. The incidence rate among the young population of Australia has decreased to about half of what it was in the 1980s. Australia's widespread SunSmart education programs and other sun protection measures are to blame for this particular decline (Arnold et al., 2022; Collins et al, 2024).

Geographical distribution

Geographically, there are significant differences in the number and pace of skin cancer cases. Australia has the highest incidence rate for both males and females for melanoma, followed by Western Europe, North America, and Northern Europe. The lowest rates are seen with most regions of Africa and Asia, less than 1 per 100000 person-years. The highest mortality rates due to melanoma are seen in New Zealand and Australia (Arnold et al., 2022). In North America, the incidence of melanoma among white people has increased by 3% yearly from 1996 to 2006. The peaks are observed in people 65 years and older. NMSC cases make up more than 5.4 million per year (Linares et al., 2015). In Europe, according to the European Academy of Dermatology and Venereology (EADV), the number of NMSC cases has increased by 3–8% annually in recent decades, and the ten to twenty-five new cases of malignant melanoma are reported for every 100,000 people (Roky et al., 2025). The greatest age-standardized rates of melanoma worldwide are found in Australia and New Zealand (Oceania). In contrast to the ongoing rise in the majority of European nations, incidence rates in Oceania and North America seem to have stabilized recently (Arnold et al., 2022).

General Overview of Skin Cancer Pathogenesis

Pathogenesis of Non-Melanoma Skin Cancers (BCC, SCC, Actinic Keratosis)

BCC is the most commonly seen form of skin cancer. It develops slowly and shows local invasion (Linares et al.,2015). It is most commonly seen on sun-exposed areas such as the ears, face, and dorsum of the hands. It is rarely seen on palmoplantar surfaces and never on mucosa (Apalla et al., 2017; Linares et al.,2015). Most cases of BCC have a good prognosis; rarely some of them progress to a more advanced form with very few treatment options and no formal treatment algorithms.

UVR is mostly responsible for the formation of BCC. In most cases, a mutation in the Hedgehog signaling pathway is seen. Mutations in the PTCH1 gene, which codes for the sonic hedgehog receptor, cause cancer by preventing cell cycle arrest and differentiation, which leads to unchecked cell proliferation and the development of hyperplasia (Al-Matouq, 2025, p. 9). Hedgehog signaling pathway inhibitors show complete remission in locally infiltrated cases of BCC (Apalla et al., 2017). The canonical (β-catenin-dependent) Wingless (WNT) signaling pathway and P53, as commonly seen with SCC, are two more frequently impacted pathways in BCC (Kashyap et al., 2022).

Squamous cell carcinoma stems from the uncontrolled proliferation of keratinocytes. The risk factors for SCC are similar to BCC, with the addition of chronic inflammation due to skin damage and epidermolysis bullosa syndromes (Linares et al., 2015). SCC progression follows a complex and multi-factorial route. Several mutations lead to precancerous lesions such as actinic keratosis (AK) and Bowen's disease (BD). BD is an intraepithelial form of SCC. They are both characterised by atypical keratinocytes at various sites of the epidermis. In about 3-5% of lesions, infiltration through the basement membrane and progression to SCC is seen (Souto et al., 2022).

DNA damage brought on by UV radiation exposure is the primary cause of cutaneous squamous cell cancer. The degree of lifetime sun exposure is correlated with the occurrence of tumors (Lazar, 2021). DNA damage response pathways are triggered by substantial DNA damage caused by acute UVR exposures. As a molecular mediator, p53 is crucial to the way cells react to UVR. In UVR-exposed keratinocytes, higher amounts of p53 were seen. p53 functions as a transcriptional regulator in cells, facilitating cell cycle arrest, initiating DNA damage repair, and promoting apoptosis. But different signaling pathways, such as phosphoinositide 3-kinase (PI3K), serine/threonine protein kinase AKT, and mammalian (or mechanistic) target of rapamycin (mTOR) (PI3K/AKT/mTOR) can act against the functions of p53 and eventually give rise to carcinomas (Manning and Cantley, 2007; Carvalho et al., 2024). The balance between those mechanisms is the determining factor for the outcome on UV-irradiated cells.

The pre-malignant lesions, such as AK, shed light on the underlying mechanisms of SCC, genetics, and acquired defects. One of the major consequences of UVR exposure is the accumulation of defects, especially TP53. The mutant p53 plays a pivotal role in carcinogenesis. p53 is thought to be one of the first agents to be affected by UVR and lead to the formation of tumors. TP53 mutations were reported to be highly common in AK lesions of light-skinned individuals, and it was also detected in more than 30% of BCC and 90% of SCC cases, affirming the influence of mutated TP53 in skin cancer sites (Kumar et al., 2015; Carvalho et al., 2024). The mutations regarding TP53 are mostly seen at pyrimidine dimers, proving the UVR effect. In normal cells, the damage caused by UVR is checked at cell cycle checkpoints by kinases of RAD3-related (ATR) and Ataxia Telangiectasia Mutated (ATM) pathways. These kinases control p53 status and expression by signals. To prevent oncogenic transformations, p53 stops the cycle at the G1 checkpoint by activating p21 and blocking cyclin-dependent kinase (CDK)/cyclin E complex activation. UVR originated reactive oxygen species (ROS) cause apoptosis signal-regulating kinase1 (ASK1) to become active, which in turn triggers p53. Depending on the amount and type of damage, p53 either facilitates DNA repair or initiates apoptosis. If the protective function of p53 is lost by a mutation, DNA repair is done by alternative ways, which are labile. Thus, both the p53 mutations and DNA

damage are passed down to next-generation cells (Lazar, 2021).

Cell division cycle 25 (CDC25) phosphatases are known to be a close proximity to NMSCs. They are the controllers of apoptosis and the cell cycle. Normally, ATM phosphorylates the effector checkpoint kinases (Chk1 and Chk2) to activate the downstream signaling pathway. CDC25 is phosphorylated by Chk1/2. UVR activates ErbB2, which is an upstream regulator of a DNA damage cell cycle checkpoint. The PI3K/Akt pathway is signaled by ErbB2 activation and inhibits phosphorylation of Chk1 on Ser280. This limits Chk2 and further inhibits the phosphorylation of CDC25, resulting in long-lasting activity that can trigger and support tumorigenesis. Excess amounts of CDC25 promote anti-apoptosis and work in favor of survival in skin cancer (Reddy et al.,2010; Al-Matouq, 2025).

Another common mutation seen with SCCs is the RAS oncogene. The RAS protein holds an important place in the regulation of cancers. When the RAS-related proteins accumulate, they can sustain tumor formation by hyperproliferation, invasion, metastasis, and anti-apoptotic effect (Al Mahi and Ablain, 2022).

Malignant Melanoma

Melanoma is an aggressive neoplasm stemming from the malignant transformation of melanocytes. Melanocytes are the cells that produce melanin pigment; they are found in the epidermis, eye, and many internal organs (Al Mahi and Ablain, 2022). Melanomas can occur in normal skin or from existing nevi (Litvin et al., 2025). Even though melanoma is responsible for 2% to 5% of skin malignancies, it is the reason for the majority of deaths (Linares et al., 2015). The prognosis depends on the location of the tumor and the status and depth of metastasis. The survival chance is high if the diagnosis is made in the early stages and the tumor is surgically resected. In case of metastasis, the prognosis of 5 years is less than 5% (Zeng et al., 2023). There are four main types of skin cancer: superficial spreading melanoma (75% of cases), nodular melanoma (15-30% of cases), lentigo maligno melanoma, and acral lentiginous melanoma (the least common subtype) (Lee et al.,2020; Linares et al., 2015). A mix of environmental and genetic factors contributes to the development of melanoma. UV radiation is closely associated with all skin cancers as the reason for acquired mutations. For melanomas, the effect of UVR is not as straightforward as for other skin cancers. Current studies state sunburns in early ages as the primary factor. But since melanomas can be seen in non-sun-exposed parts of the skin and also dark skinned individuals, other factors are thought to have a greater risk (Lazar, 2015).

10-15% of melanomas are passed down through autosomal dominant genes with varied penetrance. High-penetrance genes such as *CDKN2A*, *CDK4*, BAP1, and medium-penetrance genes such as MC1R are found to be related to familial cases (Flori et al.,

2025; Lazar, 2015). Also, light complexion as well as a tendency to get sunburns rather than tanning is related to a higher likelihood of having melanoma. It is reported that white folks are more than 20 times more likely than African Americans to develop melanoma (Linares et al., 2015).

A great proportion of melanomas present hyperactivation of the RAS/MAPK pathway in relation to an oncogenic driver (Al Mahi and Ablain, 2022). The most frequently seen oncogenic site is v-Raf murine sarcoma viral oncogene (BRAF), which is a part of the MAP/ERK pathway controlling cell development and proliferation. BRAF mutations are commonly seen in dysplastic nevus, inducing a senescence-like state that, when overcome, progresses to melanoma (Paluncic et al.,2016). The PTEN tumor suppressor is frequently lost in melanomas with BRAF mutations, which results in increased PI3K/AKT/mTOR pathway activation. Mutations in the NRAS tumor suppressor gene also contribute to the progression of melanoma. NRAS mutations activate both the MAPK and the PI3K/AKT pathway, in contrast to BRAF mutations that solely activate the MAPK pathway (Vuković et al., 2019).

Another critical factor in melanoma progression is the inactivation of tumor suppressor genes. The CDKN2A gene mutation is seen in almost half of autosomal dominant familial melanoma cases. Three distinct tumor suppressors are encoded by the complex gene CDKN2A. Of these, loss of p16/INK4a is unmistakably linked to human melanoma, and experimental data also suggest that loss of p14/ARF plays a part. p16/INK4a strengthens the retinoblastoma protein (RB) tumor suppressor's capacity to stop cells in the G1 phase of the cell cycle. Inactivation of CDKN2A tumor cells bypasses the checkpoint and leads to melanoma progression (Al-Matouq, 2025; Zaidi et al., 2009). To understand the progression and prognosis of melanoma, radial and vertical growth concepts become essential. The radial growth phase describes the horizontal progression. In this stage, the tumor stays within the epidermis and superficial dermis and does not metastasize. The vertical growth phase shows invasion towards the dermis and a subclone signaling metastasis (Lazar, 2021).

Ultraviolet radiation

It is commonly stated that chronic exposure to ultraviolet radiation is the primary cause of skin cancer (Linares et al., 2015).

UVR is a form of radiation naturally occurring in solar rays and artificially produced in tanning beds. While it is categorised as a primary carcinogen for skin cancer and other environmentally affected diseases, it is also the primary factor for vitamin D synthesis and different endorphins in the skin. Malignant melanoma, basal cell carcinoma, and squamous cell carcinoma are all attributed to UVR both molecularly and epidemiologically. Also, genetic factors, most importantly the MC1R gene, play a role in UVR-related skin cancer

risk since it is connected to the UVR protection and lightness of skin (D'Orazio, 2013). Early research showed that skin pigmentation protected against skin cancer and that UV radiation caused DNA damage that resulted in mutations (Al-Matouq, 2025)

UV radiation stands between the wavelengths of visible light and gamma rays. UVR is divided into three parts based on its wavelengths: UVA, UVB, and UVC. Solar UVR is mostly UVA and a lesser portion of UVB, showing differences according to geographic and climatic reasons. UVA are the wavelengths between 315–400 nm. It can reach the dermis and the subcutaneous tissue beneath. UVB ranges from 280 to 315 nm and has less penetrating ability compared to UVA. It gets mostly absorbed at the epidermal layer. The carcinogenic activity is mostly attributed to UVB, even though UVA makes up the majority of solar UVR. UVC has the shortest wavelengths and it gets mostly absorbed at the stratosphere layer (Al-Matouq, 2025; Carvalho et al., 2024). The effects of different wavelengths are not distinct and mostly seen overlapping each other.

An elevated risk of all main forms of skin cancer is evidently associated with indoor tanning machines, also known as tanning beds, which are a substantial artificial source of ultraviolet radiation (UVR). Tanning beds emit UVR at a much higher concentration than solar light by producing both UVA and UVB rays. The usage of such devices takes a toll on skin health due to the cumulative effects of UVR (D'Orazio, 2013). It is stated that there is no safe level of usage for tanning beds. Even though recent data show that tanning bed usage has decreased in the last decade, the lifetime effects are still visible (Diehl et al., 2024).

The effects of UVR are mainly seen with DNA damage, making itself visible through point mutations. The damage occurs mainly as direct photochemical damage or indirect oxidative damage. With direct photochemical damage, DNA directly absorbs UVB rays, which results in molecular changes that produce certain photoproducts. Cyclobutane pyrimidine dimers (CPDs) and 6–4 photoproducts (6–4 PPs) on DNA are the most commonly seen lesion types. If those lesions are not reversed, they can lead to certain mutations, most commonly seen in p53 and other skin cancer-related genes. With indirect oxidative damage, the reactive oxygen species (ROS) such as superoxide anions and hydrogen peroxide caused by both UVA and UVB play a great role. ROS can damage lipids, proteins, and DNA subunits. This damage can give rise to mutations such as guanine to thymine transversion, leading to oncogene activation or tumor suppressor gene repression (Al-Matouq, 2025; Carvalho et al., 2024). ROS can also activate signaling pathways related to inflammation and cancer formation by influencing intracellular antioxidant molecules (Kobaisi et al., 2021; Carvalho et al., 2024).

While NMSC types BCC and SCC are mostly associated with sun exposure and mostly seen on sun-exposed sites of the body, such as the face, head, and neck, there seems to

be different takes on causality between sun exposure and melanoma occurrence (Holick, 2014). Even though UVR is stated as the main environmental trigger in melanoma development, current data show that intermittent intense exposure to UVR, such as sunburns, is more crucial in early life (Al-Matouq, 2025; Bulliard et al., 2007; Elwood and Gallagher, 1998).

Genetic predisposition

In the pathophysiology of skin cancer, MC1R, other genes, and signaling pathways play a key role in the development of skin cancer. MC1R is known by its effect on pigmentation of skin and, less often, for its non-pigmentation role of DNA repair (Liu et al., 2016). MC1R triggers a signaling cascade to increase intracellular cyclic adenosine monophosphate (cAMP) and thus produce photoprotective eumelanin pigment. The production process gets stimulated by ligands like alpha-melanocyte-stimulating hormone-like ligands, whose levels increase after UV exposure (Sturm et al., 2003). Eumelanin is much more effective at UV protection than pheomelanine, which is prooxidant (D'Orazio et al., 2013). Some MC1R variants are categorised as "RHC (Red Hair Color)" due to loss-of-function mutations, making them associate more with red hair, fair skin, freckles, and a poor tanning response (Guida et al., 2022). The mutations reduce the function of the gene and increase the eumelanin ratio. Since pheomelanine is less UV protective, the MC1R mutations become susceptible to developing melanoma and NMSCs (Tagliabue et al., 2015). Also, it was seen that MC1R mutations could increase the penetrance of high-risk skin cancer-related genes like CDKN2A (Fargnoli et al., 2010).

Skin cancer is generally not considered to be genetically transferred through family members, although family members carry similar skin characteristics, which determines the likelihood of skin cancer development. Most cases of melanoma are sporadic, but around 10 to 15% of them are seen to occur due to inheritance of autosomal dominant traits with variable penetrance. Key risk factor genes for melanoma are CDKN2A, CDK4, BAP1, and MC1R variants (Manganelli et al., 2021; Gosman et al., 2023). For NMSCs, a positive family history of skin cancer is accepted as an important factor for BCC. Rather than extrinsic factors, genetic susceptibility plays a greater role in the development of BCCs. Nevoid Basal Cell Carcinoma Syndrome (NBCCS) or Gorlin Syndrome is an autosomal dominant multisystem disorder. It manifests with multiple BCCs on the skin. Similarly, Bazex-Dupre-Christol syndrome, Rombo syndrome, xeroderma pigmentosum, and Bazex syndrome are also associated with early onset of multiple BCCs (Schierback et al., 2019).

Microbiome

The skin microbiome, composed of a diverse selection of bacteria, fungi, and viruses, and the balance between them, plays a significant role in the pathophysiology of skin

cancer. The skin microbiota competes for space and nutrients, which makes them a part of the physical barrier of protection. Commensal bacteria help in keeping the skin pH levels stable and produce antimicrobial peptides, preventing pathogen invasion (Kotak, 2021). The microbiome interacts with T cells to promote their functions, help regulate inflammation, and immune tolerance (Pandey, 2021). A change or imbalance in the microbial community, known as dysbiosis, can cause a change in immunological responses, which can lead to persistent inflammation and possibly cancer.

Chronic inflammation is associated with the occurrence and development of several cancers. Additionally, the inflammatory setting triggered by dysbiosis in the skin tumor site reduces the effectiveness of cancer immunotherapy and suppresses the body's inherent anti-tumor immunity (Tang & Wang, 2016).

Atopic dermatitis is one of the known precursor lesions of skin cancer. Excess amounts of Staphylococcus aureus are known to be a major contributing factor for atopic dermatitis, which raises the risk for especially squamous cell carcinoma (Kullander et al., 2009; Pandey, 2021). Also, a decreased proportion of Malassezia and Cutibacterium species has been observed in actinic keratosis and squamous cell carcinoma sites. It is theorized that the loss of protective effect by commensal bacteria may have contributed to carcinogenesis (Woo et al.,2022).

Human Papillomavirus (HPV) is a well-known oncovirus known to be connected to a number of cancers. Although it is most recognized for its involvement in cervical cancer, some forms of HPV are also linked to skin carcinogenesis, especially when combined with other risk factors like immunosuppression and UV exposure. Because HPV oncoproteins E6 and E7 inhibit DNA repair and cause death, UV-damaged cells can multiply unchecked. (Kotak, 2021).

Conclusion

Skin cancer is one of the most frequently diagnosed cancers globally and represents one of the greatest medical challenges. The incidence rates continue to increase in many parts of the world. The rising numbers are not only related to rising UV exposure and lifestyle changes, but also a part of the overdiagnosis and improved detection phenomenon. With the help of new technologies such as artificial intelligence and advances in diagnosis with generalized usage of dermatoscopes in clinics, the detection of lesions has become easier, and the diagnostic sensitivity has increased. Longer lifespans and more common access to healthcare services compared to the past also had a role in the increasing number of diagnoses. Still, the sheer amount of cases generates a huge burden on the healthcare systems.

New studies on the pathogenesis of skin cancer cases allow us to better understand the

molecular background of disease progression and risk factors. Current studies show that cumulative exposure to ultraviolet radiation (UVR) is directly linked to the pathogenesis of skin cancer. Prevention and early diagnosis are essential to lowering the morbidity and death linked to skin cancer. Given that UVR is the strongest modifiable risk factor for skin cancer progression, sun protection plays an important role in prevention strategies.

It is recommended to minimize outdoor activities during peak ultraviolet (UV) radiation hours, typically between 10 a.m. and 4 p.m. Wearing protective clothing that covers exposed skin, along with wide-brimmed hats and UV-blocking sunglasses, provides additional defense against harmful rays. Regular application of a broad-spectrum sunscreen that protects against both UVA and UVB rays, with frequent reapplication throughout the day, is also crucial (Collins et al., 2024). Avoiding artificial sources of UV exposure, such as tanning beds and sunlamps, further reduces skin damage risk. Finally, engaging in routine skin examinations and early screening—especially for individuals at high risk—supports early detection and significantly improves treatment outcomes.

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