



## Mini Review

## The dark side of the Sun: Affected demographics of skin cancers.

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**Publication data:**  
Submitted: May 24 ,2021  
Accepted: September 22 ,2021  
Online: November 30, 2021

This article was subject to full peer-review.



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### Summary

Basal cell carcinoma (BCC) and squamous cell carcinoma (SCC) are the most common skin malignancies. BCC and SCC are affecting the corresponding cells in the epidermis where tumorigenesis starts. The onset of these diseases is caused by sun exposure and ultraviolet radiation (UVR). However, it seems that there are more vulnerable groups within the population. Several other leading factors have been described in the literature. Elderly, male and fair-skinned individuals may have significantly increased skin cancer risk. Understanding the characteristics of these cancers epidemiology may allow their early detection and ensure better medical and surgical management.

### Keywords:

Skin cancer risk; Ultraviolet radiation; Sun exposure; Sun protection; Review.

Skin cancer is the most common carcinoma affecting millions worldwide. BCC is affecting approximately 3.6 million people in the United States, while SCC is targeting 1.8 million Americans every year [1,2]. Chronic sun exposure and ultraviolet radiation (UVR) are directly involved in tumorigenesis initiation and progression [3]. The photodamage is cumulative and dose-dependent [4]. Ultraviolet A with its wavelength of 315 to 400 nm is more associated with keratosis and photocarcinogenesis [5]. BCC and SCC clinical presentations are variable. Lesions frequently present as shiny translucent skin-colored or pigmented bump generally in the head and neck areas [6].

The aim of this review was to provide a comprehensive study of skin cancers risk factors to highlight the most important prognostic indicators for this disease.

Skin types are classified according to the Fitzpatrick scale which assess the response of different types of skin to UV light. It classifies the skin colors into six phototypes from the light pale white to dark brown [7]. Photodamage following chronic sun exposure seems to be more important and less reversible for skin type with less epidermal melanin level [8]. Colored skin epidermal barrier filter twice as much UVR as in white skin [9]. However, the correlation between photoprotection and melanin pigmentation involves more complex mechanisms, and no skin type is immune to cancer [10]. Moreover, some other recent reports confirmed that the risk to develop BCC in highly UV-exposed skin was doubled independently of histological subtype, tumor localization and Fitzpatrick phototype and [11]. Non melanoma skin cancers are significantly more frequent for phototype II and III in old female patients [12].

There are specific dermoscopic characteristics in skin of color for benign neoplasms. However, there is a lack of data about specific features for skin cancers. Further descriptive studies are needed to better characterize specific predisposing factors in this skin phototype [13].

Actinic keratosis (AK) is a severe grade of skin damage. It is considered as precancerous condition and represents a reliable marker of chronic photodamage as well as skin microbiome modifications which may start immediately with UVR exposure [14]. Photodamage is a progressive phenomenon. Severe alteration of the skin texture may be observed within 30 years of UVR exposure [15]. There are several epidemiological and molecular data suggesting that skin cancer is predominantly a disease of the elderly. More than half of skin cancer-related deaths are observed in patients of more than 65 years old [16]. Intrinsic skin aging process implies random mutations-related cell damages. Excessive free radicals production during metabolic processes may accelerate the implementation of precancerous cutaneous lesions [17]. Predominance of male sex in skin cancer patients has been widely cited in the literature. However, confrontation of this evidence with the geographic, ethnic, and sociocultural factors should be considered [18]. Several studies demonstrated that dermoscopic, histologic and hormonal characteristics enhance photodamage repair in female. This may decelerate the photodamage-precancerous lesions-skin cancer filiation sequence in female patients [19,20]. However, molecular and genetic comparative studies are to be conducted to rule out clearly the involvement of gender in skin tumorogenesis. A recent south African comparative study found that the incidence of Cutaneous melanoma in Black Africans is about 10% of that in Whites. There was no difference in age and sex distribution. Independent risk factors list included sun exposure, skin trauma, human immunodeficiency virus infection, albinism, and genetic predisposition [21].

Some groups of patients are genetically predisposed to skin cancer. The most common syndromes associated with BCC are: Gorlin-Goltz syndrome, Rombo syndrome, and Bazex-Dupré-Christol syndrome. Multiple SCC is usually associated to xeroderma pigmentosum, Rothmund-Thomson syndrome and Bloom syndrome. Malignant melanoma can be inherited, as in familial atypical multiple mole melanoma syndrome [22,23]. Chronic UVR exposure seems to be higher in male workers in sunny countries. In these same conditions we could note the lack of social coverage and health insurance which make the access to specialized health facilities more difficult. The skin cancer diagnosis is always delayed which is significantly interfering with the prognosis.

Demographics and other epidemiologic factors should be studied in correlation with patients individual characteristics and social specificities as well.

In our experience according to short cohort observational study results, we noted that fair-skinned patients (Fitzpatrick I and II) were more likely at risk than the other phototypes. Age >65 and male gender were the other significant skin cancer risk factors in our study.

The data heterogeneity related to skin cancer risk assessment explain partially the absence of prevention strategy guidelines [24,25]. Based on the evidence that skin cancer is an environmental cancer rising on photo-damaged skins due to UVR exposure, all the prevention efforts were focused on sunscreens industry over the last decade [26]. Sunscreens are objectively a considerable part of the prevention strategy. In addition to its role in UVR filtration, sunscreen may reduce the p53 oncogene mutation [27]. Sunscreen's clinical effectiveness is related to its ability to reduce DNA damage, immune system modulation and free radical generation. However, there are still several gaps in research and knowledge regarding safety, efficacy, and overall public benefit perception. In the United States the Food and Drug Administration (FDA) is only approving zinc oxide and titanium dioxide for UVA filtration. Other non-mineral sunscreens may be more efficient but less stable [28-30].

This raises the concern about the ideal molecule needed and if ever the progress of the sunscreen industry is really based on clinical evidence. The establishment of screening strategy may allow early cancers diagnosis and enhance the prognosis. The primary prevention should be focused on high-risk groups. The risk assessment must consider all personal and environmental factors to elaborate a personalized protection plan.

### Key takeaways

- Skin cancers are of the most common malignancies. The incidence may be still underestimated due to the absence of effective screening strategy and the limited access to dermatology specialized consultation for a considerable part of high-risk population.
- The chronic sun exposure and photodamage caused by UVR significantly increase the risk of skin cancer. However, several personal predisposing conditions must be considered for an objective risk assessment.
- Melanin is an UVR filter. This may explain the vulnerability of fair skin to UVR induced damages. The incidence of cancer in skin of color might be higher in male sun-exposed workers and in southern African countries.
- Prevention of this entity is almost totally based on sunscreens dermo-protection. More efforts could be done in the sensibilization and primary prevention of people at risk.

**Conflict of interest:** none

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