Research & Reviews: Reports in Cancer and Treatment

Epidemiological Study and Risk Factors of UV-induced Skin Cancer

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Commentary

Received: 08-Jul-2022, Manuscript No. RCT -22-68548; **Editor assigned:** 13-Jul-2022, PreQC No. RCT-22-68548 (PQ); **Reviewed:** 28-Jul-2022, QC No. RCT-22-68548; **Revised:** 04-Aug-2022, Manuscript No. RCT-22-68548 (R); **Published:** 12-Aug-2022, DOI: 10.4172/Rep cancer Treat.6.4.003.

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DESCRIPTION

Basal Cell Carcinoma (BCC), Squamous Cell Carcinoma (SCC), and melanoma are the three main types of skin cancer, and there is strong evidence that they are all brought on by sun exposure. The risk of each increases with increasing ambient solar radiation; the highest densities are on the most sun-exposed parts of the body and the lowest on the least exposed; they are associated in people with total (primarily SCC), occupational (mainly SCC), non-occupational or recreational sun exposure (mainly melanoma and BCC); and a history of sunburst. Based on indirect inferences from studies in animals, the action spectrum of solar radiation for skin cancer, the action spectrum for dipyrimidine dimers, and evidence that presumed causative mutations for skin cancer arise most frequently at dipyrimidine sites. It is believed that UV radiation specifically causes these skin cancers. If the incidence of skin cancer is to be decreased, sun protection is crucial. The epidemiological data suggest that increasing intermittency of exposure should be avoided when implementing sun protection, that achieving sun protection as early as possible in life will have the greatest impact, and that it will likely have an impact later in life, especially in those who had high levels of childhood solar radiation exposure.

The incidence of skin cancer has reached epidemic levels, with melanoma and Non-Melanoma (basal and squamous cell carcinoma) Skin Cancer (NMSC) now being the most prevalent cancer types in white populations. Recent population-based studies from Australia indicate that males have a basal cell carcinoma incidence rate of over 2% and females have a squamous cell carcinoma incidence rate of 1%.

All people are exposed to the carcinogen known as sunlight. The primary epidemiologic risk factor for skin squamous cell carcinoma is its UV component. If tumours had UV-specific mutations, those stages of the multiple

stages of tumour progression would be revealed. The presence of a CC--TT double-base change, which is only known to be caused by UV light, in three of the tumours points to UV light's involvement in these p53 mutations.

A UV-like occurrence of mutations that only occur at dipyrimidine sites, including a high frequency of C--T substitutions, also points to UV as a possible cause. Internal malignancies with p53 mutations do not exhibit these UV-specific mutations. The dipyrimidine specificity also suggests that cytosine-containing dipyrimidine photoproducts are oncogenic photoproducts. These findings, in our opinion, point to a carcinogen-related process in a gene that contributes to the development of human cancer.

Among the most prevalent cancers in humans are Non-Melanoma Skin Cancers (NMSCs). Avoiding UV radiation from both natural and artificial sources as well as wearing sunscreen and photoprotective clothing is currently the best ways to prevent them. These strategies, though, haven't been able to stop the rise in skin cancer incidence over the past few years.

A research suggests that the prostaglandin synthesis enzyme Cyclooxygenase-2 (COX-2) may play a role in the pathogenesis of NMSC. In preclinical studies, animals that are genetically deficient in the COX-2 enzyme or that have been given pharmacological COX-2 inhibitors experience a significant reduction in tumour development when compared to control mice when exposed to a UV-induced skin carcinogenesis protocol. It is known that UV radiation increases COX-2 expression in human skin, and several epidemiological studies in humans support the idea that this enzyme is directly involved in the development of UV-induced skin cancer. According to recent research, medications that inhibit COX-2 expression may stop the growth of NMSCs. Therefore, pharmacological treatments that block COX-2 could be efficient NMSC chemopreventive treatments.

UV-induced skin cancers are not acknowledged as an occupational disease, despite the fact that it is widely accepted that exposure to UV light can cause malignant skin tumours. There are many workplaces where UV light exposure, whether natural or artificial, occurs, making the development of occupational skin cancers conceivable. Some cases of skin cancer brought on by UV exposure have recently been recognised by a special clause in the occupational disability rules.

The type of occupational UV exposure, look into prevention options, and examine the information on work-related UV-induced skin tumours. We draw the conclusion that for squamous cell carcinoma the epidemiological proof of an at least doubled risk (RR>2) due to occupational UV radiation can be given after reviewing recent publications. These epidemiological findings are supported by the obvious dose response relationship. There must be an attributive UV radiation due to occupational factors of >40% for the individual risk assessment. Squamous cell carcinoma should be recognised and compensated as an occupational disease in those circumstances.