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Commentary

Photo Carcinogenesis and Skin Penetration Prevention Strategies

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DESCRIPTION

Skin cancer may result from photo carcinogenesis. It entails a chain of events in which UV radiation drives tumour suppressor genes and proto oncogenes to mutate. Photo carcinogenesis is usually triggered by a photo carcinogen, which produces free radicals that cause cells to die.

The basic principles of UV-induced carcinogenesis are summarised in this overview, as well as current diagnostic and treatment techniques. Basal cell and squamous cell carcinomas are the most common keratinocyte-derived skin neoplasms. Cutaneous melanoma is less common yet has a high death rate. Sun exposure and DNA-repair deficits are common risk factors for all three tumour types. UV-induced DNA damage causes mutations, which result in the activation of oncogenes or the silence of tumor-suppressor genes, according to a multistep model of cancer development. As a result, the cellular mutator phenotype becomes even more susceptible to mutation acquisition. DNA repair, particularly the nucleotide excision repair (NER) pathway, prevents the production of mutations and the development of skin cancer. The NER-defective condition xeroderma pigmentosum is a good example of this. As a result, primary skin cancer prevention measures include reducing DNA photodamage through sun protection. Skin cancer screening is a secondary prevention strategy. This includes using a naked eye examination, an epiluminescence microscope, or digital epiluminescence microscopy. Confocal laser scan microscopy is a more advanced technology.

Carcinogenesis of uv-induced skin tumors

The electromagnetic spectrum is divided into two parts: ionising and non-ionizing. It spans a vast range of all potential electromagnetic fields. Ionizing radiation has sufficient energy to disrupt chemical bonds in molecules, resulting in the formation of ions. Non-ionizing radiation, on the other hand, does not have enough energy to generate charged ions yet can cause molecular excitation. Non-ionizing radiation is recognised to pose health hazards and has been linked to a variety of skin problems, including cancer.

Optical radiation and electromagnetic fields are the two primary sections of the non-ionizing spectrum, with the latter being further split by radiofrequency (microwave, very high frequency and low frequency radiowave). The optical spectrum is separated into ultraviolet (UV), visible, and infrared wavelengths. The effects of UV radiation on photocarcinogenesis.

Skin penetration

UVC is mutagenic, yet it does not reach the earth's surface because the stratospheric ozone layer blocks it almost completely. As a result, the UV light reaching the earth's surface is primarily UVA (90-95 percent) and to a lesser extent UVB (5-10 percent), with UVA passing through the ozone layer while UVB is absorbed by the layer.

Longer wavelengths penetrate deeper skin layers, and UV radiation penetrates the skin in a wavelength-dependent manner. In contrast to UVB, which is almost fully absorbed by the epidermis, the less energetic UVA rays penetrate deeper into the dermal compartments.

Although UVA is the most common component of solar UV radiation reaching the earth's surface, it is significantly less carcinogenic than UVB and causes skin photoaging (solar elastosis) mostly through dermal fibre breakdown. Despite the fact that UVB radiation accounts for a small percentage of total solar radiation, it is substantially more carcinogenic at much lower concentrations than UVA radiation. Because UVB is the major chromophore that absorbs the most light, it has a direct mutagenesis effect on DNA. UV photon energy absorption by DNA diminishes with increasing wavelength (in the UVA range); as a result, UVB radiation is thought to be the leading cause of skin cancer. UVA radiation, on the other hand, plays an important role in photocarcinogenesis, causing DNA damages through indirect mechanisms.

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