PIGMENTATION AND THE SUN

The most important non-genetic contributing factor to skin pigmentation is sun exposure.

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Excessive, unprotected exposure to solar radiation can lead to accelerated photoageing, carcinomas and disorders of skin pigmentation. Hyperpigmented lesions may be of aesthetic concern alone, or represent premalignant and malignant lesions. A number of preventative measures can be employed to reduce the risk of hyperpigmentation and premalignant skin lesions.[1]

Sunlight comprises ultraviolet (UV) radiation, visible light (VL) and infrared (IR) radiation. UV light induces DNA damage and free radical production in the epidermis, which initiates a tanning response in the skin.[2] Although it is well documented that skin pigmentation may occur in response to UV radiation, the effects of VL and IR on the skin are still under scrutiny. Recent literature has suggested that visible light (VL) does in fact induce pigmentation[3] and plays a role in worsening certain pigmentary disorders despite the use of sunscreens with UV protection.[4]

IR is perceived as heat energy by human skin and has been shown to cause temporary erythema due to a vasodilatory thermal effect. IR also contributes to photoageing. It has been proposed that increased temperature due to IR is associated with oxidative stress and free radical formation.

A distinction exists between tanning (melanisation) and pigmentation. Delayed skin tanning is an adaptive, physiological response of the skin to UV exposure (UVA and UVB). UV light and VL up-regulate melanin synthesis within the epidermis. Increased quantities of melanin allow increased absorption of UV rays by the skin, thereby, offering some degree of protection against subsequent UV damage. Tanning usually occurs three – five days after exposure to sunlight. Delayed tanning may disappear over time.[5]

The process of pigmentation is biphasic. Initially, on exposure to UVA and visible light, immediate pigment darkening (IPD) occurs. This may last up to two hours. Subsequent persistent pigment darkening (PPD) may occur. This may take up to 24 hours. IPD and PPD are due to oxidisation of existing melanin and redistribution of melanin-containing melanosomes from melanocytes to keratinocytes located more superficially in the epidermis.[5] Melanin is shed with the stratum corneum cells during the desquamation process.

Skin phototype determines the risk for pigmentation due to sun exposure.[5] Lower Fitzpatrick phototypes (1 and 2) tan poorly and burn more easily and, therefore, require more protection against the harmful effect of the sun. These phototypes are more at risk for carcinomas. Higher Fitzpatrick phototypes (3 - 6) tan more easily and are more at risk for hyperpigmentation following sun exposure.

Disorders of hyperpigmentation include postinflammatory hyperpigmentation, melasma, solar lentigines, and lichen planus pigmentosus. The likelihood of pigmentation occuring following excessive sun exposure depends on genetics, behavioural and geographic factors, and the presence of concomitant diseases and even the use of certain medications.

Solar lentigo is a well-circumscribed patch of darkened skin that occurs due to local proliferation of melanocytes and accumulation of melanin within keratinocytes. Solar lentigines (SL) result from UV exposure. They may vary in size and depth of colour and may scale. These pigmented patches are occasionally referred to as sun spots. SL occurs in both light and dark skins but tend to be more numerous in fair skinned individuals.

Sunlight may also worsen existing disorders of pigmentation such as melasma. The cause of melasma is complex, involving melanin overproduction by melanocytes. This melanin is either taken up by the keratinocytes (epidermal melanosis) and/or deposited in the dermis (dermal melanosis). Although there is a genetic predisposition and hormonal link associated with melasma, sun exposure is the most important avoidable risk factor. Other pigmented lesions may include Aktinic keratosis, pigmented basal cell carcinomas and malignant melanoma. Aktinic or solar keratoses are considered precancerous lesions or an early form of squamous cell carcinoma. These lesions present as scaly patches, which may be pigmented. They occur in response to UV light. Fair-skinned patients with sunburn history and excessive time spent outdoors for work or recreation are particularly at risk. Actinic keratosis may also occur in patients with a defective immune system and other signs of photoaging.

PREVENTION

Education and implementation of photoprotection programmes remain integral to reducing adverse effects of solar radiation including pigmentation.

Safe sun practices should be actively promoted by healthcare practitioners in an effort to lower the incidence of sunlight-induced skin damage. Healthy photoprotection habits acquired in childhood and adolescence may modify behaviours in later years.

To prevent hyperpigmentation, protection using adequate sunscreens on exposed areas is needed. To date, no efficient protection from IR light exists, but topical antioxidants may be able to provide some protection.[6] Common sunscreen ingredients include metal oxides, iron oxides, and antioxidants. Physical barriers against harmful radiation include clothing with high UPF (UV protection factor), sunglasses and tinted windows in cars and homes.

TREATMENTS

Hyperpigmentation treatments often focus on:

- Accelerating epidermal turnover
- Down-regulating tyrosinase
- Reducing melanocyte proliferation and production of melanin
- Inhibiting inflammation.

Chemical peels with glycolic, salicylic and/or lactic acid may be used to accelerate epidermal cell turnover and remove pigment in the superficial layers of the epidermis. Microdermabrasion achieves a similar result. Care must be exercised with chemical peels to prevent post-inflammatory hyperpigmentation. Microneedling can be utilised to aid delivery of active ingredients which inhibit pigment formation.

Tyrosinase is an enzyme which catalyses a rate-limiting step in melanin biosynthesis. Down-regulating this enzyme with the use of tyrosinase inhibitors decreases melanin synthesis. Topical tyrosinase inhibitors include phenolic agents such as Hydroquinone and non-phenolic agents including tretionoin, adapalene, niacinamide, liquorice extract, arbutin, vitamin C and kojic acid. These are popular constituents in topical applications treating pigmentation. Hydroquinone use is usually limited to three months of application and ideally used in a concentration of 4% or less in order to avoid the development of exogenous ochronosis.[7,8]

Antioxidants in the skin include superoxide dismutase, glutathione peroxidase and catalase. These are depleted in the skin with prolonged exposure to UVR. Antioxidants act as inhibitors of reactive oxygen species and may down-regulate hyperpigmentation and UV-induced melanogenesis. Topical and systemic antioxidants, therefore, play a positive role in preventing hyperpigmentation. Corticosteroids inhibit inflammation in the skin and may be used as an adjuvant in the treatment of pigmentation. A combination of these therapies with frequent sunscreen use is often necessary. MC

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