# SUN PROTECTION

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While sunlight has numerous benefits to human skin, the most important being vitamin D biosynthesis, excessive sun exposure may manifest with an array of skin conditions. It is known to accelerate the signs of aging, cause sunburn and increase skin cancer risk.

**Solar radiation comprises a spectrum of light of varying wavelengths including visible light (VL), ultraviolet (UV) radiation and Infrared (IR) radiation.**

Fifty percent of solar radiation is Infrared in the form of heat energy and is perceived as warmth. Even a single overexposure to IR is sufficient to cause heat pain, heat urticaria and cardiovascular collapse.

Visible light is that portion of light which can be detected by the naked eye and comprises 45% of solar radiation. Visible light has been reported to induce oxidative stress in human skin. VL is also thought to cause mutations by generating reactive oxygen species such as superoxide anion, hydrogen peroxide and the hydroxyl radical. Nucleotides are highly susceptible to free radical injury. Recent studies on the effects of visible light on the skin have suggested that shorter wavelengths of visible light could play a role in worsening certain pigmentary disorders following sun exposure, despite the use of sunscreens with UV protection 3.

UV light makes up 5% of sunlight and cannot be seen or felt. It can, however, penetrate the skin. The high energy of UV radiation induces acute and chronic skin damage after short periods of exposure. Most of the harmful effects of sunlight on the skin are caused by UV rays.

Solar UV radiation can be subdivided into UVA, UVB and UVC according to their wavelengths. As the atmospheric ozone layer absorbs UVC, this wavelength of light does not reach the earth's surface. Ambient sunlight is predominantly UVA (95%) and UVB. Each UV component of solar radiation exerts different, distinct effects on the skin.

The longer wavelength of UVA (320 – 400nm) allows it to penetrate deeply into the dermis of the skin. UVA generates reactive oxygen species that damage DNA via indirect photosensitising reactions. UVB (290 – 320nm) is almost completely absorbed by the epidermis, with comparatively little reaching the dermis. UVB induces a cascade of cytokines, vasoactive and neuroactive mediators in the skin. The end result is an inflammatory response, which causes reddening and sunburn. UVB affects DNA in epidermal cells directly. Both UVA and UVB may contribute to carcinogenesis through different mechanisms.

The intensity of UVB varies according to season, altitude, distance from the equator and time of day. The amount of UV light the skin encounters depends on the intensity and duration of solar radiation as well as the usage of UV-protective clothing, shade and sunscreens.

UV induces damage response pathways in keratinocytes. Doses of UV radiation above the damage response threshold stimulate keratinocyte apoptosis. Several hours after UV

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exposure, the damage response signals diminish and epidermal keratinocyte proliferation ensues (hyperkeratosis). This is mediated by a variety of epidermal growth factors. This proliferation increases epidermal thickness. Epidermal hyperplasia serves as a protective mechanism against future UV penetration.

In addition to epidermal hyperkeratosis, melanisation of the skin (tanning) occurs as an adaptive response to UV exposure. UV light up-regulates production of melanin in the epidermis of the skin. This UV-mediated skin darkening is a physiological tanning response which protects the skin against subsequent UV damage. Defects in this pathway are linked with increased cancer susceptibility.

Melanisation occurs in two phases. In the initial phase, skin darkening occurs as a result of redistribution of and/or molecular changes to existing epidermal melanin. Delayed skin darkening is due to increased melanin synthesis and transfer of melanin to keratinocytes. This type of tanning begins several hours to days after UV exposure.

DNA damage in keratinocytes up-regulates transcription of the pro-opiomelanocortin gene, which encodes production of melanocyte stimulating hormone (α-MSH). α-MSH binds to melanocortin-1 receptors (MC1R) on melanocytes in the basal layer of the epidermis. MC1R signaling protects the skin from UV damage in 2 ways. It generates cAMP, which increases tyrosinase and other melanogenic enzymes. This enhances production of melanin in the epidermis. Epidermal melanin functions to inhibit UV penetration into the skin by absorbing UV radiation, thus acting as a 'natural sunscreen'. This decreases mutagenesis and skin cancer risk. UV-induced pigmentation via MC1R signaling also directly influences UV resistance of melanocytes by enhancing DNA repair and oxidative resistance.

This system allows pharmacologic manipulation of cutaneous cAMP to reduce UV sensitivity and cancer risk. Raising cAMP levels in the skin can be accomplished either by stimulating its production or by inhibiting its degradation. Alternatively, α -MSH or MC1R peptide ligand agonists can be used to target melanocytes but might be less effective in individuals with inherited MC1R signaling defects.

Signaling defects in the MC1R may be found in fair-skinned individuals and individuals with 'red hair colour' alleles. These individuals accumulate more mutations from UV exposure due to less efficient DNA repair in melanocytes. This inefficient repair of DNA can lead to malignancy. People with red hair colour alleles have up to a four-fold increased lifetime risk of melanoma and other skin cancers.

Of the two chemical forms of melanin, eumelanin is far more efficient at blocking UV photons than phaeomelanin. Eumelanin is a dark pigment expressed in more pigmented individuals. Phaeomelanin is a light-coloured pigment that results from incorporation of cysteines into melanin precursors. Phaeomelanin levels are similar in both dark- and light-skinned individuals. It is the amount of epidermal eumelanin that determines skin complexion, UV sensitivity and cancer risk. Skin containing more eumelanin will allow less UV radiation to penetrate its epidermis and will suffer fewer damaging effects of UV exposure including skin malignancy.

The Fitzpatrick Scale is a semi-quantitative scale made up of six phototypes that describe skin color by basal complexion, melanin level, inflammatory response to UV radiation and cancer risk. Minimal erythematous dose (MED) is defined as the lowest dose of UVB radiation that causes inflammation in the form of redness and oedema in the skin (sunburn) 24–48 hours post exposure. Fair-skinned individuals require less UVB exposure to cause sunburn and are, therefore, more UV sensitive. These individuals have a lower MED and a higher risk for melanoma and other types of skin cancer.

There has been a significant increase in recreational UV exposure as the result of outdoor leisure activities as well as tanning for cosmetic purposes. The American Academy of Dermatology opposes the use of tanning devices and aims to ban the sale of such devices in the USA. The World Health Organisation (WHO) has classified UV radiation from tanning devices as carcinogenic to humans. The UV output from indoor

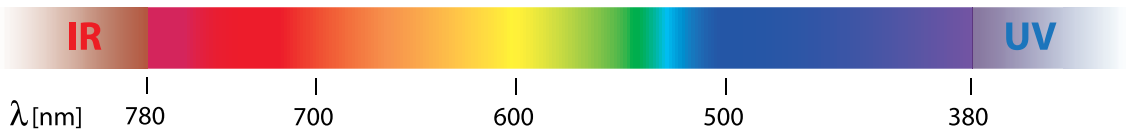
tanning machines can be up to ten times more powerful than sunlight. A review of seven studies found a 75% increase in the risk of melanoma in those who had been exposed to UV radiation from indoor tanning before the age of 35.

UV radiation is the most important modifiable risk factor for skin cancer, which is the most common malignancy in humans. UV exposure has been linked to basal cell carcinoma, squamous cell carcinoma and malignant melanoma. Genetic factors also influence risk of UV-mediated skin disease. The incidence of skin cancer increases with age. This reflects a latency between carcinogen exposure and cancer formation.

Malignant melanoma of the skin is the deadliest form of skin cancer. This skin cancer usually arises from epidermal melanocytes and has a tendancy towards metastasis. Melanoma is notoriously difficult to treat once it has spread beyond its original site and, although its incidence is low, it accounts for approximately three quarters of all deaths from skin cancer. The incidence of melanoma is highest in areas with large numbers of fair-skinned individuals living in warm, sunny climates. Non-melanoma skin cancers have a much higher incidence than melanoma. The two major forms are basal cell carcinomas and squamous cell carcinomas, which are both derived from epidermal cells. The majority of these keratinocyte malignancies develop in areas of skin most exposed to UV, such as on the face and arms. They are much less deadly than malignant melanoma. Most are effectively treated by local measures alone such as resection, MOHS microsurgery or cryosurgery and have superior long-term prognosis. Decreasing UV radiation exposure, both naturally from sunlight and artificially from tanning bed use, may be the single best way to reduce the incidence of melanoma and other skin cancers.

Excessive UV exposure can also result in premature skin aging - photoaging. Signs of photoaging include fine wrinkles around the eyes, mouth and forehead, pigmented spots referred to as solar lentigines, leathery skin texture and red, rough, scaly patches called actinic keratoses. Actinic keratosis is considered to be a pre-cancerous lesion which can progress to squamous cell carcinoma.

Photodermatoses are a group of skin conditions caused by an abnormal reaction to the sunlight, usually UVA. Primary photodermatoses include polymorphic light eruption, solar urticaria, hydroa vacciniforme, actinic prurigo and chronic actinic dermatitis. Secondary photodermatoses are usually a feature of systemic disease such as SLE, porphyrias and Xeroderma pigmentosum. Xeroderma Pigmentosum is a rare genetic disorder in which affected individuals are unable to repair DNA damage caused by UV light. Patients display extreme UV sensitivity with severe sunburn occuring in response to small doses of UV radiation. Malignant skin lesions develop frequently and much earlier in life than in unaffected persons.




| WAVELENGTH | | |
|---|---|---|
| **UVC** | **100 - 290 nm** | **Does not reach the surface of the earth it is absorbed by the ozone layer** |
| **UVB** | **290 - 320 nm** | **Sunburn** |
| **UVA 1** | **320 - 340 nm** | **Skin cancer<br>Wrinkles<br>Pigmentation** |
| **UVA 2** | **340 - 400 nm** | |
| **Visible light** | **400 - 700 nm** | **Worsens pigmentary disorders** |
| **Infrared radiation** | **700 - 300 nm** | **Heat pain<br>Heat Urticaria<br>Cardiovascular collapse** |

Individuals with conditions affecting melanin synthesis or melanocytes may be particularly susceptible to the harmful effects of UV radiation. Albinism is characterised by a congenital, inherited abnormality of melanin synthesis. Individuals have either reduced or absent melanin in the skin, hair and eyes. As melanin is photoprotective and absorbs UV light, an absence predisposes to UV damage including skin cancers.

Vitiligo is an autoimmune condition where absent melanocytes cause depigmentation of the skin. Although patients with vitiligo are not at increased risk for skin malignancies, they are likely to develop sunburn in areas of depigmentation. It is essential for individuals with these skin conditions to limit exposure to sunlight, wear protective clothing and use a broad spectrum sunscreen.

## SUNSCREENS

Topical sunscreens provide high protection against the UV portion of solar radiation. The ingredients in sunscreens are either chemical absorbers or physical blockers of UV radiation. Chemical absorbers may absorb either UVA or UVB or both, preventing absorption by the skin. Physical blockers reflect or scatter UV radiation away from the skin.

Zinc dioxide and titanium dioxide both absorb UV radiation and reflect photons in the visible light range. As they are photostable and do not degrade when exposed to sunlight, they are often combined with less photostable chemical absorbing sunscreens. Newer sunscreens have reduced the white appearance of these metal oxides when applied to the skin and improved their efficacy as UV absorbers.

Worldwide, sunscreens are rated for their sun protection factor. SPF is mainly a measure of a sunscreens ability to shield against UVB rays. SPF is a ratio of the dose of UV radiation over time required to cause a barely detectable sunburn on a person treated with sunscreen compared with the dose required to cause sunburn in untreated skin. If it takes 10 minutes to burn without a sunscreen and 300 minutes to burn with the sunscreen, then the SPF would be 30. The ratio assumes application of an amount of 2mg / cm2.

There is no internationally agreed upon method of measuring UVA protection, however, in vitro, persistent pigment darkening (PPD) may be used to determine the UVA protection factor. Critical wavelength testing may also be used.

**Low protection SPF <15**
**Medium protection SPF 15 - <30**
**High protection 30 - <60**
**Very high protection is >=60**

Broad spectrum sunscreens refer to protection against UVA and UVB rays, where the UVA protection factor is at least 1/3 of the labelled SPF. These sunscreens should protect against a minimum critical wavelength of 370nm. Although sunscreens may be water resistant, they can be rubbed off the skin surface, for example, with a towel after swimming. It is essential to apply sunscreen 30 minutes before outdoor activities and at least every two hours thereafter, especially following swimming or drying with a towel.

## SUN EXPOSURE SAFETY TIPS

- Minimise UV exposure by limiting time outdoors between 10 am and 4 pm.
- Use sunscreens with a sun protection factor (SPF) >15.
- Sunscreen should be applied repeatedly especially when sweating or swimming
- Use sunblocks that offer protection from both UV-A and UV-B rays
- Apply sunscreen to lips, ears, around eyes, neck, scalp, hands and feet
- Wear closely woven clothing that covers the arms and legs
- Clothing with an UV protection factor greater than 40 blocks UV radiation better than sunscreens
- Wear a broad brimmed hat to protect the head, face, ears and neck. If a baseball cap is worn, ensure sunscreen is applied on the ears and neck.
- Wear UV-protective sunglasses to protect the eyes
- Stay in the shade or carry an umbrella as shade can reduce UV exposure by 50%
- Reduce artificial tanning
- Sunless, topical self-tanning products are a safer option but offer little sun-blocking UV protection. Use an additional sunscreen.
- Avoid getting a sunburn. More than five sunburns doubles your risk of skin cancer.

## HEALTH AWARENESS TIPS

- Self skin examination should be done from head to toe at least once a month. A health care professional should examine your skin annually.
- Changes in moles (growth, irregularity, asymmetry, colour changes, elevation, pain, itching) should be reported to a healthcare professional. Skin cancers are much more easily treated when caught early.
- Some sun exposure is essential for vitamin D production. This is also needed for calcium metabolism and bone health. Twenty to thirty minutes of UVB exposure daily is required for vitamin D3 production in the skin. Regular use of sunscreen does not contribute to vitamin D deficiency. MC

*References available on request.*

Answer online at **www.medicalacademic.co.za.** Go to page 62 for step-by-step instructions

# Sun protection

3 CPD POINTS

**1** **Sunlight consists of**
A. Visible light only
B. UV and visible light
C. UV and Infrared
D. Visible light, UV light and Infrared

**2** **What proportion of solar radiation is made up of visible light?**
A. 30%
B. 45 %
C. 95%
D. 60%

**3** **The wavelength of UV radiation is**
A. 290 – 400 nm
B. 100 – 290 nm
C. 400 – 700 nm
D. 700 – 800 nm

**4** **UVB intensity is affected by**
A. Altitude
B. Time of day
C. Distance from the equator
D. All of the above

**5** **Choose the correct statement**
A. Tanning is a photoadaptive response to UV exposure due to increased melanin biosynthesis
B. Tanning in a sunbed has more health benefits than risks
C. MC1R receptors are found on dermal cells
D. MC1R signaling defects reduce risk of skin malignancies

**6** **MC1R signaling protects skin from UV damage by**
A. Producing keratin in the epidermis which absorbs UV radiation
B. Producing pigmentation which inhibits DNA repair
C. Producing melanin in the epidermis which absorbs UV radiation and enhancing DNA repair following UV-induced DNA damage
D. Producing pigmentation which causes oxidative stress

**7** **Eumelanin is**
A. less efficient at blocking UV radiation than phaeomelanin
B. A light pigment expressed in fair skinned persons only
C. A dark pigment which determines UV sensitivity
D. The only type of pigment found in the skin

**8** **The most important modifiable risk factor for skin cancer is**
A. Melanin concentration in the skin
B. High Vitamin D level
C. Excessive UV radiation exposure
D. Diet

**9** **Malignant melanoma**
A. Has the highest incidence of any cancer in the world
B. Is unlikely to metastasize
C. Is not related to solar radiation
D. is more common in fair-skinned individuals

**10** **A sunscreen's SPF is a measure of the sunscreens ability to shield against**
A. UVA rays
B. UVB rays
C. Infrared
D. Visible light

**11** **Titanium dioxide**
A. Is photounstable
B. Is a chemical UV absorber
C. Improves UV penetration into the skin
D. Should never be combined with less photostable chemicals

**12** **Application of sunscreen**
A. Impairs vitamin D synthesis in the skin
B. Should be performed 12 hourly
C. Should be done 30 minutes before outdoor activity and 2 hourly thereafter
D. Is not necessary from 10am to 4pm

**13** **Photodermatoses**
A. Occur due to an abnormal reaction with sunlight
B. Include vitiligo and albinism
C. Include Atopic dermatitis and Psoriasis
D. Are never associated with systemic disease

**14** **Basal cell carcinoma**
A. has a lower incidence than melanoma
B. Is more common in sun-exposed areas of the body
C. Is the deadliest form of skin cancer
D. All of the above.

**15** **Solar lentigo**
A. Is a premalignant skin condition
B. Is a sign of photo-aging
C. Can progress to squamous cell carcinoma
D. Is a secondary photodermatosis

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