THE ADVERSE EFFECTS OF THE SUN ON THE SKIN - A REVIEW

L. Scerri, M. Keefe

ABSTRACT

Solar radiation causes a variety of biologic effects in the skin which are predominantly harmful. The only recognised beneficial effect is that of endogenous photosynthesis of vitamin D from its precursors in the skin, the importance of which is greatly diminished by a well balanced diet. Acute excessive exposure to ultraviolet radiation (UVR) causes sunburn, whereas chronic overexposure is responsible for the process of photoageing and skin cancer. This sun related degenerative process and neoplastic transformation may be effectively minimised by an increased public awareness of the harmful effect of the sun.

Keywords: sun, ultraviolet, photoageing, skin cancer, melanoma

Introduction

The sun emits electromagnetic radiation over a very broad spectrum which is more or less arbitrarily divided into different wavebands (Table I). The shorter wavelengths are more susceptible to atmospheric absorption and scatter. In fact, shorter wave UVC is virtually all absorbed by the ozone layer in the stratosphere, such that it does not reach the surface of the earth. This is of great benefit to the human race as the shorter UV wavelengths are the most harmful. The amount of longer wave UVA reaching the earth exceeds that of shorter wave UVB by 10 to 100 times. Past accounts have focused on UVB as the UVB in humans, and UVB tumorigenesis in animals. It has been possible to induce elastosis in hairless mice with 3000 J/cm² from a UVA lamp, which closely approximates the UVA intensity of terrestrial sunlight. As with erythema, it requires a thousand fold more UVA to produce elastosis compared to UVB.

Although clouds and air particles both contribute to atmospheric scatter and absorption of UVR, the most important filtering medium is the ozone layer. There is currently serious concern about its depletion by man made chlorofluorocarbons (CFC's). The American Environment Protection Agency, in 1989 predicted that without controls...

Table I - Portion of solar electromagnetic spectrum of concern to dermatologists

<table>
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<tr>
<th>Ultraviolet light</th>
<th>Visible light</th>
<th>Infrared light</th>
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<tbody>
<tr>
<td>UVC 200-290nm</td>
<td>UVB 290-320nm</td>
<td>UVA 320-400nm</td>
</tr>
<tr>
<td>400-700nm</td>
<td>0.7-1000μm</td>
<td></td>
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</tbody>
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in CFC production, a 40% depletion of ozone would occur by the year 2075\textsuperscript{1}. The agency further emphasised that for every 1% decrease in ozone, there will be a compounded 2% increase in the more damaging shorter UVB wavelengths reaching the earth's surface. This is predicted to result in an additional 1 to 3% increase per year in non-melanoma skin cancer. With a general consensus, most of the world's nations agreed to phase out the use of CFC's by the end of the century.

**Skin Ageing**

The deterioration in appearance and texture of the human skin with increasing age is a "fact of life" which is generally regarded as inevitable. The process underlying this deterioration is, however, caused not by time alone, but also by external elements, particularly chronic exposure to solar UVR. Therefore, we currently recognise two forms of skin ageing:

a) **Intrinsic ageing**

Chronological ageing, otherwise known as intrinsic ageing, refers to the changes that occur naturally in the skin with the passage of time. This process is generally not preventible. It is characterised by a gradual decline in overall skin function (Table II) which results from progressive atrophy throughout the three layers of the skin\textsuperscript{8,9}. The outward skin changes related to intrinsic ageing are:

- Thinning
- Dryness
- Pallor
- Laxity
- Hair depigmentation
- New growth formation:
  - benign
    - seborrheic keratosis
    - fibroepitheliomas (skin tags)
    - cherry angiomas
    (Campbel de Morgan spots)
  - sebaceous hyperplasia
  - malignant
    - basal cell carcinoma *
    - squamous cell carcinoma *

* far more common in sun damaged skin

b) **Photoageing**

For a long time photoageing was regarded as being simply an accelerated form of intrinsic ageing. However, it has become increasingly recognised that intrinsic skin ageing and photoageing have strikingly different morphological and histological characteristics\textsuperscript{10}. One may readily appreciate the difference by, for example, comparing forearm skin and buttock skin in an elderly farmer. Unlike intrinsic ageing, photoageing is preventible and only partially reversible. If sun damaged skin is protected from the sun, natural repair processes can operate and it has been demonstrated that topical retinoin can enhance this repair process\textsuperscript{11}. The histological picture of photoaged skin is one of hyperactive, chronically inflamed skin with disordered proliferation, turnover and differentiation of cells. Its most striking feature is the massive hypertrophy and disorganisation of elastic tissue\textsuperscript{10}. The external signs produced by these changes are:

- Thickening and coarseness
- Exaggerated fine and coarse wrinkling
- Elastosis manifested as pebbly, yellowish change with loss of elasticity
- Solar comedones
- Pigmentary mottling
- Telangiectasia
- Purpura
- Solar keratoses - although these lesions demonstrate dysplastic changes in the basal epidermal layer, the risk of malignant transformation is less than 0.1%.
- Exaggerated risk of developing skin cancer

**Table II** - Physiological skin functions which decline with intrinsic ageing

- Decreased barrier function against physical, chemical and microbial attack
- Decreased tissue regeneration
- Decreased immune responsiveness
- Decreased sweat and sebum production
- Decreased melanin production
- Impaired thermoregulation
- Decreased sensory perception

**Photocarcinogenesis**

Skin cancer is the most common human malignancy\textsuperscript{13}. There is considerable epidemiological and experimental evidence to incriminate UVR as the most important cause of epidermal malignancy\textsuperscript{14,15}. Both squamous cell carcinoma and basal cell carcinoma (grouped as non-melanoma skin cancer) are much commoner in light-exposed parts of the body\textsuperscript{16}. Non-melanoma skin cancer is strongly linked, in a dose dependant fashion, with prolonged
cumulative exposure to UVR, and in fact it tends to develop late in life. Lentigo maligna appears to have a similar causal relationship with UVR.

UVR also predisposes to the far more serious malignant melanoma. The causal relationship here is somewhat different. In fact, malignant melanoma commonly arises on the covered skin, and tends to present any time after puberty, although rare cases of prepubertal melanoma have also been reported. The 'typical' patient with superficial spreading or nodular melanoma is economically well off, has an indoor office-based occupation for forty-eight weeks of the year, but is an avid sun-bather for the holiday periods of the year. In other words, the intermittent exposure of pale, normally protected skin to intense burning sunlight appears to be an important factor. There is also evidence that over-exposure to the sun in early childhood constitutes another important risk factor for the development of melanoma. It has been shown that the lifetime risk to develop cutaneous malignant melanoma in the white population in the USA is 0.7%, while the corresponding figure for the black population is 0.07%. This rising incidence is thought to reflect an increase in sun exposure, due to changes in life-style in recent times.

Factors Influencing Susceptibility to Adverse Effects of UVR

a) Quality and quantity of solar radiation
This varies according to season, geographic latitude, altitude above sea level and time of day. These factors affect the thickness of the atmospheric layer which solar radiation has to penetrate, which in turn has a bearing on both the quantity of total UVR as well as the ratio of UVA to UVB reaching the earth's surface. UV filtering is least when the sun is closest to the earth, that is in summer, at higher altitudes, in equatorial regions and around midday.

b) Skin type
The skin type is dictated by the degree of melanisation of the skin (Table III). Melanin effectively absorbs UVR and hence protects the skin against both acute and chronic adverse effects of the sun:

   i) Pigmentation is inversely related to the susceptibility to sunburn. This is painfully obvious to the over-enthusiastic British tourist who makes a quick dash south for a sunny holiday. The discomfort, redness and swelling of the skin starts within a few hours, followed by days of blistering and peeling that can ruin a holiday.

   ii) Fairer individuals with type 1 and 2 skin, are highly susceptible to photoageing, the signs of which may sometimes be evident even in their twenties. Features of photoageing tend to appear later and are usually less severe in darker-skinned individuals.

   iii) The degree of melanisation is inversely related to the incidence of skin cancer. This applies to both melanoma and non-melanoma skin cancer. It has been shown that the lifetime risk to develop cutaneous malignant melanoma in the white population in USA is 0.7%, while the corresponding figure for the black population is 0.07%.

Table III - Classification of skin type

<table>
<thead>
<tr>
<th>Type</th>
<th>Description</th>
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<tbody>
<tr>
<td>1</td>
<td>Never tans, always burns</td>
</tr>
<tr>
<td>2</td>
<td>Tans with difficulty, burns easily</td>
</tr>
<tr>
<td>3</td>
<td>Tans easily, burns rarely</td>
</tr>
<tr>
<td>4</td>
<td>Always tans, never burns</td>
</tr>
<tr>
<td>5</td>
<td>Genetically brown skin (Asian)</td>
</tr>
<tr>
<td>6</td>
<td>Genetically black skin (Negroid)</td>
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c) Genetic factors
The risk of induction of skin cancer and photodamage by UVR is greatly increased in patients with the rare genodermatoses, Xeroderma Pigmentosum, as a result of an impaired DNA repair mechanism. The Atypical Mole Syndrome, previously known as Dysplastic Naevus Syndrome is a recognised phenotype which predisposes to melanoma. It may be familial or non-familial. Large numbers of 'normal' pigmented naevi confer a 20 to 30-fold increased risk of developing melanoma. There is a further 4 to 10-fold increased risk in the presence of at least 2 clinically atypical naevi, and a 100 to 400-fold increased risk, if a family history of melanoma is present.

d) Occupation and behaviour
Outdoor workers in sunny climates such as farmers, fishermen and builders are all subject to almost constant sun exposure throughout the day for many decades of their working lives. This renders them very susceptible to developing skin cancer and greater degrees of photoageing. Individuals indulging in recreational activities such as outdoor summer sports, habitual sunbathing, skiing and mountain climbing...
constitute another important risk group.

As if solar radiation is not enough, we are seeing the mounting popularity of habitual exposure to artificial UVR in the form of sunbeds and tanning parlours. The tanning industry is rapidly growing in many developed countries. In USA more than one million people use commercial tanning facilities every day, the biggest users being adolescent and young adult females. Most tanning parlours falsely claim that their sunbeds emit only UVA which does not cause sunburn in the short term, nor skin ageing or skin cancer in the long term. They also claim that the tan they produce protects against sunburn, premature ageing and skin cancer when the same individual is later exposed to natural sunlight. It is however, known that UVA-induced tanning is much less protective against harmful UVB rays than UVB-induced tanning. Some UVA lamps generate over five times more UVA per unit time than does solar UVA radiation reaching the equator. At these doses, "pure UVA" is likely to have adverse biologic effects. With improper maintenance, a UVA source tends to emit varying degrees of UVB. Even 1% UVB emission from a UVA source can cause a significant increase in the risk of skin cancer.

Preventive Strategies

An effective way of promoting uniform sun protection is through a well targeted educational programme. The American Skin Cancer Foundation has issued a useful set of guidelines on sun protection, which are summarised in Table IV. Two out of every three people spending their lives in Australia develop a skin cancer. This stimulated the Australian authorities to set up the Slip Slap Slop Campaign and the Sun Smart Programme in the late eighties. The salient features of these campaigns were:

1. Education and advice through the media, circulated leaflets and posters in public places
2. Modification of uniforms of school children and outdoor workers
3. Environmental adaptation to improve the provision of shade in public places by, for example, introducing more shade-producing trees in parks and gardens
4. Reducing cost of sunscreens
5. Individuals intending to make use of sunbeds must first sign a declaration form outlining the dangers of UVR.

Besides having a preventive role, public education campaigns may also be useful for promoting early detection of skin cancer. Following the work of Doherty and Mackie in Glasgow, which demonstrated the need for public education in the recognition of melanoma at an earlier stage, seven Cancer Research Campaign-funded centres were designated in the UK, in

Table IV - Sun protection guidelines by the American Skin Cancer Foundation

| 1. Minimise sun exposure between 10.00 am and 2.00 pm when the sun is strongest |
| 2. Wear a hat, long-sleeved shirt and long pants when out in the sun. Choose tightly woven materials for greater protection |
| 3. Apply a sunscreen before every exposure to the sun. The sunscreen should be reapplied every two hours, and especially after swimming or perspiring heavily, since products differ in their degree of water resistance. A sunscreen with an SPF of 15 or more, and which protects against both UVB and UVA is recommended |
| 4. Use a sunscreen during high altitude activities such as mountain climbing and skiing |
| 5. Do not forget to use a sunscreen on overcast days. The sun's rays are as damaging to the skin as they are on sunny days |
| 6. Individuals at high risk of skin cancer (outdoor workers, fair-skinned individuals and persons who already had skin cancer) should apply sunscreens daily |
| 7. Beware of reflective surfaces such as sea, sand, snow and concrete! Sitting in the shade does not guarantee you protection from sunburn |
| 8. Avoid tanning parlours |
| 9. Keep infants out of the sun. Begin using sunscreens on children at 6 months of age, and then allow sun exposure with moderation |
order to further assess this issue. Southampton was one such centre (Fig. I). Pigmented lesion clinics were set up concurrently in order to cater for the increased workload resulting from the educational campaign. Most centres reported picking up an increased proportion of thin melanomas as opposed to thick melanomas in response to heightened public and professional awareness.\textsuperscript{30,31,32,33}

Unfortunately, the concept that 'brown is beautiful' is still very prevalent in the western world, and the sunbed industry is still flourishing. However, now that the negative effects of the sun on the skin are clearer than ever before, one hopes that continuing educational programmes will eventually raise public awareness on a universal basis, to a level that will significantly minimise this unnecessary insult to our skin.

References:


20. Holman CDJ, Armstrong BK, Heenan PJ, et al. The causes of malignant melanoma: results from the


