Journal of Pakistan Association of Dermatologists 2007; **17**: 46-51.

Review Article

Solar radiation: cutaneous hazards and their prevention

Tauseef Kamran, Saghir Asghar Khan

Department of Dermatology, Fatima Jinnah Medical college/Sir Ganga Ram Hospital, Lahore.

Abstract The solar radiation consists of three types of radiation, visible, infrared and ultraviolet radiation (UVR). There is daily and seasonal variation in the prevalence of ultraviolet radiation. The depth of penetration of solar radiation is directly proportional to its wavelength. Its effects on the skin are UV-induced erythema, UV-induced pigmentation and cellular responses, which include damage to DNA, epidermal hyperplasia and thickening of stratum corneum and cutaneous immunosuppression responsible for various cutaneous cancers in humans. It can also lead to various photo-induced and photo-aggravated dermatoses. Avoiding mid day exposure (between 11-3) to sunlight, use of appropriate clothing including hats, and regular use of sunscreen having SPF of 30 or more, covering both UVA and UVB, may be helpful in preventing hazards of solar radiation.

Key words
Solar radiation, ultraviolet radiation

Introduction

Sunlight consists of three types of radiation,[1] one of which is visible radiation, in the spectral rage of 400-700nm (violet through red). It causes stimulation of the retina and has little biological activity. Beyond 700nm is infrared radiation, which is radiant heat. Below 400nm is ultraviolet radiation.

Ultraviolet radiation (UVR)

It can be divided into following types,[2] as shown in **Figure 1**.

(a) *Vacuum ultraviolet radiation*
It is the shortest wavelength of ultraviolet radiation in the range of 100-200 nm. Its photons are absorbed by air and therefore can be used experimentally only in a vacuum. These are of no interest in medical photobiology.

(b) *UVC radiation*
It is also known as short wave UV radiation in the range of 200-280 nm. It is not found on the surface of the earth. It is filtered out by ozone layer and water vapours in the atmosphere. It is also known as "germicidal radiation" since it is used to kill microorganisms.

(c) *UVB radiation*
Also known as middle wave UV radiation in the wavelength of 280-320nm. It is the most biologically active waveband of UV radiation in sunlight. It is responsible for erythemal reaction following exposure to the sun, also termed as "sunburn UV radiation".

Address for correspondence
Dr. Tauseef Kamran,
H#12, St#19,
Dil Muhammad Road, Lahore
E mail: dr_t_k@yahoo.com

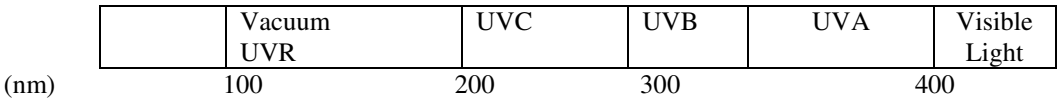

Figure 1 Ultraviolet radiation spectrum.

(d) *UVA radiation*
Also known as long wave UV radiation, in the wavelength of 320 to 400 nm. It is less biologically active than UVB and partially responsible for sun-induced erythema. It is further subdivided into UVAI or far UVA in the range of 340-400nm and UVAII or near UVA in the range of 320-340nm.

Daily and seasonal variation of UVR

The emission spectrum of UVR, measured at the earth surface, varies considerably with the time of the day and season of the year. Short wavelengths are more prone to scatter than longer wavelengths. In early morning and late afternoon sunlight contains mainly UVA. Between 11.00 am to 3.00 pm, UVB is the main constituent because at this time sun is high and radiation passes vertically. Thus in temperate climates, early morning and late afternoon sunlight contains very little UVB and it is not very erythemogenic or melanogenic, while the ratio of UVA and UVB is high.

Clouds greatly diminish the amount of UVB radiation but there is fairly small reduction in the amount of UVA radiation. Therefore as much of 80% UVR may still penetrate the cloud cover.

Penetration of solar radiation into skin

When solar radiation strikes the skin, part is reflected back from the surface or deeper layers, part is absorbed in various layers, and part is inwardly transmitted to successive layers of the cells until the energy of the incident beam has been dissipated. The depth of penetration of radiation is directly proportional to its wavelength, as shown in **Figure 2**.

Cutaneous effects of UVR

a. ***UV-induced erythema***
Erythema is a reflection of the vascular response to UV radiation.[3] Immediate erythema is a faint reddening of the skin beginning shortly after the start of the exposure and fading within 30 minutes. Delayed erythema appears after a latency period of 2-6 hours, peaks at 12 to 16 hours and subsides over the next few days. It is also known as sunburn erythema. An increase in vascular permeability accompanies both phases and is seen as edema or blistering following large exposure. These changes are largely caused by UVB radiation. The initial phase involves the release of prostaglandins and histamine, leukotriene b4 and later phase involves polymorphs.[5]

b. ***UV-induced pigmentation***
The melanocytic response to UV radiation is also biphasic. Immediate pigment darkening is seen mainly with UVA radiation. It occurs within minutes of exposure and fades within an hour. It involves movement of melanosome in to the keratinocytes out of melanocytes and photochemical alteration of melanin.

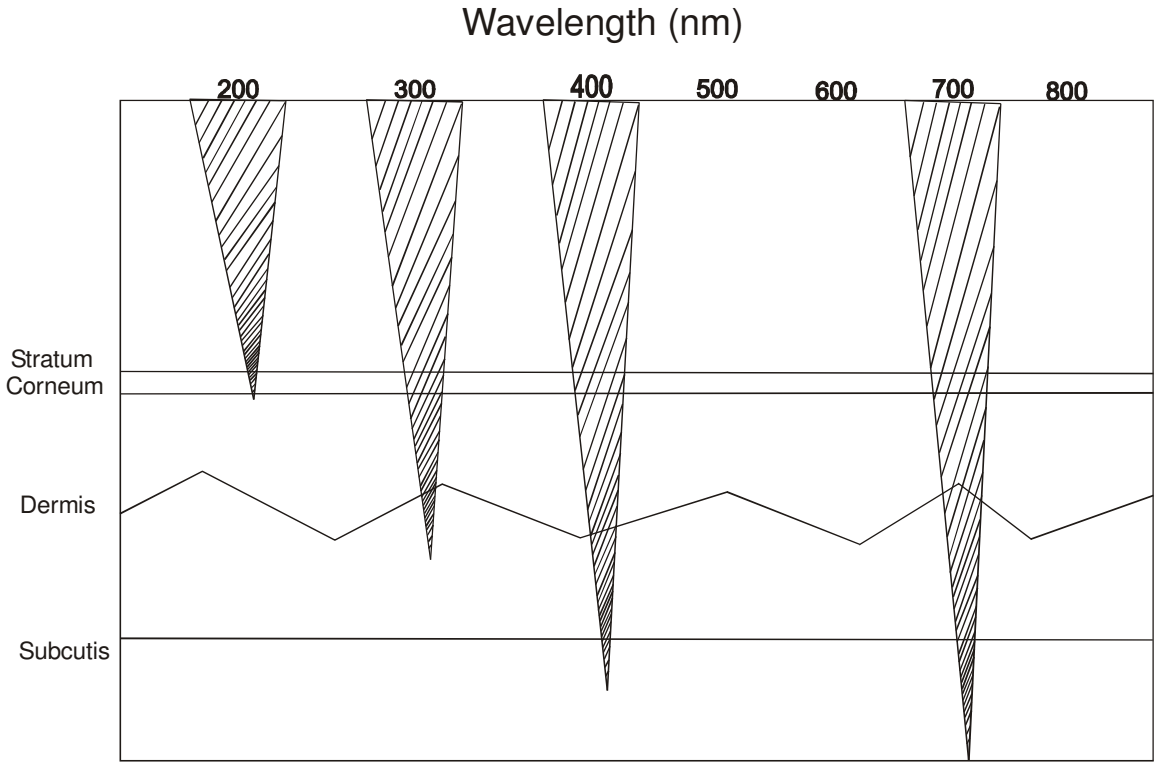

Figure 2 Wavelengths penetration into skin.

Delayed pigmentation also known as suntan, appears within days of exposure and lasts weeks or months and involves production of new melanin, caused mainly by UVB.[6] Melanogenesis is wavelength dependent. UVA radiation produces dark pigmentation limited to basal layer while UVB radiation produces a lighter pigmentation and is distributed throughout the epidermis. UVC produces little or no pigmentation.

c. ***Cellular response to radiation***

(i) *Effect on DNA*

DNA is the most important target for radiation-induced damage, which inhibits cell division and if extensive, leads to cell death.[7] UV-induced damage to RNA and protein may also be important but it is poorly understood.

(ii) *Effect on epidermal cell kinetics*

UV exposure causes epidermal hyperplasia and associated thickening of stratum corneum, is a major defense against further UV damage. Epidermal hyperplasia varies with wavelength. It is more marked after exposure to UVC, less marked after exposure to UVB, and minimal after exposure to UVA radiation.

d. *Effect on the immune system*

Skin is an important component of the immune system. Langerhans cells in the epidermis are of macrophage-monocyte lineage, and function as antigen presenting cells. Furthermore lymphocytes are normally resident in the skin, thus important component of immune system are in the range of exposure to UV radiation. A UV-induced alteration in immune function results in generation of suppressor T lymphocytes, causing immunosuppression.

Table 1 Major groups of light sensitive reactions.

Systemic or metabolic disease
Prophyria
Xerodema pigmentosum
Systemic or topically applied drugs
Sulphonamides
Tetracyclines
Phenothiazines
8-Methoxypsoralen
Oral contraceptives
Cosmetics and toiletry products
Skin diseases aggravated by sun
Discoid lupus erythematosus
Rosacea
Pemphigus
Psoriasis
Atopic eczema
Skin diseases of unknown cause
Polymorphic light eruption
Hutchinson's summer prurigo
Solar urticaria
Persistent light reaction and actinic reticuloid

This may be responsible for the development of skin cancer in humans.[8]

e. ***Effect on vitamin D synthesis***

UVB radiation in moderate doses rapidly converts epidermal 7-dehydrocholesterol in to pre-vitamin D3, which is then isomerized over several days to vitamin D3, and transported by plasma vitamin D-binding protein into the circulation.[9]

f. ***Photoaging of the skin***

Long-term recurrent exposure to sunlight may lead to photoaging of the skin, which is gradual deterioration of cutaneous structure and function. These changes include: cutaneous fine and coarse wrinkling, dryness, coarseness, telangiectasia yellowness, mottled pigmentation, laxity, loss of tensile strength and comedones.[10]

g. ***Photocarcinogenesis***

Chronic UVB and to a lesser extent UVA exposure are responsible for the induction of most non-melanoma skin cancers. UVA may play an important role in development of melanoma.[11] The possible mechanisms are mutation of the p53 tumor suppressor gene and alternation in immune surveillance.[12]

Photo-induced and photoaggravated dermatoses

There are certain cutaneous disorders, which can be induced or aggravated by exposure to solar radiation. A detailed list is given in **Table 1**.[13]

Photoprotection

Photoprotection is helpful in preventing the harmful cutaneous effects of solar radiation, and can be achieved by following ways.

(a) Sun avoidance

It is the most desirable form of photoprotection. Mid-day exposure (between 11 am to 3pm) should be avoided, especially in tropical or subtropical areas,[14] because sun passes vertically at this time and there is less filtration of UV radiation. Window glass absorbs most of the radiation below 320 nm, however considerable amount of UVA radiation still passes through glass.[15] Special plastic films containing UVA shields as an interleaf or overlays are available, in some parts of the world. Shades availability in recreational areas is also desirable.

(b) Clothing

It can be an excellent form of sun protection.[16] The most important determinant is tightness of the weave. Fabric type and thickness is less important than regular weave. Protection drops significantly

when the fabric becomes wet. Color plays a minor role, with dark colors protecting better than light colors. The FDA defines clothing with SPF rating as medical advice.

Hats are the most important articles of clothing. A 4-inch wide circumferential brim is required to cover entire face and neck.

(c). Sunscreen

The regular use of sunscreen has been shown to reduce actinic keratosis, solar elastosis and squamous cell carcinoma.[17] Their effects on preventing melanoma are less clear. Drug photosensitization and photo-induced or photo aggravated dermatoses may be prevented with sunscreen use.[18]

Sunscreen having SPF of 30 or more, covering both UVA and UVB radiation should be used. Adequate application to all exposed skin surfaces including the lips is necessary.[19] It should be applied 15-30 minutes prior to sun exposure to allow sufficient time for a protective film to develop. It should be reapplied after prolonged swimming or vigorous activity. The use of sunglasses that screen out both UVA and UVB should be encouraged to protect periorbital skin and eyes.

Sunscreen alone provides insufficient protection from UVR. They represent only one component of a total programme of photoprotection, which includes sun avoidance and use of appropriate clothings.

Conclusion

Solar radiation has many harmful effects on skin, eye and immune system. These hazards can be prevented only by using total programme of photoprotection, which include, sun avoidance, use of appropriate clothing including hats and regular use of sunscreen having SPF of 30 or more covering both UVA and UVB.

References

1. Arnold HL, Odom RB, James WD. Dermatoses due to physical factors. In: **. *Andrew's Diseases of the Skin, 9th edition*. Philadelphia: W.B. Saunders; 2000. p. 21-49.
2. Warwick L. Morison. Electromagnetic radiation. In: **. *Phototherapy and Photo-chemotherapy of Skin Disease, 2nd edn*. New York: Raven Press; 1991. p. 10-26.
3. Solaer NA. Acute effects of ultraviolet radiation on the skin. *Semin Dermatol* 1990; **9**: 11-5.
4. Rhodes LE, Belgi G, Parslen R *et al*. Ultraviolet B-induced erythema is mediated by nitric oxide and prostaglandin E2 in combination. *J Invest Dermatol* 2001; **117**: 880-5.
5. Hawk JLM, Murkly GM, Holden CA. The presence of neutrophils in human cutaneous ultraviolet-B inflammation. *Br J Dermatol* 1988; **118**: 27-30.
6. Eller MS, Yaar M. Gilchrest BA. DNA damage and melanogenesis. *Nature* 1994; **372**: 413-4.
7. C1ydesdale GJ, Dandie GH, Muller HK. Ultraviolet light induced injury: immunological and inflammatory effect. *Immunol Cell Biol* 2001: **79**: 547-68.
8. Kelly DA, Young AR, McGregor JM *et al*. Sensitivity to sunburn associated with susceptibility to ultraviolet radiation-induced suppression of cutaneous cell-mediated immunity. *J Exp Med* 2000; **191**: 561-6.
9. Holick MF, Maclanghlin JA, Parrish JA *et al*. The photochemistry and photobiology of vitamin D3. In: Regan JD, Parrish JA, eds. *The Science of Photomedicine*. New York: Plenum Press; 1982. p. 195-218.
10. Herschenfeld RE, Gilchrest BA. The cumulative effects of ultraviolet

radiation on the skin; photoaging. In: Hawk JLM, ed. *Photodermatology*. London: Arnold; 1999. p. 69-89.
11. Wang SQ, Setlow R, Berwick M *et al*. Ultraviolet A and melanoma: a review. *J Am Acad Dematol* 2001; **44**: 837-46.
12. Brasha DE, Ziegler A, Jonason T *et al*. Sunlight and sunburn in human skin cancer: p53, apoptosis, and tumor promotion. *J Invest Dermatol Symp Proc* 1996; **1**:136-42
13. Marks R, Dykes P, Motley R. *Clinical Signs and Procedures in Dermatology, 1st edn*. London: Martin Dunitz; 1993. p. 64-70.
14. Moloney FJ, Collins S, Murphy GM. Sunscreens: safety, efficacy and appropriate use. *Am J Clin Dermatol* 2002; **3**: 185-91.
15. Kullavanijaya P, Lim HW. Photoprotection. *J Am Acad Dermatol* 2005; **52**: 937-56;-quiz 959-62.
16. Menter JM, Hatch KL. Clothing as solar radiation protection. *Curr Probl Dermatol* 2003; **31**: 50-63.
17. Naylor MF, Farmer KC. The case for sunscreens. A review of their use in preventing actinic damage and neoplasia. *Arch Dermatol* 1997; **133**: 1146-54.
18. Deleo V. Sunscreen use in photodermatoses. *Dermatol Clin* 2006; **24**: 27-63.
19. Kaidbey KH. The photoprotective potential of the new superpotent Sunscreen. *J Am Acad Dermatol* 1990; **22**: 449-52.

Authors Declaration

Authors are requested to send a letter of undertaking signed by all authors along with the submitted manuscript that:

The material or similar material has not been and will not be submitted to or published in any other publication before its appearance in the *Journal of Pakistan Association of Dermatologists.*

Journal of Pakistan Association of Dermatologists 2007; **17**: 46-51.