FEATURES

Ultraviolet Radiation: Human Exposure and Health Risks

Thomas D. Tenkate, M.App.Sc.

Abstract

This article provides an overview of human exposure to ultraviolet radiation (UVR) and associated health effects, as well as risk estimates for acute and chronic conditions resulting from UVR exposure. For most people, the main source of UVR is the sun. Adverse health effects include photokeratitis, erythema, pterygium, some types of cataracts, basal and squamous cell carcinomas, and malignant melanoma. Human exposure is influenced by the following factors: type of occupation, protective measures employed, types of recreational activities undertaken, and personal behavior. Acute conditions may result within 30 minutes of noontime sun exposure, and the minimum risk estimate for nonmelanoma skin cancer (NMSC) in a person 70 years of age is two to three percent. Risks for NMSC are increased for outdoor workers and those participating in recreational sun exposure, but can be significantly reduced if sunscreen is used during childhood.

Introduction

Ultraviolet radiation (UVR) is one portion of the electromagnetic radiation (EMR) spectrum. EMR consists of oscillating electric and magnetic fields that can be propagated both in free space and in matter (1). The main groupings of the EMR spectrum (in order of increasing wavelength) are as follows:

- cosmic and gamma rays,
- X-rays,
- ultraviolet radiation,
- visible radiation,
- infrared radiation,
- radar, and
- radio frequency.

Ultraviolet, visible, and infrared radiation are collectively known as optical radiation because these wavelengths have effects on the eye. A number of schemes are used to divide the optical radiation section of the EMR spectrum. A frequently used photobiological scheme classifies UVR into three divisions:

1. UVC = 100 to 280 nanometers (nm),
2. UVB = 280 to 315 nm, and
3. UVA = 315 to 400 nm.

The interaction of EMR with matter takes the form of absorption, transmission, reflection, refraction, and diffraction. In most cases, one of these effects will dominate. Each effect is, however, always present to some extent (1). Energy can produce an effect within matter only when it is absorbed. When non-ionizing radiation (such as UVR) is absorbed by a molecule, either it affects the electronic energy levels of the atoms in the molecule, or it changes the rotational, vibrational, and transitional energies of the molecule. In biological systems, this energy transfer produces electron excitation, which can result in dissociation of the molecule, dissipation of the excitation energy in the form of fluorescence or phosphorescence, formation of free radicals (i.e., photochemical injury), and degradation into heat (i.e., thermal injury) (2).

Ultraviolet radiation and other forms of EMR are emitted by many sources and are primarily produced by the following processes:

- incandescence,
- electrical/gaseous discharge (such as in arc welding), and
- lasers (3).

The major source of UVR at the earth's surface is the sun, which is an example of an incandescent source. The wavelengths and relative intensities of solar radiation reaching the surface of the earth are affected by a num-

FIGURE 1

Absorption of Ultraviolet Radiation by Media of the Eye*

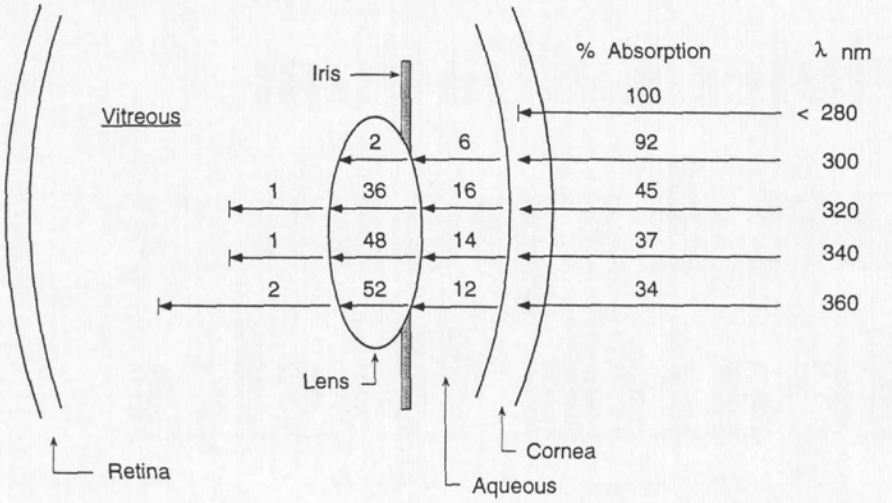


*Figure adapted from Matelsky (2). Data are from Boettner and Wolter (8).

ber of factors, including absorption, scattering, and reflection. Ozone, which is found in the stratosphere, has a peak concentration between an altitude of 20 and 30 kilometers (km). Its absorption band is centered on 250 nm and extends to 350 nm. Ozone thus effectively eliminates all UVC radiation and about half of the UVB radiation from reaching the earth's surface (4). Other meteorological factors that contribute to the attenuation of UVR include the presence of cloud cover, air pollution, haze, and scattered clouds (5).

The aim of this article is to provide an overview of human exposure to UVR and the associated health effects, as well as to present risk estimates for acute and chronic conditions that may result from UVR exposure. The substantial reduction in health risk that can be achieved through preventive actions will also be demonstrated.

Health Effects

Because of the non-ionizing nature of UVR, its interaction with animals—humans in particular—is limited to the skin and eyes. The type and extent of the damage that radiation does to the eye depends on the energy absorbed, the wavelength of radiation, and the duration of exposure (6). When exposed to optical radiation, the various media of the eye act as a series of filters, each component absorbing certain wavelengths to varying degrees (7). A schematic representation of the UVR absorption characteristics of the human eye is provided in Figure 1.

The complex structure of the skin and the presence of structures such as hair follicles, sweat glands, and sebaceous glands make it difficult to determine the exact path that optical radiation travels within the tissue. The presence of optically absorbing molecules (pigments) also affects the penetration of different wavelengths in the skin. For wavelengths less than 300 nm, epidermal thickness, aromatic amino acids, nucleic acids, urocanic acid, and melanin are the major factors that influence skin penetration and absorption. The relative importance of each of these factors depends on wavelength and varies between skin sites and individuals. For wavelengths that range from about 350 to 1,200 nm, melanin is the major absorber of radiation in the epidermis, especially at the shorter wavelengths.

At a cellular level, UVR exposure results in photochemical modification of the genetic material (DNA). Most of this damage is accurately and efficiently repaired by the cell. However, if the amount of damage is too great, some of the alterations to the DNA remain as permanent mutations. These mutations are thought to be one step in the mechanism of cancer formation. DNA absorption of UVR is dictated by the component nucleic acids of the DNA. A peak occurs around 260 nm (in the UVC range). Absorption drops sharply in the UVB range, and absorption is undetectable at wavelengths longer than 320 nm (i.e., in the UVA range) (5).

Eye conditions in which UVR has been implicated as a primary or contributing cause and the affected media are as follows:

- nodular band keratopathies, pinguecula, pterygium, photokeratitis, and epidermal carcinoma (cornea);
- photoconjunctivitis (conjunctiva);
- cataracts (lens); and
- solar photoretinitis, cystoid macular edema, and age-related macular degeneration (retina) (7).

The skin's acute response to UVR exposure is, in general, a reparative and protective reaction (6). UVR exposure has only two beneficial effects on the skin: synthesis of vitamin D3 and maintenance of the ability of the skin to sustain repeated UVR exposures (i.e., skin thickening). The acute reactions of the skin to UVR exposure are erythema, sunburn, tanning, and photosensitization.

In its mildest form, sunburn consists of a reddening of the skin (erythema) that appears up to eight hours after exposure to UVR and gradually fades within a few days. In its most severe form, it results in inflammation, blistering, and peeling of the skin. The main factors that determine whether a dose of UVR will induce erythema are the wavelength of the radiation, length of exposure, and the skin type and the pigmentation of the subject. UVA, UVB, and UVC radiation are all able to induce erythema. The most effective wavelengths, however, are between 250 nm and 290 nm (5). In addition to acute effects on the skin, UVR is responsible for some conditions that may not be evident for many years. The main conditions are skin aging and skin cancer. Both result from cumulative exposure to UVR. Skin cancers are the most frequently detected malignant tumors in humans. Tumors of three main types are associated with sunlight (and in particular UVR). The first two types are squamous cell carcinoma (SCC) and basal cell carcinoma (BCC), both of which are referred to as nonmelanoma skin cancer (NMSC). The third type is malignant melanoma (9).

Exposure Assessment

Human Exposure Levels

For the vast majority of people, the sun is the single largest source of exposure to UVR. Therefore the major factor influencing overall exposure is whether the person is an indoor or outdoor worker. The results of various field studies indicate that, excluding recreational

exposure, indoor workers receive about two to four percent of the annual ambient dose on a horizontal plane, and outdoor workers receive annual doses three to five times greater than those received by indoor workers (10–12). Indoor workers on sun-seeking holidays may, however, receive as much solar UVR during a two-week summer vacation as they receive in the remaining 50 weeks of the year while they go about their normal activities (10,11). The exposure necessary to result in a barely perceptible erythema in unacclimatized skin is called minimal erythemal dose (MED) and is equivalent to an erythemally weighted radiant exposure of 200 joules per square meter (J/m^2) (13). Representative annual minimal erythemal doses for various exposure scenarios are displayed in Table 1.

TABLE 1

Representative Minimal Erythemal Doses (MEDs) for Various Exposure Situations*

Situation	Annual MEDs
Outdoor worker	270
Indoor worker (excluding weekend exposure)	90
Sunbathing holiday in the Mediterranean for two weeks in summer	100
UVA sunbed (low-pressure fluorescent lamps, 30 sessions of 30 minutes each)	20

*Data are from Diffey (14).

Human exposures to artificial sources can occur in workplaces through photo processes, UVR sterilization, and welding arcs. Other sources include medical exposure for the treatment of certain conditions (such as psoriasis) and the use of sunbeds. Welding arcs produce a substantial UVR emission and probably pose the greatest occupational hazard. The danger was illustrated in a recent study in which personal exposure levels of welders were measured at up to 6,000 times the maximum permissible exposure limit (MPE) outside the clothing, and around five times the MPE within welding helmets (15).

Human Exposure Variables

A number of factors influence the amount of solar UVR to which people are exposed. A major influence is ambient solar UVR levels, which vary according to latitude and season. At a single site these levels also are constantly changing throughout the day. For example, at noon when the sun is overhead, the UVR level at 300 nm is 10 times greater than the UVR level three hours earlier (nine a.m.) or three hours later (three p.m.). This difference means that an untanned person with fair skin may become sunburnt within 25 minutes at noon (depending on latitude and time of the year) but would have to lie in the sun for at least two hours to receive the same dose after three p.m. (5).

Geometry of exposure also plays a key role in the actual UVR dose an individual receives. Studies on the anatomical distribution of solar UVR have shown that vertical surfaces of the body receive about half of the dose received by the vertex (top of head) (11,16). Similar studies on the distribution of solar UVR over the face have indicated that the nose and forehead receive the highest doses, and that a brimmed hat can reduce face exposure by a factor of at least two and eye exposure by a factor of four to five (17,18). Ocular UVR exposure levels are influenced by a number of factors, including angle of the sun, natural squint reflex and aversion mechanisms, and facial anatomy (19).

For skin exposure, certain biologic and genetic factors can increase sensitivity to UVR. These factors include medical conditions such as xeroderma pigmentosum, and genetic influences such as albinism and skin type. For example, persons with Skin Type I (who always burn, often peel, and never tan) are far more sensitive to UVR than are persons of Skin Type VI (who never burn and tan easily).

Other factors that influence personal exposure are

- reflection from surfaces (such as snow, sand, and water);
- use of protective measures (e.g., sunglasses);
- shade provided by trees and other structures; and, most important
- individual behavior (e.g., intentional exposure at times of peak UVR) (11,20).

Protective Measures

The risks to health associated with exposure to UVR from both natural and artificial sources can be substantially reduced through appropriate control and protective measures. As UVR exposure occurs externally, simple measures can be taken to reduce the exposures received. A high degree of protection can be afforded by protective clothing (including hats); UVR-protective eye wear (e.g., welding helmets, face shields, goggles, sunglasses, spectacles); and the application of sunscreen lotion to exposed skin. The degree of protection can, however, be reduced by personal behavior that increases UVR exposure, as well as through the ingestion of photosensitizing drugs or photoallergic reactions to chemicals or cosmetics that come in contact with the skin. Therefore, education is also an important control measure (5).

Clothing

The use of protective clothing is one of the simplest means of reducing UVR exposure. The degree of protection offered by clothing depends on the ability of UVR to penetrate through the fabric. Fabrics that are visually opaque tend to be more highly absorbent of UVR; however, the structure or weave of the fabric is the most important factor in determining its protective value. Color and thickness have been found to be poor guides to the UVR-protective properties of garments (5,21).

Hats

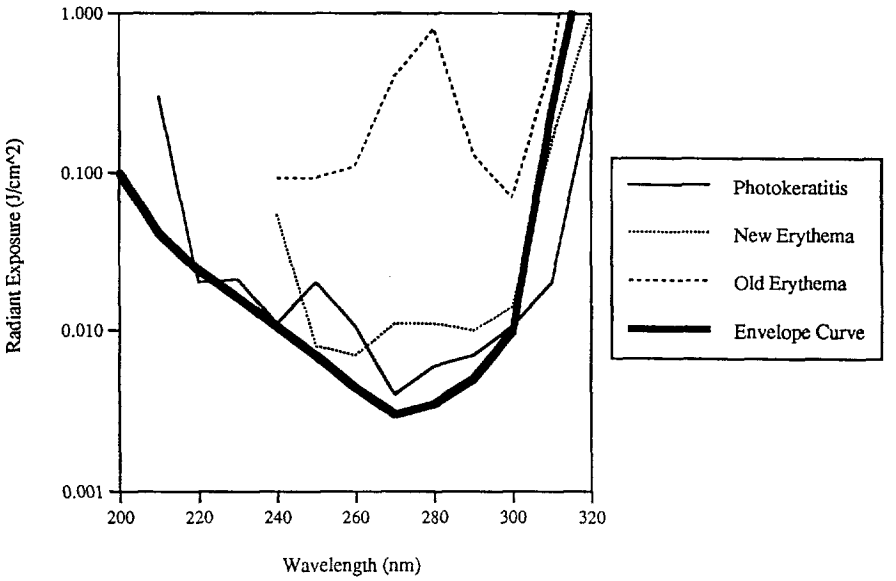
Various design features have a marked influence on the protection provided by hats. For example, hats with small brims provide negligible protection at all head sites. Baseball-style caps offer good protection to the nose but are relatively ineffective at other sites on the face. For reasonable protection of the nose and cheeks, hats with wide brims (greater than 7.5 centimeters) have been found to be necessary (22).

Sunscreens

Sunscreens are topical preparations with physical and chemical properties that attenuate the transmission of solar UVR into the skin by absorption, reflection, or scattering. Physical sunscreens (sunblocks), which may contain zinc oxide or titanium dioxide, function by reflection and scattering and provide protection against a broad spectrum of UVR. Even though concerns have been raised about the safety of some ingredients and the actual protectiveness of sunscreens, the use of broad-

FIGURE 2

UVR Envelope (Hazard) Curve, with Photokeratitis and Erythema Action Spectra*



*Based on data from Sliney (25).

spectrum sunscreens with an SPF of at least 15 is still considered an effective means of personal protection (5).

Eye Protection

Sunglasses and spectacles provide protection from exposure to solar UVR; however, the amount of attenuation is related more to design (size and shape) and wearing position than to the transmission properties of the lens (23,24). Maximum protection is provided by goggle style or wraparound sunglasses that also provide side protection. In addition, sunglasses should be worn so that the frame is against the wearers' forehead, as it has been found that substantial amounts of UVR (up to 45 percent) can reach the eyes through this pathway (24).

Occupational Protection

Protection of workers in an occupational setting consists of administrative controls, engineering controls, and the use of personal protective equipment (PPE). Administrative controls include job rotation to minimize exposure time. Engineering controls include substitution of high-UVR sources with ones that produce lower levels of UVR, the use of low-reflectant surfaces, and the use of UVR-absorbing screens and barriers. The range of PPE available is considerable and includes safety spectacles, goggles, face shields, and welding helmets.

UVR Exposure Standards

The most widely recognized standard for UVR exposure to eyes and skin was proposed in 1971 by the American Conference of Governmental Industrial Hygienists (ACGIH). The standard was based on action spectra for the main UVR acute effects (i.e., erythema and photokeratitis). The threshold data for the acute effects of erythema and photokeratitis were combined on one graph, and an envelope (or hazard) curve was drawn around the collective data so that a single-envelope action spectrum could be applied to both skin and eye exposure (25) (Figure 2).

The envelope curve does not, however, account for repeated exposures. Because tanning and thickening provide increased protection, the envelope is overly conservative for skin exposure. The cornea does not have this same capability, so the standard must be considered the limit for eye exposure. Nevertheless, facial anatomy and natural aversion reflexes combine to provide the eye with some protection, especially if the UVR source is overhead, as is the sun. Because of a comparative lack of data about the chronic effects of UVR, the envelope curve concentrates on the elimination of risk from acute effects. The assumption is that chronic exposure at the exposure limit would contribute slightly to the overall risk from UVR (26).

The envelope curve provides weighting factors that represent the effectiveness of different wavelengths in producing adverse effects. When these weighting factors are combined with the measured spectral irradiance of the source, an effective irradiance is determined. From this value, permissible exposure times can be calculated (27).

Risk Assessment Model for Skin Cancer

The application of multivariate analysis to the epidemiology of skin cancer has shown that, for a group of subjects with a given genetic susceptibility, age and ambient UVR exposure are the two most important factors in determining relative risk (28). The following simple power relationship was developed to express cumulative risk in terms of these factors (13,29):

$$\text{Risk} \propto (\text{Annual Solar UVR Dose})^{\beta}(\text{age})^{\alpha}$$

or

$$I = \gamma A H^{\beta} a^{\alpha}$$

where

I = cumulative incidence of NMSC as the total number of cases per 100,000 up to age a years;

A = the fraction of the body surface normally exposed (e.g., face and hands);

H = annual carcinogenic-effective exposure (expressed in MED) at the skin surface; and

α, β, and γ = numerical constants associated with the age dependence of the cumulative incidence, the biological amplification factor, and the genetic susceptibility of the population, respectively.

This formula was found to be inadequate in accounting for changes in annual exposure (as is experienced in occupational exposure to UVR), so a new expression was developed to estimate the risk of NMSC at a particular age T (13,30):

$$\text{Risk} \propto (\text{Cumulative UVR Dose at Age } T)^{\beta-1} \sum (\text{Annual Dose at Age } T - t)t^{\alpha-\beta}$$

or

$$I = \gamma A H_e^{\beta} a^{\alpha}$$

where

$H_e = H + H_o(a - a_s)/a$

H = annual exposure from day to day natural UVR exposure,

H_o = annual dose (in MED) from occupational exposure, and

a_s = age at which occupational exposure began.

The additional risk of NMSC as a result of occupational exposure, defined as relative cumulative incidence (RCI), may be determined as follows (13):

$$RCI = [H_c/H]^{\beta}$$

The values of α and β are normally derived from surveys of skin cancer incidence and UVR climatology. In calculations of risk estimates, 5 and 2 are often used as exemplary values for α and β, respectively (31).

Risk Characterization

As outlined above, the risks of adverse health effects from exposure to UVR are influenced by many factors. These factors include

- whether the individual is an indoor or outdoor worker;
- exposure to artificial sources of UVR such as welding arcs and sunbeds;
- protective measures employed (e.g., hats, sunglasses, sunscreen);
- behavior (e.g., sun exposure at the peak UVR irradiance times);
- recreational activities (e.g., sunbathing, skiing);
- amount of shade provided and used during outdoor activities;
- age of individual (especially UVR exposure at certain UVR-sensitive ages);
- biologic or genetic sensitivity to UVR; and,
- for artificial sources of UVR (in particular those in occupational settings), the spectrum and intensity of the source, distance from the source, and any specific engineering, administrative, or protective-equipment control measures employed.

Because of the range of adverse effects associated with UVR, the risks for acute and chronic conditions will be assessed separately.

Acute Conditions

Because the ACGIH exposure standard is based on the action spectra and threshold doses for erythema and photokeratitis, and because it also has a built-in safety factor, the risks associated with acute effects will be addressed in relation to the permissible exposure time (PET) provided in the standard. Table 2 shows PETs for the sun and a number of artificial sources at exposure distances representative of normal human exposure. The values in Table 2 are based on representative spectral irradiance data for these sources. It should be noted that the ACGIH standard is for occupational exposure of eight hours per day and should be interpreted within these constraints.

The data in Table 2 indicate that welding arcs produce a substantial UVR emission and illustrate the need for adequate protection of all workers in a welding environment. The PET for the sun would be exceeded after 15 minutes of exposure. Within this exposure time, acute effects would not, however, be expected because of the safety factor built into the standard. The PET only provides an indication of exposure. Depending on the season, latitude, and skin type, the time for which an individual could be exposed without developing acute effects could be much higher. The PET for the sun does indicate that acute effects can be experienced in a relatively short time and that exposure to solar UVR for extended periods should be avoided.

TABLE 2

Permissible Exposure Times (PETs) for Various Sources

Source	Reference	Measurement Distance (meters)	PET (seconds)
Welding 1[a]	1	1	2.31
Welding 2[a]	1	1	2.40
Welding 3[a]	1	1	42.35
Phototherapy lamp, Type A, unenclosed[b]	32	1	<120.00
UVR curing unit[c]	33	0.8	<7,200
Sun, Melbourne, Spring, 12:10[d]	34	ground	883.00

[a]Welding 1 = gas metal arc welding, steel, 250A, O_2 + Ar shield;
Welding 2 = gas metal arc welding, aluminum, 300A, Ar shield; and
Welding 3 = gas tungsten arc welding, steel, 250A, Ar shield. Data are from Sliney and Wolbarsht (1).
[b]Data are from Diffey and Langley (32).
[c]Data are from Surakka et al. (33).
[d]Data are from Sydenham (34).

TABLE 3

Risks of Nonmelanoma Skin Cancer (NMSC) for Various Exposure Situations*

Exposure Situation	Risk at Age 70 (%)
Indoor worker, no sunbathing	2–3
Outdoor worker, no sunbathing	7.4–11.1
Indoor worker, sunbathing 2 wks/yr	10–15
Outdoor worker, sunbathing 2 wks/yr	37–55.5
Indoor worker, sunbathing 4 wks/yr	20–60
Outdoor worker, sunbathing 4 wks/yr	74–100

*Based on data from Diffey (14).

Chronic Conditions

Because knowledge about the action spectrum for the induction of melanoma is limited, a risk analysis for this condition could not be conducted. Risk assessment for NMSC is, however, possible, and the risks associated with a number of exposure situations have been calculated. The risks of solar UVR exposure for fair-skinned Caucasians in Europe are shown in Table 3, assuming that $\alpha = 5$, $\beta = 2$, and indoor workers have no occupational exposure to UVR (14).

As a specific occupational example, it has been calculated that during a working life of

REFERENCES

1. Sliney, D.H., and M. Wolbarsht (1980), *Safety with Lasers and Other Optical Sources: A Comprehensive Handbook*, New York: Plenum Press.
2. Matelsky, I. (1968), "The Non-Ionising Radiations," In *Industrial Hygiene Highlights, Volume 1*, L.V. Cralley, L.J. Cralley, and G.D. Clayton. eds., Pittsburgh: Industrial Hygiene Foundation of America, pp. 140-178.
3. McKinlay, A.F. (1991), "Characteristics of Optical Radiation Sources and Their Potential to Cause Ocular Injury," In *Vision and Visual Dysfunction, Volume 16*, J. Marshall, ed., London: MacMillan Press, pp. 1-21.
4. Boal, T.J., and G.J. Rouch (1989), "Introduction," In *Health Effects of Ozone Layer Depletion*, Canberra, Australia: National Health and Medical Research Council, pp. 1-7.
5. *Environmental Health Criteria 160: Ultraviolet Radiation* (1994), Geneva: World Health Organization.
6. Parrish, J.A., R.R. Anderson, F. Urbach, and D.G. Pitts (1978), *UVA: Biological Effects of Ultraviolet Radiation with Emphasis on Human Responses to Longwave Ultraviolet*, New York: Plenum Press.
7. Marshall, J. (1991), "The Effects of Ultraviolet Radiation and Blue Light on the Eye," In *Vision and Visual Dysfunction, Volume 16*, J. Marshall, ed., London: MacMillan Press, pp. 54-66.
8. Boettner, E.A., and J.R. Wolter (1962), "Transmission of the Ocular Media," *Investigative Ophthalmology*, 1(6):776-783.
9. *Solar and Ultraviolet Radiation* (1992), *Monographs on the Evaluation of Carcinogenic Risks to Humans, Volume 55*, Lyon: International Agency for Research on Cancer.
10. Diffey, B.L., O. Larko, and G. Swanbeck (1982), "UV-B Doses Received During Different Outdoor Activities and UV-B Treatment of Psoriasis," *British Journal of Dermatology*, 106:33-41.
11. Holman, C.D.J., I.M. Gibson, M. Stephenson, and B.K. Armstrong (1983), "Ultraviolet Irradiation of Human Body Sites in Relation to Occupation and Outdoor Activity: Field Studies Using Personal UVR Dosimeters," *Clinical and Experimental Dermatology*, 8:269-277.
12. Larko, O., and B.L. Diffey (1983), "Natural UV-B Radiation Received by People with Outdoor, Indoor, and Mixed Occupations and UV-B Treatment of Psoriasis," *Clinical and Experimental Dermatology*, 8:279-285.
13. Diffey, B.L. (1988), "The Risk of Skin Cancer From Occupational Exposure to Ultraviolet Radiation in Hospitals," *Physics in Medicine and Biology*, 33:1187-1193.
14. Diffey, B.L. (1987), "Analysis of the Risk of Skin Cancer from Sunlight and Solaria in Subjects Living in Northern Europe," *Photodermatology*, 4:118-126.
15. Tenkate, T.D., and M.J. Collins (1997), "Personal Ultraviolet Radiation Exposure of Workers in a Welding Environment," *American Industrial Hygiene Association Journal*, 58:24-29.
16. Diffey, B.L., A. Davis, and M. Kerwin (1977), "The Anatomical Distribution of Sunlight," *British Journal of Dermatology*, 97:407-410.
17. Diffey, B.L., A. Davis, and T.J. Tate (1979), "Solar Dosimetry of the Face: The Relationship of Natural Ultraviolet Radiation Exposure to Basal Cell Carcinoma Localisation," *Physics in Medicine and Biology*, 24(5):831-939.
18. Roy, C.R., G. Elliot, and H.P. Gies (1988), "Solar Ultraviolet Radiation: Personal Exposure and Protection," *Journal of Occupational Health and Safety* [Australia and New Zealand], 4:133-139.
19. Sliney, D.H. (1983), "Biohazards of Ultraviolet, Visible, and Infrared Radiation," *Journal of Occupational Medicine*, 25(3):203-206.
20. Diffey, B.L., and P.J. Saunders (1995), "Behavior Outdoors and its Effects on Personal Ultraviolet Exposure Rate Measured Using an Ambulatory Datalogging Dosimeter," *Photochemistry and Photobiology*, 61:615-618.

Continued on page 15

40 years, a hospital worker exposed daily (250 days per year) to UVR in phototherapy departments (physiotherapy and dermatology) will incur an additional risk of NMSC of around 25 percent relative to nonexposed workers (13). Additional risk posed by recreational sunbed use, defined as the use of a UVA solarium for 10 sessions per year, was found to be negligible (14).

Other investigators have also calculated numbers for NMSC that account for age, gender, sun affinity, and geographic location (35). Assumptions used in these calculations were as follows:

- UVR exposure is age dependent within two separate intervals: childhood (less than 18 years of age) and adulthood. Doses within these intervals are constant, and childhood dose is influenced by the sun affinity ratio and the sun protection factor.
- Sun affinity ratio (SAR) is defined as the ratio of annual sun exposure before the age of 18 years to annual sun exposure after the age of 18 years. For example, the average child is assumed to have three times the annual sun exposure of an average adult.
- The sun protection factor (SPF) is the use or the nonuse of an SPF-15 sunscreen, which was deemed to have an effective SPF of 7.5 or 2, depending on use and application factors.

Results of the calculations indicate that for individuals 55 years of age, the number of NMSCs per 100,000 ranges between 1,614 and 26,999 (1.6 percent and 27 percent) for men, and 1,655 and 24,434 (1.6 percent and 24 percent) for women. Increased sun affinity produces increased incidence. Increased incidence also has been associated with increased geographic solar UVR exposure, determined according to latitude for three cities in the United States (Seattle, San Francisco, and Albuquerque). It has also been found that significant reductions in NMSC could be achieved by the use of sunscreen during childhood. For example, persons with average SARs (i.e., SAR = 3) who as children achieve an effective SPF of 7.5 could expect an 84 percent reduction in the risk that they will develop NMSC before the age of 55 years. Reductions of between 43 percent and 60 percent could also be achieved, even if the effectiveness of the sunscreen was reduced to an SPF of 2 (35).

The issue of increased solar UVR caused by a depletion of stratospheric ozone and the possible resulting increase in NMSC also has been well investigated and well documented. For adults, ozone depletion at current rates is

estimated to result in a small increase in lifetime risk of NMSC (less than five percent) if there are no changes in climate, outdoor UVR exposure, behavior, or clothing habits. The lifetime risk of NMSC for today's children, however, is estimated to be 10 to 15 percent greater than if there were no ozone depletion. If the production and use of ozone-depleting substances is reduced in accordance with the Montreal Protocol, the increased risk of NMSC is likely to be less than estimated (36).

Conclusion

The sun is the main source of UVR to which most people are exposed; other sources include welding arcs, sunbeds, and a range of UVR applications in industry. Experimental studies have shown that UVR causes DNA damage by direct photochemical effects and by oxidative effects. This UVR-induced damage is one step in the mechanism by which UVR can cause cancer. Squamous cell carcinoma, basal cell carcinoma, and malignant melanoma have been associated with UVR exposure. UVR also has been shown to be responsible for the acute conditions photokeratitis and erythema, as well as for the chronic eye conditions pingueculum, pterygium, and cataracts of some types.

Human exposure to UVR is influenced by a number of factors, including occupation, use of protective measures, types of recreational activities undertaken, and personal behavior (e.g., intentional exposure at peak periods of solar UVR). On the basis of the ACGIH exposure standard for UVR, permissible exposure times for various activities have been found to range from a few seconds for some types of welding arcs to around 15 to 20 minutes for noontime sun exposure (depending on season and latitude). For an indoor worker 70 years of age who undertakes no sunbathing, the risk of developing NMSC has been estimated at between two and three percent. The risk has been found to be substantially increased for outdoor workers. It also increases with recreational sun exposure. Exposure during childhood has been found to lead to higher risks than the same exposure later in life. The effective use of sunscreens (especially during childhood) has been found to significantly reduce the risk of NMSC.

Corresponding Author: *Thomas D. Tenkate, M.App.Sc., School of Public Health/Department of Environmental Health Sciences, University of Alabama at Birmingham, 317 Ryals Bldg., 1665 University Blvd., Birmingham, AL 35294-0022.*

NEHA's New Zip Code Now Mandatory

NEHA's zip code changed from 80222 to **80246** last July. Mail not addressed with the new zip code will be returned to sender after July 1st, 1998.

REFERENCES

Continued from page 14

21. Robson, J., and B.L. Diffey (1990), "Textiles and Sun Protection," *Photodermatology, Photoimmunology, and Photomedicine,* 7(1):32-34.
22. Diffey, B.L., and J. Cheeseman (1992), "Sun Protection with Hats," *British Journal of Dermatology,* 127:10-12.
23. Rosenthal, F.S., A.E. Bakalian, and H.R. Taylor (1986), "The Effect of Prescription Eyewear on Ocular Exposure to Ultraviolet Radiation," *American Journal of Public Health,* 76(10):1216-1220.
24. Rosenthal, F.S., A.E. Bakalian, C. Lou, and H.R. Taylor (1988), "The Effect of Sunglasses on Ocular Exposure to Ultraviolet Radiation," *American Journal of Public Health,* 78(1):72-74.
25. Sliney, D.H. (1972), "The Merits of an Envelope Action Spectrum for Ultraviolet Radiation Exposure Criteria," *American Industrial Hygiene Association Journal,* 33:644-653.
26. Sliney, D.H. (1987), "Unintentional Exposure to Ultraviolet Radiation: Risk Reduction and Exposure Limits," In *Human Exposure to Ultraviolet Radiation: Risks and Regulations,* W.F. Passchier and B.F.M. Bosnjakovic, eds., Amsterdam: Elsevier, pp. 425-437.
27. *Documentation of the Threshold Limit Values and Biological Exposure Indices* (1991), 6th ed., Cincinnati: American Conference of Government Industrial Hygienists.
28. Fears, T.R., J. Scotto, and M.A. Schneiderman (1977), "Mathematical Models of Age and Ultraviolet Effects on the Incidence of Skin Cancer Among Whites in the United States," *American Journal of Epidemiology,* 105:420-427.
29. Schothorst, A.A., R. Schouten, H. Slaper, and D. Suurmond (1985), "UVB Dose in Maintenance Psoriasis Phototherapy Versus Solar UVB Exposure," *Photodermatology,* 2:213-220.
30. Slaper, H., and J.C. van der Leun (1987), "Human Exposure to Ultraviolet Radiation: Quantitative Modelling of Skin Cancer Incidence," In *Human Exposure to Ultraviolet Radiation: Risks and Regulations,* W.F. Passchier and B.F.M. Bosnjackovic, eds., Amsterdam: Elsevier, pp. 155-171.
31. Diffey, B.L. (1991), "Solar Ultraviolet Radiation Effects on Biological Systems," *Physics in Medicine and Biology,* 36(3):299-328.
32. Diffey, B.L., and F.C. Langley (1986), *Evaluation of Ultraviolet Radiation Hazards in Hospitals,* Report 49, London: Institute of Physical Sciences in Medicine.
33. Surakka, J., T. Fischer, L.A. Nylander-French, and G. Rosen (1997), "Assessment of Ultraviolet Radiation Exposure in the Wood Surface Coating Industry," *Applied Occupational and Environmental Hygiene,* 12:261-270.
34. Sydenham, M.M. (1995), A Study of Ultraviolet Radiation Exposure at the Human Eye, Ph.D. thesis, Brisbane, Australia: Queensland University of Technology.
35. Stern, R.S., S.G. Baker, and M.C. Weinstein (1986), "Risk Reduction for Nonmelanoma Skin Cancer with Childhood Sunscreen Use," *Archives of Dermatology,* 122:537-545.
36. Diffey, B.L. (1992), "Stratospheric Ozone Depletion and the Risk of Non-Melanoma Skin Cancer in a British Population," *Physics in Medicine and Biology,* 37(12):2267-2279.