Ultraviolet Radiation: Human Exposure and Health Risks


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-
Absorption of Ultraviolet Radiation by Media of the Eye

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 keratopathy, 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 (3). 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).

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.
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/m²) (13). Representative annual minimal erythemal doses for various exposure scenarios are displayed in Table 1.

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 (13).

**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 unattended 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. (3).

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 (3).

**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 (3,21).

**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-
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):

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

\[I = \gamma A H^p a^q\]

where

- \(I\) is cumulative incidence of NMSC as the total number of cases per 100,000 up to age \(a\) years;
- \(A\) is the fraction of the body surface normally exposed (e.g., face and hands);
- \(H\) is annual carcinogenic-effective exposure (expressed in MED) at the skin surface; and
- \(\alpha, \beta, \gamma, p, q\) are 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):

\[
\text{Risk} \propto (\text{Cumulative UVR Dose at Age } T)^{\beta - 1} \sum (\text{Annual Dose at Age } T - t) e^{-\beta t} a^q
\]

where

- \(H = H + H (a - a)/a\)
- \(H\) is annual exposure from day to day natural UVR exposure,
- \(H\) is annual dose (in MED) from occupational exposure, and
- \(a\) is age at which occupational exposure began.

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

$$\text{RCI} = |H/H|^\beta$$

The values of $\alpha$ and $\beta$ 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 $\alpha$ and $\beta$, 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.

**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...
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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 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 non-time 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.

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