Health and Environmental Effects of Ultraviolet Radiation
A Scientific Summary of Environmental Health Criteria 160
Ultraviolet Radiation (WHO/EHG/95.16)

Introduction

A monograph entitled Environmental Health Criteria 160 "Ultraviolet Radiation" was published in 1994 by the World Health Organization, jointly in collaboration with the United Nations Environment Programme and the International Commission on Non-Ionizing Radiation Protection. The monograph was the result of an in-depth review of the scientific literature and was primarily concerned with the effects of ultraviolet (UV) radiation exposure on human health and the environment. The penultimate draft was subjected to a WHO Task Group for final peer review prior to publication. Such a review was considered particularly timely in view of the consequences of increasing levels of UV at the surface of the earth resulting from depletion of stratospheric ozone.

The purpose of this document is to provide a summarised form of the monograph, allowing the subject to be reviewed by a more general readership, and to update activities that have resulted from the recommendations of the Task Group that reviewed the monograph.

Exposure to UV occurs from both natural and artificial sources. The sun is the principal source of exposure for most people. Solar UV undergoes significant absorption by the atmosphere. With depletion of the stratospheric ozone people and the environment will be exposed to higher intensities of UV. The consequences of this added UV exposure are considered so serious that it was a major topic for discussion at the World Environment Conference, held in Rio de Janeiro in 1992. In Agenda 21, adopted by the Conference, it was specifically recommended to "undertake, as a matter of urgency, research on the effects on human health of the increasing ultraviolet radiation reaching the earth's surface as the consequence of depletion of the stratospheric ozone layer." It is this issue that underscores the current need to better understand the potential health and environmental risks of UV exposure. INTERSUN, global UV project, is WHO’s response to the need to disseminate information about the health and environmental hazards of excessive UV exposure. INTERSUN has developed a document entitled "UV Protective Measures" in response to the need to educate the public and workers on measures they can take to reduce their UV exposure, and has been involved in the development of a Solar UV Index, an index related to daily UV exposure, reported with the news and weather, that facilitates a continuing educational process about possible health effects and measures to reduce UV exposure. More details on these programmes are given at the end of this text.

Summary of the major health concerns

Skin cancer and cataracts are important public health concerns. The social cost of these diseases, such as death, disfigurement and blindness, can be overwhelming both in terms of human suffering and the financial burden. Solar UV exposure is known to be associated with various skin cancers, accelerated skin aging, cataract of the lens of the eye and other eye diseases, and possibly has an adverse effect a person's ability to resist infectious diseases. Most of these health concerns could be avoided by reducing exposure to solar UV.

The United Nations Environment Programme has estimated that over 2 million non-melanoma skin cancers and 200,000 malignant melanomas occur globally each year. In the event of a 10% decrease in stratospheric ozone, with current trends and behaviour, an additional 300,000 non-melanoma and 4,500 melanoma skin cancers could be expected world-wide.

Some 12 to 15 million people in the world are blind because they have cataracts. WHO has estimated that up to 20% of cataracts or 3 million per year could be due to UV exposure to the eye. It has been estimated that for each 1% sustained decrease in stratospheric ozone there would be an increase of 0.5% in the number of cataracts caused by solar UV (van der Leun et al 1989). In the United States alone, it costs the US Government $US 3.4 billion for 1.2 million cataract operations per year. Substantial savings in cost to health care can be made by prevention or delay in the onset of cataracts.

Ultraviolet radiation

UV is one of the non-ionizing radiations in the electromagnetic spectrum and lies within the range of wavelengths 100 nm to 400 nm (see figure 1). The short wavelength limit of the UV region is often taken as the boundary between the ionizing radiation spectrum (wavelengths < 100 nm) and the non-ionizing radiation spectrum. UV can be classified into UVA (315 - 400 nm), UVB (280 - 315 nm) and UVC (100 - 280 nm) regions, although other conventions for UVA, UVB and UVC wavelength bands are in use.
Most artificial sources of UV, except for lasers, emit a spectral continuum of UV containing characteristic peaks, troughs and lines. These sources include various lamps used in medicine, industry, commerce, research and the home.

Since UV is normally absorbed over a surface it can be measured as a radiant exposure, the incident UV energy divided by the receptor surface area in joules per square metre ($J \ m^{-2}$). UV can also be measured as an irradiance, the incident power divided by the receptor surface area in watts per square metre ($W \ m^{-2}$).

**Biological effectiveness of UV**

UV-induced biological effects depend on the wavelengths of the radiation emitted by the source. Thus, for a proper determination of hazard it is necessary to have information on the spectral (range of wavelength) emissions. These consist of spectral irradiance ($W \ m^{-2} \ nm^{-1}$) measurements from the source. The total irradiance ($W \ m^{-2}$) is obtained by summing over all wavelengths emitted. The biological or hazard weighted irradiance ($W \ m^{-2} \ effective$), commonly called the effective UV irradiance or dose rate (exposure), is determined by multiplying the spectral irradiance at each wavelength by the biological or hazard weighting factor (which quantifies the relative efficacy at each wavelength for causing the effect) and summing over all wavelengths. Such factors or weighting functions are obtained from action spectra.

**Action Spectrum and Minimum Erythemal Dose**

An action spectrum is a graph that provides information on the effectiveness of the UV wavelengths in producing a biological effect, e.g. erythema. It is the reciprocal of the radiant exposure required to produce the given effect at each wavelength. Figure 2 depicts the ICNIRP-CIE action spectrum for erythema. All the data in such curves are normalized to the most effective wavelength(s). By summing the biologically effective irradiance over the exposure period, the biologically effective radiant exposure ($J \ m^{-2} \ effective$) can be calculated.

For UV induced erythema, the action spectrum adopted by the International Commission on Non-Ionizing Radiation Protection (ICNIRP), International Commission on Illumination (CIE), the International Electrotechnical Commission (IEC) and various national bodies, is a composite curve obtained by statistical analysis of many research results on the minimum radiant exposure of UV at different wavelengths necessary to just cause erythema.
The most commonly used quantity for describing the erythemal potential of an exposure to UV is the number of minimum erythemal doses (MEDs) represented by the exposure. An MED is the radiant exposure of UV that produces a just noticeable erythema on a previously unexposed skin. It corresponds to a radiant exposure of monochromatic radiation at the maximum spectral efficacy for erythema (around 300 nm) of approximately 150 to 2000 J m$^{-2}$ effective, depending on skin type. Values of 200 - 300 J m$^{-2}$ effective are commonly used as the value of 1 MED for comparative safety purposes for white skin.

**Cellular and Molecular Studies**

To produce any change, UV must be absorbed by a biological molecule. This involves absorption of a single photon by the molecule and the production of an excited state in which one electron of the absorbing molecule is raised to a higher energy level. The primary products caused by UV exposure are generally reactive species or free radicals which form extremely quickly but which can produce effects that can last for hours, days or even years. DNA is the most critical target for damage by UVB and UVC. Figure 3 indicates the formation of pyrimidine dimers in DNA. While a considerable amount of knowledge is available concerning the interaction of UV with nucleic acids, controversy exists as to which lesion constitutes the most important type of pre-mutagenic damage.

Cell death, chromosome changes, mutation and morphological transformations are observed after UV exposure of procaryotic and eucaryotic cells. Many different genes and several viruses (including HIV) are activated by UV exposure. The genes activated by UVB and UVC are different from those activated by UVA. Studies of DNA repair defective disorders have clearly established a link between UV induced DNA damage in skin and various types of cancer.

**Animal Studies**

**Skin cancer**

Solar UV exposure has been shown to produce cancers in domestic and food animals. In experimental animals UV causes predominantly squamous cell carcinomas (SCCs). UVB is most effective at producing SCCs, although they are produced by UVA but at much higher intensities, similar to the levels needed for erythema and tanning. The effectiveness of UVC is unknown except at one wavelength (254 nm). At this wavelength the effectiveness is less than UVB.
Melanomas are much less common and only two animal models have been found for induction of melanoma by UV alone. An initial action spectrum determined for a type of hybrid fish indicates a peak in the UVB range but also shows a high level of effectiveness in the UVA. Basal cell carcinomas are rare in animals.

**Immune response**

Exposure to suberythemal doses of UV have been shown to exacerbate a variety of infections in rodent models. UV affects infections both at the site of exposure and at distant sites. Recent work indicates that systemic infections without skin involvement may be affected. Enhanced susceptibility appears to result from T-helper cell activity. The mechanisms associated with this suppression appear to be the same as those identified with suppression to contact and delayed type hypersensitivity responses. Suppression of these immune responses appears to be mediated by release of soluble mediators from UVB exposed skin which alters the antigen presentation by Langerhans and other cells so that they fail to activate TH1 cells. The resulting immune suppression is antigen specific, can occur regardless of whether or not antigen is applied at the site of exposure, and is relatively long lasting. UV exposure also prevents the development of protection immunity to a variety of infections in mice and rats.

**Effects on the eye**

Many studies in experimental animals have demonstrated that UV exposure can cause both acute and delayed effects such as cataract, photokeratitis, damage to the corneal epithelium and various retinal effects. Studies of photochemical retinal injury in aphakic monkeys have shown that the retina is six times more vulnerable to photochemical damage from UV than the visible wavelengths.

**Health Effects on Humans**

**Skin**

The degree of damage that UV produces in skin will depend on the incident intensity and wavelength content (UVA or UVB), and on the depth of penetration of these wavelengths into the skin (see figure 4). Acute effects on the skin consist of solar erythema, "sunburn", which, if severe enough, may result in blistering and destruction of the surface of the skin with secondary infection and systemic effects, similar to those resulting from a first or second degree heat burn. Although UVC is very efficiently absorbed by nucleic acids, the overlying dead layers of skin absorb the radiation to such a degree that there is only mild erythema and, usually, no late sequelae, even after repeated exposures. Much less is known about the biological effects of UVA. However, doses of UVA, which alone may not show any biological effect, can, in the presence of certain environmental, consumer and medicinal chemical agents,
Chronic skin changes due to UV consist of skin cancer (both melanoma and non-melanocytic), benign abnormalities of melanocytes (freckles, melanocytic naevi and solar or senile lentigines), and a range of other chronic injuries resulting from UV exposure to keratinocytes, blood vessels and fibrous tissue, often described as "photoaging" (solar elastosis). The much increased rates of skin cancer in patients with xeroderma pigmentosum, who have a deficiency in the capacity to repair UV-induced DNA damage, suggest that direct UV damage of the DNA may be a step in the cause of these cancers. This suggestion has also been supported by the observation of UV specific mutations of the p53 tumour suppressor gene in a proportion of patients with non-melanocytic skin cancer. Oxidative and immune suppressant effects may also contribute to the capacity of UV to cause skin cancers.

Cancer of the lip is much more common in fair than dark skin populations and is associated with outdoor work. However possible confounding with tobacco and alcohol use has not been adequately controlled in any study, and so it is not possible at present to associate directly solar UV exposure in the cause of this cancer.

Strong epidemiological evidence exists that sun exposure causes cutaneous melanoma and non-melanocytic skin cancer. Their incidence is less in darker than light skin groups living in the same geographical area. Risk of skin cancer decreases with increasing pigmentation. The anatomical site most seen for squamous cell carcinoma (SCC) is the head and neck, areas most exposed to the sun. Incidence of both melanoma and non-melanocytic skin cancer are increased in areas of high ambient solar UV radiation.

The worldwide incidence of malignant melanoma has continued to increase. Cutaneous melanoma is the result of neoplastic transformation of melanocytes, the pigment producing cells in the epidermis. Four basic categories of melanoma have been identified in humans: superficial spreading melanoma, nodular melanoma, lentigo malignant melanoma (also known as Hutchinson's melanotic freckle), and unclassified melanoma.

Melanoma is strongly related to frequency of recreational exposure to the sun and to history of sunburns. The evidence that risk of melanoma is related to intermittent exposure to UV, especially in childhood, is inferred from the locations of the melanomas over the body (larger numbers on irregularly exposed sites), higher occurrence in indoor than outdoor workers, and higher levels of exposure during childhood (prior to 15-20 years of age).

There is suggestive evidence that exposure to sunlamps may increase the risk of melanoma, but the studies conducted so far have not consistently controlled confounding factors.

**Immune system**

A number of studies suggest that UV exposures at environmental levels suppress immune responses in both rodents and man. In rodents this immune suppression results in enhanced susceptibility to certain infectious diseases with skin involvement and some systemic infections. Mechanisms associated with UV-induced immunosuppression and host defence mechanisms which provide for protection against infectious agents, are similar in rodents and man. It is therefore reasonable to assume that exposure to UV may enhance the risk of infection and decrease the effectiveness of vaccines in humans. However additional research is necessary to substantiate this.

**Eye**

UV exposure of the eye depends on many factors: ground reflection, degree of brightness in the sky leading to activation of the squint reflex, the amount of atmospheric reflection and the use of eyewear. In addition, the target for UV-induced damage will depend on the wavelength of the incident radiation as shown in figure 5.

The acute effects of UV on the eyes consist of the development of photokeratitis and photoconjunctivitis, which are unpleasant but usually reversible and easily prevented by appropriate eyewear. Chronic effects on the eye consist of the development of pterygium and squamous cell cancer of the conjunctiva and cataracts. A review of the studies suggests that there is sufficient evidence to link acute ocular exposure to photokeratitis but our knowledge of the effects of chronic exposure is less certain. While there is sufficient evidence that cortical and posterior subcapsular cataracts (PSC) can be caused by UVB in laboratory animals, there is limited evidence to link cortical and PSC cataracts in humans to chronic ocular exposure to UVB.
Insufficient information is available to separate out the other factors contributing to cataract formation, or to state the proportion of cataracts which can be attributed to UVB exposure. There is also limited evidence to link the development of climatic droplet keratopathy and pterygium, but insufficient evidence to link uveal melanoma with UV exposure.

**Environment**

Increased levels of UV due to ozone layer depletion may have serious consequences for living organisms. A 10% reduction in ozone could lead to as much as a 15-20% increase in UV exposure depending on the biological process being considered. While the impact on human health, crop production, fisheries etc. is largely unknown, adverse effects of increased exposure to UVB have been reported on plant growth, photosynthesis and disease resistance. Further, the impact of increased UV levels on aquatic ecosystems (the major contributor to the earth's biomass) may be substantial (see figure 6).
Phytoplankton, at the base of the aquatic food chain, serves as food for larvae of fish and shrimp. These in turn are consumed by fish, which subsequently provide an essential food source for many human beings and other animals. A significant reduction in phytoplankton from increased UVB exposure will directly affect the human and animal marine food source.

**Guidelines on Exposure Limits**

Guidance on exposure limits for UV are described in chapter 13. International guidelines published by the International Commission on Non-Ionizing Radiation Protection define exposure limits (ELs) below which it is expected that nearly all people may be repeatedly exposed without adverse effects. The ELs are intended to be used to evaluate potentially hazardous exposures from, for example, solar radiation, arcs, gas and vapour discharges, fluorescent lamps and incandescent sources. The ELs are generally below levels which are often used for the UV exposure of patients required as part of medical treatment and below levels associated with sunbed exposure. ELs are not intended to apply to exposure of pathologically photosensitive individuals, to people concomitantly exposed to photosensitising agents or to neonates.

**Protective Measures**

Typical protection and control measures for reducing UV exposure include the containment of UV sources, and methods for personal protection including the use of sunscreen preparations, clothing, eye and skin protection, and behavioural modifications.

While topical application of sunscreen is a preferred method of absorbing UVB, some preparations do not absorb the longer wavelength UVA effectively. Moreover, some have been found to contain ingredients that are mutagenic in sunlight. There is still much research necessary before the impact on health of increased levels of UVA will be known. In the meantime people using sunscreens should use only those with the highest sun protection factor (SPF) and be aware that they are for their protection from the sun and not for tanning purposes. Use of wide brimmed hats, protective clothing and UV absorbing eye glasses is still the best personal protection against the adverse effects of UV exposure.

With increasing levels of solar UV resulting from depletion of the ozone layer, and the continuing rise in the level of melanoma worldwide, people should become more aware of their UV exposure and take appropriate precautions. These precautions include staying out of the sun during the period around noon (the period when the UV levels are highest), or wearing UV protective clothing, hats and sun glasses. Broad spectrum (UVB and UVA protective) sunscreens should be used when other means of protection are not feasible. These sunscreens should be used to reduce exposure rather than lengthen the period of exposure to the sun. Protection of young children is particularly important for the prevention of long-term consequences of UV exposure. In general behavioural patterns must change to protect against increasing solar UV levels.

**Education**

**UV Protective Measures Publication**

This INTERSUN publication provides information to people on how to protect themselves from the potentially harmful effects of exposure to ultraviolet radiation (UV). Following a detailed review of the scientific literature conducted by a WHO Task Group meeting, a number of adverse health effects resulting from exposure to UV have been identified that need to be addressed through further research and more particularly through educational programmes for people most exposed to UV.
The purpose of this document is to provide information to the general public and workers on the various health hazards known to be associated with excessive exposure to UV and measures that can be taken to reduce this exposure to acceptable levels.

**Solar UV Index**

A joint initiative of the World Meteorological Organization, UNEP and WHO has resulted in the drafting of a report that outlines the details of a solar UV index whose use will be encouraged in the radio and TV news and weather programmes, and in the print media, worldwide. The solar UV index is related to the UV exposure a person would receive outdoors. It identifies periods of the day when UV exposure may be intense and provides a tool to educate the public and outdoor workers about the hazards of excessive UV exposure and what precautions may be necessary to avoid such exposure. The initiative for this project is being taken by the WHO Collaborating Centre in the German Government’s Institute for Radiation Hygiene.
Further reading


For further information on any of the material above, please contact:

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