INTERNATIONAL COMMISSION ON NON-IONIZING RADIATION PROTECTION

ICNIRP GUIDELINES

ON LIMITS OF EXPOSURE TO ULTRAVIOLET RADIATION OF WAVELENGTHS BETWEEN 180 nm AND 400 nm (INCOHERENT OPTICAL RADIATION)

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GUIDELINES ON LIMITS OF EXPOSURE TO ULTRAVIOLET RADIATION OF WAVELENGTHS BETWEEN 180 NM AND 400 NM (INCOHERENT OPTICAL RADIATION)

The International Commission on Non-Ionizing Radiation Protection*

INTRODUCTION

SINCE THE publication of the ICNIRP *Guidelines on UV Radiation Limits* (ICNIRP 1996),† recent research has made it appropriate to update the guidelines for protection. While no significant changes are made in the values, the biological basis can be strengthened, and the limitations on use can be clarified.

A document titled *Environmental Health Criteria 160, Ultraviolet Radiation* (UNEP 1994), was published in 1994 under the joint sponsorship of the United Nations Environment Programme (UNEP), ICNIRP, and the World Health Organization (WHO). The document contains a review of the biological effects reported from exposure to ultraviolet radiation (UVR) and serves as the scientific rationale for the development of these guidelines. In addition, the International Agency for Cancer Research (IARC) published a monograph on UVR in 1992 (IARC 1992) and published a monograph on sunscreens more recently (IARC/WHO 2001). Furthermore, the National Radiological Protection Board (NRPB) has recently published a scientific review of the health effects of UVR (NRPB 2002). Reviews of relevant UVR biological action spectra were published in a monograph on the measurement of optical radiation hazards (ICNIRP/CIE 1998). The important publications that relate most directly to the guidelines [some of which have appeared since the Environmental Health Criteria (EHC) document was drafted] are referenced in the rationale (Appendix).

The purpose of these guidelines is to provide basic principles of protection against non-coherent ultraviolet radiation, so that they may serve as guidance to the various international and national bodies or individual experts who are responsible for the development of regulations, recommendations, or codes of practice to protect workers and the general public from the potentially adverse effects of UVR.

The Committee recognized that when standards or exposure limits (ELs) are established, various value judgments are made. The validity of scientific reports has to be considered, and extrapolations from animal experiments to effects on humans have to be made. Costs vs. benefit analyses are necessary, including economic impact of controls. The limits in these guidelines were based on the scientific data, and no consideration was given to economic impact or other non-scientific priorities. However, the limits represent conditions under which it is expected that nearly all individuals may be repeatedly exposed without acute adverse effects and, based upon best available evidence, without noticeable risk of delayed effects (see paragraph on Special Considerations). Although a single set of limits can apply for exposure of the eye, it is not possible to provide a single exposure limit that applies to all skin phototypes. Additional guidance is required for applying guidelines for skin protection.

ICNIRP Subcommittee IV (Optical Radiation) prepared the initial update of these guidelines after an extensive review of the current scientific evidence. The IRPA Associate Societies as well as a number of competent institutions and individual experts were consulted in the preparation of the guidelines and their cooperation is gratefully acknowledged.

In its review of the whole database, ICNIRP noted that a substantial number of studies have been published since 1989, when the last detailed rationale for the guidelines was published, and since the UNEP/ICNIRP/WHO EHC was published in 1994. Many of the biological effects, where only tentative data were available in 1994, have now been clarified. In particular, the understanding of UVA-induced damage to DNA by indirect mechanisms, the involvement

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† The initial guidelines were published in Health Phys 49:331–340; 1985, amended in Health Phys 56:971–972; 1989, and reconfirmed by ICNIRP in Health Phys 71:978; 1996.

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of new mechanisms for cell protection against the harmful effects of photosensitized reactions, and the participation of UVA in the chain of events believed to play a role in melanocytic and non-melanocytic skin cancer provide a better understanding of the risk of human exposure to UVR. There is further evidence for the importance of early life (childhood and adolescence) irradiation for melanocytic skin cancer (IARC/WHO 2001) and probably for basal cell carcinoma (Kricker et al. 1995; Gallagher et al. 1995a, b). There has been significant improvement in the understanding of the complex chain of events involved in photocarcinogenesis, e.g., the discovery of a UVR signature at the molecular level (i.e., the p53 gene mutation) (Mukhtar and Elmets 1996; IARC 1992). Progress has also been made in standardizing several action spectra including those for photocarcinogenesis and erythema by the International Commission on Illumination (CIE 1999, 2000, 2002).

It was noted, however, that a number of issues still need further research before a more complete health risk assessment can be made. These include the modulation of the immune system by both UVA and UVB and their interaction with several chromophores; the apparent role of UVA in the development of melanocytic skin cancer; and the role of both UVA and UVB in the development of different types of cataract (UNEP 1994). The International Agency for Research on Cancer (IARC) of the WHO recently reviewed the impact of sunscreens (IARC/WHO 2001).

ICNIRP concludes that, while significant clarification has occurred with respect to health risk assessment from exposure to UVR, recent data do not provide any results suggesting that the exposure limit values contained in Table 1 of the 1989 guidelines need to be amended. This conclusion is supported by a review conducted by the National Radiological Protection Board (NRPB 2002). Thus, ICNIRP reaffirms the 1989 guidelines on exposure limits to UVR as valid for current use. ICNIRP will continue to monitor the scientific literature and amend the guidelines on exposure limits as necessary.

BACKGROUND

Ultraviolet radiation (UVR) occupies that portion of the electromagnetic spectrum from at least 100 to 400 nanometers (nm). In discussing UVR biological effects, the International Commission on Illumination (CIE) has

Table 1. UV exposure limits and spectral weighting function.

λ[a] (nm)	EL[d] (J m^{-2})	EL[d] (mJ cm^{-2})	$S(\lambda)$[b]	λ[a] (nm)	EL[d] (J m^{-2})	EL[d] (mJ cm^{-2})	$S(\lambda)$[b]
180	2,500	250	0.012	310	2,000	200	0.015
190	1,600	160	0.019	313[c]	5,000	500	0.006
200	1,000	100	0.030	315	1.0×10^4	1.0×10^3	0.003
205	590	59	0.051	316	1.3×10^4	1.3×10^3	0.0024
210	400	40	0.075	317	1.5×10^4	1.5×10^3	0.0020
215	320	32	0.095	318	1.9×10^4	1.9×10^3	0.0016
220	250	25	0.120	319	2.5×10^4	2.5×10^3	0.0012
225	200	20	0.150	320	2.9×10^4	2.9×10^3	0.0010
230	160	16	0.190	322	4.5×10^4	4.5×10^3	0.00067
235	130	13	0.240	323	5.6×10^4	5.6×10^3	0.00054
240	100	10	0.300	325	6.0×10^4	6.0×10^3	0.00050
245	83	8.3	0.360	328	6.8×10^4	6.8×10^3	0.00044
250	70	7	0.430	330	7.3×10^4	7.3×10^3	0.00041
254[c]	60	6	0.500	333	8.1×10^4	8.1×10^3	0.00037
255	58	5.8	0.520	335	8.8×10^4	8.8×10^3	0.00034
260	46	4.6	0.650	340	1.1×10^5	1.1×10^4	0.00028
265	37	3.7	0.810	345	1.3×10^5	1.3×10^4	0.00024
270	30	3.0	1.000	350	1.5×10^5	1.5×10^4	0.00020
275	31	3.1	0.960	355	1.9×10^5	1.9×10^4	0.00016
280[c]	34	3.4	0.880	360	2.3×10^5	2.3×10^4	0.00013
285	39	3.9	0.770	365[c]	2.7×10^5	2.7×10^4	0.00011
290	47	4.7	0.640	370	3.2×10^5	3.2×10^4	0.000093
295	56	5.6	0.540	375	3.9×10^5	3.9×10^4	0.000077
297[c]	65	6.5	0.460	380	4.7×10^5	4.7×10^4	0.000064
300	100	10	0.300	385	5.7×10^5	5.7×10^4	0.000053
303[c]	250	25	0.120	390	6.8×10^5	6.8×10^4	0.000044
305	500	50	0.060	395	8.3×10^5	8.3×10^4	0.000036
308	1,200	120	0.026	400	1.0×10^6	1.0×10^5	0.000030

[a] Wavelengths chosen are representative; other values should be interpolated (see Eqns. 2a–c).
[b] Relative spectral effectiveness.
[c] Emission lines of a mercury discharge spectrum.
[d] EL for a monochromatic source, but also limited by a dose-rate of 10 kW m^{-2} (1 W cm^{-2}) for durations greater than 1 s as well in order to preclude thermal effects.

divided the UV spectrum into three bands. The band 315 to 380–400 nm is designated as UVA, 280 to 315 nm as UVB, and 100 to 280 nm as UVC (CIE 1987, 1999). Wavelengths below 180 nm (vacuum UV) are of little practical biologic significance since they are readily absorbed in air. Ultraviolet radiation is used in a wide variety of medical and industrial processes and for cosmetic purposes. These include photocuring of inks and plastics (UVA and UVB), photoresist processes (all UV), solar simulation (all UV), cosmetic tanning (UVA and UVB), fade testing (UVA and UVB), dermatology (all UV), and dentistry (UVA). Even though the principal operating wavelengths for most of these processes are in the UVA, almost always some shorter wavelength (UVB and UVC) radiation and violet light are emitted as well. Many industrial applications employ arc sources for heat or light (e.g., welding), which also produce UVR as an unwanted admixture for which control measures may be necessary. While it is generally agreed that some low-level exposure to UVR benefits health (UNEP 1994; Preece et al. 1975; Clemens et al. 1982; Holick 2000; Webb et al. 1988, 1989; MacLaughlin and Holick 1985), there are adverse effects (de Gruijl 1997; UNEP 1994; ICNIRP/CIE 1998) that necessitate the development and use of ELs for UVR. However, the development of UVR EL poses a real challenge to achieve a realistic balance between beneficial and adverse health effects.

Until 1980, it was generally thought that the most significant adverse UVR health effects resulted from exposures at wavelengths below 315 nm; but today these effects are recognized to be produced at longer wavelengths (UVA) at substantially higher doses. At one time, wavelengths below 315 nm were collectively known as "actinic radiation," when it was thought that these effects occurred only in the UVB and UVC. This guideline has been limited to wavelengths greater than 180 nm where UVR is transmitted through air. The most restrictive limits are for exposure to radiation having those wavelengths less than 315 nm.

PURPOSE AND SCOPE

The purpose of this document is to provide guidance on maximal limits of exposure to UVR in the spectral region between 180 nm and 400 nm. The limits represent conditions under which it is expected that nearly all individuals may be repeatedly exposed without acute adverse effects and, based upon best available evidence, without noticeable risk of delayed effects (see paragraph on Special Considerations). These EL values for exposure of the eye or the skin may be used to evaluate potentially hazardous exposure from UVR; e.g., from arcs, gas and vapor discharges, fluorescent lamps, incandescent sources, and solar radiation. The limits do not apply to lasers that emit UVR. Most incoherent UVR sources are broadband, although single emission lines can be produced from low-pressure gas discharges. These values should be used as guides in the control of exposure to both pulsed and continuous sources where the exposure duration is not less than 1 μs. These ELs are below levels that would be used for UV exposures of patients required as a part of medical treatment or for elective cosmetic purposes. These ELs are exceeded for exposed skin by noonday summer sunlight overhead at 0–40° latitude within 5–10 min. The ELs should be considered absolute limits for direct exposure of the eye and "advisory" for skin exposure because of the wide range of susceptibility to skin injury depending on skin type. The ELs should be adequate to protect lightly pigmented individuals.

BASIC CONCEPTS

This document makes use of the spectral band designations of the CIE. Unless otherwise stated, UVA is from 315 to 400 nm, UVB is from 280 to 315 nm, and UVC is from 100 to 280 nm (CIE 1984, 1987). It should be noted that some specialists follow this general scheme but take the dividing line between UVA and UVB at 320 nm. The UVR exposure should be quantified in terms of an irradiance E (W m^{-2} or W cm^{-2}) for continuous exposure or in terms of a radiant exposure H (J m^{-2} or J cm^{-2}) for time-limited (or pulsed) exposures of the eye and skin. The geometry of exposure to UVR is very important. For example, the eyes (and to a lesser extent the skin) are anatomically protected against UVR exposure from overhead sources such as the sun overhead (Sliney 1995; UNEP 1994). The limits should be applied to exposure directed perpendicular to those surfaces of the body facing the radiation source, measured with an instrument having cosine angular response (UNEP 1994). For highly non-uniform irradiation the irradiance and radiant exposure need not be averaged over the area of a circular measurement aperture smaller than 1 mm in diameter for pulsed exposures and 3.5 mm for lengthy exposures.

These ELs should be used as guides in the control of exposure to UV sources and as such are intended as limits for non-therapeutic and non-elective exposure. The ELs should be considered as absolute limits for ocular exposure. The ELs were developed by considering lightly pigmented populations (i.e., white Caucasian) with greatest sensitivity and genetic predisposition for skin cancer. Exposure during sun bathing and tanning under artificial sources may well exceed these limits but

exposed individuals should be advised that some health risk is incurred from such activity. Eye protection is always required during therapeutic exposures. Nevertheless, occasional exposures to conditioned skin may not result in adverse effects. The rationale for the UVR exposure limits is provided in the Appendix.

EXPOSURE LIMITS

For the EL for both general and occupational exposure to UVR incident upon the skin or eye within an 8-h period, the following applies.

Exposure of the eyes

Ultraviolet radiant exposure in the spectral region 180 to 400 nm incident upon the unprotected eye(s) should not exceed 30 J m^{-2} effective spectrally weighted using the spectral weighting factors contained in Table 1, and the total (unweighted) ultraviolet radiant exposure in the spectral region 315 to 400 nm should not exceed 10^4 J m^{-2}.

Exposure of the skin

For the most sensitive, non-pathologic, skin phototypes (known as "melano-compromised"), ultraviolet radiant exposure in the spectral region 180 to 400 nm upon the unprotected skin should not exceed 30 J m^{-2} effective spectrally weighted using the spectral weighting factors contained in Table 1. This limit should be considered a desirable goal for skin exposure to minimize the long-term risk, but it must be recognized that this limit is difficult to achieve in sunlight and judgment must be used in its practical application. It has a very substantial safety factor for dark skin phototypes (known as "melano-competent") and more generally for individuals who have been conditioned by previous, repeated exposures (known as "melano-adapted," i.e., tanned).

To determine the effective irradiance of a broadband source weighted against the peak of the spectral effectiveness curve (270 nm), the following weighting formula should be used:

$$E_{eff} = \Sigma\, E_\lambda \cdot S(\lambda) \cdot \Delta\lambda, \quad (1)$$

where:

E_{eff} = effective irradiance in μW cm^{-2} (μJ s^{-1} cm^{-2}) or W m^{-2} (J s^{-1} m^{-2}) normalized to a monochromatic source at 270 nm;
E_λ = spectral irradiance from measurements in μW cm^{-2} nm^{-1} or W m^{-2} nm^{-1};
$S(\lambda)$ = relative spectral effectiveness (unitless); and
$\Delta\lambda$ = bandwidth in nanometers of the calculation or measurement intervals.

Permissible exposure time in seconds for exposure to UVR incident upon the unprotected skin or eye may be computed by dividing 30 J m^{-2} by the value of E_{eff} in W m^{-2}. The maximal exposure duration may also be determined using Table 2, which provides representative exposure durations corresponding to effective irradiances in W m^{-2} or μW cm^{-2}.

Values of $S(\lambda)$ for wavelengths that are not listed in Table 1 may be interpolated through the application of the following three formulas (Wester 2000). The three simple mathematical expressions apply in the range only from 210–400 nm:

For the region

$$210 \le \lambda \le 270 \text{ nm } S(\lambda) = 0.959^{(270-\lambda)} \quad (2a)$$

For the region

$$270 < \lambda \le 300 \text{ nm } S(\lambda) = 1 - 0.36x\left(\frac{\lambda - 270}{20}\right)^{1.64} \quad (2b)$$

For the region

$$300 < \lambda \le 400 \text{ nm } S(\lambda) = 0.3 \times 0.736^{(\lambda-300)} + 10^{(2-0.0163\lambda)}. \quad (2c)$$

The formulae interpolate between and substitute with reasonable accuracy the points along the action spectrum.

SPECIAL CONSIDERATIONS

These EL values are intended to apply to UVR exposure of the working population, but with some precaution also apply to the general population. However, it should be recognized that some rare, highly

Table 2. Limiting UV exposure durations based on exposure limits.

Duration of exposure per day	Effective irradiance	
	E_{eff} (W m^{-2})	E_{eff} (μW cm^{-2})
8 h	0.001	0.1
4 h	0.002	0.2
2 h	0.004	0.4
1 h	0.008	0.8
30 min	0.017	1.7
15 min	0.033	3.3
10 min	0.05	5
5 min	0.1	10
1 min	0.5	50
30 s	1.0	100
10 s	3.0	300
1 s	30	3,000
0.5 s	60	6,000
0.1 s	300	30,000

photosensitive individuals exist who may react adversely to exposure at these levels. These individuals are normally aware of their heightened sensitivity. Likewise, if individuals are concomitantly exposed to photosensitizing agents (Fitzpatrick et al. 1974; Johnson 1992), a photosensitizing reaction can take place. It should be emphasized that many individuals who are exposed to photosensitizing agents (ingested or externally applied chemicals, e.g., in cosmetics, foods, drugs, industrial chemicals, etc.) probably will not be aware of their heightened sensitivity. Phototoxic reactions apply to all individuals and depend upon the quantity of photosensitizing chemicals and the UVR exposure, whereas photoallergic reactions will be observed for much lower quantities of the substance in sensitized individuals. Lightly pigmented individuals conditioned by previous UVR exposure (leading to tanning and hyperplasia) and heavily pigmented individuals can tolerate skin exposure in excess of the EL without erythemal effects. However, repeated tanning may increase the risk for those persons later developing signs of accelerated skin aging and even skin cancer. Such risks should be understood prior to the use of UVR for medical phototherapy or cosmetic exposures.

PROTECTIVE MEASURES

Protective measures will differ depending upon whether the UVR exposure results from sunlight or from artificial sources. The use of hats, eye protectors, clothing, and sun-shading structures are practical protective measures to reduce sunlight exposure. When these measures are inadequate, topical sunscreens should be applied to the skin. However, the value of sunscreens has been questioned, and an IARC Working Group on the Evaluation of Cancer-Preventive Agents concluded that there was inadequate epidemiological evidence in humans for a cancer-preventive effect of topical use of sunscreen formulations against cutaneous malignant melanoma, or basal-cell carcinoma, despite the experimental evidence in animal studies (IARC/WHO 2001).

When exposure is to artificial sources, as in some industrial hazard situations, engineering control measures are preferable to protective clothing, goggles, and procedural safety measures. Glass envelopes for arc lamps will filter out most UVB and UVC. Where lengthy exposure to high power glass-envelope lamps and quartz halogen lamps will occur at close proximity, additional glass filtration may be necessary (McKinlay et al. 1989). Light-tight cabinets and enclosures and UVR absorbing glass and plastic shielding are the key engineering control measures used to prevent human exposure to hazardous UVR produced in many industrial applications such as the fade testing of materials, solar simulation, photoresist applications, and photocuring. For arc welding, cabinets are not practical. Shields, curtains, barriers, and a suitable separation distance are used to protect individuals against the UVR emitted by open-arc processes such as arc welding, arc-cutting, and plasma spraying. Dynamic-filter welding helmets and see-through curtains have improved the safety of welding operations in recent decades. There is a need for operational rules to protect potentially exposed individuals. Operators should be trained to follow these general rules properly. Ventilation may be required to exhaust ozone and other airborne contaminants produced by UVC radiation.

MEASUREMENT

UV measurements for health risk evaluation are sometimes of value for indoor exposure assessment. However, they are generally not routinely performed for outdoor exposure conditions, except with regard to the use of the Global UV Index (ICNIRP/WHO/WMO/UNEP 2002; Gies et al. 1995).

Although direct-reading UVR radiometers exist, attempts to produce relatively inexpensive field safety survey meters that respond directly to UVB and UVC radiation [following the $S(\lambda)$ function] have not been fully successful. However, relatively expensive instruments exist which respond to UVB and UVC radiation according to the relative spectral effectiveness, $S(\lambda)$. Spectroradiometric measurements of the source which can then be used with the $S(\lambda)$ weighting function to calculate E_{eff} are often necessary for measurements more accurate than those with simple, direct-reading safety meters. Whichever measurement technique is applied, the geometry of measurement is important. All the preceding ELs for UVR apply to exposures that are measured with an instrument having a cosine-response detector oriented perpendicular to the most directly exposed surfaces of the body when assessing skin exposure. The detector is oriented along (or parallel to) the line(s) of sight of each exposed individual when assessing ocular exposure. The use of UV film badges makes it possible to integrate UV exposure on specific body sites which move with respect to the UVR source (Diffey et al. 1977; Saunders and Diffey 1995); however, the spectral response of such film badges still does not accurately follow $S(\lambda)$.

For outdoor exposure, environmental UVR measurements may be of limited use for individual dose assessment because of geometrically changing exposure

conditions and human behavioral considerations. Personal dosimeters must properly take into consideration the exposed sites of the individual, time of exposure, sun angle, etc. The Global UV Index can be a useful tool in educating persons who are outdoors as to the changing level of overhead UVR. It is, however, not very predictive of ocular exposure since it is a measure of the overhead UVR incident on a horizontal surface. Ocular exposure is highly dependent upon ground reflectance factors and the upper lid and brow-ridge block most overhead UVR (Sliney 1995).

CONCLUDING REMARKS

Greater attention should be paid to the potential hazards of UVR exposure. The increasing socially driven solar exposure as well as the increasing use of artificial UVR sources is a cause for concern. In many populations, skin cancer incidence continues to rise, due in large part to a poor appreciation of the risk among the general population. Reduction of risk by avoidance of needless sunlight exposure and by physical means of protection should be an important public health goal. Improved educational programs are needed for school children, for outdoor workers and the general public. The present understanding of injury mechanisms and long-term effects of exposure to UVR is incomplete, and awaits further research. The above guidelines will be subject to periodic review and amendment as appropriate.

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During the preparation of these guidelines, the composition of the International Commission on Non-Ionizing Radiation Protection was as follows:
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APPENDIX: RATIONALE FOR THE LIMITS OF EXPOSURE TO UVR

Background

Comprehensive reviews of UVR effects have been published in conjunction with the United Nations Environment Program and the World Health Organization (UNEP 1979, 1994), and the interested reader is referred to those documents in particular. The CIE and ICNIRP also reviewed UVR effects and action spectra in a monograph on optical radiation measurements (ICNIRP/CIE 1998). In addition, the International Agency for Cancer Research (IARC) published a monograph on UVR in 1992 (IARC 1992) and published a monograph on sunscreens more recently (IARC/WHO 2001). Furthermore, the National Radiological Protection Board (NRPB) has recently published a scientific review of the health effects of UVR (NRPB 2002). The following discussion is a brief review of those physical and biological factors used to derive the UVR guidelines.

General approach

Life has evolved under the daily exposure to solar radiation. Although UVR is only about 5% of the solar spectrum that reaches the earth's surface, it plays a significant biological role since individual photon energies are the greatest within the optical spectrum. These shorter-wavelength, higher energy photons have sufficient energy to produce photochemical alterations that may initiate biological effects that are potentially injurious (sometimes referred to as "actinic effects"). Both beneficial and unwanted photobiological effects result from UVR exposure. The critical organs for UVR exposure are the eye and the skin since they may be readily exposed.

The approach taken in the development of these guidelines was to limit exposure to preclude any significant acute photobiologic effects and reduce the risks for delayed effects from chronic exposure as much as possible, based upon the best available evidence. Thresholds for observed bioeffects vary strongly with wavelength. Consequently, various spectral dose-response relationships and time-dependent dose-response relations have been developed. In photobiology, the term "action spectrum" refers to the relative spectral effectiveness of different wavelengths in eliciting a biological effect. Available data on the action spectra and dose response curves for each delayed effect were reviewed with the goal of estimating risk at exposure levels below those producing acute effects. Furthermore, the dose and action spectra for beneficial effects, such as vitamin-D synthesis, were examined to assure that the exposure guidelines did not lead to an inadequate level of exposure.

Both the acute skin response (erythema) and long-term risk of skin cancer appear to be related to DNA damage (de Gruijl and van der Leun 1994; Cesarini 1996; Kelly et al. 2000). In theory, only a single UV photon is required to alter directly a single DNA molecule (or indirectly through free-radical production). However, DNA repair mitigates most single-molecule events. An enormous number of DNA lesions are produced in one single cell nucleus at the basal epidermal layer in skin irradiated at one minimal erythemal dose. The three major categories of photo-lesions induced in single cell in culture (hamster ovary mesenchyme) have been identified and calculated after exposure to a solar simulated source. The delivered dose corresponded to the dose received on the skin basal layer from 2 h exposure to a UV-index 6 (Paris mid-summer). The total number of lesions was around 100,000 photolesions in a single cell for one standard erythema dose (SED) (Perdiz et al. 2000). In practice, laboratory techniques exist to detect UVR-induced changes down to the cellular and molecular level (e.g., Marrot et al. 1998). These can detect DNA lesions and errors in repair (mutations). UVR exposure produces specific types of DNA damage that result in "UV signature" mutations, which can be detected in skin carcinomas (Brash et al. 1991). Such mutations in the p53 gene can serve as a biomarker for past exposure and future carcinoma risk (Nakasawa et al. 1994; Ziegler et al. 1994). The commonly experienced "sunburn" (erythema) may be used as a marker for the presence of substantial UVR-induced DNA damage (Young and Walker 2000). Through DNA damage, erythema is related to skin cancer; and a knowledge of erythema-dose response and action spectra are of value for developing guidance that might reduce risk from skin cancer.

Acute responses of the skin—Erythema

Erythema (i.e., the reddening of the skin as in sunburn) is the most commonly observed direct effect observed in the skin following exposure to UVR. UVR-induced erythema results from photochemical damage—principally to DNA—that leads to a cascade of molecular events resulting in redness due to an increased blood content of the skin by dilatation of the superficial blood vessels. Unlike the erythema from a thermal insult, the erythema from UVR is delayed by some (1–6) hours after the exposure. The duration of the delay is reduced as the exposure dose increases.

Erythema action spectra have been the subject of experimental and theoretical interest for over 70 y (Urbach 1998); and upon first review, there appear to be many different, and even contradictory, action spectra. The apparent differences relate to different methods of assessment, types of monochromatic sources, clinical endpoint, time of assessment, etc. The International Commission on Illumination (CIE) reference action spectrum $E(\lambda)$ as shown in Fig. A1, is routinely used today to convert absolute UV exposure levels into erythemally effective irradiance (CIE 1998; McKinlay and Diffey 1987), but this is based upon specific reference conditions. An historical perspective is useful to understand the differences in published action spectra and the significance of these differences.

Hausser and Vahle (Hausser 1928; Hausser and Vahle 1927) were the first to document quantitatively erythema as a wavelength-dependent effect in the late 1920's. By 1935, the CIE had recommended an early "standardized" erythemal action spectrum based upon several studies using a limited number discrete monochromatic emission lines of the mercury lamp (e.g., 254 nm, 280 nm, 297 nm, 303 nm, 313 nm, etc.), and this limited the full spectral detail (Coblentz et al. 1931; Hausser 1928; Luckiesh et al. 1930; Urbach 1998). With the development of xenon-arc lamps and their use with

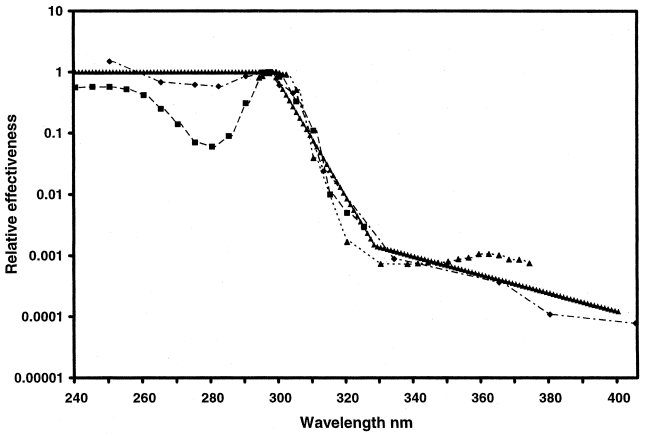

Fig. A1. Erythemal action spectra. The CIE (1998) reference action spectrum for erythema in human skin (solid line), an erythema action spectrum (Anders et al. 1995) determined using dye lasers (triangles), and the CIE (1935) action spectrum (squares) are shown with the action spectrum of human skin adapted from Parrish et al. (1982) for 8 h after irradiation (diamonds). If measured at 24 h, the MED differs below 300 nm.

monochromators in the 1960's, several groups conducted studies to fill in the missing spectral information (Everett et al. 1965; Freeman et al. 1966; Berger et al. 1968), but these appeared to differ somewhat from the "classical" studies of the 1930's—particularly at wavelengths less than 300 nm. The short-wavelength variations were the result of different end-points (Urbach 1998; Hausser 1928). The use of high-pressure xenon-arc lamps and xenon-mercury arc lamps with monochromators filled in missing spectral regions, but also introduced a new problem, since the 5–10 nm spectral bandwidths used in these studies lowered the spectral resolution. Later, highly monochromatic studies with lasers were able to refine the spectral detail in the 300–320 nm spectral region where the action spectrum was rapidly changing (Anders et al. 1995). These and other quantitative studies have confirmed that the erythemal threshold varies with anatomical site, wavelength, and time between exposure and assessment (Willis et al. 1972; van der Leun and Stoop 1969; Paul and Parrish 1982; Parrish et al. 1978; McKinlay and Diffey 1987; CIE 1999). In addition, the variation in published threshold values results from differences in the clinical definition and estimate of "minimal erythema" and radiometric measurement techniques. Despite the steeper slope between 300 nm and 315 nm found in laser studies (Anders et al. 1995), Diffey concluded from a mathematical analysis of the action spectra obtained with monochromators and lasers that the CIE reference action spectrum was a valid predictor of the erythemal effectiveness of different wavelengths of ultraviolet radiation (Diffey 1998).

Exposure to UVA alone can produce erythema, but only at very high radiant exposures (i.e., at >10 J cm^{-2}, i.e., 100 kJ m^{-2}) as shown by more recent studies (Diffey et al. 1987; Parrish et al. 1982; Anders et al. 1995). Multiple-wavelength exposures (as within broad-spectrum exposures such as from sunlight) are additive in producing erythema. Deviations from additivity have been reported, such as photoaugmentation (Willis et al. 1972), or the opposite effect, photoprotection (Van der Leun and Stoop 1969; Paul and Parrish 1982), but their importance is considered marginal.

Individuals can be grouped into one of six sun-reactive skin types as shown in Table A1 (Fitzpatrick 1975). These skin types fall into three more significant groups: melano-compromised, melano-competent, and melano-protected (ICNIRP 2003). Individual susceptibility to both acute and delayed effects varies markedly with skin phototype and exposure history. Skin pigmentation ("tanning") and "conditioning" (thickening of the stratum corneum and tanning) may result in an increase of an individual's minimal erythemal doses (MED) by a factor of at least four for a UVB source (Gange et al. 1986). For individuals with melanocompromised skin, sunburn and tanning are obtained for a single exposure dose, and there is no tanning without burning. However, for individuals with melanocompetent skin, a significant tan may be

Table A1. Skin phototypes.

Phototype	Skin response to sunlight	Typical appearance
I	Burns easily and severely (painful burn); tans little or none and peels	People with very fair skin, blue eyes, freckles; unexposed skin is white
II	Usually burns easily and severely (painful burn); tans minimally or lightly, also peels	People most often with fair skin, red or blond hair, blue, hazel or even brown eyes; unexposed skin is white
III	Burns moderately and tans	People with white skin when unexposed; generally dark hair
IV	Burns minimally, tans easily	People with white or light brown unexposed skin, dark hair, dark eyes
V	Rarely burns, tans easily and substantially	People with brown skin
VI	Never burns and tans profusely	People with black skin

obtained without burning—most notably for UVA wavelengths. Skin color and other phenotype characteristics (hair color, eye color, and freckles) are associated with the susceptibility to sunburn (Andreassi et al. 1987; Azizi et al. 1988). Because the MED varies with each individual, the CIE standard erythema dose (SED) unit was introduced for objective UVR dosimetry of skin effects (CIE 1998). Erythemal thresholds as reported in studies for untanned, lightly pigmented skin, range from about 1.5 to 3 SED, i.e., 15 to 30 mJ cm^{-2} as weighted by the CIE standard action spectrum for erythema (CIE 1998; Everett et al. 1965; Freeman et al. 1966; Parrish et al. 1982; Cox et al. 1990; Diffey 1994). The ICNIRP guideline values are therefore approximately 2 to 4 times less than these MED values. Fig. A1 also illustrates the results of one study of the variation of erythema action spectrum (Parrish et al. 1982). The six sun-reactive skin types shown in Table A1 (Fitzpatrick 1975; Andreassi et al. 1987) were based on a personal history of response to 45–60 min of exposure to midday summer sun in early summer.

There are anatomical differences in erythemal sensitivity. The face, neck, and trunk are two to four times more sensitive than the limbs (Olson et al. 1966). There is no difference in sunburn susceptibility between sexes. Although there have been suggestions that erythemal sensitivity may change with age, and that young children and elderly people are said to be more sensitive (Hawk and Parrish 1982), quantitative studies of erythemal sensitivity in subjects of these age groups have not confirmed this (Cox et al. 1990).

The MEDs in a given spectral waveband and for a normal population have a positively skewed distribution (Mackenzie 1983). Values for the MED should therefore be expressed as the median, or geometric mean, rather than the arithmetic mean. Examples of MEDs determined in a population of 252 subjects (skin types I, II and III) are given in Table A2 (Diffey 1994).

Cellular damage can be detected at levels below the MED. At approximately 0.1 MED, it is possible to detect activation of p53 protein and p21 gene expression, which indicate a cellular response (Ponten et al. 1995). At approximately one-third MED sunburn cells and immune suppressive effects can be detected (Jeevan et al. 1995; Kelly et al. 1998). Table A3 summarizes the cellular responses to increasing dose levels at different MED values.

Table A2. Examples of minimal erythemal doses.

Central wavelength nm	Bandwidth (FWHM[a]) nm	Median MED J cm^{-2}	95% range J cm^{-2}
300	5	0.027	0.015–0.051
320	10	1.9	1.0–3.4
330	15	5.6	3.1–10
350	30	19	11–35
370	30	27	16–47
400	30	62	38–102

[a] Full-width at half-maximum.

Long-term effects on the skin

Chronic exposure to the UVR in sunlight accelerates the skin aging process and increases the risk of developing skin cancer (NRPB 2002). The solar spectrum is greatly attenuated by the earth's ozone layer, limiting terrestrial UV to wavelengths greater than approximately 290 nm. The UVB irradiance at ground level is a strong function of the sun's elevation angle in the sky. This results from the change of UV attenuation with atmospheric path length (time of day and season). Several ecological epidemiologic studies showed that the incidence of skin cancer is strongly correlated with latitude, altitude, and cloud cover (UNEP 1979). Exact quantitative dose-response relationships have not yet been established although fair-skinned melanocompromised individuals, especially of Celtic origin, are much more prone to develop skin cancer. Since the discovery by Valverde and associates of polymorphism in the alpha-melanocytic stimulating hormone (α-MSH) receptor associated with red-haired phenotypes and extreme photosensitivity, it has also been shown that polymorphisms in this receptor are an important risk determinant for all types of skin cancers (Bastiaens et al. 2001; Rees 2000; Valverde et al. 1996).

Prior to 1970, skin cancer was typically a disease of outdoor workers such as farmers and seamen routinely exposed to sunlight (Urbach et al. 1974), however, because of the change in social activity, it has become a disease of the general public whose exposure is largely intermittent from recreational exposure (Cesarini 1996). This change is important in interpreting epidemiological studies of skin cancer because of the different exposure patterns. Only a few quantitative studies have examined

Table A3. Dose response values.

Exposure level	Effect	Reference
0.1 MED	*p*53 and *p*21 activated	Ponten 1995
0.3 MED	Sunburn cells just detectable	Cesarini 1996
0.3 MED	Immunosuppressive effect in melanocompromised individuals	Kelly 1998
0.5 MED	Modification and depletion of Langerhans cells	Cooper 1992
1.0 MED	20 sunburn cells/cm^2	Cesarini 1996
1.0 MED	Immunosuppressive effect in melanocompetent individuals	Kelly 1998
2 MED	150 sunburn cells/cm^2	Cesarini 1996
3 MED	400–500 sunburn cells/cm^2	Cesarini 1996
6–10 MED	Blistering	Everett 1965

indoor working populations chronically exposed to artificial sources of UVB to determine whether there is an increased skin cancer risk in this occupational environment. Squamous cell carcinoma is the most common type in the outdoor working population. This is localized at exposed sites (e.g., hands and back of the neck) and this is suggestive of the importance of total cumulative exposure. Studies of the incidence of melanoma in outdoor workers show a lower incidence than for indoor workers (Armstrong and Kricker 1993; IARC 1992; UNEP 1994).

Types of skin cancer

The three common forms of skin cancer, listed in ascending order of severity, are basal cell carcinoma (BCC), squamous cell carcinoma (SCC), and malignant melanoma (MM). SCC is also known as spindle-cell carcinoma. Around 90% of skin cancer cases are of the non-melanoma variety (BCC and SCC) with BCCs being approximately four times as common as SCCs. The overall lifetime risk of any type of skin cancer varies with ethnic status and geography, but, as an example, the cumulative lifetime risk of developing MM is 1:90 for a white American. This risk increases to 1:7 for SCC and BCC in the same population (Parkin et al. 1997).

Exposure to UVR is considered to be a major etiological factor for all three forms of skin cancer (IARC 1992). For basal cell carcinoma and malignant melanoma, neither the wavelengths involved nor the exposure pattern that results in risk have been established with certainty; whereas for squamous cell carcinoma, UVB and probably UVA are implicated and the major risk factors seem to be cumulative lifetime exposure to UV radiation and a poor tanning response.

Squamous cell cancer

The evidence that exposure to solar radiation is the predominant cause of squamous cell cancer in man is very convincing. These cancers occur almost exclusively on sun-exposed skin such as the face, neck and arms, and the incidence is clearly correlated with geographical latitude, being higher in whites in the more equatorial areas of the world (Kricker et al. 1994). Recent epidemiological studies and a randomized trial suggest that sun exposure in the 10 years prior to diagnosis may be important in accounting for individual risk of SCC (Gallagher et al. 1995a; English et al. 1996; Green et al. 1999).

Basal cell cancer

The relationship between basal cell carcinoma and sunlight is less compelling, but the evidence is sufficiently strong to consider it also to be a consequence of exposure to sunlight. Whilst SCC is strongly related to cumulative lifetime exposure to sunlight, this relationship is not so convincing for BCC (Gallagher et al. 1995b), and it may be that intermittent sun exposure and perhaps exposure in childhood and adolescence may be critical for establishing adult risk for BCC (Kricker et al. 1995; Gallagher et al. 1995b).

Action spectrum for non-melanoma skin cancer

At present, an action spectrum for skin cancer can only be obtained from animal experiments. The most extensive investigations to date are those from groups at Utrecht and Philadelphia. These workers exposed a total of about 1,100 hairless albino mice to 14 different broad-band ultraviolet sources and by a mathematical optimization process derived an action spectrum referred to as the Skin Cancer Utrecht-Philadelphia (SCUP) action spectrum (de Gruijl and van der Leun 1994). The SCUP action spectrum is that for skin tumor induction in hairless mice, a species with a thinner epidermis than humans. By taking into account differences in the optics of human epidermis and hairless albino mouse epidermis, the experimentally determined action spectrum for tumor induction in mouse skin can be modified to arrive at a postulated action spectrum for human skin cancer (de Gruijl and van der Leun 1994). The resulting action spectrum resembles the action spectrum for erythema (Fig. 1). The CIE has recently published a "standardized" action spectrum based upon this work (CIE 1999).

Malignant melanoma

During the past 40 years or so there has been an increase of the order of a doubling in each decade in the incidence of cutaneous malignant melanoma in white populations in several countries. There exists an inverse relationship between latitude and melanoma incidence; and this, plus many other factors, has been taken as evidence for a possible role of sunlight as a cause of malignant melanoma. However, this pattern is not always consistent. In Europe, for example, the incidence and the mortality rates in Scandinavia are considerably higher than those in Mediterranean countries. This inconsistency may reflect ethnic differences in constitutional factors and customs. Also, the unexpectedly low incidence in outdoor workers, the sex and age distribution, and the anatomical distribution have pointed to a more complex association (Armstrong and Kricker 1994).

There is now growing evidence that intermittent sun exposure, mainly from recreational activities, is associated with increased risk of developing malignant melanoma. Several studies have found that a history of sunburn is associated with risk for melanoma development, although in these studies a potential for recall bias

exists (Elwood and Jopson 1997) and may be confounded by skin type. Studies of migrants have led to the suggestion that sun exposure in childhood and adolescence is a particularly critical period in terms of melanoma risk.

Action spectrum for melanoma

At one time, the only data that existed for an action spectrum for melanoma induction were those obtained from irradiating hybrids of a small tropical fish with different wavelengths of UVR (Setlow et al. 1993). This fish action spectrum suggested that all wavelengths of UV radiation could be important in melanoma, unlike non-melanoma skin cancer; however, at least one attempt to replicate this action spectrum was unsuccessful (Anders et al. 1994). More recent studies in transgenic mice (Noonan et al. 2001) and in *monodelphis domestica* (Robinson et al. 2000) indicate that neonatal UV exposure is most significant. In contrast to small UVB doses, Robinson and colleagues also found that large doses of UVA to neonates could not produce tumors (Robinson et al. 2000). Melanoma incidence is also extremely high in xeroderma pigmentosa (X-P) patients, who lack the capacity to repair UVB induced damage (Kraemer 1997). The weight of current evidence now suggests that UVB is the primary risk factor for MM.

Ocular effects—Photokeratoconjunctivitis

Short-wavelength UVR ($\lambda < 300$ nm) is strongly absorbed by the cornea and conjunctiva. Excessive exposure of these tissues causes photokeratoconjunctivitis, commonly referred to as "welder's flash," "arc-eye," etc. Several research groups have characterized the course of ordinary clinical photokeratitis (Pitts 1993) and the cellular changes in ocular tissues (Ringvold et al. 1982). The latent period varies inversely with the severity of exposure ranging from ½ to 24 h but usually occurs within 6–12 h. Conjunctivitis tends to develop more slowly and may be accompanied by erythema of the facial skin surrounding the eyelids. The individual has the sensation of a foreign body or sand in the eyes and may experience photophobia, lacrimation, and blepharospasm to varying degrees. The acute symptoms last from 6 to 24 hours and discomfort usually disappears within 48 h. Although exposure rarely results in permanent ocular injury, the individual is visually incapacitated during this 48-h period. Pitts and Tredici (1971) reported threshold data for photokeratitis in humans for 10 nm wavebands from 220 to 310 nm (Pitts 1993). The guideline ELs between 200 nm and 305 nm are about 1.3 to 4.6 times less than the threshold for minimal change. The maximum sensitivity of the human eye was found to occur at 270 nm. The wavelength response (action spectrum) between 220 and 310 nm does not vary as greatly as in the case of erythema with the thresholds varying from 4–14 mJ cm^{-2}. Sliney and colleagues used an excimer laser to determine the photokeratitis threshold at 193 nm (Sliney et al. 1991). Corneal injury from UVA wavelengths requires levels exceeding 10 J cm^{-2} (Hamerski 1969; Pitts 1993; Sherashov 1977; Tapaszto and Vass 1969; Zuclich and Kurtin 1977; Zuclich 1980; Cullen and Perera 1994).

Cataract

Wavelengths above 295 nm can be transmitted through the cornea and are absorbed by the lens. Pitts et al. (1977) have shown that both transient and permanent opacities of the lens (cataracts) can be produced in rabbits and primates by exposure to UVR having wavelengths in the 295–320 nm band. Similar findings were reported for the rat (Soderberg 1990). Thresholds for transient opacities ranged dramatically with wavelength, from 0.15 to 12.6 J cm^{-2}. Thresholds for permanent opacities were typically twice those for transient opacities (Pitts 1993). Experimental methods cannot readily show a threshold, since a measure of increased scatter is difficult when there is a background level of scattering (Michael et al. 1998). The action spectrum for UVR induced cataract was recently confirmed in the rat by use of a quantitative criterion for light scattering (Merriam et al. 2000). Opacities from chronic exposure at lower levels has been very difficult to show experimentally (Jose and Pitts 1985; Zigman 1993). However, several epidemiological studies show an association between the incidence of cortical opacities with ambient UVB exposure (Hiller et al. 1977; Taylor et al. 1988; West et al. 1998). Sasaki has shown a clear correlation of different forms of cataract with latitude, but does not explicitly link this with the change of UVR exposure with latitude (Sasaki et al. 2002), although it was speculated that both temperature and UVR could be etiologic factors (Sliney 2002). A number of biochemical studies of the effects of UV irradiation of lens proteins have led to the theory that UVA radiation is a causal factor in cataract (Pirie 1971; Roberts 2001; Young 1994). However, it has been difficult to link UVA radiation with cataract either epidemiologically or experimentally.

Retinal effects

The cornea and crystalline lens normally sufficiently shield the retina from acute effects from UVR exposure. Normally, less than 1% of UVA reaches the retina, shorter UVB wavelengths being totally attenuated except in neonates (UNEP 1994). Upon removal of the crystalline lens, Ham and colleagues (Ham et al. 1982) demonstrated acute retinal injury (photoretinitis) at levels of the

order of 5 J cm^{-2} at the retina. Photoretinitis at these wavelengths is covered by the ICNIRP guidelines for exposure to incoherent optical radiation (ICNIRP 1997).

Envelope action spectrum

Clearly, the development of UVR exposure limits for workers and the general population must consider two risks. These are the risks of acute and chronic injury to both the eye and skin. The literature indicates that thresholds for injury vary significantly with wavelength for each effect. In the UVB and UVC regions, an action spectrum curve can be drawn which envelops the threshold data for exposure doses (radiant exposures) in the range of reciprocity (Schmidt 1964; Zuclich 1980) for acute effects obtained from recent studies of minimal erythema and keratoconjunctivitis. Reciprocity means that irradiance E and exposure duration t have a reciprocal relation, and a constant product of E and t (i.e., exposure) results in a given effect. This EL curve does not differ significantly from the collective threshold data considering measurement errors and variations in individual response (Sliney 1972; Sliney and Wolbarsht 1980). Although the safety factor is minimal for just-detectable increases in corneal scatter, it is believed to range from 1.5 to 2.0 for acute keratitis. The curve is also well below the acute UVB cataractogenic thresholds (Merriam et al. 2000; Pitts 1993). Repeated exposure of the eye to potentially hazardous levels of UV is not believed to increase significantly the protective capability of the cornea as does skin tanning and thickening of the stratum corneum [although some recent studies show a detectable change in threshold (Ringvold 1997)]. Thus, this EL is more readily applicable to the eye and must be considered a limiting value for that organ (Sliney 1972). Any accumulation of UVB and UVC exposures causing photokeratitis is limited to about 48 h since the outer corneal epithelial layers are replaced in about 48 h by the normal repair process of this tissue. Some slight additivity of UVA exposures exists beyond 48 hours because of the deeper penetration of UVA rays (Zuclich 1980). The additivity factors were considered in deriving the magnitude of the safety factor built into the guidelines. On the basis of acute effects, the safety factor for UVA guidelines is large, varying from about 7 at 320 nm to more than 100 at 390 nm.

Because of the wide variations in threshold values and exposure history (conditioning) among individuals, these guidelines should only be used as a starting point for evaluating skin hazards (Despres 1978; Gezondheidsraad 1978; NRPB 2002; Sliney and Wolbarsht 1980; UNEP 1994). The envelope guideline has some margin of safety to protect all but the most sensitive individuals. An exact value for this margin cannot be given, but for lightly pigmented persons (i.e., melanocompromised skin phototypes I and II) it varies from about 3 to 20 depending on the spectral composition of the radiation. Since there may be more than one target molecule (chromophore) involved in erythema (and therefore more than one erythemal action spectrum), the effect of radiations of two widely differing wavelengths in the 180 nm to 315 nm range may not be simply additive. The EL should be used with caution in evaluating sources such as the sun and fluorescent lamps, having a rapidly increasing spectral irradiance in the 300–315 nm range. Large errors can arise because of the difficulty in making accurate spectral measurements of such sources in this region (Sliney and Wolbarsht 1980; ICNIRP/CIE 1998).

The EL may not provide adequate protection for photosensitive individuals or for normal individuals exposed concomitantly to chemical, pharmaceutical, or phyto-photosensitizers, and special precautions must be taken for such cases (Dahaw-Barker 1987; Ferguson 1998).

Based upon current knowledge, the EL should prevent significant acute effects and reduce the magnitude of chronic skin effects by limiting life-long UV exposure. The action spectrum for each type of skin cancer is still debated, although most research suggested that at least squamous-cell carcinoma is probably related (both directly and indirectly) to UV-induced molecular damage to DNA, and the action spectrum is similar to that of the erythemal action spectrum. Indeed, a Technical Committee of the CIE has proposed a tentative action spectrum for photocarcinogenesis (CIE 1999). The Dutch Health Council (Health Council of the Netherlands 1986) was the first to propose envelope limits similar to the guidelines developed for acute daily exposure (up to 10 y duration) and reduced levels for longer periods to protect against chronic effects. These should have the same action spectrum in the UVB and UVC. In many cases, occupational exposure to UVB adds to an individual's non-occupational exposure to solar UVB.

It is worthy of note that in addition to the direct hazard of UV exposure, very intense UVC sources (particularly of wavelengths less than 230 nm) may also produce hazardous concentrations of ozone and nitrogen oxides from the air and of phosgene gas in the presence of degreasers; thus, many UV germicidal lamps now have quartz-glass envelopes that block wavelengths below ~230 nm.

UVA radiation effects

Studies of skin and ocular injury action spectra (Fig. A2) in the UVA spectral region (315–400 nm) show very similar thresholds for acute injury (Anders et al. 1995; McKinlay and Diffey 1987; Parrish et al. 1982; Pitts et al. 1977; Zuclich 1989). These data are sufficient to define the relative spectral effectiveness, $S(\lambda)$, for exposure guidelines up to 400 nm. However, if radiant energy were to be delivered to the skin or ocular tissues sufficiently fast for a substantial temperature

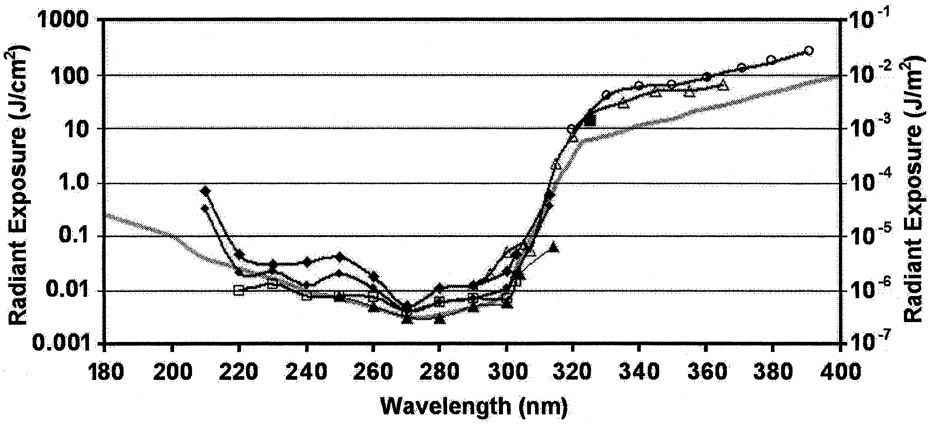

Fig. A2. Ocular action spectra. The ICNIRP UV guideline for exposure is depicted by the shaded, solid line. The data for primate cornea of Pitts and Tredici (1971) are symbolized by a line with a closed circle, ●, of Kurtin and Zuclich (1978) by a line containing an open circle, ○, and of Zuclich and Taboada (1978) by a line containing a closed square, ■. The data for rabbit cornea of Pitts and Tredici (1971) are represented by a line containing a closed diagonal square, ◆, and of Pitts et al. (1977) by a line containing an open triangle, △. The human cornea data of Pitts (1973) are shown by a line with an open square, □, and human conjunctiva data (Cullen and Perera 1994) by a line with a closed triangle, ▲, but the outlier data point at 320 nm apparently resulted from thermal effects or experimental problems, as it is totally inconsistent with environmental experience. Each data point was plotted after adjustment for spectral bandwidth used for each exposure (Sliney and Wolbarsht 1980). A single 193-nm laser threshold point 1 J cm^{-2} is not shown.

increase, a thermal effect could result (Sliney and Wolbarsht 1980) at radiant exposures less than those required for photochemically induced injury. However, few industrial sources emit sufficient intensity in the UVA spectral region to cause adverse biologic effects, and only lasers may place tissues at thermal risk. Nevertheless, a limit of 1 J cm^{-1} will protect against such effects.

There is a lack of evidence that the low levels of UVA (of the order of 1–3 mW cm^{-2} or less) experienced in sunlight or found in most indoor work environments present a hazard to either skin or eye. However, the hypothesis originating from in vitro studies that UVA may be one causative agent for cataract (Roberts 2001), suggests the need for caution with regard to chronic low-level UVA ocular exposure. The EL for UVA should protect against potential photochemical injury; however, experimental threshold data are lacking. In the absence of experimental data, the Commission recommends a more cautious approach for chronic ocular exposure.

In recent years there has been a rapidly growing population of individuals who have had one or both crystalline lenses surgically removed as part of cataract surgery. Most of these patients have received artificial intraocular lenses of plastic. (Such individuals are frequently referred to as "pseudophakics"). Aside from a few with implants that were not designed to absorb UVA to simulate the crystalline lens, or persons with no implant ("aphakics"), all of these patients will be adequately protected against retinal injury from UVA exposure at the EL (Mainster 1986). Those without UV-absorbing IOLs should be fitted with UVA protective eyewear if working with sources of UVA radiation.

■ ■