



ISBN 3 900 734 94 1

COMMISSION INTERNATIONALE DE L'ÉCLAIRAGE
INTERNATIONAL COMMISSION ON ILLUMINATION
INTERNATIONALE BELEUCHTUNGSKOMMISSION

CIE COLLECTION

in Photobiology and Photochemistry

1999

Contents:

- 134/1 CIE TC 6-26 report: Standardization of the terms UV-A1, UV-A2 and UV-B
- 134/2 CIE TC 6-30 report: UV protection of the eye
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- Abstracts of CIE Publications prepared within Division 6

CIE 134 - 1999

UDC: 612.014.481-06

Descriptor: Optical radiation effects on humans

CIE 134 - 1999

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CIE TC 6-26 report**STANDARDIZATION OF THE TERMS UV-A1, UV-A2 AND UV-B****TERMS OF REFERENCE**

To develop standardized terms for UV-A1 and UV-A2 and to examine whether there is a necessity for changing the boundaries of UV-A and UV-B.

HISTORY OF THE TC

The terms UV-A1 and UV-A2 have been introduced into the scientific and medical literature within the last few years, and the use of these terms has spread quite rapidly throughout different branches of photobiology and photomedicine. During the Melbourne meetings of the Division 6 in July 1991, J.P. Césarini was assigned the task to find a chairman for a technical committee having the task to prepare a report on the status of UV-A1, UV-A2, to examine the boundary between UV-A and UV-B, and eventually prepare a standard for these terms.

Prof. Jean Cadet, a specialist in the photobiology of DNA and the products of photoaddition and photodegradation, was contacted to be the chairman. After a meeting with him, he decided not to take over the proposed task stating that "no action spectrum for any DNA events can help to define clear-cut biological differences between UV-A1 and UV-A2. Hence, Dr. Césarini assumed the chairmanship and sent a letter explaining the future goals of the technical committee to the members listed below on May 4, 1992, calling for a meeting during the International Dermatology Congress in New York (June 17, 1992). A second meeting was held at the 20th Meeting of the ASP at Marco Island, FL (June 20-24, 1992) and the American Academy of Dermatologists meeting on December 4, 1992 in Washington DC.

Several members of the TC also met in Chicago on June 29, 1993 and sketched an outline for a draft report. J. P. Césarini prepared the initial draft and forwarded it to the different members for comments. A final editorial meeting of the TC was held in Vienna on September 4, 1996.

The following members of TC 6-26 "Standardization of the terms UV-A1, UV-A2 and UV-B" took part in the preparation of this Technical Report. The Committee comes under CIE Division 6.

Pr. J. Barth	Germany	Dr. J. Cadet	France
Dr. J. P. Césarini	France, chairman	Pr. T. B. Fitzpatrick	USA
Dr. A. McKinlay	U.K.	Pr. M. Mutzhas	Germany, deceased
Dr. M. Pathak	USA	Dr. M. Peak	USA
Dr. D. Sliney	USA	Pr. F. Urbach	USA

STANDARDIZATION OF THE TERMS UV-A1, UV-A2 AND UV-B**SUMMARY**

The terms UV-A, UV-B and UV-C were introduced in the 1930's by CIE Committee 41 on Ultraviolet Radiation as a short-hand notation for photobiological spectral bands. It was never intended that the bands were exclusive for different effects. The bands have been in widespread use in different medical fields and scientific research. UV-A and UV-B were divided at 315 nm by the CIE. In recent decades, some photodermatologists and others have used different dividing lines such as 320 nm without recognizing the importance of maintaining an international standardized terminology. Because the terminology is used in many fields, this report recommends that the 315 nm division between UV-A and UV-B be maintained. However, recent research has clearly shown a difference in the photobiological interaction of long and short wavelength UV-A radiation with DNA. This led to a further division of UV-A into UV-A1 and UV-A2 with a dividing line at approximately 340 nm. While this division may be of

value, the committee does not recommend officially to split UV-A into these two sub-bands at this time. Further research may justify a dividing line different from 340 nm in the future.

NORMALISATION DES TERMES UV-A1, UV-A2 ET UV-B

RESUME

Les termes UV-A, UV-B et UV-C ont été introduits dans les années 30 par le Comité 41 de la CIE sur le rayonnement ultraviolet, comme abbréviations désignant les bandes spectrales photobiologiques. Il n'avait jamais été déclaré que ces bandes étaient spécifiques pour des effets différents. Ces bandes ont été utilisées couramment dans divers domaines de la médecine et dans la recherche scientifique. La CIE a fixé la limite entre l'UV-A et l'UV-B à 315 nm. Dans les dernières décennies, des photodermatologues ont utilisé d'autres valeurs pour cette limite comme p.ex. 320 nm, sans tenir compte de l'importance de garder une terminologie internationale normalisée. Comme la terminologie est utilisée dans plusieurs domaines, ce rapport recommande de garder la division entre UV-A et UV-B à 315 nm. Cependant, des recherches récentes ont démontré clairement une différence dans l'interaction photobiologique entre le rayonnement UV-A de courtes et de grandes longueurs d'onde avec l'ADN. Ceci a conduit à une nouvelle division de l'UV-A en UV-A1 et UV-A2, la ligne de partage se situant à 340 nm environ. Bien que cette division ait un certain intérêt, le comité ne recommande pas officiellement, pour le moment, de couper l'UV-A en ces deux sous-domaines. A l'avenir des recherches supplémentaires pourraient justifier une autre ligne de partage qu'à 340 nm.

NORMUNG DER BEZEICHNUNGEN UV-A1, UV-A2 UND UV-B

ZUSAMMENFASSUNG

Die Begriffe UV-A, UV-B und UV-C wurden in den 30er Jahren vom CIE Komitee 41 "Ultraviolette Strahlung" als Kurzbezeichnung für photobiologische Spektralbereiche eingeführt. Es war jedoch damit nicht beabsichtigt, die Bedeutung dieser Bereiche auf bestimmte Wirkungen einzuschränken.

Die Verwendung dieser Spektralbereiche ist in verschiedenen medizinischen Gebieten und in der naturwissenschaftlichen Forschung weitverbreitet. UV-A und UV-B wurden durch die CIE bei 315 nm voneinander abgegrenzt. In den letzten Jahrzehnten haben einige Photodermatologen und andere Fachleute unterschiedliche Grenzen zwischen UV-A und UV-B, beispielsweise 320 nm, benutzt, ohne die Bedeutung der Beibehaltung international normierter Terminologie zu berücksichtigen. Da die Bezeichnungen UV-A und UV-B in vielen Gebieten verwendet werden, empfiehlt dieser Bericht, die Grenze von 315 nm zwischen UV-A und UV-B beizubehalten.

Jüngste Forschungen bezüglich der Wechselwirkung mit DNA haben jedoch einen deutlichen Unterschied zwischen lang- und kurzwelliger UV-A Strahlung gezeigt. Dies hat zu einer weiteren Teilung von UV-A in UV-A1 und UV-A2 mit einer Grenze bei etwa 340 nm geführt. Obwohl diese Unterteilung ihre Berechtigung haben könnte, empfiehlt das Komitee gegenwärtig nicht, die Teilung von UV-A in diese zwei Unterbereiche zu normieren. Künftige Untersuchungen könnten eventuell ergeben, daß die Trennung bei einer anderen Wellenlänge als bei 340 nm sinnvoll ist.

BACKGROUND

Introduction

The internationally accepted definitions of UV-B (280 nm - 315 nm) and UV-A (315 nm - 400 nm) originated in the early 1930s and were established around 1935 by CIE Committee 41 on Ultraviolet Radiation [1-3]. However, in recent years a vast number of publications related to several fields of photobiology have been defining the wavelength of

320 nm as the dividing line between UV-A and UV-B. The international consensus has eroded such that many photodermatologists and photobiologists (particularly in the US) have made 320 nm the dividing line in their publications. Still other specialists have attempted to adjust the lower limit of UV-B without recognizing the long history of the CIE definitions.

Three points are independently discussed in the technical report:

- 1 - The lower limit of UV-B,
- 2 - The boundary between UV-B and UV-A, and
- 3 - The opportunity to split the UV-A band in 2 parts.

The purpose of the CIE band notations

Although useful, it should be kept in mind that these photobiological spectral bands are merely a "short-hand" notation, and they can be used to make general (but not absolute) statements about the relative spectral effectiveness of different part of the UV spectrum in producing effects. The dividing lines, while not arbitrary, were certainly never intended to represent fine dividing lines between wavelengths which may or may elicit a given biological effect. One should always provide a wavelength band or spectral emission curve for the UV source being used and not rely totally on these spectral terms. There have also been authors who use 320 nm rather than the CIE defined dividing line of 315 nm to divide UV-A from UV-B. When this is done, the authors should explain parenthetically the wavelength band limits. Good practice in research publications for photobiologists and scientists who describe different effects should refer to specific action spectra and source spectra if at all possible.

Historical perspective

The internationally accepted definitions of UV-B (280 nm - 315 nm) and UV-A (315 nm - 400 nm) were established by the former Committee on Photobiology of the CIE around 1935. These limits were based largely on studies of UV erythema at that time which had determined a dual peak action spectrum for erythema which had secondary maximal value of 0,58 at 248 nm, a primary maximal value of 1,00 at 297 nm and a null of 0,06 at 280 nm. This earlier CIE standard erythema action spectrum reached a value of 0,01 at 315 nm. (Later, the standard erythema action spectrum values were extended down to a value of 0,003 at 325 nm; and today, a different standardized action spectrum is used). Additionally, during the 1930's, Coblentz (USA) worked to develop sharp cut-off filters at 280 nm, 315 nm and 400 nm for practical measurements, and the availability of these cut-off filters and the Hg emission wavelengths solidified these dividing lines. UV-B was meant to be the spectral band where erythema was most demonstrable for the least energy. Lamps without UV-B present were effectively incapable of producing erythema. With the advent of very intense UV-A sources in recent years, this dividing line has come into question.

However, as early as 1938, when Hausser [4] first suggested 320 nm, a number of publications related to photobiology used the 320 nm wavelength as the division between UV-A and UV-B. In several of its most recent documents, the CIE firmly reaffirmed 315 nm as the dividing line between UV-A and UV-B [3].

1. LOWER LIMIT OF UV-B

The lower limit of UV-B has been officially defined at 280 nm since about 1935. However, some plant photobiologists and, to a lesser extent, researchers dealing with ozone depletion, have favoured a lower limit of UV-B set at 290 nm. This position is also sustained by some photobiologists who have been dealing with human erythema action spectrum, who argue that because of epidermal absorption, that the highest activity of UV-B erythema is at 290 nm. Furthermore, at sea level, no radiation from sunlight is readily detectable below 290 nm (although, by photon counting techniques, the terrestrial solar irradiance extends at best to 285 nm). In the opinion of J. van der Leun [6], and several other authors, the limit of 280 nm should not be changed. They argue that it is important to maintain the UV-B lower limit at 280 nm since electrical engineers and lighting engineers who have to be concerned about the spectra of several sources, health authorities who have to deal with the consequences of UV

radiation on human exposure under both natural and artificial conditions, need to include the entire spectrum of UV-B. For plant physiologist, the problem may be different. Taking into account the many varied special interest groups, each with slightly different suggestions, it is clear that not all would be happy with a change. The Committee also had to consider the adverse impact of changing a standard definition, when a large number of older publications followed the official CIE definition.

The Committee proposes to retain the lower limit of UV-B at 280 nm for the purpose of calculation of action spectra and for occupational and public health requirements.

2. BOUNDARY BETWEEN UV-B AND UV-A

Several papers call attention to the effect of different wavelengths upon DNA absorption and damage. The types of lesions induced in DNA varies with wavelength. Peak and Peak (1984) [5-6] have stated that most action spectra for biological effects recorded in human tissue, change their slope in the region of 330 nm. This is true for erythema in humans, mutagenicity and lethality of *Escherichia Coli*, etc. This indicates that two mechanisms of action can be involved. The shorter wavelength photons induce pyridine photoproducts by direct photon absorption by the nucleic acids. This effect is dominant in the UV-C and UV-B up to 315 nm. The longer wavelength photons produce oxidative DNA damage though non-nucleic acid sensitizers with triplet oxygen involvement. For this reason, Peak and Peak [5] suggested in 1986 that the dividing line between UV-B and UV-A be moved to a longer wavelength, such as 327 nm, 335 nm or 347 nm depending upon no oxidative, equal effect or no direct effect for a constant radiant exposure. At the International Congress for Photobiology held in Kyoto (1992), the boundary between UV-A and UV-B (reported in the meeting summary by M.J. Peak and J. C. van der Leun, [6]) was recommended to be 347 nm - where all of the effects of UV-A are indirect; whereas, at 320 nm and below, all the effects are direct.

It is important to recognize that the above considerations do not consider the impact of the illuminating source, and implicitly assume an equal spectral irradiance at all wavelengths. This is hardly the case with most lamps and with solar radiation. Furthermore, the long history of the 315 nm dividing line with a large literature using 315 nm (or even 320 nm) argues against extending the dividing line to 327 nm, 335 nm or 347 nm.

This Committee, after assessing the past history, the different user communities and the photochemistry, concludes that the upper limit of UV-B should remain at 315 nm where no effect of UV-A can be recorded.

3. OPPORTUNITY TO SPLIT THE UV-A BAND IN TWO PARTS

M.J. Peak and J.C. van der Leun [6] clearly establish that UV-A at 340 nm and below, induces purely direct lesions in the DNA. For that reason, it can be reasonably proposed to recognize 340 nm as a landmark within the UV-A band. For the purpose of specific work dealing with different human photobiological events, the terms previously proposed by T. B. Fitzpatrick [7], as UV-A1 (longest wavelengths) and UV-A2 (shorter wavelengths) can be used, but should be clearly defined in the protocols and in the publications.

The Committee does not want to officially split the UV-A into two parts at this time, but it seems reasonable to consider the effects of UV-A between 315 nm and 340 nm on one hand, and between 340 nm and 400 nm on the other hand.

4. COMMENTS

Throughout its work, this Committee received suggestions and recommendations from scientists and engineers in a variety of disciplines who use these bands in describing their work. As recommendations were received it became clear that relative to specific problems and specific interests no consensus in changing limits was possible, as for some disciplines longer wavelength limits were desired, for others, shorter. Photodermatologists, photobiologists, physicists, lighting & optical engineers, plant physiologists, ecologists, meteorological physicists among others use these limits. With all of these factors taken into

consideration, it became clear that fundamental changes in the dividing lines were not advisable.

5. RECOMMENDATIONS

The Committee proposes to retain the lower limit of UV-B at 280 nm for the purpose of calculation of action spectra and for occupational and public health requirements.

This Committee, after assessing the past history, the different user communities and the photochemistry, concludes that the upper limit of UV-B should remain at 315 nm where no effect of UV-A can be recorded.

The Committee does not want to officially split the UV-A into two parts at this time, but it seems reasonable to consider the effects of UV-A between 315 nm and 340 nm on one hand, and between 340 nm and 400 nm on the other hand. In the meantime, if scientists wish to use the terms UV-A1 and UV-A2, they are advised to define these terms at the outset of their work.

No changes in the International Lighting Vocabulary are required for these terms. [8]

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CIE TC 6-30 report**UV PROTECTION OF THE EYE**

The following members of TC 6-30 "Dosimetry of UV exposure of the human cornea" took part in the preparation of this technical report. TC 6-30 comes under CIE Division 6 "Photobiology and Photochemistry".

P. Gies	Australia	D.G. Pitts	USA
F.S. Rosenthal	USA	C. Roy	Australia
J. Silver	UK	D. Sliney	USA
H.R. Taylor	Australia	C.F. Wong	Australia, chairman

UV PROTECTION OF THE EYE**SUMMARY**

This report provides a review on the current methods for UV dosimetry for the human cornea. The contents include a human action spectrum, the dosimeters, the methodology for calibration and the calculation of cumulative exposures. Based on the existing data, the levels of protection for the UV-A band and the UV-B band are recommended. A set of guidelines for ocular protection is proposed to assist medical practitioners in providing eye protection for patients.

PROTECTION DE L'ŒIL CONTRE LE RAYONNEMENT UV**RESUME**

Ce rapport fournit un aperçu des méthodes actuelles pour la dosimétrie UV de la cornée humaine. Il contient un spectre d'action pour l'homme, les dosimètres, la méthodologie pour l'étalonnage et le calcul des expositions cumulatives. Sur la base des données existantes, des niveaux de protection pour le domaine des UV-A et celui des UV-B sont recommandés. Une série de guides pour la protection oculaire est proposée pour aider les médecins à fournir une protection oculaire à leurs clients.

SCHUTZ DES AUGES VOR UV-STRALUNG**ZUSAMMENFASSUNG**

Dieser Bericht bietet einen Überblick über die aktuellen Methoden der Beurteilung der UV-Strahlungsexposition des menschlichen Auges und der möglichen schädlichen Wirkungen. Der Bericht enthält Angaben über die Wirkungsfunktion, Dosimeter, Kalibrierverfahren für Dosimeter und die Berechnung der kumulativen Bestrahlung. Basierend auf den vorliegenden Daten werden Schutzniveaus für den UV-A- und den UV-B-Bereich empfohlen. Richtlinien für den Augenschutz werden vorgeschlagen, um Ärzte bei der Auswahl des Augenschutzes für Patienten zu unterstützen.

1. INTRODUCTION

The waveband of ultraviolet radiation (UVR) extends from about 100 nm to 400 nm. It can be divided into three bands: UV-C - 100 nm to 280 nm; UV-B - 280 nm to 315 nm, and UV-A - 315 nm to 400 nm. The waveband between 100 nm and 200 nm seldom exists in our environment. This report will be confined to the waveband between 200 nm and 400 nm.

A summary of the ocular response thresholds (Cogan et al 1956; Pitts et al 1969; Pitts 1970; Zuclich et al 1976; Pitts et al 1977; Zuclich et al 1977; Kurtin et al 1978; Cullen 1980;

Ringvold 1980; Zuclich 1980; Ringvold et al 1982; Ringvold 1983; Henton 1983, 1984; Blumthaler 1987; Cullen et al 1990) to the ultraviolet radiation as it varies with wavelength and the level of radiant exposure is given in table 1.1.

Table 1.1 Ocular response thresholds to UVR

Ocular Media	Cornea	Conjunctiva	Lens	Retina
Waveband (nm)	200 - 320 mainly UV-C and UV-B	270 - 310 mainly UV-B	295 - 320 mainly UVB	310 - 380 mainly UV-A
Threshold measured at the plane of the cornea ($\text{J}\cdot\text{m}^{-2}$)	40	25	1500	3600
Wavelength (nm)	270	270	300	320

Exposure of UV radiation has been suggested to be linked to a number of eye diseases, which include: photokeratitis - corneal and adnexal damage (Widmark 1889, 1892, 1893; Verhoeff et al 1916; Pitts 1971, 1974; 1985, 1989; Zuclich 1977, 1984; Schive 1984); climatic droplet keratopathy - a deposition of altered proteins in the superficial cornea leading to opacification (Taylor 1980a, 1989); pterygium - the fleshy growth on the normally clear cornea (Cameron 1965, Taylor 1980, Moran et al 1984); cataract - opacities usually located in the anterior cortical and posterior subcapsular regions of the crystalline lens (Pitts et al 1969; Pitts 1970; Duke-Elder et al 1972; Chatterjee 1973; Zuclich et al 1976; Pitts et al 1977; Taylor 1980; Hiller et al 1977; Hollows 1981; Weale 1982; Brilliant et al 1983; Hiller et al 1983; Cullen et al 1984; Mitchell et al 1986; West et al 1986; Taylor et al 1988; Collman et al 1988; Bochow et al 1989); and retinal damage, which varies from a focal lesion to a generalized retinitis depending on the exposure conditions (Widmark 1889; Goodeve 1945; Wald 1945; Ham et al 1982; Mainster 1987). While the link between UVR exposure and ocular disease may be controversial, the results suggest that UVR is harmful. Just as UVR is known to accelerate aging of the skin, it apparently accelerates aging in ocular tissue as well.

Reliable data on acute effects such as photokeratitis, cataractogenesis and retinal damage are available, but data are lacking to construct an action spectrum for chronic effects such as cataract which may result from years of exposure because of the problem with uncertainty in UV dosimetry (Sliney 1994). Recent developments have provided several methods which may improve the accuracy for the measurement of UV dose to the eye.

2. HUMAN ACTION SPECTRUM

Sufficient data (Cogan et al 1956; Pitts et al 1969; Pitts 1970; Pitts et al 1977; Kurtin et al 1978) is available on the cornea to provide the spectral distribution for the threshold radiant exposure (fig. 2.1) for photokeratitis. Also included in fig. 2.1 are the results on the primate cornea and the rabbit lens for comparison. The data points are for 5 nm and 10 nm spectral bandwidth exposures, with the result that some threshold points are shifted from that resulting from true monochromatic irradiation.

The human actinic action spectrum (IRPA, 1989) for the assessment of the exposure to human eye is presented in tables 2.1-2.3. A graphical representation is provided in fig. 2.2. Note that the graph in fig. 2.2 is approximately proportional to the reciprocal of the threshold radiant exposure given in fig. 2.1, with the impact of the 5-nm and 10-nm bandwidths for the exposure data taken into account.

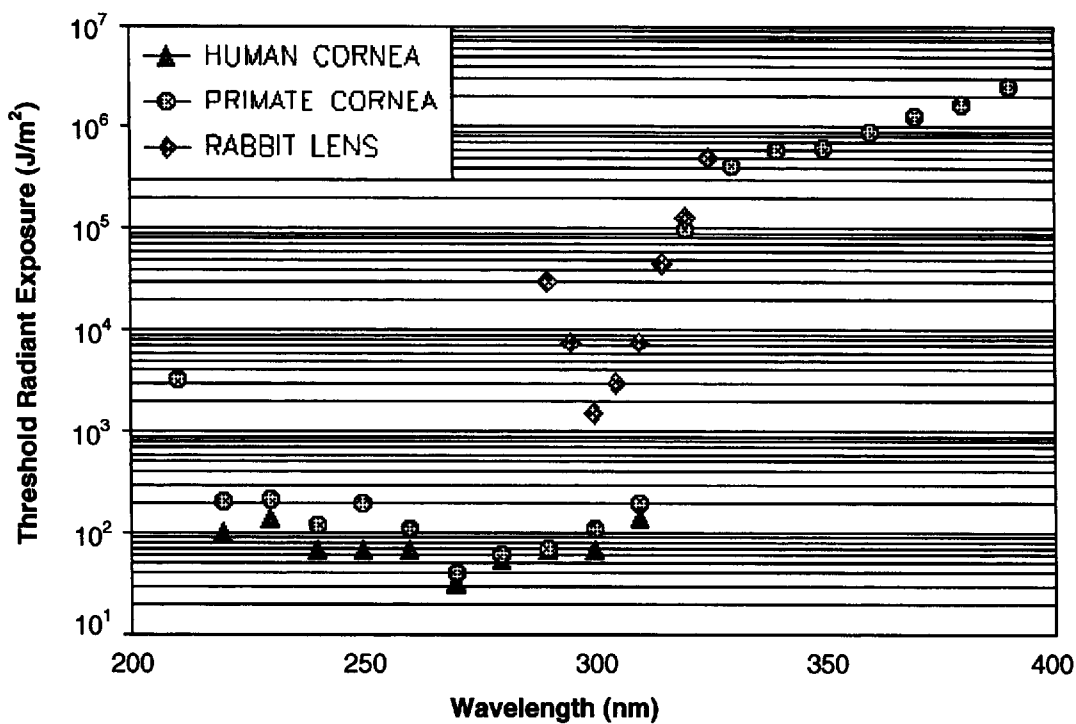


Fig. 2.1 Threshold radiant exposure

Table 2.1 Relative spectral efficiency, $A(\lambda)$, in UV-C band

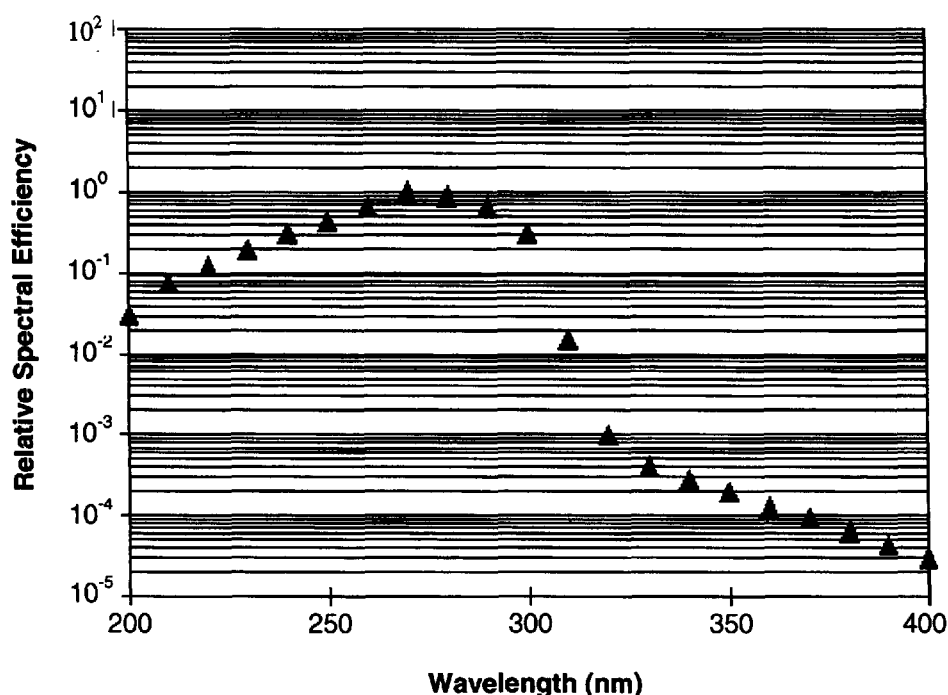
Wavelength (nm)	Spectral actinic efficiency $A(\lambda)$
200	0,030
210	0,075
220	0,120
230	0,190
240	0,300
250	0,430
260	0,650
270	1,000

Table 2.2 Relative spectral efficiency, $A(\lambda)$ in UV-B band

Wavelength (nm)	Spectral actinic efficiency $A(\lambda)$
280	0,880
290	0,640
300	0,300
310	0,015

Table 2.3 Relative spectral efficiency, $A(\lambda)$ in UV-A band

Wavelength (nm)	Spectral actinic efficiency $A(\lambda)$
320	0,0010
330	0,00041
340	0,00028
350	0,00020
360	0,00013
370	0,000093
380	0,000064
390	0,000044
400	0,000030

**Fig. 2.2** Human action spectrum

3. UV EXPOSURE TO THE EYE

The exposure of the eye to ultraviolet radiation depends mainly on the exposure at the cornea. The exposure to the interior structure of the eye is affected by the transmission of ultraviolet radiation through ocular media. The cornea absorbs almost all radiation below 290 nm. About 1%- 3% UV at 320 nm reaches the retina in younger eyes. The exposure at the cornea is affected by a number of factors. These factors include: the eyelid, the pupil, exposure duration, and protective devices.

Exposure to the corneal surface is affected by the eyelid. Unless extremely intense sources are used the transmittance of the eyelid in the UV-B region is negligible. The pupil and the lid opening control the amount of UV radiant energy that reaches the ocular structures behind the cornea. The size of the pupil is affected by factors such as accommodation, intensity of light and drugs. The diameter of the pupil can vary from about 1,5 mm to about 8,0 mm. The radiant energy passing through the pupil varies as the illumination changes from

the dark adapted state to the light adapted condition. Exposure duration is also an important factor for estimating the amount of UV radiant energy which is applied to the eye. Corneal exposure to UVR may be influenced by eyewear and/or headwear (Rosenthal et al 1985). It has been shown that one of the important factors in determining personal exposure in a given environment is whether protective devices are worn (Rosenthal et al 1986).

3.1 Biologically relative effective exposure

Biologically effective exposure for a particular type of photobiological effect may be used to assess the effect of the exposure to ultraviolet radiation. In determining the biologically effective dose, the action spectrum relevant to the biological effect should be used. The spectral irradiance varies with the source. For examples, the shortest wavelength in solar radiation is about 290 nm. Most artificial light sources such as lamps do not emit below 315 nm without a quartz envelope open arc process. Light sources such as the quartz halogen lamp or welding arcs (Mariutti et al 1988) can emit wavelengths shorter than 290 nm. The biologically effective irradiance $E_{\text{euv}}(t)$ for the eye due to the spectral irradiance, $E_{\lambda}(t)$, is given by

$$E_{\text{euv}}(t) = \sum_{\lambda=200}^{400} E_{\lambda}(t) \cdot A(\lambda) \cdot \Delta\lambda \quad (3.1)$$

where $A(\lambda)$ is the actinic action spectrum as defined in the previous section and $\Delta\lambda$ is the wavelength integration step. For an exposure period of T , the integrated biologically effective exposure is

$$H = \sum_{t=0}^T E_{\text{euv}}(t) \cdot \Delta t \quad (3.2)$$

where Δt is the sampling interval for $E_{\text{euv}}(t)$. The actinic action spectrum presented in section 2 can be used to calculate the effective dose that would affect the eye.

3.2 Dosimeters

A dosimeter suitable for estimation of corneal dose must satisfy the following conditions:

- Its spectral response resembles the actinic action spectrum.
- The dose-response curve is monotonic in the working region.
- The effect is additive and it is independent of the dose rate.
- The effect is stable against the change in temperature and humidity.
- The dosimeter should be portable, rugged and economical.

Polysulphone and Allyl Diglycol Carbonate (CR-39) are the two types of dosimeters (Rosenthal et al 1985; Barth et al 1990; Sydenham et al 1991; Wong et al 1992) which can be used to assess the ocular exposure from solar radiation. But CR-39 does not have the required responsivity in the waveband shorter than 280 nm, therefore it would not be suitable for the estimation of actinic dose from artificial light sources. Both polysulphone and CR-39 have a wave band sensitive to UV-B (Diffey 1989; Wong et al 1989). The relative spectral responsivity curves for polysulphone (40 μm thick) and CR-39 (etched in 6N KOH at 80°C for 3 h) are compared in fig. 3.1.

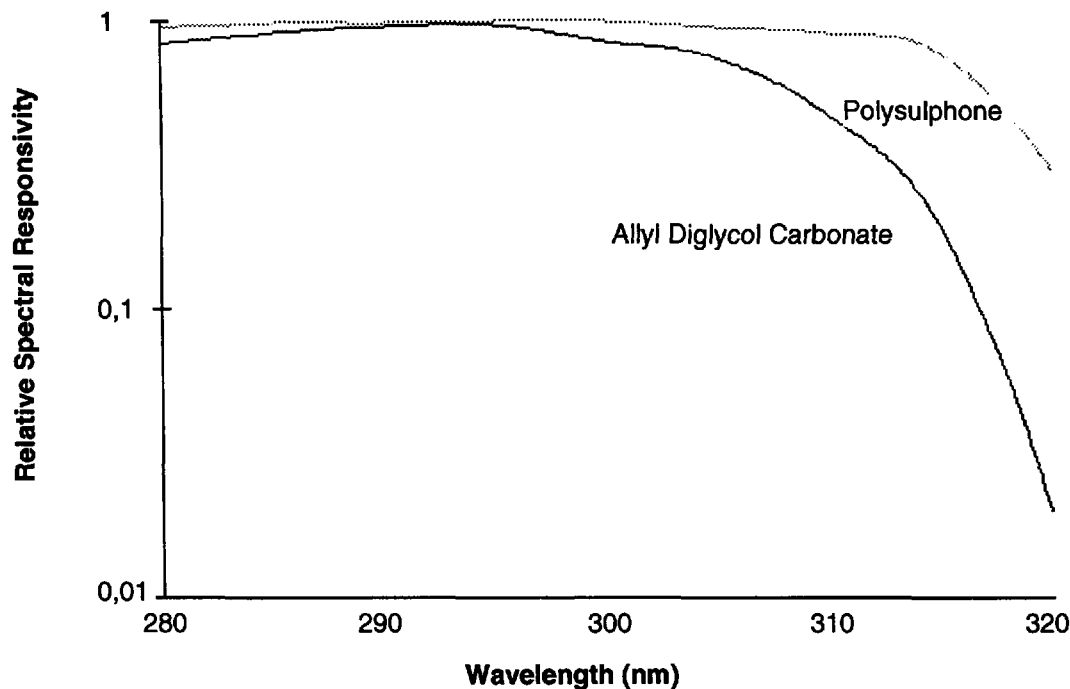


Fig. 3.1 Relative spectral responsivity curves

3.2.1 Measurements of corneal exposure

Corneal exposure can be estimated using suitable dosimeters (Diffey 1989; Wong et al 1989). These experiments require attaching the dosimeter to the human body. The human subject has to wear the dosimeter at a suitable site for a selected period of time. The dosimeter badges can be attached to headwear or eyewear worn by the participants. Contact lenses or sunglasses can also be fabricated from either polysulphone or CR-39 to be placed in front of the eye for estimating the corneal exposure.

3.2.2 Irradiating the dosimeters

Both Polysulphone and CR-39 can be fabricated into portable dosimeters. Most Polysulphone films that are useful for measurements of human exposure are of a thickness less than 40 μm . It would require some kinds of support for mounting the dosimeter. Usually, the film is mounted on a projector slide frame. The overall size of the dosimeter is greater than 12 mm x 16 mm. On the other hand, the thickness of the CR-39 is greater than 0,5 mm. It can be fabricated as a self-supporting dosimeter with a minimum size of about 10 mm x 10 mm or even less as required for special purposes. The dosimeters are attached to the appropriate sites of the human body for exposure. The minimum exposure time for Polysulphone is about a few hours in a horizontal plane in the midday sun. For CR-39, the minimum exposure time is about one week. The working ranges of uncovered dosimeters are given in table 3.1.

Table 3.1 Working exposure range for polysulphone and CR-39

Dosimeter	Range of erythema dose ($\text{kJ}\cdot\text{m}^{-2}$)
Polysulphone	1 to 12 $\text{kJ}\cdot\text{m}^{-2}$
CR-39 (etched in 8N KOH at 80°C for 2,5 hours)	5 to 15 $\text{kJ}\cdot\text{m}^{-2}$

3.2.3 Processing

After exposure, Polysulphone should be kept in the dark for at least 24 hours to avoid dark reaction. However, the CR-39 dosimeter should be treated with a suitable chemical (e.g. 6N KOH) in order to reveal the effect. The spectral absorbance of the dosimeter is measured with a spectrophotometer. The conditions for processing these dosimeters are summarized in table 3.2. After these procedures, the change of absorbance in the dosimeters is determined.

Table 3.2 Processing conditions for polysulphone and CR-39

Dosimeters	Polysulphone	CR-39
Developing time	> 2 hours	--
Concentration of etchant	--	6N - 8N KOH
Etching temperature	--	60°C - 80°C
Etching time	--	1 hour - 3 hours
Washing time	--	10 minutes
Wavelength for absorbance measurements	330 nm	400 nm

3.2.4 Calibration

Since the spectral responsivity curves of the dosimeter are not identical to the human ocular hazard action spectrum, the calibration data would be affected by the variation in the source spectrum. Dosimeters should be calibrated against the source in order to obtain accurate dose assessment of the exposure to the source. A UV-spectroradiometer fitted with an integrating sphere or a diffuser should be used for calibration. The spectroradiometer should have a resolution of 1 nm and a sensitivity of about $1 \text{ mW} \cdot \text{m}^{-2}$ at 300 nm. The scanning time in the entire UV waveband (200 nm - 400 nm) should not exceed five minutes. The dosimeter is placed at a distance of about 100 mm from the entrance port of the spectroradiometer with its surface parallel to, and at the same level as, the plane containing the entrance port. Care must be taken to ensure that the UV-irradiance at the site of the dosimeter is the same as that at the entrance port. The dosimeter is exposed for a selected time interval. During the same time interval, the source spectrum is measured every ten minutes or for a time interval as short as possible. A range of exposure is applied to dosimeters and they are processed in the way as described in section 3.2. The biologically effective doses are calculated with the source spectra. The results yield a functional relation between the change in absorbance and the dose.

3.3 Short term exposure and annual exposure

If the duration of exposure is less than two weeks, then corneal dose can be determined directly using suitable dosimeters. The wearing of these dosimeters may be inconvenient. It is necessary to design a suitable package in order to minimize the problem. The minimum duration of exposure has to be estimated for each type of dosimeter using the calibration graphs. As a guideline, the use of polysulphone would require an exposure of about one day in the summer sun in order to give measurable effects and using CR-39 would require a minimum exposure of about one week if the detector is etched in 8N KOH at 80°C for 3 hours. It should be pointed out that the dosimeters should not be exposed too long because a saturation of the dosimeter may occur.

The effective corneal exposure for long term exposure has been estimated by a number of authors (Rosenthal et al 1988, 1991). These authors used a mathematical model for estimating the effective ocular exposure from ambient UVR exposure. Let N be the number of activities (A) and N' be the number of months (M). The effective ocular exposure $H_{0,\text{eff}}$ for a subject performing N activities in N' months can be computed as

$$H_{o,eff} = \sum_{M=1}^{N'} \sum_{A=1}^N H_{PAE}(A, M) \cdot FOE(A, M) \quad (3.3)$$

where $H_{PAE}(A, M)$ is the monthly personal ambient exposure for a subject performing activity A in month M;

$FOE(A, M)$ is the ratio of effective ocular exposure to personal ambient exposure for a subject performing activity A in month M.

In order to determine the effective ocular exposure for a subject, a group of suitable participants with similar activities was selected for survey. The monthly personal ambient exposure was estimated from the published data of monthly ambient exposure which was measured using Robertson-Berger meters (Berger 1976). The data of the days per weeks that a given activity is performed would be required. The second part of the data can be obtained by administering a suitably designed questionnaire to the participants. The ratio, FOE , of effective ocular exposure to personal ambient exposure was determined (Rosenthal et al 1988) by attaching suitable dosimeters to selected sites.

4. EYE PROTECTION

The survey shows that exposure to UVR may induce permanent ocular damage. Proper eye protection against UVR will require both the recommendations for the level of protection and the use of protective devices. While the application of protective devices will certainly reduce the exposure to the eye but the cost factor may have to be taken into account.

4.1 Recommendations for the level of protection

For acute effect, the level of protection for the eye can be derived from the data presented in table 1.1. Based on the published data of threshold radiant energy and the exposure limits for the skin and corneal protection (Sliney 1982; Pitts 1985; ACGIH 1985; IRPA 1989; NH&MRC 1989), the recommended levels of protection for monochromatic sources are summarized in tables 4.1 - 4.3.

Table 4.1 Daily exposure limit for UV-C band

Wavelength (nm)	Daily exposure limit ($J \cdot m^{-2}$)
200	1000
210	590
220	100
230	130
240	76
250	70
260	46
270	25