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in Photobiology and Photochemistry

Contents:

- 106/1 Determining Ultraviolet Action Spectra
- 106/2 Photokeratitis (reprint)
- 106/3 Photoconjunctivitis (reprint)
- 106/4 A Reference Action Spectrum for Ultraviolet Induced Erythema in Human Skin (reprint)
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CIE RESEARCH NOTE

DETERMINING ULTRAVIOLET ACTION SPECTRA

ABSTRACT:

DETERMINING ULTRAVIOLET ACTION SPECTRA

Background information on photobiological action spectra is provided as a general introduction to the five previously published research notes that appeared in the CIE Journal and are now conveniently republished as a **CIE COLLECTION in Photobiology and Photochemistry**. This note also provides current information on international guidance related to exposure to ultraviolet radiation, which supersedes earlier CIE recommendations regarding action spectra for photokeratitis and photoconjunctivitis.

ZUSAMMENFASSUNG:

BESTIMMUNG ULTRAVIOLETTER WIRKUNGSFUNKTIONEN

Hintergrundinformation über photobiologische Wirkungsfunktionen wird als allgemeine Einleitung zu den fünf Research Notes gegeben, die im CIE Journal erschienen sind und jetzt als **CIE COLLECTION in Photobiology and Photochemistry** neuerlich veröffentlicht werden. Dieser Artikel bietet auch aktuelle Informationen über internationale Richtlinien bezüglich ultravioletter Bestrahlung, die frühere CIE Empfehlungen über Wirkungsfunktionen für Photokeratitis und Photokonjunktivitis ersetzt.

RESUME:

DETERMINATION DES SPECTRES D'ACTION ULTRAVIOLETS

D'autres informations sur les spectres d'action photobiologiques sont données comme introduction générale aux cinq Research Notes parus dans le CIE Journal, qui sont maintenant publiés comme **CIE COLLECTION in Photobiology and Photochemistry**. Cet article fournit également une information actuelle sur les guides internationaux concernant le rayonnement ultraviolet, remplaçant les recommandations précédentes de la CIE portant sur les spectres d'action pour photokératite et photoconjonctivite.

1 INTRODUCTION

The purposes of this brief introductory note are to provide the reader with the fundamental concepts characteristic of all photobiological effects, to explain the difficulties of obtaining action spectra, to clarify the limitations of biological action spectra, and to update CIE recommendations regarding the use of "standard" photobiological actions spectra. This report is intended to provide background information which should be useful in interpreting the conclusions of two previous CIE research notes (106/2 and 106/3) [1, 2].

2 ACTION SPECTRA

In any quantitative description of a photobiological effect, it is absolutely critical to obtain the *action spectrum*. The action spectrum describes the relative effectiveness of monochromatic optical radiation to elicit a given biological response. The most familiar photobiological action spectrum is the CIE visual response function, V_λ , which forms the basis for the system of photometric units. However, photobiological action spectra exist for photokeratitis (an ultraviolet-induced inflammatory response of the cornea of the eye), erythema (sunburn), etc. In addition, an action spectrum is useful in optimizing the use of ultraviolet radiation (UVR) in any photochemical effect, such as UVR-curing of inks or paints.

This CIE Research Note was prepared by Division 6 Director, D. Sliney [US Army Environmental Hygiene Agency, Aberdeen Proving Ground, MD 21010-5422 USA].

Photochemical (including photobiological) effects have a fundamental characteristic that the interaction at the molecular level results from one photon interacting with one molecule to alter or break the molecule into two new molecular species. Since a certain minimum photon energy Q_v is required to produce the molecular alteration, any photochemical process necessarily will have a long-wavelength cut-off $\lambda_{\text{cut-off}}$ where photon energies are less than a critical value Q_{crit} and insufficient to cause the molecular change of interest, since:

$$Q_v = hc/\lambda \quad [1]$$

where h is Planck's Constant (approximately $6,6 \times 10^{-34}$ J·s) and c is the velocity of light (approximately 3×10^8 m/s).

3 BUNSEN-ROSCOE LAW

A photochemical reaction will also exhibit *reciprocity* between irradiance (exposure dose rate) and exposure duration. This is termed the "Bunsen-Roscoe Law." That is, a given radiant exposure in J/m² is required to elicit a given response regardless of exposure duration over a wide range of exposure durations. Repair mechanisms, recombination over long periods and photon saturation for extremely short periods will lead to reciprocity failure. The product of irradiance E in W/m² and exposure duration t is the radiant exposure H in J/m², i.e.,

$$H = E \cdot t \quad [2]$$

While both E and H may be defined over the entire optical spectrum, it is necessary only to weight these quantities over the extent of the action spectrum for photochemical effects.

4 EFFECTIVE IRRADIANCE AND RADIANT EXPOSURE

In the same manner that spectroradiometric measurements of a light source can be used to calculate photometric quantities such as illuminance by spectrally "weighting" the spectral irradiance by V_λ , so too can a source spectrum be "weighted" by other action spectra to calculate an "effective irradiance," which may be used to estimate the exposure duration necessary to produce the given effect. With modern computer spread-sheet programs, one can readily develop a method for spectrally weighting a source spectrum by a large variety of photochemical action spectra. These computations can be tedious, but straightforward and take the form:

$$E_{\text{eff}} = \sum E_\lambda \cdot A_\lambda \cdot \Delta\lambda \quad [3]$$

where A_λ may be any action spectra of interest. One then can compare different sources to determine relative effectiveness of the same irradiance from several lamps for a given action spectrum.

5 PHOTOCHEMICAL VS. THERMAL EFFECTS

It is not uncommon for photochemical effects to be confused with thermal effects. For example, erythema ("sunburn") occurs after a critical radiant exposure as a result of a photochemical injury mechanism, rather than thermal injury, although one frequently senses warmth when exposed to sunlight. Two key factors distinguish a photochemical process from a thermal process. Thermal injury is a *rate process* and is dependent upon the volumic absorption of energy across the spectrum. By contrast, photochemical effects occur only over a given spectral range in accordance with an action spectrum and for a given threshold radiant exposure "dose."

6 PHOTOKERATITIS AND PHOTOCONJUNCTIVITIS

In the first two of the following CIE Research Notes, the action spectra for photokeratitis (inflammation of the cornea) and photo-conjunctivitis (inflammation of the surrounding conjunctiva) are reprinted. These two Research Notes provide a good review of the literature and present the range of published data [1, 2]. Both notes conclude with a recommended action spectrum for use by lighting engineers for each effect based upon these reviews. The Note, *Photokeratitis*, erroneously attributed support for the recommended action spectrum to the World Health Organization [3]. The Note, *Photoconjunctivitis*, proposes an action spectrum based on no biological data from ocular studies, but on an hypothesis that the conjunctiva should behave as the skin deprived of *stratum corneum*. Both notes implicitly assume that each effect has only one ("correct") action spectrum. This seems to be a very reasonable first assumption. However, because of different biological (or clinical) endpoints used to define the effect, multiple action spectra actually exist!

The confusion over multiple action spectra arises because the photobiologist must define an observational threshold for the effect in terms of the "grade" or degree of severity of the tissue response and must also specify the time of assessment. Furthermore, different optical examination aids and biochemical markers may be used in the assessment. All of these factors can lead to a different threshold at each wavelength; hence, a different action spectrum [3]. When the physical scientist is faced with what appears to be different action spectra from conflicting sources, he or she is most likely to assume that one action spectrum is in error because of improper radiometric measurements or other physical errors. This variation of action spectra based upon the time of assessment (e.g., 4 hours, 8 hours, 24 hours, 48 hrs, etc.) and intensity of skin redness was first shown very clearly for erythema by Hausser in 1929 [3, 4], and the lack of understanding of differing endpoints led to conflicting action spectra being published in the 1960s by several authors [5, 6]. For this reason, it has been necessary to derive a CIE reference erythema action spectrum [7], which is the third of the following republished notes. On the other hand, CIE Division 6 can no longer recommend the reference action spectra for ocular effects that were published in 1986. Since that time, other international bodies have recommended exposure limits which are more restrictive than in the Research Notes [8].

The differences in action spectra for photokeratitis exist largely because of different biological endpoints and also different responses for human and animal species used in the studies. The endpoint and threshold radiant exposures used by Cogan and Kinsey [9] and Pitts [10] differed. Pitts used a very sensitive threshold criteria which was a slight change in the number of vacuoles in the corneal epithelium (outermost layer of the cornea); whereas, Cogan and Kinsey used a more severe inflammatory response to term "threshold." Hence, it is not surprising that a lower threshold was reported by Pitts compared to Cogan and Kinsey. The latter action spectrum is more indicative of what the average person would sense as an uncomfortable keratitis; whereas, the Pitts' action spectrum probably provides the most sensitive endpoint for showing any effect. Hence the groups which have defined exposure limits (ELs) for safety have used the Pitts' action spectrum and thresholds for defining safety [8, 11]. As noted in the original CIE Research Note [1], the actual exposure of the human eye out-of-doors is frequently very close to the threshold dose reported by Pitts [10], but this does not disprove the data of Pitts. One must consider the eye's squint and the generally low reflectance of most terrain which greatly limit the chances of developing threshold photokeratitis [12].

New sources of UVR now exist which are being used to further define action spectra. The Argon-fluoride excimer laser was recently used to determine the threshold for photokeratitis at 193 nm for the rabbit cornea [12]. The threshold for a very mild form of photokeratitis characterized by a mild superficial corneal haze in the rabbit cornea has been found to lie between 1.0 and 1.5 J/cm² at 193 nm.

Further studies regarding repeated ocular exposures of the cornea and studies of lenticular damage all point to the need for limiting UVR exposure of the cornea below the levels cited in the 1986 Research Notes [1, 2], because of potentially additive effects [13-16].

7 CONCLUSIONS

The action spectra recommended previously [1, 2] probably define acute, clinically significant injury of the cornea and conjunctiva, and may be used to estimate the likelihood of experiencing this effect when exposed in a single incident to a source containing UVR. However, the reader is advised to consult other international guidelines for repeated human exposure to UVR. The UVR EL values promulgated by the

International Non-Ionizing Radiation Committee (INIRC) of the International Radiation Protection Association (IRPA) [8] should be consulted. Additionally, one should be aware that these limits should not be applied without restrictions to any general population which includes photo-sensitive individuals and children.

The cornea absorbs nearly all UVR with wavelengths shorter than about 295 nm and the principal acute effect at these short optical wavelengths is photokeratitis. Cataracts seem to be mainly limited to exposure to UVR in the wavelength range 295-325 nm [20, 21]. Fortunately, the cornea has good regenerative capacity [13-20], since the human cornea is typically exposed outdoors to levels just below the threshold for photokeratitis [20].

The CIE reference spectrum for erythema [7] is currently being submitted as an international standard and CIE Division 6 continues to recommend the action spectrum for use in describing erythema.

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CIE RESEARCH NOTE

PHOTOKERATITIS*

Abstract:

Ultraviolet radiation may cause photokeratitis, a painful inflammation of the cornea of the eye.

The spectral responsivity for photokeratitis - the action spectrum - follows a characteristic curve, the maximum of which lies at 288 nm according to Cogan and Kinsey [4], at 270 nm according to Pitts et al. [5]. The threshold dose is between 50 and 100 J/m².

The threshold exposure times are evaluated, within the range of validity of Bunsen-Roscoe law, for some sources. The data obtained by various authors are compared and the discrepancies are discussed. As a final statement, the CIE Division 6 recommends the use of the data of Cogan and Kinsey [4].

*Photokératite**Résumé:*

Le rayonnement ultraviolet peut provoquer la photokératite qui est une inflammation douloureuse de la cornée de l'oeil, apparaissant lorsqu'une dose trop forte est appliquée. La réponse en fonction de la longueur d'onde -spectre d'action- suit une courbe caractéristique, dont le maximum se situe à 288 nm, d'après Cogan et Kinsey [4] et à 270 nm, d'après Pitts et al. [5]. La dose seuil est environ 50 à 100 J/m².

Les temps d'exposition pour un effet seuil ont été évalués, en restant dans le domaine de validité de la loi de Bunsen-Roscoe, pour un certain nombre de sources. Les résultats obtenus en utilisant les données des divers auteurs sont comparés et les désaccords sont discutés. En conclusion, la Division 6 de la CIE recommande d'utiliser les données de Cogan et Kinsey [4].

*Photokeratitis**Zusammenfassung:*

Ultraviolet-Strahlung verursacht Photokeratitis, eine schmerzhaft Entzündung der Augenhornhaut, wenn zu starke Bestrahlungen verabreicht werden.

Die spektrale Wirkungsfunktion für Photokeratitis - das Wirkungsspektrum - folgt einer charakteristischen Kurve, deren Maximum nach Cogan und Kinsey [4] bei 288 nm und nach Pitts

et al. [5] bei 270 nm liegt. Die Schwellenbestrahlung beträgt ungefähr 50 bis 100 J/m².

Die Schwellenbestrahlungszeiten werden innerhalb des Gültigkeitsbereiches des Bunsen-Roscoeschen Gesetzes für einige Lichtquellen ermittelt. Die von verschiedenen Autoren erhaltenen Daten werden verglichen und die Diskrepanzen diskutiert. Als schliessliche Feststellung empfiehlt die CIE Division 6 den Gebrauch der Daten von Cogan und Kinsey [4].

1 AIM OF THE WORK

This paper aims at surveying the experiments performed to determine the relative spectral responsivity (i.e. action spectrum) of the cornea, by considering the acute effect of "photokeratitis". Having compared the results obtained by different authors, the responsivity function ($s(\lambda)_{ker,rel}$) seems to be an appropriate indication of how the cornea of the human eye weights the radiant flux $P(\lambda)$ emitted by the source, according to the model

$$\int P(\lambda) s(\lambda)_{ker,rel} d\lambda$$

A further step consists in examining the quantitative estimates of radiant exposure needed to reach the threshold for photokeratitis, e.g. by considering the wavelength where the spectral responsivity is a maximum, to decide what value to recommend.

Third, if reference is made to the application of the Bunsen-Roscoe law, or time-intensity reciprocity, the exposure thresholds are evaluated, for a number of sources.

The data are intended for use by Illuminating Engineers, who may have to evaluate the corneal "actinic" effectiveness of sources of various kind (natural or artificial, for general lighting or special purposes). The numerical data are derived in controlled laboratory conditions. Practical situations may deviate to a greater or lesser extent. Hence, our data represent "broadly safe" limits.

2 INTRODUCTION

Photokeratitis is an inflammation of the cornea, following over-exposure to ultraviolet radiation.

The symptoms are a sensation of "sand in the eyes", accompanied by varying degrees of

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photophobia, tears (lacrimation), and spasm of eyelids (blepharospasm).

These acute symptoms usually last for 6 to 24 hours, and almost total recovery occurs within 48 hours. Exposure rarely results in permanent damage. The radiation reaching the earth's surface [1] is not normally an ultraviolet hazard to man, because of the filtering action of earth's atmosphere mainly due to the presence of ozone. In part, the protection is afforded by the facial anatomy, i.e. the eye orbit and the eyelids.

Photokeratitis runs a characteristic clinical course. After exposure, there is a period of latency as short as 30 minutes or as long as 24 hours, depending upon the severity of exposure, but is typically 6 to 12 hours.

Many cases of photokeratitis have been reported following exposure to ultraviolet radiation from welding arcs, high pressure mercury lamps, electronic flash lamps, lasers and reflected solar radiation from snow and sand [2].

3 THE CORNEA

Normally, the living cells of the human skin are protected by the dead cells of the horny layer of the epidermis. This is not the case with the cornea which has no superficial protective layer corresponding to that of the skin.

The cornea consists of five tissue layers (epithelium, Bowman's membrane, stroma, Descemet's membrane and endothelium). The external epithelial layer is composed of a stratified squamous epithelium.

The normal epithelium contains no blood vessels or blood derived cells, but is rich in many fine nerve fibers. The stroma is avascular and composed of collagen fibers interspersed with fibrocytes. The stroma constitutes approximately 85% of the corneal thickness [3].

4 STUDIES ON ACTION SPECTRA

Two of the earliest authors who reported systematic experiments in an attempt to determine the action spectrum of photokeratitis were Cogan and Kinsey in 1946, who used rabbits [4]. The eyes of albino rabbits were exposed to separate bands of ultraviolet radiation from a high pressure mercury quartz lamp, in order to determine the threshold dose at different wavelengths.

The spectral range of their double quartz monochromator was from 240 to 316 nm and the band width was approximately 20 nm. As the threshold for photokeratitis Cogan and Kinsey used a granular appearance within the corneal epithelium as viewed by transillumination with a slit lamp and biomicroscope. The granules were presumed to be swollen epithelial cells.

Associated with this granular appearance were a stippling of the surface of the cornea seen in reflected light and a varying amount of fluorescein "take". Their action spectrum shows a peak at 288 nm and a long wavelength limit somewhere between 306 and 326 nm. The energy necessary to elicit a threshold reaction in the cornea at the peak of sensitivity is of the order of 150 J/m² (Fig. 1; Table 1).

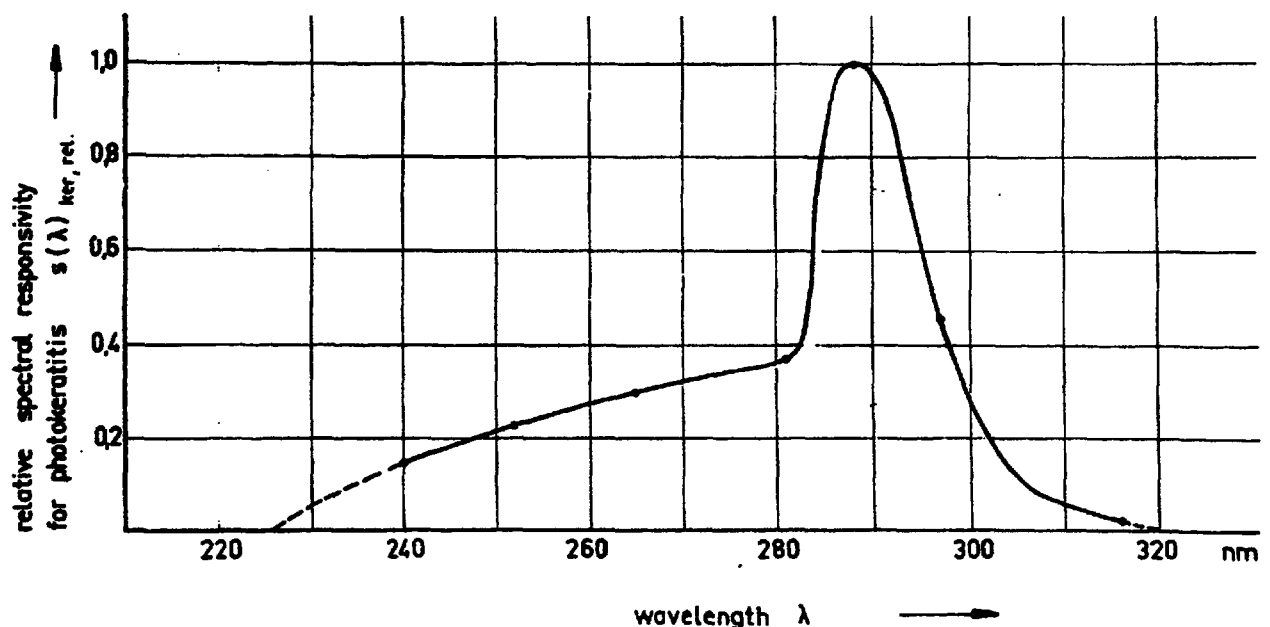


Figure 1.

Table 1. Photo-keratitis exposure $H_{s,ker}$ and relative spectral efficiency $s(\lambda)_{ker,rel}$ for rabbit eyes according Cogan and Kinsey [4].

Wavelength λ in nm	Exposure thresholds $H_{s,ker}$ in J/m ²	Relative spectral efficiency $s(\lambda)_{ker,rel}$
240	1000	0,15
252	660	0,227
265	500	0,30
281	400	0,375
288	150	1,000
297	330	0,455
305	1320	0,114
316	(6700)	(0,0224)

The energy necessary to produce photokeratitis in man was determined by Cogan, Kinsey and Drinker (see footnote Nr.3 in [4]) to be approximately two thirds that necessary in rabbits.

Pitts [1] reported thresholds for photophthalmia based on the exposure of the eyes of pigmented rabbits to ultraviolet radiation from 200 to 300 nm (in 10 nm steps).

The ultraviolet action spectrum ranged from 210 to appr. 310 nm. Peaks at 270 nm, with a threshold of 54 J/m² to produce photophthalmia

were observed. All lesions induced by ultraviolet radiation appeared to be confined to the external epithelial layer of the cornea.

In 1972 Pitts and Gibbons [5] published data of experiments made with a 5000 watt xenon-mercury high pressure lamp. Human and animal exposures were made to establish the photokeratitis threshold and action spectrum over the wavelength range 220 nm to 310 nm. The radiant exposure threshold at 270 nm was 50 J/m² for the rabbit and 40 J/m² for the primate and man.

The curve for the rabbit shown in Fig. 2 is double-peaked with maxima at 230 nm and 270 nm. The curve for the primate eye is tri-peaked with maxima at 220 nm, 240 nm and 270 nm, where the human data show a rather shallow curve with a maximum at 270 nm.

Seven criteria were used to determine the threshold for keratitis:

- epithelial debris (small glistening bodies in the corneal tear layer)
- epithelial haze (irregular, crackled appearance of the corneal anterior surface)
- epithelial granules (small, white, discrete, round spots located deep in the epithelial layer)
- photophobia (an avoidance response or verbal response indicating that the slit lamp bothered the subject)
- symptomatology and corneal light scatter.

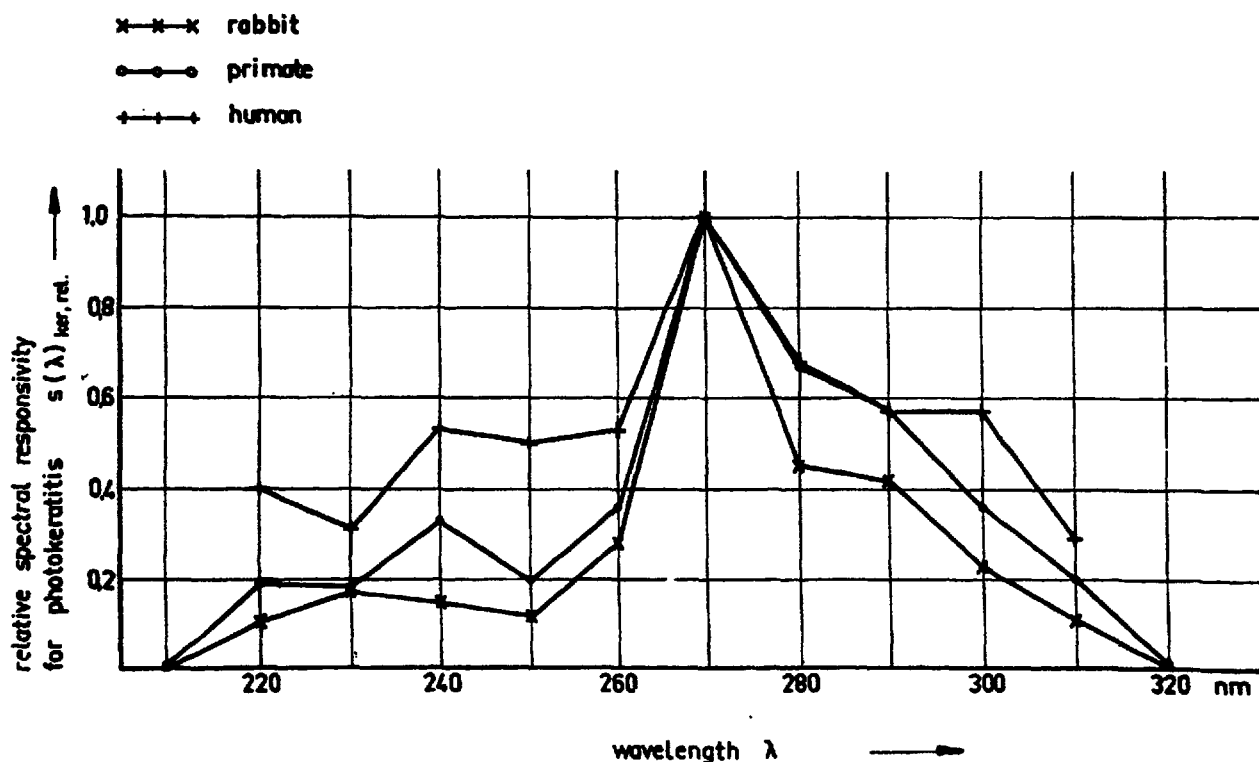


Figure 2.

Earlier, Pitts and Kay in 1969 [7] and Pitts in 1970 [6] sought to establish experimental thresholds for photokeratitis in rabbits, primates and man. The maximum sensitivity was again at 270 nm and the radiant exposure threshold at 270 nm was 50 J/m² for man, 60 J/m² for the monkey and 110 J/m² for the rabbit.

As a result of observations at above threshold intensities it was felt that the reaction of the cornea to ultraviolet radiation at wavelengths between 220 and 250 nm was different from that to wavelengths between 250 and 310 nm. For wavelengths below 250 nm, signs and symptoms occurred soon after exposure, and symptoms always returned to normal after approximately 14 h. For exposures to wavelengths longer than 250 nm, symptoms did not generally occur until 9 to 11 h after exposure, and visual acuity remained below normal for 24 h after exposure. The differences observed were attributed to differences in absorption of the radiation of different wavelengths.

Radiation of the shorter wavelengths is absorbed in the outer epithelial layers of the cornea, which undergo rapid change, whereas radiation of longer wavelengths is absorbed in the deeper epithelial layers which show delayed changes because the cells are more susceptible. Thus the lesions produced by shorter wavelengths are rapidly repaired while for radiation of longer wavelengths there is a delayed and more marked response [8].

Table 2 contains a summary of photokeratitis thresholds for rabbits, primates and man with the relevant action spectra [5]. The data shown in Table 2 refer to the basic research, which might be defined as "classical". In 1977 Pitts, Cullen and Hacker [12] published data for UV-threshold exposure for the rabbit cornea causing reversible damage. This data cover also the UV-A spectral range. The requested information is displayed in Table 4.

Table 2. Photo-keratitis exposure threshold $H_{s,ker}$ and relative spectral efficiency $s(\lambda)_{ker,rel}$ for rabbits, primates and humans according Pitts and Gibbons [5].

Wavelength λ, nm	Rabbit		Primate		Human	
	$H_{s,ker}, J/m^2$	$s(\lambda)_{ker,rel}$	$H_{s,ker}, J/m^2$	$s(\lambda)_{ker,rel}$	$H_{s,ker}, J/m^2$	$s(\lambda)_{ker,rel}$
210	7000	0.007	3270	0.012	-	-
220	460	0.11	210	0.19	100	0.40
230	300	0.17	220	0.18	130	0.31
240	330	0.15	120	0.33	76	0.53
250	410	0.12	200	0.20	80	0.50
260	180	0.28	110	0.36	75	0.53
270	50	1.00	40	1.00	40	1.00
280	110	0.45	60	0.67	59	0.68
290	120	0.42	70	0.57	70	0.57
300	220	0.23	110	0.36	70	0.57
310	470	0.11	200	0.20	140	0.29
320	6000	0.008	3500	0.011	-	-
330	50000	0.001	-	-	-	-

Table 3. Exposure time thresholds $t_{s,ker}$ in minutes for photo-keratitis the rabbit and the human eye under global radiation (sun + sky) and radiation from several fluorescent lamps.

Author	Global radiation		Fluorescent lamps* at 1000 Lx		
	Bener 1972 [9]	Dehne u. Kasten 1983 [10]	L 40 W/15 (daylight)	L 40 W/25 (universal white)	L 36 W/21 (neutral white)
Cogan and Kinsey (rabbit) 1946 [4]	24.1	14.75	$1.9 \cdot 10^3$	$2.9 \cdot 10^3$	$9.0 \cdot 10^3$
Pitts (rabbit) 1969 [2]	2.9	2.2	334	505	$1.33 \cdot 10^3$
Pitts and Gibbons (human) 1972 [5]	1.9	1.2	125	173	613
TLV-curve NIOSH 1975 [11]	19.5	9.7	900	$1.54 \cdot 10^3$	$6.05 \cdot 10^3$

*Osram-brand, Germany

Table 4. UV-threshold exposure for the rabbit cornea according Pitts, Cullen and Hacker [12].

Wavelength nm	Corneal threshold radiant exposure reversible damage, $\text{J} \cdot \text{m}^{-2}$
295	200
300	500
305	700
310	550
315	22500
320	75000
335	109900
365	425500

5 DISCUSSION

It must be noted that the investigators had experimental difficulties in accurately determining the obviously extreme slope of the action spectrum in the 300 to 315 nm range.

In addition, thresholds that were averaged over 10 nm intervals weighted against a source spectrum rapidly increasing in this region can lead to error, since sufficiently narrow wavelength intervals could not be used [8].

However, the exposure time thresholds derived from the Pitts (et al.) data are much shorter than those obtained by using the data obtained by Cogan and Kinsey. Indeed, the acceptance of these data leads to the challenging question why we do not develop photokeratitis in our everyday outdoor exposure. On the one side one might infer that the protection of our facial anatomy is indeed very powerful. On the other side one might suspect that when data obtained in different laboratories are compared, the role of calibration uncertainties is critical.

6 STATEMENT

Division 6 of the CIE recommends the use of the action spectrum for photokeratitis determined by Cogan and Kinsey and adopted by WHO, and recommends the use of 100 J/m^2 for the threshold exposure (threshold dose) at a wavelength of 288 nm.

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CIE RESEARCH NOTE

PHOTOCONJUNCTIVITIS*

Abstract:

Ultraviolet radiation can cause photoconjunctivitis, a painful inflammation of the conjunctiva, the tissue coating the eyelid and part of the eyeball.

The spectral responsivity of photoconjunctivitis - the action spectrum - follows a characteristic curve, whose maximum lies, according to Schulze, at 260 nm, and resembles closely the absorption spectrum of DNA damage. The threshold radiant exposure (threshold dose) is about 50 J/m².

The opinion expressed by several authors, that the same action spectrum should exist for photoconjunctivitis and photokeratitis, cannot be shared by CIE Division 6, since the cornea and the conjunctiva of the human eye are different from both anatomical and histological standpoints.

Photoconjonctivite**Résumé:**

L'exposition à une dose trop importante de rayonnement ultraviolet peut provoquer la photoconjonctivite qui est une inflammation douloureuse du tissu recouvrant la partie interne des paupières et une partie du globe oculaire.

La réponse en fonction de la longueur d'onde de la photoconjonctivite - spectre d'action - suit une courbe caractéristique, dont le maximum se situe à 260 nm, d'après Schulze, et ressemble de près au spectre d'action pour les dommages au DNA. La dose seuil est environ 50 J/m².

L'opinion exprimée par plusieurs auteurs, que les spectres d'action pour la photoconjonctivite et pour la photokeratite devraient être identiques ne peut être partagée par la Division 6 de la CIE, puisque la cornée et la conjonctive de l'œil humain sont trop différentes du point de vue anatomique et histologique.

Photoconjunctivitis**Zusammenfassung:**

UV-Strahlung kann Photoconjunctivitis verursachen, eine schmerzhaft Entzündung der Bindehaut zwischen Augenapfel und Augenlid, wenn zu starke UV-Bestrahlungen appliziert werden.

*This CIE Research Note was prepared by TC 6-03, chairman B. Steck, c/o Hellux, C.A. Schaefer KG., P.O.Box 1380, 3014 Laatzen 1, Germany, on behalf of CIE Division 6: Actinic Effects.

Die spektrale Empfindlichkeit für Photoconjunctivitis - das Wirkungsspektrum - folgt einer charakteristischen Kurve, deren Maximum nach Schulze bei 260 nm liegt und dem Absorptionsspektrum der DNS-Schädigung ähnlich ist. Die Schwellenbestrahlung (Schwellendosis) liegt bei 50 J/m².

Die Meinung, die von mehreren Autoren geteilt wird, daß für Photoconjunctivitis und Photokeratitis das gleiche Wirkungsspektrum existieren sollte, kann von der CIE Division 6 nicht geteilt werden, da die Hornhaut des menschlichen Auges und die Bindehaut zu unterschiedlich sind vom anatomischen und histologischen Standpunkt.

1 INTRODUCTION

Photoconjunctivitis, a painful inflammation of the conjunctiva, may come about by accidental exposure of the eye to ultraviolet radiation. This inflammation appears after a latency period of about 5 to 10 hours. At high radiant exposures, pain results and both vision and sleep may be impaired. The inflammation fades away, according to its severity, after a time between 10 hours and several days [1]. The conjunctiva, that is the membrane extending from the eyelid margin to the corneal margin (or limbus) may be exposed directly to ultraviolet radiation reaching the eye. On the other hand, in the case of the skin, the deeper layers of the living cells taking part in the metabolism, are protected by the filtering action of the horny layer (stratum corneum).

The present paper aims at surveying the attempts performed to determine the relative spectral sensitivity (or action spectrum) of the conjunctiva. The sensitivity function $s_{co,rel}(\lambda)$ which indicates how the conjunctiva of the human eye weights the radiant flux, is specified. Quantitative estimates of the radiant exposure needed to produce threshold photoconjunctivitis are considered.

Practically, the methodology followed here parallels that followed in our previous paper on photokeratitis [2]. This paper is intended for the use of Illuminating Engineers, faced with the evaluation of the "actinic effectiveness" of sources of various kinds. The numerical data are derived in controlled laboratory conditions. Practical situations may deviate to a greater or less extent. Hence, our data represent "broadly safe" limits.

2 DATA

The experimental data related to the action spectrum of photoconjunctivitis are rather scarce. In the present paper we mainly refer to data produced by authors like Henschke, Schulze and Roessler, as compared with some data on erythema and with the absorption spectrum of DNA.

3 RELATIVE SPECTRAL RESPONSIVITY (ACTION SPECTRUM) AND THRESHOLD EXPOSURE

Up to the present time, relatively few systematic experiments have been carried out on the action spectrum of radiation (spectral responsivity) in producing photoconjunctivitis.

From experiments and physical tests made by Hamperl, Henschke and Schulze [3] on the primary event of the production of erythema of the skin by UV radiation it has been shown that for radiation below 315 nm a reduction of nucleic acid takes place and that the target of the radiation is the nucleic acid of the cell nuclei.

By superposition of the absorption spectrum of the nucleic acid and the transmission spectrum of the horny layer acting as a filter, Hamperl, Henschke and Schulze constructed an action spectrum very similar to the action spectrum of erythema found by Hausser and Vahle [4] (see Figure 1).

Tronnier [5] has also made measurements of the spectral transmission of different skin layers of 25 μm thickness and converted the results to 75

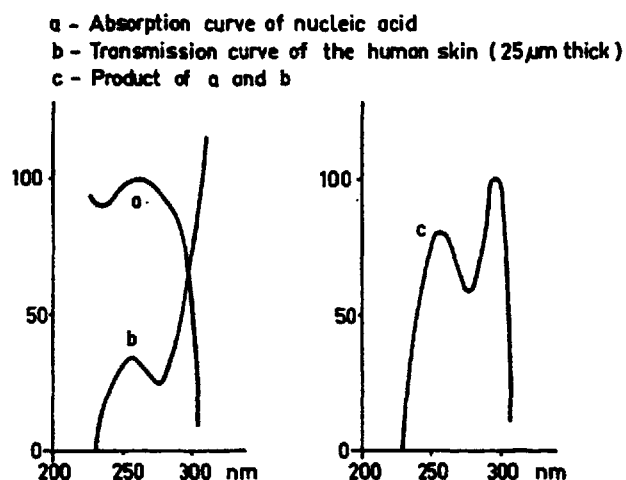


Fig. 1. Absorption spectrum of nucleic acid (a) transmission spectrum of the stratum corneum (b) and the curve gained by superposition (multiplication) which resembles very much the erythema curve by Hausser and Vahle [4].

μm thickness of the stratum corneum. He found that the characteristic minimum of the action spectrum as proposed by the CIE-UV-Committee coincides with the minimum of the spectral transmittance curve of a horny layer of 75 μm thickness.

In further investigation Tronnier [7] has proved that the horny layer not only acts as a filter for UV radiation but also takes an active part in producing an erythema by transporting effective substances acting on the subpapillary vessels.

If the horny layer is partly or completely removed e.g. by stripping, the erythema progresses faster but not so fast as one would assume in normal skin which is only filtered by an optical filter equivalent to the horny layer (see Figure 2).

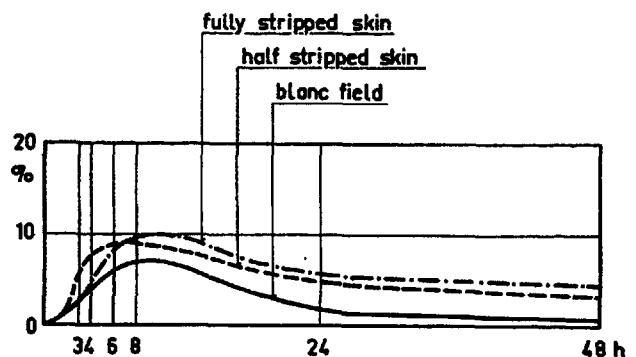


Fig. 2. Time course of erythema in relation of different thickness of the horny layer [7].

Parrish et al. have also explained the erythema action spectrum by a superposition of the spectral transmittance for fair Caucasian stratum corneum and the absorption spectrum of DNA (experiment made with sheared calf thymus DNA in phosphate buffer). The product of these two curves strongly resembles the measured action spectra and supports (but does not prove) that DNA is the primary chromophore involved in erythema induction for wavelength less than 320 nm [8].

The changes observed in the conjunctiva evoked by UV radiation correspond more or less to those in the rete Malpighi and in the basal cell layer of the skin during UV erythema [9].

As the protecting stratum corneum is absent in the conjunctiva, Henschke and Schulze [10] advocate the interpretation that the relative spectral sensitivity of photoconjunctivitis corresponds to the absorption spectrum of the thymus nucleic acid and the absorption spectrum for DNA damage.

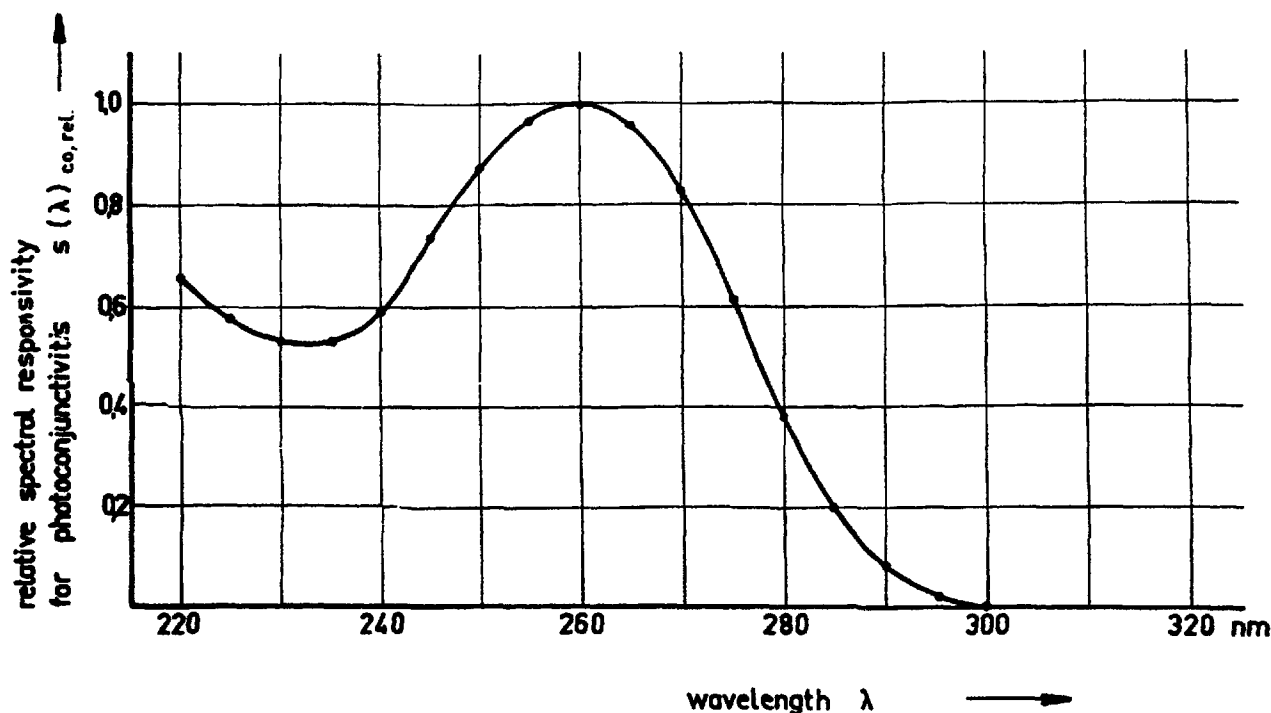


Fig. 3. Relative spectral responsivity $s(\lambda)_{co,rel}$ for photoconjunctivitis according Schulze [10].

This suggestion is supported by experiments made with high pressure mercury lamps, the spectral power distribution of which was deliberately varied by using UV filters (Schott WG-filters). The absorption limit of these filters varied over a range of wavelengths. An action spectrum for photoconjunctivitis has been constructed by determination of the threshold exposure using the various filters and by calculating the differences. Schulze has specified the values of the action spectrum for photoconjunctivitis as shown in Figure 3 and Table 1 [11].

In their experiments on photoconjunctivitis, Henschke and Schulze [10] tested whether conjunctivitis would decrease more rapidly than erythema if UV radiation below 280 nm was filtered out.

For these experiments, as source the so-called UV-Normal after Roessler [12] and a self ballasted high pressure mercury lamp with filter-bulb known under the trade name Ultravitalux were used.

The results of these experiments on volunteer subjects were that the UV-Normal at a distance of 50 cm (without reflector) after an exposure time of 10 seconds provoked no distinct conjunctivitis, but a 15 second exposure produced complaints by the subjects. After an exposure time of 14 minutes with the Ultravitalux lamp in a reflector, at a distance of 50 cm, distinct symptoms were produced. After double exposure time (28 minutes) the conjunctivitis was very strong.

Table 1. Relative spectral responsivity for photoconjunctivitis of the human eye according Schulze. Values are gained from results of experiments on conjunctivitis of the human eye by Henschke and Schulze [10], the action spectrum of killing bacteria and the CIE-erythema action spectrum.

Wavelength λ , nm	Relative responsivity for photo- conjunctivitis, $s(\lambda)_{co,rel.}$
220	0.66
225	0.58
230	0.53
235	0.53
240	0.59
245	0.74
250	0.87
255	0.97
260	1.00
265	0.95
270	0.83
275	0.61
280	0.38
285	0.20
290	0.08
295	0.028
300	0.007
305	0.0014
310	0.00013

Henschke and Schulze drew the following conclusions:

1. As the symptoms provoked by the UV-Normal compared with those provoked by the Ultravitalux lamp were not very distinct even with double and threefold exposure times, the gradient of the effectiveness as a function of the dose, i.e. the gradation is steeper for the Ultravitalux than for the UV-Normal.
2. The ratios of the effectiveness for conjunctivitis to erythema for the UV-Normal is 10.7 to 1, and for the Ultravitalux 0.24 to 1. This effectiveness for conjunctivitis of the UV-Normal which is more than 40 times stronger than that of the Ultravitalux lamp results in action spectra different for conjunctivitis and for erythema. The short wave UV (UV-C) is extremely effective in producing conjunctivitis. The action spectrum for conjunctivitis is similar to the absorption spectrum of the nucleic acid. The maximum of the spectral effectiveness for photoconjunctivitis is at about 260 nm and the threshold exposure (threshold dose) at this wavelength is about 50 J/m².

Calculations using the spectral sensitivity $S_{co,rel}(\lambda)$ and the threshold exposure $H_{es,co}(\lambda) = 50 \text{ J/m}^2$, as proposed by Schulze [11] with natural and artificial light sources, show that photoconjunctivitis in lighting installations due to the UV contents of artificial light sources like fluorescent lamps does not occur. The threshold exposure time in a lighting installation producing an illuminance of 1000 lux would be more than 1000 hours.

Under natural sunlight (global radiation, sun plus sky), the threshold exposure time is 12 hours and more. These values seem to be realistic (Table 2).

Table 2. Exposure time thresholds $t_{s,co}$ in hours resp. in minutes or seconds for photoconjunctivitis under global radiation (sun+sky) and radiation of several fluorescent lamps at an illumination level of 1000 lx.

Global Radiation	$t_{s,co}$
Bener 1972 [13]	31.7 h
Dehne/Kasten 1983 [14]	12.8 h
Fluorescent Lamps* at 1000 lx	
L 40W/15 (daylight)	1060 h
L 40W/25 (universal white)	1700 h
L 36W/21 (neutral white)	9000 h
UV-radiation sources at 1.0 m distance	
UV-Normal [12]	42 s
Ultra-Vitalux*	45 min

*Osram-brand, Germany

Calculations with the spectral power distribution of the Ultravitalux lamp and the UV-Normal [12] result in threshold exposure times of 45 minutes and of 42 seconds, respectively.

4 PHOTOCUNJUNCTIVITIS AND PHOTOKERATITIS

According to early medical nomenclature, the term conjunctiva denotes the tissue covering the eye from the eyelid margin, including the cornea. Thus, the same label included various histologically differentiated tissues. In this respect, it may appear reasonable that some authors assumed that the action spectrum for photokeratitis is identical to that for photoconjunctivitis. Accordingly, the term photo-keratoconjunctivitis has been coined.

The above opinion cannot be shared by CIE Division 6, as the cornea of the human eye and the conjunctiva consist of different tissues and different cells. The most obvious difference is that the conjunctiva is interlaced by several blood vessels and is nourished by blood, whereas the cornea is absolutely free from blood vessels.

5 CONCLUSIONS AND RECOMMENDATIONS

As long as no new data related to the action spectrum and the threshold exposure for photoconjunctivitis are available, CIE Division 6 recommends the use of the values of Schulze according to Figure 3 and Table 1, and the threshold value of 50 J/m² exposure at 260 nm.

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