Ocular effects of ultraviolet radiation from 295 to 365 nm.

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A 5,000 watt Xe-Hg source and a double monochromator were used to produce 6.6 nm. full band-pass ultraviolet (UV) radiation. Pigmented rabbit eyes were exposed to the 6.6 nm. band-pass UV radiant energy in 5 nm. steps from 295 to 320 nm. and at random intervals above 320 nm. Corneal and lenticular damage was assessed and classified with a biomicroscope. Corneal threshold radiant exposure (H_C) rose very rapidly from 0.022 Jcm.$^{-2}$ at 300 nm. to 10.99 Jcm.$^{-2}$ at 335 nm. Radiant exposures exceeding 2 × H_C resulted in irreversible corneal damage. Lenticular damage was limited to wavebands above 295 nm. The action spectrum for the lens began at 295 nm. and extended to about 315 nm. Permanent lenticular damage occurred at radiant exposure levels approximately twice the threshold for lenticular radiant exposure. The importance in establishing both corneal and lenticular damage criteria is emphasized.

Key words: ultraviolet radiation, corneal damage, lenticular damage, cataract, action spectrum, anterior uveitis, rabbits, biomicroscopy, ultraviolet thresholds.

With the advent of proposed standards[1] to protect the health and safety of workers exposed to ultraviolet (UV) radiant energy, it becomes increasingly important to have research data from which valid criteria and effective standards can be established. The ocular effects of UV radiation from 200 to 300 nm. in 10 nm. wavebands have been recently researched[2-7] and the data have provided adequate criteria for protection. Conversely, the wavelength range from 300 to 400 nm. has received little attention despite keratinization of the cornea and lenticular opacities from UV exposure being reported since the early part of this century. More recently, the environmental impact of aerosols and supersonic transport pollutants re-emphasizes the need for research in the UV portion of the electromagnetic spectrum.[8]

The purpose of this research was to establish the effects of UV radiation on the eye in the 300 to 400 nm. wavelength range.

Methods

The source was a 5,000 watt xenon-mercury high-pressure lamp, powered by a 10 kW., DC power supply regulated to ±0.5 percent and capable of delivering from 0 to 80 amp. at 25 to 65

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Table I. UV threshold data for the rabbit cornea and lens

Wavelength (nm.)	*Threshold radiant exposure reversible damage ($Jcm.^{-2}$)*				*Time to reverse*
	Corneal threshold	*Exposure duration (sec.)*	*Lens threshold*	*Exposure duration (sec.)*	
295	0.020	130	0.75	7,339	24 hr.
300	0.050	137	0.15	661	3 days
305	0.070	182	0.30	880	7 days
310	0.055	201	0.75	4,892	2 wk.
315	2.250	3,882	4.50	6,374	1 wk.
320	7.500	23,217	> 8.00		—
335	10.990	18,581	>15.00		—
365	42.500	16,647	>70.00		—

v. to the arc electrodes. The lamp housing was cooled by two blowers. The radiation from the source was focused at the monochromator entrance slit by the housing optics. A 10 cm. quartz-enclosed water chamber was placed between the focusing lenses and the monochromator to remove the infrared radiation. The exit optical beam was focused by a quartz lens with a beam size of 1.6 by 1.8 cm. at the plane of the cornea. The desired UV waveband was obtained with a Czerny Turner double-grating monochromator (Model 25-100; Jarrell Ash, Div. Fisher Scientific, Waltham, Mass.). The gratings were blazed at 300 nm. and grooved with 1,180 grooves per millimeter, allowing an approximately 5.0 nm. band-pass. Entrance, intermediate, and exit slits were set to pass a nominal full band-pass of 6.6 nm. Full band-pass did not exceed 7.0 nm. for all wavebands used in these experiments. The double monochromator system was aligned with a helium-neon laser and the wavelength counter was calibrated with a mercury source. Exposure durations were set with a Gerbands electronic shutter (Ralph Gerbands Co., Inc., Arlington, Mass.) controlled by an H-P Model 5330B preset counter. The preset counter allowed exposure durations of any desired length with millisecond accuracy. A 580/585 radiometer (EG&G, Inc., Salem, Mass.) was used to measure the UV source. The EG&G radiometer was cross-calibrated against an Eppley 16 junction thermopile (Eppley Laboratory, Inc., Newport, R. I.) traceable to an NBS UV standard source. The radiometer was placed in the position occupied by the animal's cornea during exposure.

Healthy adult pigmented rabbits of 4 to 7 lb. were used for the experimental animals. Prior to exposure, each eye was examined with the biomicroscope. Animals with anomalies of the anterior segment of the eye (cornea, anterior chamber, iris, or lens) were rejected. The animals were restrained without anesthesia in a rabbit holder especially designed to permit biomicroscopic examination. The cornea was centered normal to the optical beam with the monochromator set at 450 nm. The eyes were exposed in 5 nm. waveband steps in the wavelength range from 290 to 365 nm. An irradiated eye was not used in further experimentation.

Ocular damage criteria used to determine corneal damage were epithelial debris, epithelial stippling, epithelial granules, epithelial haze, epithelial exfoliation, stromal haze, stromal opacities, and endothelial disturbances. Anterior chamber signs included flare and cells. The crystalline lens criteria were subcapsular opacities, capsular and stromal haze, stormal opacities, and increased prominance of the anterior suture. Lenticular subcapsular opacities were small, discrete, white dots located in the anterior epithelium just beneath the capsule of the lens. Lenticular capsular and cortical haze were the result of increased scatter of the lens capsule and the cortex. Lenticular cortical opacities appeared to be migrations and coalescing of the subcapsular opacities into "clumps." Criteria for the iris were the presence of the anterior chamber signs, changes in clarity of the iris stroma, and sluggish pupillary response.

Two observers independently determined the criteria status and classification of each eye. The severity of the reaction of the eye to the radiant exposure for the corneal criteria was indicated as negative (-), probably positive but not certain (±), positive (+), moderately positive (++), severely positive (+++), and extremely positive (++++). If five or more corneal criteria were positive (+), the eye was classified as above threshold (+). Three to four positive corneal criteria were classified as threshold (±). Fewer than three positive corneal criteria resulted in a below-threshold classification (-). Any lens or anterior chamber signs resulted in a positive (+) classification. The lowest radiant exposure resulting in an above-threshold classification terminated the experiment for that waveband. Conventional statistical rounding procedures were used. All data were rounded to two significant figures.

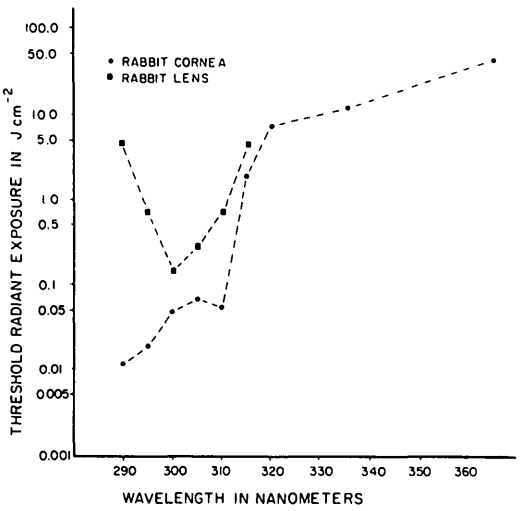

Fig. 1. The action spectra of radiant exposure for corneal and lens thresholds for the rabbit. The symbols are as follows: dashed lines represent the rabbit cornea threshold (●); rabbit lens thresholds are represented by (■). The rabbit lens threshold is reversible damage.

Results and comments

UV exposures were made on 106 pigmented rabbit eyes. The threshold radiant exposure data for the cornea (H_C) and the lens (H_L) are summarized in Tables I and IV. H_L levels have not been achieved at wavebands 320, 335, and 365 nm.

Fig. 1 shows the H_C's and H_L's for the rabbit in the 295 to 365 nm. wavelength range. At 300 nm and above, the lens and cornea curves are relatively parallel up to about 320 nm. From the shape of the curves, it appears that the H_C's and H_L's will become almost equal above 320 nm. The action spectrum previously established for the H_C extended from 210 nm. to about 315 nm.; however, corneal damage was produced at longer wavelengths in this study. The action spectrum for the lens begins at 295 nm. and may extend to about 335 nm.; however, the most effective wavelength range for producing lenticular opacities was from 295 to 315 nm. An important finding in this study was the relatively low radiant exposures in the 295 to 315 nm. wavelength range required to produce lenticular opacities. The sharp rise in the H_L below 300 nm. was probably due to the absorption characteristics of the cornea, since complete absorption of the UV radiation occurs at 290 nm. and below. The radiant exposure required to produce a threshold response at 295 nm. was five times the threshold value at 300 nm. (0.75 vs. 0.15 Jcm.$^{-2}$), which supports establishing the lower wavelength of the lens action spectrum at 295 nm. Further support was shown by the fact that radiant exposures at 290 nm. of 3.0 Jcm.$^{-2}$ did not result in lenticular opacities.

Corneal changes followed a consistent pattern of increased involvement as the radiant exposure was increased. At H_C and above, there was a consistent increase in epithelial debris. The granules observed at wavelengths between 290 and 300 nm. coalesced to form a network following higher radiant exposures. Epithelial damage, stippling, and haze were detected within 2 hr. following exposure. Stromal damage, haze, and opacities appeared within 24 hr. after exposure. Endothelial changes were noted within 4.5 hr. after exposure. Exposures of $2 \times H_C$ usually resulted in irreversible damage to the cornea.

Severe corneal reactions were accompanied by secondary anterior uveitis. Anterior uveitis is characterized by ciliary injection, aqueous flare, and membranous inflammatory by-products deposited on the corneal endothelium (Fig. 2). Anterior uveitis made it difficult to observe the lens or its capsule. The inflammation usually regressed spontaneously within approximately 2 days. Table II provides information on the wavelength, radiant exposure, time after exposure, and time to recover from anterior uveitis. It is apparent from Table III that no anterior uveitis was induced by UV radiant exposures above 315 nm. Lenticular involvement was initially considered secondary to the anterior uveitis; however, the appearance of the characteristic UV-induced lens changes without and prior to the anterior uveitis negated this hypothesis.

At 295 nm. the radiant exposure required

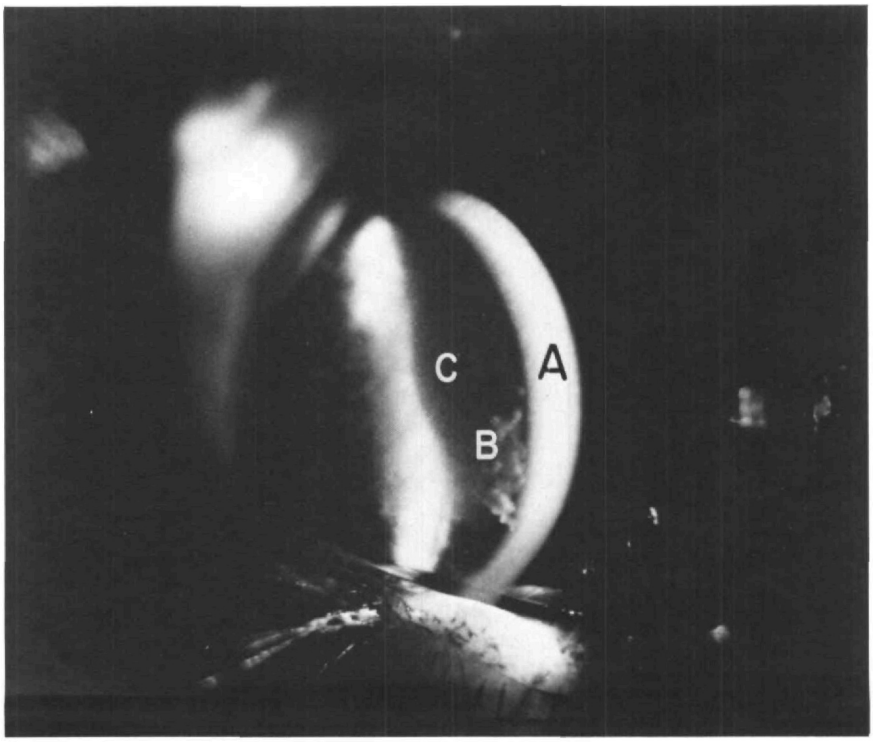

Fig. 2. An optical section of the anterior segment of the rabbit eye which demonstrates anterior uveitis: cornea. *A;* membraneous inflammatory by-products on the corneal endothelium, *B;* and aqueous flare, *C*. Animal H080R, 305 nm., radiant exposure 0.5 Jcm.$^{-2}$, 27 hours after exposure.

Table II. Anterior uveitis

Wavelength	*Radiant exposure (Jcm.$^{-2}$)*	*Exposure duration (sec.)*	*Time after exposure (hr.)*	*Recovery (hr.)*
295	0.75	7,339	1	24
300	0.50	1,886	24	48
305	0.30	880	24	48
310	1.00	4,036	2	24
315	No anterior uveitis found			
320	No anterior uveitis found			
335	No anterior uveitis found			

to produce a positive lenticular disturbance also produced an immediate corneal reaction with epithelial haze, granule formation, stippling, and anterior stromal haze over the entire irradiated area. The epithelium stained extensively with sodium fluorescein, confirming the immediate response. The severity of the reaction increased to complete exfoliation of the irradiated area at 20 hr. after exposure. The anterior stromal haze of the cornea also increased as the radiant exposure increased. The anterior chamber, iris, and lens were difficult to assess with radiant exposures exceeding 0.75 Jcm.$^{-2}$ Severe anterior uveitis was found 1 hr. after exposure with a radiant exposure of 0.75 Jcm.-2 and regressed within 24 hr.

Radiant exposures above 0.3 Jcm.$^{-2}$ at 300 nm. ($2 \times H_L$) resulted in immediate corneal damage. The animal displayed extreme photophobia. Permanent damage resulted to the corneal epithelium, stroma, and endothelium. There was a thickening of the cornea and dense posterior stromal striation. The iris was swollen and showed a sluggish pupillary response. The anterior

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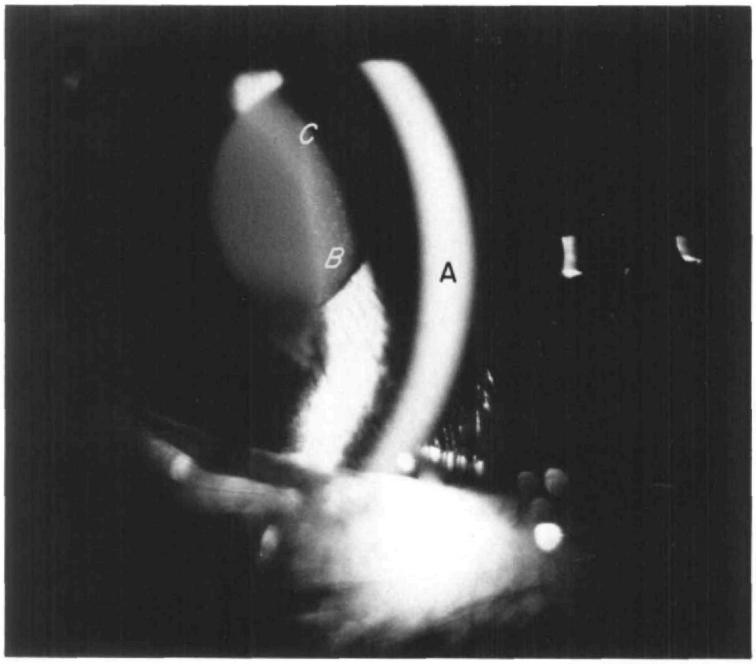

Fig. 3. Ultraviolet lenticular damage of the rabbit eye. The optical section demonstrates the cornea, *A;* dot-like, discrete, anterior subcapsular opacities, *B;* and the anterior suture line, *C,* a vertical whitish line bisecting the pupil. Animal HOCR, 310 nm., and 1.5 Jcm.$^{-2}$ radiant exposure, 24 hours after exposure ($2 \times H_L$).

chamber demonstrated a slight flare and a few cells were found in the aqueous. The corneal thickening indicated an interference with the normal metabolism of the endothelium. Minor anterior uveal changes were found at a 0.2 Jcm.$^{-2}$ radiant exposure which returned to normal within 3 days. Below 0.2 Jcm.$^{-2}$ anterior uveal changes were not found.

At 305 nm. radiant exposures above 0.3 Jcm.$^{-2}$ resulted in granules, opacities, stippling, and fluorescein staining of the corneal epithelium. In addition, the stroma of the cornea was hazy and developed opaque striae after about 8 days. Within 24 hr. there were severe fibrinous endothelial deposits. An aqueous flare was noted within 4 hr. following exposure but this reduced in severity within 24 hr. All signs of anterior uveal inflammation disappeared within 8 days. There was an exfoliation of iritic tissue within 24 hr. after exposure at the 1.0 Jcm.$^{-2}$ radiant exposure. Below 0.3 Jcm.$^{-2}$ no anterior uveal changes were found at 300 nm. There was a general increase in corneal damage as the radiant exposure was increased above the corneal radiant threshold of 0.04 Jcm.$^{-2}$

Radiant exposures of 1.5 Jcm.$^{-2}$ at 310 nm. ($2 \times H_L$) produced a very minor aqueous flare which disappeared within 48 hr. Permanent lenticular opacities and stromal opacities were also found at this level of radiant exposure (Fig. 3). The exposure also resulted in a slight area of endothelial exfoliation with an increase in corneal thickening limited to the area of exfoliation. There was an increase in corneal involvement as the radiant exposure exceeded the H_C value of 0.047 Jcm.$^{-2}$

At 315 nm. no anterior uveal or aqueous changes were found with radiant exposures up to 7.0 Jcm.$^{-2}$ The general pattern of epithelial haze, and stromal haze increased in severity as the radiant exposure increased, with a generalized, diffuse fluorescein staining of the epithelium.

There is no description of UV-induced lenticular damage in the literature from which lenticular damage criteria could be established. The first biomicroscopic signs of lenticular damage were a loss or reduc-

Table III. Transient lenticular opacities

Wavelength (nm.)	*Radiant exposure ($Jcm.^{-2}$)*	*Exposure duration (sec.)*	*Appearance of lens opacities (hr.)*	*Disappearance of lens opacities*
295	0.75	7,339	2	24 hr.
300	0.15	661	12	3 days
305	0.30	880	24	7 days
310	0.75	4,892	24	2 wk.
315	4.50	6.374	48	1 wk.

Table IV. Permanent lenticular opacities

Wavelength (nm.)	*Radiant exposure ($Jcm.^{-2}$)*	*Exposure duration (sec.)*	*Appearance of lens opacities (hr.)*	*Performance* of lens opacities*
295	1.0	8,064	2	Permanent
300	0.5	1,866	24	Permanent
305	0.5	1,528	24	Permanent
310	1.5	6,053	24	Permanent
315	6.0	8,721	24	Permanent

*Lenticular opacities present 1 month after exposure.

Table V. Comparison of monochromator thresholds with laser thresholds

	Corneal threshold	*Lenticular threshold*
Pitts and Cullen, 335 nm.	10.99 $Jcm.^{-2}$ Reversible damage	>15.0 $Jcm.^{-2}$
Ebbers and Sears, He-Cd laser, 325 nm.	0.8 $Jcm.^{-2}$ Reversible damage	6.5 $Jcm.^{-2}$ Permanent cataracts
MacKeen, Sears, and Fine, He-Cd laser, 325 nm.	—	28.80 $Jcm.^{-2}$ Permanent cataracts
Zuchlich and Connolly:		
CW Krypton-ion laser 350.6 + 356.4 ≅ 1:3	≅ 60-70 $Jcm.^{-2}$	(retinal lesions)
Nitrogen laser	≅ 10 $Jcm.^{-2}$	≅ 1 $Jcm.^{-2}$

tion of "orange-peel" appearance of the anterior capsule and an increased prominance of the vertical anterior suture line. These two biomicroscopic signs regressed to normal within 24 hr. after exposure. As the radiant exposure approached H_L, many small, discrete white dots appeared in the anterior subcapsular epithelium of the lens (Fig. 3). The anterior subcapsular opacities appeared similar to the corneal epithelial granules. Table III presents the wavelength, the radiant exposure necessary to produce the lenticular opacities, the time of appearance, and the time of disappearance of the lenticular opacities after exposure.

Suprathreshold exposures resulted in permanent lenticular opacities. The change from the small, discrete, white, anterior subcapsular dots into the permanent opacities developed as follows. The fine discrete opacities coalesced and migrated posteriorly into the anterior cortex of the lens. At the same time, an increase in the anterior lens cortical haze was detected. The permanent opacities usually developed in proximity to the anterior suture line. Vacuoles were occasionally observed in addition to the permanent opacities. Table IV gives the wavelength radiant exposure and the time of appearance of the permanent lenticular opacities following exposure. Table IV demonstrates that permanent lenticular opacities were not induced until the radiant exposure reached approximately 2 × H_L. An animal observed at regular intervals

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for 3 months after exposure showed neither nuclear nor posterior subcapsular opacities.

Discussion

A comparison of our monochromator threshold data with laser threshold data is provided in Table V. Zuclich and Connolly[9] postulated corneal damage to be photochemical and lenticular damage to be thermal from laser exposures. These damage differences were based on the reciprocity relationship ($I \times t = k$) for corneal damage and the lack of reciprocity for lenticular damage. Table I shows that the H_C for noncoherent, long-exposure UV radiation at 335 nm. was 10.99 Jcm.$^{-2}$ and at 365 nm. the H_C was about 42.5 Jcm.$^{-2}$ The H_C of 10.99 Jcm.$^{-2}$ at 335 nm. compares favorably with the nitrogen laser (337.1 nm.) corneal threshold of 8.4 ± 3.3 Jcm.$^{-2}$.[10, 11]

The data of Zuclich and Connolly show that the argon laser (351.1 and 363.8 nm.) lenticular threshold was 76 Jcm.$^{-2}$ for a 4 sec. exposure and was 19 ± 1.8 Jcm.$^{-2}$ for 1 sec. exposures for a corneal irradiance of 19 Wcm.$^{-2}$. These differences in responses could be due to wavelength rather than thermal causes. Fig. 1 demonstrates the action spectra for the production of corneal and lenticular damage found in this study. The curve indicates that corneal threshold damage at 365 nm. should be approximately one magnitude above the corneal threshold damage at 335 nm.; therefore, the increased radiant exposure required to produce lenticular damage at the higher wavelengths could be due partly to the relative effectiveness of the 365 nm. wavelength in producing a photochemical reaction rather than being thermal in nature. However, the high-power short duration of the laser exposures may induce thermal changes. The rate of delivery may affect damage which would make the comparison of the laser with the broad-band, long-duration exposures more difficult.

Ham et al.[12] have reported retinal damage from the short wavelengths of the visible spectrum (488, 457.8, and 441.6 nm.) produced by the laser. They reported that the temperature rise for wavelengths below 500 nm. was too small to account for the retinal lesions. Thus, the retinal damage may be induced by photochemical changes. The radiant energy necessary for threshold retinal damage was less than for longer wavelengths.

It is assumed that both the corneal damage and the lenticular damage found in this study are photochemical. This assumption is based on the fact that the corneal and lenticular damage produced the similar granular appearance. Additionally, corneal damage and lenticular damage were not obtained at threshold radiant exposure levels until after a rather long latency, whereas thermal damage usually occurs immediately after the tissue has been exposed. It may be that the low-power, continuous, long-duration exposures provided a radiant exposure which affected the lens in both a photochemical and thermal response.

Several conclusions can be raised relative to the biochemical studies.[13-26] Near-UV light in the 300 to 400 nm. wavelengths from the sun or artificial lights is transmitted by the cornea and maximally absorbed by the crystalline lens. Most of the research has been in vitro and on the mouse and dogfish. The corneas of these animals most probably do not possess the same transmittance characteristics as the rabbit or human cornea and thus could not afford the same protection against the near-UV light. However, studies on human cataractous lenses demonstrate that some of the biochemical changes found for certain cataracts may be equivalent to those found in the animal studies. The human lens does absorb most of the near-UV light in the 320 to 370 nm. bandwidth, which may cause photo-oxidation of isolated lens proteins, induce pigmentation, and cause an increase in crosslinking to take place. The question of whether lens protein is directly photo-oxidized by near-UV light, or is mediated by sensitizers inherent in the lens, or if free amino acids are photo-oxidized and bound covalently to the pro-

teins, has not been conclusively demonstrated.

Biochemical research indicates that exposure of the eye to near-UV light for sufficient periods of time with a low irradiance comparable to the irradiance level of sunlight may interfere with the synthesis of lens proteins, may catalyze insoluble lens protein, and may result in chromatic changes in the lens. Although the basic mechanism remains to be found, the evidence clearly demonstrates that both in vitro and in vivo exposure to the near-UV light can enhance cataractous changes in the crystalline lens.

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