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Effects of Ultraviolet Light on the Eye: Role of Protective Glasses

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Global atmospheric changes such as depletion of ozone in the stratosphere are thought to lead to increased levels of ultraviolet radiation on earth. This can have adverse effects on human health, and long-term effects of ultraviolet light on the eye are of increasing concern. Ultraviolet light exposure to the eye has been associated with cataract formation and retinal degeneration. In both cases, it is hypothesized that ultraviolet light can initiate formation of free radicals, which can cause protein modification and lipid peroxidation. Several procedures can be recommended to prevent ultraviolet light damage to the eye, such as the use of suitable protective glasses when outdoors.

Introduction

Several effects of exposure of the eye to excessive light have been well established, including photokeratitis, erythema of the eyelid, cataract, solar retinopathy, and retinal damage (*1,2*). This paper discusses the role of ultraviolet (UV) light exposure in cataract formation and retinal degeneration.

Global atmospheric changes such as stratospheric ozone depletion may increase the level of UV radiation on earth (*3*). This would contribute to enhanced chronic exposure of the skin and eye tissues of humans to UV light (*1,3*). Hazardous effects of UV light on the eye were already recognized in 1920 by van der Hoeve (*4*). Biochemical changes that take place in eye tissues during light exposure are discussed, with emphasis on alterations in the lens and the retina.

One mechanism that is thought to play an important role involves generation of oxygen radicals, which can cause lipid peroxidation and protein modification. This process is also thought to be related to aging and many human diseases, including cancer, atherosclerosis, and arthritis (*5,6*).

Lipid peroxidation has received an enormous amount of attention, and Esterbauer illustrated the enormous increase in the numbers of papers published in this field between 1972 and 1985 (*7*). Unfortunately, most of the research exploring the role of lipid peroxidation in human disease states is based on the measurement of malondialdehyde (MDA) by its reaction with thiobarbituric acid (TBA). This method, often called the TBA test, is nonspecific and not very sensitive (*8*).

During previous studies on lipid peroxidation in retinal degeneration, highly sensitive and specific gas chromatography–mass spectrometry methods were developed for detecting phospholipid peroxides and aldehydic products of lipid peroxidation (*9–12*). It was found that these products of lipid peroxidation are present in vitamin E-deficient and -supplemented rat retinal tissues (*13–15*). Oxidized fatty acids in membrane phospholipids significantly alter the permeability of cell membranes, which may contribute to malfunction of cells and eventually to cell death (*16*). Vitamin E is an antioxidant known to inhibit lipid peroxidation, and vitamin E deficiency can cause structural changes in membranes (*17*). In addition, aldehydic products of lipid peroxidation, especially 4-hydroxyalkenals, are very reactive and combine with amino and sulfhydryl groups of proteins (*18,19*). Inactivation of proteins due to modification by 4-hydroxyalkenals is another mechanism by which retinal damage may occur through lipid peroxidation (*13*).

Cataract

A cataract is an opacity of the lens, which can vary in degree of density and is usually associated with aging (senile cataract) (*20*). There are three major types of senile cataract: *a*) nuclear cataract in the nucleus of the lens; *b*) cortical cataract in the surrounding cortex; and *c*) posterior subcapsular cataract, which occurs beneath the posterior capsule of the lens. Other causes of cataract are associated with systemic diseases (*21*), such as diabetes, galactosemia (*22*), microwave or ionizing radiation (*23*), trauma, the use of photosensitizing drugs (*24*), and UV light (*1,20,25*).

It is thought that reactive oxygen species and free radicals (*26*) may play a role in the formation of cataracts by oxidation of lipids (*27*) and modification of proteins (*28*). Lipid peroxidation can be inhibited by various antioxidants such as vitamin C and vitamin E, the glutathione system, superoxide dismutase, and catalase (*29*). These antioxidants deplete in the lens with increasing age (*30*), which is thought to contribute to cataract formation due to increased oxidative stress (*31*). It is known that antioxidants are decreased (vitamin C) or absent (glutathione) in the cataractous lens. Recently, Jacques et al. reported that the oxidant systems may play a role in cataractogenesis (*32,33*). They found that individuals with high plasma levels of two or more vitamins that influence the antioxidant status (vitamin C, vita-

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min E, carotenoids) appear to have a reduced risk of cataract (*33*). There appeared to be no difference in the levels of erythrocyte superoxide dismutase, glutathione peroxidase, and glucose-6-phosphate dehydrogenase enzymes between subjects with and without cataracts (*33*). These results suggest that the level of lens antioxidants could perhaps be influenced by the plasma levels of antioxidants.

The decrease of antioxidant levels in the human lens could lead to an increase of lipid peroxidation products. Bhuyan et al. (*27*) found an increase of malondialdehyde in the human lens with age. However, Borchman et al. found no changes with age in the concentration of a phospholipid malondialdehyde adduct, which was also measured as an indicator of lipid peroxidation (*34*). They argue that hydrolysis of the adduct may explain its constant levels. Perhaps the specific gas chromatography–mass spectrometry methods for measurement of lipid peroxidation products (*9–12*) may provide a better tool to test the hypothesis that lipid peroxides increase in the human lens with age.

Alteration of lens proteins is another biochemical mechanism that has been proposed following UV radiation (*28,35,36*). These biochemical changes can occur through a direct effect of UV light or by virtue of photosensitivity (*35,37*). Studies on animals and humans have shown several effects. A 90% decrease in Na^+K^+-ATPase activity was found following 20 hr of *in vitro* exposure to UV radiation of 365 nm at 5 mW/cm^2. Chronic exposure to UV light was found to lead to abnormal lens epithelial cell differentiation and enhancement of aggregated proteins in mice lenses within several months, and cortical cataracts developed after 1.5 years (*38*). Grey squirrel lens have also been employed because they have a yellow near-UV absorbing pigment similar to that present in the human lens. Following UV exposure *in vivo*, aggregation of soluble proteins and enhancement of low molecular weight peptides was found. In *in vivo* studies, near UV radiation enhanced levels of insoluble protein. Fluorescence properties changed because of a decrease of the usual tryptophan, and an increase of nontryptophan fluorescence was found. Goosey et al. reported the formation of singlet oxygen as a major mechanism by which lens proteins were aggregated (*39*), and Jedziniak et al. found that singlet oxygen altered the properties of the crystallin component of the human lens, in addition to damage to several associated enzymes (*40*). Dillon et al. identified the crosslinks of insoluble proteins from brunescent cataracts as tryptophan oxidation products (*41*).

During recent years, there has been increasing evidence showing that UV radiation could be a risk factor in the formation of human cataracts. The mechanisms are unknown, but could depend on protein aggregation due to free radical reactions as discussed above. It has been proposed that chronic exposure to ambient UV radiation results in generation of a series of fluorescent chromophores. These chromophores are associated with a deepening yellow color of the lens nucleus (*37*). Furthermore, acute exposure of the lens to higher levels of UV light than normally present in the environment has been implicated in cortical opacities (*25*). A relationship between sunlight exposure and cataracts has been suggested, based on basic chemical studies (*25,26,35*).

Epidemiological studies supported the hypothesis that persons who live in geographic areas where there are long periods of sunlight (*42*) have an increased incidence of cataract (*20,43–45*). Zigman et al. reported that exposure to sunlight specifically enhances the development of dark brown cataracts in humans (*43*). Recently, more detailed epidemiological surveys have been performed, and the relation between UV light exposure and the development of senile cataract was quantified (*46,47*). These studies were based on a protocol to measure the dose of UV radiation in different occupational groups (*48*).

UV light is usually divided into three portions, UV-C (200–290 nm), UV-B (290–320 nm), and UV-A (320–400 nm). An association between cumulative exposure to UV-B radiation after age 15 and cortical cataract formation was recently reported by Taylor et al. (*46*). They did not find an association between nuclear cataract and UV-B exposure or between any type of senile cataract and UV-A exposure (*46*). Since relatively few subjects with posterior subcapsular cataracts participated, no attempt was made in their study to correlate this type of senile cataract to UV light exposure. However, Bochow et al. reported that a history of high exposure to UV-B is associated with an increased risk for development of posterior subcapsular cataract (*47*).

Treatment of cataract has been dramatically improved with modern surgical techniques. The lens is typically removed from the eye (lens extraction) and is usually replaced with an intraocular lens. Since cataracts can be treated with a high success rate, research efforts on the biochemical pathways behind cataract may have been reduced. Although cataracts are now effectively treated in Western societies, cataract is still the leading cause of blindness in third world countries (*20*). Surgical treatment is not available worldwide and operations for cataracts have nearly doubled during the past decade in wealthy countries using an increasing proportion of resources for health (*20*). Therefore, research efforts on finding the cause of cataract should be maintained, this work could eventually lead to effective treatment plans or recommendations for lifestyle changes that may prevent this disease worldwide.

Retina Degeneration

The retina is sensitive to light and can be damaged mechanically, thermally, and photochemically (*49*). Mechanical damage results from short-term high irradiance levels such as obtained with YAG laser photodisrupters. Thermal damage occurs when absorbed light raises the temperature in the retina by 10 to 20°C and also depends on short-term exposure. Photochemical damage is induced by relatively long-term exposure to lower levels of light in the UV and blue regions of the spectrum. Damaging levels and colors of light are thought to initiate chemical reactions. Kremers and van Norren suggested two classes of photochemical damage of the retina (*50*), whereas Rapp et al. found separate mechanisms for retinal damage by UV-A and mid-visible light (*51*).

To understand why photochemical reactions may occur in the retina following light exposure, it is helpful to review the structure of the retina. The retinal photoreceptors convert light into an electrical signal, which is sent to the occipital cortex. The photoreceptor outer segments consist of a plasma membrane that surrounds a pile of disc membranes. The discs are lipid bilayers containing the lightsensitive protein rhodopsin (*52*). The phospholipids of the disc membranes have a unique fatty acid composition. About 50 mol% of the fatty acid content of the disc

consist of docosahexaenoic acid (22:6ω3) (*53*), which is the most highly polyunsaturated fatty acid (PUFA) known. Susceptibility to lipid peroxidation increases in proportion to the amount of double bonds (*54*). Light exposure may also be a factor that plays a role in promoting lipid peroxidation because light of appropriate wavelength may trigger photooxidation reactions (*55,56*). Furthermore, there is an excellent oxygen supply to the photoreceptors through the choroid and retinal vessels causing a high oxygen tension in the retinal pigment epithelium (RPE) and photoreceptors (*49*).

Since high PUFA and oxygen are often combined with abundant light in the retina, Noell et al. first suggested a role for lipid peroxidation in photochemical retinal damage in 1966 (*57*). Noell later showed that light damage to the rat retina is enhanced when body and eye temperature are elevated and that the extent of damage is also related to history of light intensity of the animal before acute light exposure (*58*). Noell's work initiated many studies investigating the role of antioxidants in retinal degeneration, in which vitamin E-deficient and -supplemented animals were most often used. These studies were reviewed by Handelman and Dratz (*29*), and they showed microscopic degeneration of the photoreceptors after exposure to light when animals were reared in the dark or in low-level cyclic light. The morphological changes after light exposure were usually enhanced in vitamin E-deficient animals as compared to vitamin E-supplemented animals (*29*). Biochemical changes investigated showed that antioxidant-deficient rats accumulated fluorescent pigment thought to be characteristic of lipid peroxidation in the retinal pigment epithelium (*59*), the conjugated diene content in the retina doubled (*60*), and the polyunsaturated fatty acids in the retina decreased (*61*). These studies all suggested a role for lipid peroxidation in retinal degeneration. However, to prove that lipid peroxidation products are formed in retinas of vitamin E-deficient rats, gas chromatography–mass spectrometry (GC-MS) assays were developed and employed (*9–12*). Using these specific and sensitive methods, it was possible to show the presence of oxidized fatty acids and 4-hydroxyalkenals in rat retinas (*13–15*). The levels of these lipid peroxidation products were higher in vitamin E-deficient as compared to vitamin E-supplemented retinas, giving more support for the involvement of lipid peroxidation in retinal degeneration (*13,14*), although firm evidence is not yet available on the effect of light.

Evidence that UV light exposure has a variety of adverse effects on the retina is accumulating (*49*). Different sources of damage from excessive light are possible, including sunlight (*62,63*), arcs of electrical current (*64*), lasers (*65,66*), and operating microscopes (*67,68*). It has been proposed that lipid peroxidation is an important factor in solar radiation contributing to age-related macular degeneration (ARMD) (*62*). ARMD deserves attention because it is the major cause of blindness in the United States and Western Europe in the elderly and is likely to become a greater public health problem as the number of persons in older age groups continues to increase (*69*). ARMD is characterized by histopathological features of pigmentary disturbances, drusen, thickening of Bruch's membrane, and basal laminar deposits (*70*). It is apparently influenced by a large number of variables such as nutrition, age, family history, iris pigmentation, and solar radiation (*71*).

The role of solar radiation in ARMD was recently reviewed by Young (*62*), and the major concepts of the radiation hypothesis proposed by Young can be summarized as follows. Damaging effects of radiation are dependent on the wavelength. Because the energy content is highest at lower wavelengths, this higher energy light is more damaging to various eye tissues (*72,73*). A critical point appears to be the wavelength of 510 nm. Light below this wavelength has a photon energy of > 2.43 eV, and a sudden rise occurs in the amount of damage produced in the retina as a function of exposure to light of different wavelengths (*62,72,73*). Therefore, the blue portion of the visible region (400–500 nm) is much more hazardous than the green and red portion (500–700 nm). This phenomenon is referred to as blue light hazard (*62*).

The lens transmits light of lower wavelengths to a different extent at various ages (*74–77*). Lerman reported that after birth, nearly all of the UV light is transmitted by the lens (*77*). During childhood, lens transmittance decreases, and by the age of 25, the lens absorbs UV light almost completely, as shown in Figure 1*A*. It is interesting to correlate this result with the data on lipofuscin accumulation in the retinal pigment epithelium as a function of age shown in Figure 1*B*. Wing et al. demonstrated that there is a steady increase of lipofuscin content during the first three decades, after which it does not change significantly until the eighth decade (Fig. 1*B*) (*78*). Comparing these data provoke the hypothesis that the accumulation of lipofuscin in the RPE at younger ages is associated with exposure of the retina to UV radiation transmitted by those lenses. As lipofuscin is thought to be an end product of lipid peroxidation, this mechanism could have an important contribution to biochemical changes mediated by UV light. These data are consistent with the hypothesis by Weiter et al. that cumulative exposure to UV light before the age of 25 is a risk factor in ARMD (*79*).

Several epidemiological studies have recently been performed studying the role of light in development of retinal disease. Liu et al. showed an association between occurrence of ARMD and lens opacities in the aged (*80*). They found that the odds for having ARMD may be increased by 50% among persons with cataracts, 80% in persons with opacities that do not decrease visual acuity, and 200% among aphakic persons (*80*). They hypothesize that the weak correlation between cataracts and ARMD in their group of subjects could be due to the fact that cataracts decrease the transmission of light to the retina, providing some protection from light damage (*80*). On the other hand, the strong increase in occurrence of ARMD in aphakic persons, where the UV absorbing lens is not present, suggests that UV light exposure could be an important risk factor for ARMD (*80*).

West et al. found no association between ARMD and cumulative exposure to either UV-A or UV-B light (*81*). This study was conducted on 838 Maryland watermen from whom cumulative exposure to UV-A and UV-B light after age 15 was well documented. A weakness of this study is perhaps that the cumulative exposure to UV light before age 15 was not established; however, it could be practically impossible to obtain such data. As indicated above and in Figure 1*A*, the human retina may receive most of its cumulative UV exposure during the first 15 years of life (*77*). The results of the two studies evaluating light as a risk factor for ARMD described above do not agree, and further studies are warranted to resolve the question of whether cumulative UV light exposure during life is a risk factor for ARMD.

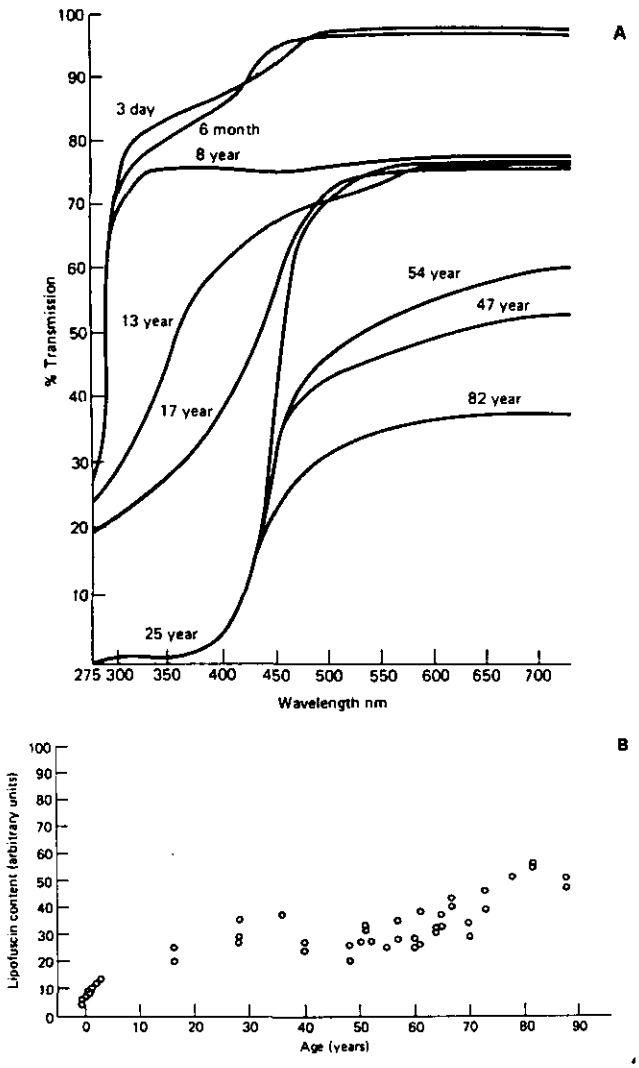

FIGURE 1. (*A*) Transmittance of the human ocular lense with age. From Lerman (*77*), with permission. (*B*) Lipofuscin accumulation in the posterior pole of human retinal pigment epithelium as a function of age. From Wing et al. (*78*), with permission.

Recently, Munoz et al. reanalyzed the data obtained from the study (*81*) on 838 Maryland watermen (*82*) to determine whether ocular exposure to blue or visible light is associated with ARMD. They found that established cases of ARMD had significantly higher exposure to both blue and visible light over the last 20 years and concluded that high levels of exposure to blue and visible light late in life may be an important factor in ARMD (*82*). Interestingly, these results differ from those by Weiter et al. (*79*) discussed above.

Prevention of UV-Light Damage to the Eye

The eye can be protected against excess sunlight exposure in many different ways (*83–85*). These include goggles with narrow slits as used by the Eskimos; hats with large brims like the stetson, the pith helmet, and the sombrero; and protective glasses. Rosenthal et al. performed an interesting study on light blocking effects using mannilin headforms containing UV-B sensitive film, fitted with brimmed baseball caps (*84*). They showed a 22 to 95% reduction in ocular exposure to light depending on the angle of the hat brim on the forehead. In addition, they found that the amount of UV-B exposure was independent of the cloud cover (*84*). This study demonstrates that a habit of wearing a hat with a brim outdoors greatly reduces the amount of sunlight exposure to the eye.

Protective glasses are commonly recommended to protect the eye from harmful effects of UV radiation (*1,3*). However, several studies showed the occurrence of UV windows in commercial sunglasses and commonly used clip-on sunglasses (*85–90*). UV transmitting windows are not desirable because tinted sunglasses may cause pupillary dilatation, which would increase the exposure of the retina to UV light when the sunglasses used have a UV window (*88*). On the other hand, it has been proposed that visible light energy in a sunny outdoor environment penetrates dark tinted glasses sufficiently to produce significant pupillary constriction (*87*). It is also reported that darker lenses do not assure sufficient blocking of low wavelength light (*88*). Protection of the retina against UV light exposure is a concept that has also received attention from ophthalmic surgeons. Intraocular lenses implanted during surgery for cataracts now have chromatophores incorporated that block UV light. This will help to counteract a sudden increase of exposure of the retina to UV light, which was blocked by the cataract before surgery.

Although several publications in professional journals have shown problems with UV transmission of sunglasses (*86–88,90*) and standards have been proposed (*88,91*), these problems are not yet eliminated. My recent experiences are illustrated in Figure 2, which shows transmission curves of several different glasses. Curve I is the transmission of a clear lens from corrective prescription glasses which are used continuously throughout the day. A UV filter was requested for these lenses when purchased, and it was said by the sales person that UV filters were present in any lens sold in that store. When the new glasses were picked up several weeks later, it was noticed that no yellow tint was present, which is often seen when a UV coat is applied. The sales manager was asked if a UV coat was present, and assurance was given that the lenses (purchased for $70 each) had a new type of transparent UV coating. The glasses were purchased and several months later a transmission curve was obtained with a Perkin Elmer 576 UV-Visible spectrophotometer. It was found that the UV-B light is blocked, but that a substantial fraction of UV-A light is transmitted (Fig. 2, curve I). This was reason to approach several local "eye care" stores, and various samples of clear lenses were borrowed that were said to have a UV absorbing coat. There was a substantial difference in UV light transmittance properties as reported before for sunglasses (*86–88,90*) and tinted lenses (*89*). Some lenses blocked all of the UV light ($<$ 400 nm), while others showed transmittance similar to curve I, Figure 2.

Curve II in Figure 2 is an average transmittance curve of several lenses coated with blue blocking filter, and it shows that some of the UV-A light is not blocked and that 10 to 20% of the blue light is still transmitted. The transmittance is equal to or higher than the 10% blue light transmittance recommended by Silver (*88*).

The third curve in Figure 2 represents the transmission of prescription sunglasses that were recently purchased. A UV

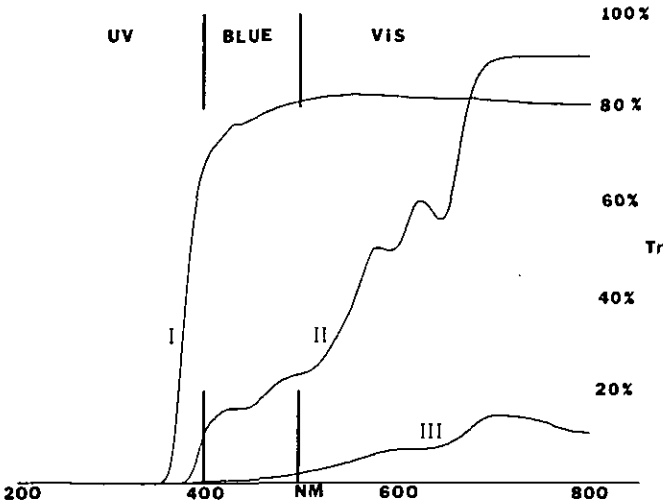

FIGURE 2. Transmittance curves from several prescription glasses. (I) Transmittance of regular prescription glasses with UV filter. (II) Transmittance of lens with blue blocker. (III) Transmittance of prescription sunglasses.

filter was added to a dark colored lens, and it was agreed that a transmission curve could be obtained before payment ($65 per lens). The results are shown in Figure 2, curve III, and demonstrate that essentially all of the UV light is blocked and that the transmission of blue light was reduced to 1 to 3%. In addition, transmission was highest at the long wavelength of the visible region at about 700 nm and decreased in the infrared region. Because infrared radiation may enhance thermal light damage to the retina, reduction of infrared radiation is also desirable (*83*). These UV protective glasses serve well for driving and outdoors sports activities.

The question remains what should be recommended to the public as far as reduction of ocular exposure to hazardous light is concerned. Should education start with children? Recently, the U.S. Council on Scientific Affairs recommended that children should be taught to use sunscreen protection early in life because skin damage accumulates over an entire lifetime (*1*), but no recommendations were made about the use of glasses or hats to protect the eyes when outdoors. To develop recommendations for children, several factors should be taken into consideration.

The ocular lens of children transmits UV light (*77*), and most UV light damage to the eye may occur in the earlier years of life. Weiter et al. showed that the incidence of ARMD is lower in myopic subjects, who had been forced to use glasses at a young age, as compared to hyperopic subjects who did not start using glasses early in life (*79*). Since the glasses filter a part of the UV light, Weiter et al. suggested an association between development of ARMD and UV light exposure in the first 25 years of life. The results of this study favor a recommendation to teach children the use of protective glasses or hats. On the other hand, Lerman et al. reported that ambient low levels of UV light assist the development of UV-absorbing chromophores in the young lens (*36*), which contribute to making the lens an efficient UV filter by age 20 to 30 (Fig. 1*A*) (*77*). This study suggests that some ambient UV exposure to eyes of children is desirable. Further evaluation of these factors is needed to establish an appropriate recommendation for protection of children's eyes against excessive UV-light exposure.

Second, it is desirable that consensus be obtained among professionals on what wavelengths of light UV protective sunglasses should block/transmit. Unfortunately, there is considerable disagreement on this subject (*46,47,62,83,92*). Recommendations to date vary from the use of ordinary glasses with plastic lenses to reduce UV-B exposure (*46,47*) to the use of glasses that block UV and blue light (*62,83,92*). In addition, there are differences in opinion about the transmittance characteristics of clear lenses and their usefulness to protect against exposure to UV light (*85, 93–97*). This is surprising because several studies have shown that there are great differences in the transmittance characteristics of tinted lenses, clip-on sunglasses, and regular sunglasses (*86–90*). These different opinions lead to different recommendations, which have caused confusion among scientific colleagues (*98*) and must be even more confusing to the public.

The differences in opinions about transmittance characteristics of plastic lenses are in part due to the fact that the wavelength range of UV-B light is defined differently by different investigators. Table 1 summarizes which wavelength ranges are attibuted to UV-A, UV-B, and UV-C in studies cited in this paper. The definitions vary substantially, and the ranges used in this paper are those most commonly used in other studies. Surprisingly, all these wavelength ranges differ from those as defined by the International Non-Ionizing Radiation Committee (INIRC) of the International Radiation Protection Association (IRPA), which proposed guidelines on limits of exposure to UV radiation of the eye and skin (*99*).

A plastic lens can therefore block nearly all the UV-B light when 310 nm is defined as the cutoff wavelength of UV-B (*85,89*), but the same lens can transmit substantial UV-B light when 320 nm is defined as the cutoff (*88*). Agreement on the wavelength range of UV-B light is desirable to establish more valid and consistent transmittance characteristics of lenses.

Upon reviewing the recent literature, it can be concluded that recent epidemiological studies showed an association between cumulative UV-B light exposure and the development of nuclear cataract (*46*) and posterior subcapsular cataract (*47*). Scientific evidence is also accumulating that there is an association between solar radiation and ARMD, also referred to as the blue light hazard (*62*). Epidemiologic surveys performed to date, studying sunlight exposure as a risk factor in ARMD show contradictory

Table 1. Wavelength ranges commonly used to define parts of the ultraviolet light.[a]

	Wavelength, nm			
Reference	UV-C	UV-B	UV-A	Near UV
(*1*)	< 290	290–320	320–400	
(*3*)	100–290	290–320	320–400	
(*20*)	—	290–320	320–400	
(*46*)	100–290	290–320	320–400	
(*27*)	< 280	280–315	315–400	
(*81*)	—	290–320	320–400	
(*85*)	—	290–310	310–350	
(*86*)	—	—	—	300–400
(*87*)	200–290	290–320	320–400	
(*89*)	—	—	—	300–400
(*100*)	100–290	290–320	320–400	
(*101*)	100–280	280–315	315–350	
(*99*)	180–315[b]			315–400

[a]A dash indicates that data were not provided.
[b]UV-C + UV-B.

results. Liu et al. reported that excessive light contributes to both lens and retinal disease (*80*), whereas West et al. reported no association between cumulative UV light and exposure after age 15 and ARMD (*81*). Further work is needed to evaluate the effects of chronic and cumulative UV and blue light exposure to the retina. Since this issue is not yet resolved, a conservative recommendation for UV protective glasses would be to use lenses that have at least a UV filter and blue blocker (e.g., curve III, Fig. 2), as this may prevent lens and retinal disease. This has been suggested before (*62,83*) and is also in agreement with the standards suggested by Silver, who noted that it has never been suggested that intermittent wearing of any lens can actually cause damage to the eye (*88*). Furthermore, recent reports on retinal damage caused by excessive visible light (*51,82*) indicate that reduction of transmittance of visible light should be considered as well. A general reduction of transmittance of light of all wavelengths including UV, blue and visible (e.g., curve III, Fig. 2) is a recommendation that could be considered after further research on this topic.

Rosenthal et al. showed that the amount of attenuation of ocular exposure to UV radiation by sunglasses is highly variable and is affected by size, shape, and wearing position (*85*). Placing the sunglasses a small distance from the forehead can triple the ocular exposure (*85*). This is important information that can help to improve education on the proper use of protective sunglasses.

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REFERENCES

1. Hendee, W. R. Harmful effects of ultraviolet radiation. J. Am. Med. Assoc. 262: 380–384 (1989).
2. Taylor, H. R. The biological effects of UV-B on the eye. Photochem. Photobiol. 50: 489–492 (1989).
3. Olson, C. M. Increased outdoor recreation, diminished ozone layer pose ultraviolet radiation threat to eye. J. Am. Med. Assoc. 261: 1102–1103 (1989).
4. van der Hoeve, J. Eye lesions produced by light rich in ultraviolet rays, senile cataract, senile degeneration of macula. Am. J. Ophthalmol. 3: 178–194 (1920).
5. Marx. J. L. Oxygen free radicals linked to many diseases. Science 235: 529–531 (1987).
6. Cross, C. E., Halliwell, B., Borish, E. T., Pryor, W., Ames, B. N., Saul, R. L., McCord, J. M., and Harman, D. Oxygen radicals in human disease. Ann. Intern. Med. 107: 526–545 (1987).
7. Esterbauer, H. L. Lipid peroxdation products: formation, chemical properties and biological activities. In: Free Radicals in Liver Injury (G. Poli, K. H. Cheeseman, M. U. Dianzani, and T. F. Slater, Eds.), IRL Press Limited, Oxford, 1986, pp. 29–47.
8. Bird, R. P., and Draper, H. H. Comparative studies on different methods of malonaldehyde determination. In: Methods in Enzymology (L. A. Packer, Ed.), Academic Press Inc., Orlando, FL, 1984, pp. 299–305.
9. Van Kuijk, F. J. G. M., Thomas. D. W.. Stenhens. R. J., and Dratz, E. A. Gas chromatography-mass spectrometry method for determination of phospholipid peroxides I: transesterification to form methyl esters. J. Free Rad. Biol. Med. 1: 215–225 (1985).
10. van Kuijk, F. J. G. M., Thomas, D. W., Stephens, R. J., and Dratz, E. A. Gas chromatography-mass spectrometry method for determination of phospholipid peroxides II: transesterfication to form pentafluorobenzyl esters and detection with picogram sensitivity. J. Free Rad. Biol. Med. 1: 387–393 (1985).
11. Van Kuijk, F. J. G. M., and Dratz, E. A. Detection of phospholipid peroxides in biological samples. Free Rad. Biol. Med. 3: 349–354 (1988).
12. van Kuijk, F. J. G. M., Thomas, D. W., Stephens, R. J., and Dratz, E. A. Occurrence of 4-hydroxyalkenals in rat tissues determined as pentafluorobenzyloxime derivatives by gas chromatography-mass spectrometry. Biochem. Biophys. Res. Commun. 139: 144–149 (1986).
13. van Kuijk, F. J. G. M. Detection of lipid peroxidation associated with retinal degeneration by gas chromatography-mass spectrometry. Thesis, University of Nijmegen, Nijmegen, the Netherlands, 1988.
14. Dratz, E. A., Farnsworth, C. C., Loew, E. C., Stephens, R. J., Thomas, D. W., and Van Kuijk, F. J. G. M. Products of in vivo peroxidation are present in tissues of vitamin E deficient rats and dogs. In: Vitamin E: Biochemistry and Health Implications (L. J. Machlin, Ed.), New York Academy of Sciences, New York, 1989, pp. 46–60.
15. Stephens, R. J., Negi, D. S., Short, S. M., Van Kuijk, F. J. G. M., Dratz, E. A., and Thomas, D. W. Lipid peroxidation and retinal degeneration. In: Oxygen Radicals in Biology and Medicine (M. G. Simic, K. A. Taylor, J. F. Ward, and C. von Sonntag, Eds.), Plenum Publishing Corp., New York, 1989, pp. 283–289.
16. Smolen, J. E., and Stephen, S. B. Permeability changes induced by peroxidation in liposomes prepared from human erythrocyte lipids. J. Lipid Res. 15: 273–280 (1974).
17. Molenaar, I., Hulstaert, C. E., and Vos, J. Membrane characteristics in vitamin E deficiency and the assessment of vitamin E status. Proc. Nutr. Soc. 32: 249–254 (1973).
18. Esterbauer, H., Zollner, H., and Scholz, N. Reaction of glutathione with conjugated carbonyls. Z. Naturforsch. 30c: 466–473 (1975).
19. Esterbauer, H. Aldehydic products of lipid peroxidation. In: Free Radicals, Lipid Peroxidation, and Cancer (D. C. H. McBrien and T. F. Slater, Eds.), Academic Press Inc., London, 1982, pp. 101–128.
20. Cheng, H. Causes of cataract. Age, sugars, and probably ultraviolet B radiation. Br. Med. J. 298: 1470–1471 (1989).
21. Ederer, F., Hiller, R., and Tattor, H. Senile lens changes and diabetes in two population studies. Am. J. Ophthalmol. 91: 381–395 (1981).
22. Stambolian, D. Galactose and cataract. Surv. Ophthalmol. 32: 333–349 (1988).
23. Lipman, R. M., Tripathi, B. J., and Tripathi, R. C. Cataracts induced by microwave and ionizing radiation. Surv. Ophthalmol. 33: 200–210 (1988).
24. Lerman, S. Photosensitizing drugs and their possible role in enhancing ocular toxicity. Ophthalmology 93: 304–318 (1986).
25. Lerman, S., Hockwin, O., and Dragomirescu, V. In vivo fluorescence photography. Ophthal. Res. 12: 303–314 (1980).
26. Borkman, R. F., and Lerman, S. Evidence for a free radical mechanism in aging and UV irradiated ocular lenses. Exp. Eye Res. 25: 303–309 (1977).
27. Bhuyan, K. C., Bhuyan, D. K., and Podos, S. M. Lipid peroxidation in cataract of the human. Life Sci. 38: 1463–1471 (1986).
28. Augusteyn, R. C. Protein modification in cataract: possible mechanisms. In: Mechanisms of Cataract Formation in the Human Lens (G. Duncan, Ed.), Academic Press, New York, 1981, pp. 71–115.
29. Handelman, G. J., and Dratz, E. A. The role of antioxidants in the retina and retinal pigment epithelium and the nature of prooxidant induced damage. Adv. Free Rad. Biol. Med. 2: 1–89 (1986).
30. Rathbun, W. B., and Bovis, M. G. Activity of glutathione peroxidase and glutathione reductase in the human lens related to age. Curr. Eye Res. 5: 381–395 (1986).
31. Simonelli, F., Nesti, A., Pensa, M., Romano, L., Savastano, S., Rinaldi, E., and Auricchio, G. Lipid peroxidation and human cataractogenesis in diabetes and severe myopia. Exp. Eye Res. 49: 181–187 (1989).
32. Jacques, P. F., Hartz, S. C., Chylack. L. T., Jr., McGandy, R. B., and Sadowski, J. A Nutritional status in persons with and without senile cataract: blood vitamin and mineral levels. Am. J. Clin. Nutr. 48: 152–158 (1988).
33. Jacques, P. F., Chylack, L. T., Jr., McGandy, R. B., and Hartz, S. C. Antioxidant status in persons with and without senile cataract. Arch. Ophthalmol. 106: 337–340 (1988).
34. Borchman, D., Yappert, M. C., Rubini, Q., and Paterson, C. A. Distribution of phospholipid malondialdehyde adduct in the human lens. Curr. Eye Res. 8: 939–946 (1989).
35. Zigman, S. Light damage to the lens. In: Clinical Light Damage to the Eye (D. Miller, Ed.), Springer-Verlag, New York, 1987, pp. 65–78.
36. Lerman, S., Megaw, J. M., and Moran, M. N. Further studies on the effects of UV radiation on the human lens. Ophthal. Res. 17: 354–361 (1985).
37. Lerman, S., Gardner, K., Megaw, J., and Borkman, R. Prevention of direct and photosensitized ultraviolet radiation damage to the ocular lens. Ophthal. Res. 13: 284–292 (1981).
38. Zigman, S., Yulo, T., and Schultz, J. Cataract indication of mice exposed to near UV light. Ophthal. Res. 6: 259–270 (1974).

39. Goosey, J. D., Zigler, J. S., and Kinoshita, J. H. Crosslinking of lens crystallins in a photodynamic system. Science 208: 422–423 (1980).
40. Jedziniak, J., Arredondo, M., and Andley, U. Oxidative damage to human lens enzymes. Curr. Eye Res. 6: 345–350 (1991).
41. Dillon, J., Spector, A., and Nakanishi, K. Identification of beta carbolines isolated from fluorescent human lens proteins. Nature 259: 422–423 (1976).
42. Johnson, F. S., Mo, T., and Green, A. E. S. Average latitudinal variation in ultraviolet radiation at the earth's surface. Photochem. Photobiol. 23: 179–188 (1976).
43. Zigman, S., Datiles, M., and Torczynski, E. Sunlight and human cataracts. Invest. Ophthalmol. Vis. Sci. 18: 462–467 (1979).
44. Hiller, R., Sperduto, R. D., and Ederer, F. Epidemiologic associations with nuclear, cortical, and posterior subcapsular cataracts. Am. J. Epidemiol. 124: 916–925 (1986).
45. Hiller, R., Giacometti, L., and Yuen, K. Sunlight and cataract: an epidemiologic investigation. Am. J. Epidemiol. 105: 450–459 (1977).
46. Taylor, H. R., West, S. K., Rosenthal, F. S., Munoz, B., Newland, H. S., Abbey, H., and Emmett, E. A. Effect of ultraviolet radiation on cataract formation. N. Engl. J. Med. 319: 1429–1433 (1988).
47. Bochow, T. W., West, S. K., Azar, A., Munoz, B., Sommer, A., and Taylor, H. R. Ultraviolet light exposure and risk of posterior subcapsular cataracts. Arch. Ophthalmol. 107: 369–372 (1989).
48. Rosenthal, F. S., Phoon, C., Bakalian, A. E., and Taylor, H. R. The ocular dose of ultraviolet radiation to outdoor workers. Invest. Ophthalmol. Vis. Sci. 29: 649–656 (1988).
49. Weiter, J. J. Phototoxic changes in the retina. In: Clinical Light Damage to the Eye (D. Miller, Ed.), Springer-Verlag, New York, 1987, pp. 79–125.
50. Kremers, J. J. M., and Van Norren, D. Two classes of photochemical damage of the retina. Lasers Light Ophthalmol. 2: 41–52 (1988).
51. Rapp, L. M., Tolman, B. L., and Dhindsa, H. S. Separate mechanisms for retinal damage by ultraviolet A and mid visible light. Invest. Ophthalmol. Vis. Sci. 31: 1186–1190 (1990).
52. Dratz, E. A., and Hargrave, P. A. The structure of rhodopsin and the rod outer segment disc membrane. Trends Biochem. Sci. 8: 128–131 (1983).
53. Stone, W. L., Farnsworth, C. C., and Dratz, E. A. A reinvestigation of the fatty acid content of bovine rat and frog rod outer segments. Exp. Eye Res. 28: 387–397 (1979).
54. Cosgrove, J. P., Church, D. F., and Pryor, W. A. The kinetics of the autooxidation of polyunsaturated fatty acids. Lipids 22: 299–304 (1987).
55. Foote, C. S. Photosensitized oxidation and singlet oxygen: consequences in biological systems. In: Free Radicals in Biology (W. A. Pryor, Ed.), Academic Press, New York, 1976, pp. 85–133.
56. Foote, C. S. Light, oxygen, and toxicity. In: Pathology of Oxygen (A. P. Autor, Ed.), Academic Press, New York, 1982, pp. 21–44.
57. Noell, W. K., Walker, V. S., Kang, B. S., and Berman, S. Retinal damage by light in rats. Invest. Ophthalmol. 5: 450–473 (1966).
58. Noell, W. K. Effects of environmental lighting and dietary vitamin A on the vulnerability of the retina to light damage. Photochem. Photobiol. 29: 717–723 (1979).
59. Katz, M. L., Stone, W. L., and Dratz, E. A. Fluorescent pigment accumulation in retinal pigment epithelium of antioxidant deficient rats. Invest. Ophthalmol. Sci. 17: 1049–1058 (1978).
60. Wiegand, R. D., Guisto, N. M., Rapp, L. M., and Anderson, R. E. Evidence for rod outer segment lipid peroxidation following constant illumination of the rat retina. Invest. Ophthalmol. Vis. Sci. 24: 1433–1435 (1983).
61. Wiegand, R. D., Joel, C. D., Rapp, L. M., Nielsen, J. C., Maude, M. B., and Anderson, R. E. Polyunsaturated fatty acids and vitamin E in rat rod outer segments during light damage. Invest. Ophthalmol. Vis. Sci. 27: 727–733 (1986).
62. Young, R. W. Solar radiation and age-related macular degeneration. Surv. Ophthalmol. 32: 252–269 (1988).
63. Sadun, A. C., Sadun, A. A., and Sadun, L. A. Solar retinopathy. A biophysical analysis. Arch. Ophthalmol. 102: 1510–1512 (1984).
64. Gardner, T. M., Ai, E., Chrobak, M., and Shoch, D. E. Photic maculopathy secondary to short circuiting of a high tension electric current. Ophthalmology 89: 865–868 (1982).
65. Marshall, J. Structural aspects of laser induced damage and their functional implications. Health Phys. 56: 617–624 (1989).
66. Ham, W. T., Jr., and Mueller, H. A. The photopathology and nature of the blue light and near UV retinal lesions produced by lasers and other optical sources. In: Laser Applications in Medicine and Biology (M. L. Wolbarsht, Ed.), Plenum Publishing Corp., New York, 1989, pp. 191–246.
67. Robertson, D. M., and Mclaren, J. W. Photic retinopathy from the operating room microscope. Study with filters. Arch. Ophthalmol. 107: 373–375 (1989).
68. Parver, L. M., Auker, C. R., and Fine, B. S. Observations on monkey eyes exposed to light from an operating microscope. Ophthalmology 90: 964–972 (1983).
69. Bressler, N. M., Bressler, S. B., and Fine, S. L. Age-related macular degeneration. Surv. Ophthalmol. 32: 375–413 (1988).
70. Young, R. W. Pathophysiology of age related macular degeneration. Surv. Ophthalmol. 31: 291–306 (1987).
71. Weiter, J. J. Macular degeneration: is there a nutritional component? Arch. Ophthalmol. 106: 183–184 (1988).
72. Ham, W. T., Jr., and Mueller, H. A. Retinal sensitivity to damage from short wavelength light. Nature 260: 153–155 (1976).
73. Ham, W. T., Jr., Mueller, H. A., Ruffolo, J. J., Jr., Guerry, D., III, and Guerry, R. K. Action spectrum for retinal injury from near ultraviolet radiation in the aphakic monkey. Am. J. Ophthalmol. 93: 299–306 (1982).
74. Sample, P. A., Esterson, F. D., Weinreb, R. N., and Boynton, R. M. The aging lens. In vitro assessment of light absorption in 84 human eyes. Invest. Ophthalmol. Vis. Sci. 29: 1306–1311 (1988).
75. Pokorny, J., Smith, V. C., and Lutze, M. Aging of the human lens. Appl. Optics 26: 1437–1440 (1987).
76. Werner, J. S. Development of scotopic sensitivity and the absorption spectrum of the human ocular media. J. Opt. Soc. Am. 72: 247–258 (1982).
77. Lerman, S. Light induced changes in ocular tissues. In: Clinical Light Damage to the Eye (D. Miller, Ed.), Springer-Verlag, New York, 1987, pp. 183–215.
78. Wing, G. L., Blanchard, G. C., and Weiter, J. J. The topography and age relationship of lipofuscin concentration in the retinal pigment epithelium. Invest. Ophthalmol. Vis. Sci. 17: 601–607 (1978).
79. Weiter, J. J., Dorey, C. K., Delori, F., and Fitch, K. Relationship of senile macular degeneration to refractive error. Invest. Ophthalmol. Vis. Sci. 28(suppl.): 119 (1987).
80. Liu, I. Y., White, L., and Lacroix, A. Z. The association of age related macular degeneration and lens opacities in the aged. Am. J. Public Health 79: 765–769 (1989).
81. West, S. K., Rosenthal, F. S., Bressler, N. M., Bressler, S. B., Munoz, B., Fine, S. L., and Taylor, H. R. Exposure to sunlight and other risk factors for an age related macular degeneration. Arch. Ophthalmol. 107: 875–879 (1989).
82. Munoz, B., West, S. K., Bressler, N. M., Bressler, S. B., Rosenthal, F. S., and Taylor, H. R. Blue light and risk of age related macular degeneration. Invest. Ophthalmol. Vis. Sci. 30(suppl.): 49 (1990).
83. Slinley, D. H. Eye protective techniques for bright light. Ophthalmology 90: 937–944 (1983).
84. Rosenthal, F. S., Safran, M., and Taylor, H. R. The ocular dose of ultraviolet radiation from sunlight exposure. Photochem. Photobiol. 42: 163–171 (1985).
85. Rosenthal, F. S., Bakalian, A. E., Lou, C. Q., and Taylor, H. R. The effect of sunglasses on ocular exposure to ultraviolet radiation. Am. J. Public Health 78: 72–74 (1988).
86. Anderson, W. J., and Gebel, R. K. H. Ultraviolet windows in commercial sunglasses. Appl. Optics 16: 515–517 (1977).
87. Ober Magnante, D. B., and Miller, D. Ultraviolet absorption of commonly used clip-on sunglasses. Ann. Ophthalmol. 17: 614–616 (1985).
88. Silver, J. The hazards of sunglasses. Towards simple standards for labelling and testing. In: Hazards of Light (J. Cronly-Dilon, E. S. Rosen, and J. Marshall, Eds.), Pergamon Press, Oxford, 1986, pp. 343–363.
89. Borgwardt, B., Fishman, G. A., and Van der Meulen, D. Spectral transmission characteristics of tinted lenses. Arch. Ophthalmol. 99: 293–297 (1981).
90. Lerman, S., and Megaw, J. Transmission characteristics of commercially available sunglasses. J. Ocul. Cutan. Toxicol. 2: 47–61 (1983).
91. Scrivener, A. B. Sunglass standards. Their development and impact on current materials. In: Hazards of Light (J. Cronly-Dilon, E. S. Rosen, and J. Marshall, Eds.), Pergamon Press, Oxford, 1986, pp. 333–342.
92. Lerman, S. Ocular phototoxicity. N. Engl. J. Med. 319: 1475–1477 (1988).
93. Taylor, H. R., West, S. K., and Munoz, B. Ultraviolet radiation and formation of cataracts. Reply. N. Engl. J. Med. 320: 1285–1286 (1989).
94. Lerman, S. Ultraviolet radiation and formation of cataracts. Reply. N. Engl. J. Med. 320: 1286 (1989).
95. McDonagh, A. F., and Nguyen, M. L. Spectacles, ultraviolet radiation, and formation of cataracts. N. Engl. J. Med. 321: 1478–1479 (1989).
96. Taylor, H. R., and West, S. K. Spectacles, ultraviolet radiation, and formation of cataracts. Reply. N. Engl. J. Med. 321: 1479 (1989).

97. Lerman, S. Spectacles, ultraviolet radiation, and formation of cataracts. Reply. N. Engl. J. Med. 321: 1479 (1989).
98. Stark, P. Ultraviolet radiation and formation of cataracts. Reply. N. Engl. J. Med. 320: 1285 (1989).
99. International Radiation Protection Association. Proposed change to the IRPA 1985 guidelines on limits of exposure to ultraviolet radiation. Health Phys. 56: 971–972 (1989).
100. Miller, S. A., and James, R. H. Variables associated with ultraviolet transmittance measurements of intraocular lenses. Am J. Ophthalmol. 106: 256–260 (1988).
101. Rosenthal. F. S., Bakalian, A. E., and Taylor, H. R. The effect of prescription eyewear on ocular exposure to ultraviolet radiation. Am. J. Public Health 76: 1216–1220 (1986).