The Long-term Effects of Visible Light on the Eye

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• The relationship between exposure to sunlight and senile cataract, age-related macular degeneration, pterygium, and climatic droplet keratopathy was examined in 838 watermen who work on the Chesapeake Bay. The presence and severity of lenticular, corneal, and macular changes were assessed by either clinical examination or from stereo macular photographs. From detailed exposure histories, ocular exposure was estimated for three bands of visible radiation—violet (400 to 450 nm), blue (400 to 500 nm), or all visible (400 to 700 nm)—as well as for UV-A (320 to 340 nm) and UV-B (290 to 320 nm). The results with each band of visible radiation were similar. Neither cortical nor nuclear cataract was associated with ocular exposure to blue or all visible radiation, but pterygium and climatic droplet keratopathy were more common with higher exposures. Compared with age-matched controls, patients with advanced age-related macular degeneration (geographic atrophy or disciform scarring) had significantly higher exposure to blue or visible light over the preceding 20 years (odds ratio, 1.36 [1.00 to 1.85]) but were not different in respect to exposure to UV-A or UV-B. These data suggest that high levels of exposure to blue or visible light may cause ocular damage, especially later in life, and may be related to the development of age-related macular degeneration.

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Cataracts occur more commonly in tropical or sunny areas than in more temperate regions.[1-3] Studies in Australian aborigines have shown a dose-response relation between the prevalence of cataracts and levels of UV-B radiation.[4,5] In the United States, data from the National Health and Nutrition Examination Survey have suggested that people with higher exposures to sunlight or UV radiation levels have an increased risk of cataract, specifically cortical cataract.[2,6] A population-based survey of watermen who worked on Chesapeake Bay showed an association between individual ocular exposure to UV-B (290 to 320 nm) and cortical cataract.[7] A statistically significant association was not seen with UV-A (320 to 400 nm) or with exposure to either type of UV radiation and nuclear cataract.

Sunlight has also been suggested as a cause of several corneal disorders, including pterygium and climatic droplet keratopathy (CDK). Pterygium occurs more commonly in tropical or sunny areas than in more temperate regions and has been ascribed to greater sun exposure, specifically to UV-B.[8-11] Climatic droplet keratopathy is an unusual corneal condition and is more often reported in harsh climates such as the Arctic or in desert areas. Ultraviolet radiation is thought to play a role in the cause of CDK.[10,12-14] In the study of watermen, pterygium and CDK were significantly associated with a broad band of UV radiation exposure (both UV-B and two bands of UV-A).[15,16]

Exposure to sunlight has long been suggested as a cause of age-related macular degeneration (AMD). Short-term exposures to longer wavelength UV and blue light can cause retinal damage in animals.[17] These changes are comparable with those seen in humans following sun gazing.[18,19] The long-term changes induced in laboratory animals by exposure to shorter wavelength visible light have some similarities to those changes seen in patients with AMD and are thought to result from photic damage induced by photochemical or photo-oxidative mechanisms.[20,21] The retina is much more sensitive to experimental damage from UV or blue light than it is from longer wavelengths of visible light,[22] even though the lens normally filters out most of the UV.[23] These observations have led to the notion that long-term exposure to solar radiation, either UV or visible, may play a role in the cause of AMD, and some observations support this. Pseudophakic eyes containing non–UV-absorbing intraocular lenses show a loss of short-wave cones compared with fellow pseudophakic eyes with UV-absorbing implants.[24] Data from the National Health and Nutrition Examination Survey suggested that an apparent increase of AMD in aphakic eyes may be due to light-induced damage.[25] However, our initial analysis of data collected from the population-based study of Chesapeake Bay watermen found no association between carefully ascertained individual ocular exposure to UV-B or two bands of UV-A and AMD.[26] We now report further analysis of the data collected during our

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study of Maryland watermen and examine the relationship between ocular exposure to visible light and the occurrence of cataract, corneal disease, and AMD.

SUBJECTS AND METHODS

Ocular Examination

Eight hundred thirty-eight watermen participated in a study of the effects of sun exposure on the eye.[7,27] Data on risk factors, including work and leisure exposure histories, were collected by personal interview at the time of examination. An ophthalmic examination was performed after pupillary dilation, and the type and severity of lens opacities were graded.[28,29] In addition to the clinical grading of cortical, nuclear, and posterior subcapsular opacities, slit photographs of the lens were graded for nuclear color. Nuclear color was graded 0 if less yellow than standard photograph 2,[28] grade 1 if the same color, and grade 2 if more yellow-brown. Any lens that had been operated on, even if lens remnants were present, was regarded as aphakic. There were 18 subjects with aphakia. The operating ophthalmologist was contacted in each case, and the preoperative diagnosis of the type of cataract was obtained for 17 of the 18 subjects.[27] Analyses were performed for each type of opacity with use of the grade for the most severely affected eye. Two men with congenital cataracts and 12 eyes with traumatic opacities were excluded.

Data on corneal status were available for all 838 watermen. Pterygium and CDK were diagnosed by clinical appearance as described previously.[15]

Stereoscopic fundus photographs were taken of 782 subjects (93%) to assess the presence and severity of macular degeneration. The most frequent reason for missing photographs occurred when watermen were interviewed and examined in their homes where photographs could not be taken.[27] A comparison of the characteristics of those with and without fundus photographs showed that those without photographs tended to be older, were more likely to report having been a smoker, and had a somewhat lower cumulative UV-B exposure.[26] For the analyses of AMD, eight subjects with bilateral aphakia were excluded, as was one with extensive retinal disease not associated with macular degeneration that precluded the grading of macular changes. Only 13 subjects (2%) had fundus photographs that could not be graded for macular change. For most of these, severe lens opacities obscured macular details. Only 46 (6%) of the remaining 769 subjects were categorized on the basis of photographs whose quality was graded as "fair." In fair photographs, one or more characteristics could not be graded.

The grading scheme used to detect the component features of macular degeneration has been described in detail elsewhere.[30] In summary, the photographs were graded for the presence of exudative (neovascular) disease, geographic atrophy, "high-risk" macular characteristics (drusen >50 μm, confluent drusen, or focal hyperpigmentation of the retinal pigment epithelium), and for the number of drusen within 1500 μm and 3000 μm circles around the fovea (Table 1). If only one eye had photographs, the individual was classified based on findings in the single eye; otherwise, he was classified according to findings in his worse eye. Previously, we have shown the absence of bias in classifying subjects based on photographs from only one eye and the reliability and validity of the grading scheme in our population.[30] Eyes with grade 4 changes (exudative disease or geographic atrophy) are termed "AMD-4."

Table 1.—The Grading of AMD*

Grade	Description
1	≥5 small drusen in central 1500 μm†
	≥10 small drusen between 1500 and 3000 μm
2	≥20 small drusen in central 1500 μm
3	Large or confluent drusen†
	Focal hyperpigmentation†
4	Exudative disease
	Geographic atrophy†

*From Bressler et al.[30] AMD indicates age-related macular degeneration.
†Reference made to standard photographs.

Ocular Exposure

The personal, ocular exposure index for visible light as well as UV-A and UV-B was constructed based interview data and laboratory and field measurements. The methods for estimating exposure to UV-B light have been described previously.[27] In brief, the personal exposure model was combined with work and leisure time histories and ambient data. For UV, ambient levels were obtained from published data. These methods were extended to visible light by computing ambient levels from the model of Green and Chai.[31] The ocular ambient exposure ratio, defined as the average fraction of ambient radiation that reached the eyes of a subject not wearing spectacles or sunglasses, had been measured for UV-B[32] and was assumed to be similar for visible radiation. Ocular exposure was corrected for the use of eyewear according to laboratory measurements of UV attenuation of typical eyewear.[33] In the case of visible light, the attenuation was assumed to be zero. Data on sunglass and corrective spectacle use were not collected separately and full transmission of visible radiation by eyeglasses has been assumed.

Based on these procedures, the yearly ocular exposure to violet light (400 to 450 nm), blue light (400 to 500 nm), and all visible light (400 to 700 nm) as well as UV-A (320 to 340 nm) and UV-B (290 to 320 nm) was calculated for each individual for each year of life after the age of 15 years. The annual exposure was expressed as a proportion of Maryland sun-years; 1 Maryland sun-year is equal to the total ambient radiation of a given wavelength at sea level in Maryland during 1 year.

Data Analyses

Univariate and contingency table analyses were performed using standard statistical procedures. Logistic regression models were used in the multivariate analysis to simultaneously examine factors associated with the different severities of AMD. All analysis was done using SAS statistical software (SAS Institute, Cary, NC). Age and exposure were continuous variables, and regression coefficients were calculated as estimates of risk. For dichotomous variables, an odds ratio was calculated. Detailed analyses for AMD were restricted to those subjects older than 65 years as no cases of AMD-4 were seen in subjects younger than this age.

The serial additive dose model analysis was used where the expected yearly exposure for each "case" was calculated based on the combined data from all "controls" who did not have the particular change of the same degree or worse and who were of the same age group (matched within 3 years of age).[34] The actual observed cumulative dose for each case was then compared with the expected dose determined from the controls. A paired t test was used to test the difference, except when there were less than 15 cases when the Wilcoxon Signed-Rank test was used.

In each analysis, the results obtained with the three different bands of light—violet, blue, and all visible—were very similar. In each case, the results were either identical or the association was minimally stronger for blue light. To avoid confusion, only the data for blue light are presented. Cumulative ocular exposure to blue light showed an expected increase with age, but also demonstrated a marked range in exposures at each age group (Fig 1).

RESULTS

Cataract

Overall, 111 watermen (13%) had some cortical lens opacity and 229 (27%) had some nuclear opacity, including those who had undergone cataract surgery. There was a progressive increase in the prevalence and severity of cortical and nuclear opacities with age.[7] Only 14 watermen were found to have posterior subcapsular cataracts, including seven who had undergone cataract surgery for this condition. The numbers with posterior subcapsular cataracts were too few (14) to provide firm age-specific prevalence rates, and no further analyses are presented on this type of cataract.

No relationship was seen among cortical opacity, nuclear opacity, or nuclear color and exposure to any of the three bands of visible light. This was true in regression models using cumulative or annual ocular exposures (Table 2), in regression models examining exposure in the last 20 years, and in the serial additive dose model (data not shown). A doubling of cumulative blue light exposure gave an odds ratio (with 95% confidence intervals) of develop-

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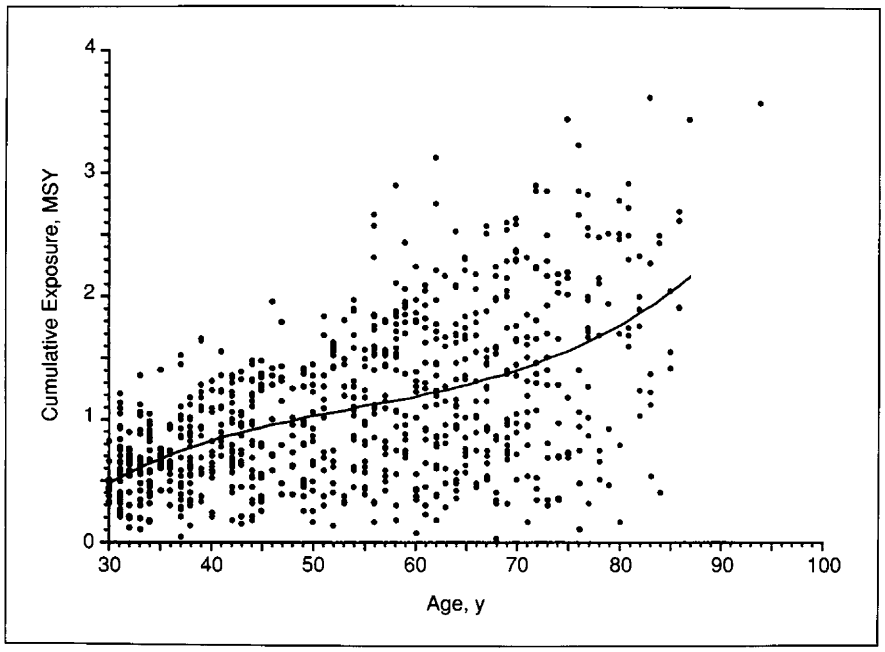


Fig 1.—Cumulative ocular exposure to blue light (440 to 500 nm) by age in 838 watermen. MSY indicates Maryland sun-year.

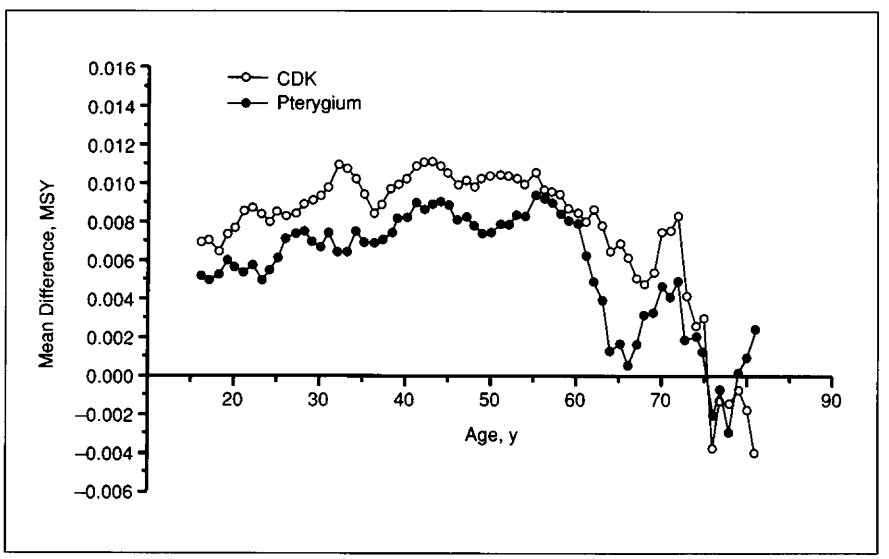


Fig 2.—Average dose difference at each year of life between annual ocular exposure to blue light among 156 watermen with climatic droplet keratopathy (CDK) and 605 age-matched controls without CDK and 135 watermen with pterygium and 687 age-matched controls without pterygium. MSY indicates Maryland sun-year.

ing cortical opacity of 1.29 (0.80 to 2.01), nuclear opacity of 1.02 (0.81 to 1.06), and nuclear color of 0.80 (0.60 to 1.07).

Corneal Disease

Overall, 140 watermen (17%) had pterygium and 162 (19%) had CDK. There was a progressive increase in the prevalence of pterygium and CDK with age.[15] In almost every case CDK was of only mild severity, and in no case was it associated with severe visual impairment. A doubling of cumulative blue light exposure increased the odds ratio for development of both pterygium (1.85 [1.43 to 2.38]) and CDK (2.79 [2.07 to 3.73]).

There was a strong and consistent association between cumulative ocular exposure and both pterygium and CDK (Table 2). The excess exposure in those watermen with these corneal changes was seen at all ages, and the serial additive dose model showed no evidence for a period of either increased resistance or increased susceptibility in later life. To the contrary, it suggested that exposure before the age of 65 years was more important in determining the risk of having either condition than exposure after this age (Fig 2).

Table 2.—Association Between Cumulative Blue and UV Light and Eye Changes

	Regression Coefficient	SE	P
Cortical opacities*			
Age	0.15	0.02	<.001
Blue†	0.37	0.34	.28
UV-A	0.54	0.36	.13
UV-B	0.71	0.35	.04
Nuclear opacities			
Age	0.17	0.01	<.001
Blue	0.03	0.17	.85
UV-A	0.03	0.17	.86
UV-B	0.02	0.17	.99
Nuclear color			
Age	0.17	0.01	<.001
Blue	−0.31	0.21	.15
UV-A	−0.29	0.21	.18
UV-B	−0.22	0.20	.27
Pterygium			
Age	0.03	0.01	.005
Blue	0.89	0.19	<.001
UV-A	0.82	0.19	<.001
UV-B	0.65	0.16	<.001
CDK‡			
Age	0.05	0.01	<.001
Blue	1.48	0.22	<.001
UV-A	1.49	0.21	<.001
UV-B	1.26	0.19	<.001
AMD-4‡			
Age	0.13	0.04	<.001
Blue	0.52	0.62	.44
UV-A	−0.12	0.55	.82
UV-B	−0.53	0.49	.28

*Grade 2 opacities or greater. Grade 1 opacities excluded from this regression.
†Log cumulative ocular blue light exposure.
‡CDK indicates climatic droplet keratopathy; AMD-4, age-related macular degeneration, grade 4.

Macular Degeneration

Age-related macular degeneration became increasingly common with increasing age (Table 3). Overall, eight watermen had AMD-4 and 84 watermen had AMD-3.

Initial logistic regression analysis did not show a significant association between the presence of AMD of any grade and lifetime cumulative ocular exposure. However, when the analysis considered the ocular exposure for only the previous 20 years, a significant association was seen between AMD-4 and blue light exposure (Tables 2 and 4; regression coefficient, 3.0; $P = .05$). This association was not seen with either UV-A or UV-B exposure, similar to our previous report.[26]

Analysis using the serial additive dose model showed on average those with AMD-4 have a 48% higher exposure to blue light during the last

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Table 3.—Age-Specific Prevalence of AMD Changes*

Age Group, y	No. (%) With AMD†			
	Grade 1 or Worse	Grade 2 or Worse	Grade 3 or Worse	Grade 4 or Worse
30-49	60 (17.8)	39 (11.5)	24 (7.1)	0 (0)
50-59	50 (32.3)	32 (20.5)	11 (7.0)	0 (0)
60-69	48 (30.6)	32 (20.3)	22 (13.8)	1 (0.6)
70-79	38 (44.7)	30 (34.5)	25 (27.5)	4 (4.3)
80-95	11 (78.6)	10 (58.8)	10 (52.7)	3 (13.7)
Total	**207** (27.6)	**143** (18.9)	**92** (12.0)	**8** (1.2)

*AMD indicates age-related macular degeneration.
†Denominator is the number of men with photographs judged readable for that sign.

Table 4.—Association Between Ocular Exposure to Blue Light Over Last 20 Years and AMD for Those Older Than 65 Years*

	Odds Ratio (95% CI)†		
	Blue	UV-A	UV-B
AMD-1, grade 1 only	1.00 (0.90-1.13)	1.04 (0.81-1.34)	1.10 (0.88-1.38)
AMD-2, grade 2 only	1.04 (0.92-1.16)	1.02 (0.76-1.37)	1.02 (0.76-1.37)
AMD-3, grade 3 only	1.05 (0.95-1.16)	0.93 (0.78-1.11)	0.93 (0.76-1.11)
AMD-4, grade 4 only	1.36 (1.00-1.85)	0.92 (0.62-1.37)	0.92 (0.62-1.37)

*AMD indicates age-related macular degeneration.
†Odds ratio estimate for the increase in exposure of 0.1 Maryland sun-year. CI indicates confidence interval.

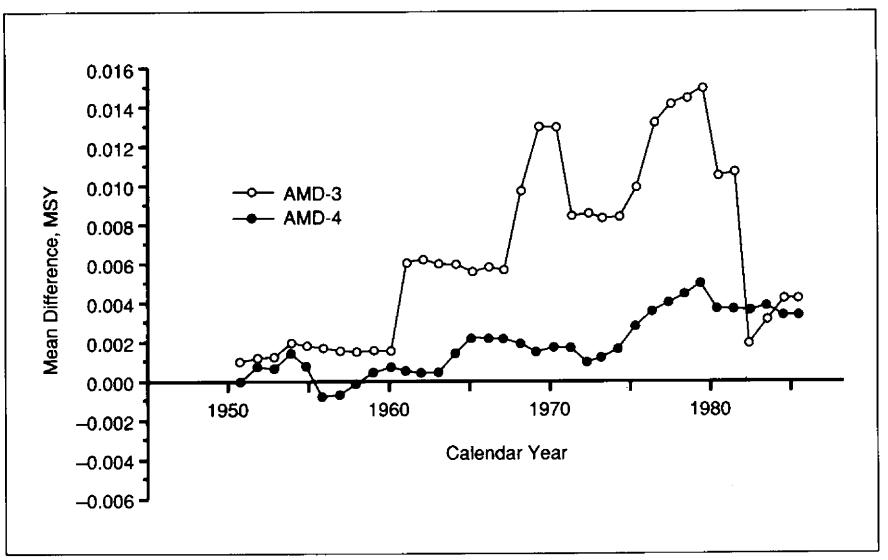


Fig 3.—Average dose difference for recent annual ocular exposure to blue light among eight watermen with age-related macular degeneration (AMD-4) and 135 age-matched controls without this degree of AMD and among 60 watermen with AMD-3 and 358 age-matched controls without that degree of AMD. MSY indicates Maryland sun-year.

20 years than their age-matched controls. Prior to 20 years (ie, before 1960), little difference was observed in terms of ocular exposure to blue light between those with AMD-4 and controls (Fig 3). Thereafter, those with AMD-4 had higher ocular exposure than controls. A similar but less marked trend was seen with the less severe changes of AMD-3.

Altogether, eight watermen had AMD-4 (Table 5). The difference between the observed ocular exposure to blue light during the previous 20 years and the expected exposure (that of their age-matched controls) was statistically significant (Wilcoxon Signed-Rank test, $P=.027$). Using the same statistical test, there was no significant difference in exposure to either UV-A or UV-B between patients with AMD-4 and controls. Two of the eight watermen with AMD-4 gave a history of having performed arc welding in the past, whereas only 18 of the 135 controls without AMD-4 had also welded. The two watermen with AMD-4 and a history of welding did not have an ocular exposure as high as the others with AMD-4. If all welders were excluded, the association between blue light exposure and AMD-4 became even stronger (Wilcoxon Signed-Rank test, $P=.015$). It may be that the assessment of ocular exposure understated the true exposure in those who had welded.

COMMENT

These data support an association between ocular exposure to visible light and an increased risk of vision-impairing AMD in a phakic population. This association was seen only in later life. No evidence was found to link visible exposure to lenticular changes, although pterygium and CDK are linked to higher exposures of solar radiation, especially in younger years.

Watermen were selected for these studies because of the consistency of their traditional work practices as they catch and collect crabs, oysters, and other seafood from Chesapeake Bay and because of the potential for a definable range of ocular exposure as they work year-round in open boats.[27] These characteristics allowed the ascertainment of their individual ocular exposures to solar radiation.

Our initial measurements and analyses were for UV.[7,15,26] Several assumptions had to be made in adapting the exposure histories to the visible spectrum. The ocular ambient exposure ratio (the fraction of ambient exposure that reaches the eye) for visible radiation was assumed to be the same as for UV. Detailed information was collected on eyeglass wear, but data on sunglass and corrective spectacle wear were not collected separately, so we have assumed full transmission of visible radiation by eyeglasses. This will overestimate the ocular exposure in those with extensive sunglass use. However, most watermen do not wear sunglasses as they get covered in spray. Most eyeglass use, especially with older watermen, was for refractive purposes and the impact of this deficiency in data collection is likely to be small. Finally, while we collected detailed exposure data for outdoor work, few data were collected for indoor work, as our initial investigations were aimed primarily at UV exposure, which does not occur indoors. We have had to assume no ocular exposure to visible radiation occurred indoors. Although a broad assumption, the indoor exposure is negligible compared with

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Table 5.—Characteristics of Eight Watermen With AMD-4 and Ocular Exposure to Blue Light Over the Previous 20 Years*

	Affected Eye					Ocular Exposure, MSY × 10^{-3}		
			Macular Change					
Age, y	Arc Welding	Visual Acuity	Disciform Scar	Geographic Atrophy	Fellow Eye	Observed	Expected	Difference
67	N	<20/100	+	...	AMD-3	580	517	63
71	N	20/80	...	+	Normal	1007	509	498
71	Y	20/80	...	+	AMD-3	452	509	−57
73	N	20/80	+	...	AMD-2	916	497	421
77	N	20/30	...	+	AMD-3	736	415	321
83	Y	20/40	...	+	AMD-3	292	317	−25
84	N	HM, HM	...	+	AMD-4 Bilateral	360	317	42
94	N	20/300, 20/300	+	+	AMD-4 Bilateral	779	545	234

*AMD indicates age-related macular degeneration; HM, hand movement.

the magnitude of exposure obtained while outdoors.[35]

In this study, we attempted to distinguish the effects on the eye of exposure to various wave bands of light. A problem with identifying a causal relationship between eye changes and a particular band of radiation is that exposure to the different wave bands may be highly correlated. For example, in our population, the correlation coefficient between cumulative exposure to blue light and cumulative exposure to UV-B was .87 ($P<.001$). This correlation is because similar characteristics of the exposure history (eg, hours spent outdoors or wearing a brimmed hat) affect exposure in all wave bands. Thus, an association of an outcome with blue light could result from either a causal effect of blue light exposure itself or a causal effect of the closely related UV-B or UV-A exposure. We examined this possibility by a parallel analysis of each outcome with each band of light (Table 4). For CDK and pterygium, significant association was found with all wave bands. One possibility is that exposure to light of all wavelengths contributes to these conditions. Alternatively, one wave band may be responsible for causing the condition (eg, UV-B) and the association with other wave bands may be due to their close correlations with UV-B.

In our analysis of AMD-4, only blue or visible light showed an association. In this analysis, greater distinction between wave bands was observed because restricting the exposure to the last 20 years reduced the correlation between blue or visible light and the UV wave bands (eg, correlation coefficient of exposure in the last 20 years for blue light vs UV-B is .73 ($P<.001$). This weaker correlation is probably due to the strong influence that eyewear use has on ocular exposure to UV-B. In later years, a greater percentage of subjects wore glasses; and under our assumption, their patterns of use reduced their ocular UV-B exposure but not visible light exposure. Our data suggest that those with AMD-4 and their controls were different in their use of eyewear with subjects more likely to wear glasses or sunglasses all the time. These data do not permit us to determine if this difference is due to differences in preexisting ocular conditions or differences in nonprescription eyewear use. Regardless of glasses use, however, for subjects to have a higher visible light exposure but similar UV-B exposure compared with controls, subjects must have spent more time outdoors.

Our previous studies showed an association between cortical cataract and UV-B but not UV-A. The lack of an association with any of the visible wavelengths further strengthens the notion that ocular exposure to UV-B can be implicated specifically as a risk factor for cortical cataract. Similarly, the lack of any association between either nuclear opacity or nuclear color and either visible light or UV makes it seem unlikely that solar radiation is an important factor in causing nuclear cataract.[7]

The lack of a significant association between ocular exposure and the presence of small drusen (AMD-1 or AMD-2) suggests that these changes may have a different cause or a different significance in the evolution of sight-destroying AMD than that which is usually assumed. It is often assumed that drusen form an early stage of AMD, first with a few small drusen followed by more numerous, larger, confluent drusen and subsequently with the development of either geographic atrophy (atrophic AMD) or neovascularization leading to a disciform scar (disciform AMD). It may be that more than one process is occurring concurrently. Small drusen are an almost ubiquitous feature of aging[30] and may, in fact, represent an epiphenomenon in much the same way as increasing nuclear color is largely independent of the development of nuclear opacity in nuclear cataract.[36] Alternatively, small drusen may represent an initiating event but visible light is required as a promoter for the later development of AMD-4.

The strong association between recent blue or visible light exposure, that is during the last 20 years, and AMD-4 is provocative. It could suggest the presence of a normal defense or repair mechanism that deteriorates with age. Although capable of handling the normal photo-oxidative stress in youth, with age such a system may no longer be able to protect the retinal structures against previously nondamaging exposures and irreversible changes start to accumulate. Because of the limitations in the assessment of ocular exposure and the small numbers of subjects, this study does not prove a causal linkage between ocular exposure to blue or visible light and AMD. However, it does support that notion and suggests that further study of this important question is indicated.

The nature of the association between solar radiation and pterygium and CDK is different from that for either cortical cataract or AMD-4. Whereas cortical cataract is associated only with UV-B and AMD-4 is associated only with visible radiation, an association has been found between each band of radiation between 290 nm and 700 nm we examined and both pterygium and CDK. In each instance, the association has been somewhat stronger for CDK, although the association was statistically significant for each condition. These differences in the association with exposure suggest that the pathophysiology of these conditions is likely to be very different. In none of these conditions is the pathophysiology well established. Climatic

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droplet keratopathy may be due to the photodenaturation of diffusible plasma proteins that accumulate in the cornea.[14] Pterygium may be due to the generation of vasoproliferative factors in a photodenatured Bowman's membrane or possibly by cells in the corneal epithelium.[37] Cortical cataract may be due to photo-oxidation of the crystallines in cortical lens fibers, the membranes of these fibers, or damage to either the lens epithelium or the lens fiber enzyme systems that repair photo-oxidative damage.[27] Macular changes are usually thought of as a result of photo-oxidative damage to either the photoreceptors themselves or damage to the retinal pigment epithelium that leads to secondary changes.[38] Recently, it has been suggested that photo-activation of hemoglobin precursors may occur in red blood cells passing through the choriocapillaris and that this could lead to the generation of reactive oxygen species.[39] This would then lead to retinal pigment epithelium damage with secondary changes in the overlying photoreceptors.

Whatever the mechanisms for the various types of ocular damage, it seems prudent to protect the eyes from unnecessary solar radiation. UV-B has been previously associated with lenticular damage; and in this study, we found evidence supporting an association between excessive ocular exposure to blue or visible light and retinal disease. Our data are not able to distinguish between a "blue light hazard" and a "visible light hazard" for the macula. Those exposed to high levels of solar radiation, such as fishermen, skiers, sunbathers, golfers, gardeners, and so forth, would be well advised to wear UV-absorbing, wrap-around sunglasses when they are out in the sun. Despite the relatively small number of subjects in this study suggesting an association between ocular exposure to blue or visible light and AMD, the use of sunglasses that also block a significant amount of broad-spectrum visible radiation should not be discouraged, especially since this use would be relatively inexpensive and without side effects. Similarly, the middle-aged and elderly would do well to reduce unnecessary exposure to intense sunlight, and the use of sunglasses and a brimmed hat should be encouraged during outdoor exposure in the middle of the day.

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