SOLAR RETINOPATHY: A PHOTOBIOLOGICAL AND GEOPHYSICAL ANALYSIS*

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INTRODUCTION

SINCE THE TIME OF PLATO, VISUAL DISTURBANCES HAVE BEEN ASSOCIATED with sun viewing. In Phaedo, Socrates advised individuals to watch an eclipse only through its reflection in water.[1] The adverse effects of solar radiation have been appreciated in studies by physicians and scientists for more than two centuries.[1] The earliest investigations on ocular damage of the retina by light were by Czerny (1867),[2] Deutschmann (1882),[3] and Widemark (1893).[4] Legendary scientists, beginning with Galileo, the father of astronomy, to Meyer-Schwickerath, the father of photocoagulation, have tragically injuried their eyes through the study of the sun or the experimental production of radiant energy. It was Moron-Salas[5] who first controlled the adverse effects of light so that it could be used for therapeutic coagulation in numerous experimental procedures on humans and rabbits in the late 1940s. These concepts were legitimized by the pioneering work of Meyer-Schwickerath,[6] who was studying them concurrently.

The first description of visual damage caused by the sun was by the Swiss physician, Bonetus, during the 17th Century.[1] Clinical cases of eclipse blindness were subsequently reported by Saint-Yves in 1722.[7] Cases of solar retinopathy reported in the literature since the 18th Century have principally been the result of eclipse viewing, when persons

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misjudged the intensity of solar radiation through relatively nonconstricted pupils. With the advent of the ophthalmoscope, the clinical manifestations of eclipse burns were described by Cords,[8] Blessing,[2] and Birch-Hirschfield[9] in 1912. Over the years, each major eclipse has tended to produce a series of cases.[10-28] Direct, sustained viewing of the sun has also been associated with solar retinopathy.[2,13,14,24,27] Some reports described patients who viewed the sun only minimally.[29,30] Finally, the macular disorder has also been rarely reported as a hazard of sunbathing without a clear history of sun gazing.[2,13,14,21] Very extensive summaries of the retinal damage created by solar radiation were published in a dissertation by Ham (1947),[31] a manuscript by Lanum (1978),[32] a book by Lerman (1980),[33] a thesis by Lawwill (1982),[34] and an edited text by Waxler (1986).[35]

This paper is the report of a series of young adult patients who developed solar retinopathy over a 2-day period in the spring of 1986 in a particular region of the United States. These patients had a history of sun exposure, without direct sun viewing. None was predisposed to solar retinopathy because of any other known risk factor except for age and refractive state of the eye. Several governmental scientific agencies provided geophysical data which were analyzed in an attempt to explain the chorioretinal radiational effects noted in these patients. Certain geophysical changes present during this 2-day period were investigated as possible risk factors in the pathogenesis of retinal damage present in these patients with solar retinopathy. On the basis of the photobiological and geophysical analysis of these cases, a multifactorial hypothesis for the pathogenesis of solar retinopathy is presented.

METHODOLOGY

Ocular histories were obtained from four patients who reported visual disturbances following sun exposure in the United States on the weekend of March 28-29, 1986. One case was seen in a patient from the western part of the Great Lakes District on March 28, 1986, and the remaining three patients were from the Greater Metropolitan Area of New York on the following day, March 29, 1986. These patients had a complete ophthalmological examination including vision testing, muscle balance testing, anterior and posterior slit-lamp biomicroscopic examinations, direct and indirect ophthalmoscopy through a dilated pupil, central and peripheral field testing, intraocular pressure determinations by applanation, fundus color and monochromatic photography, and fluorescein angiography. All patients were examined by one of the authors (LAY).

CASE REPORTS

CASE 1

A 20-year-old white woman who sunbathed in Ann Arbor, Michigan for 2 hours on March 28, 1986 noted the onset of a central scotoma in each eye 1 day later. The patient denied any direct sun gazing, the use of sunglasses, the wearing of a hat, the use of systemic or topical drugs, and a previous ocular or medical history. The patient also experienced a severe sunburn on the day of sun exposure. An examination by a local retinal specialist on April 2, 1986 revealed a visual acuity of 20/80 OD and 20/40 OS. A relative central scotoma and metamorphopsia were noted bilaterally on Amsler grid testing. The anterior slit-lamp examination was normal in each eye. The fundus examination revealed a yellowish-gray disturbance in the foveal area of each eye, more prominent in the right eye, which was her dominant one. Some greyish discoloration to the pigment epithelium was evident surrounding the more conspicuous yellowish lesions.

The patient was examined in New York City on July 21, 1986, approximately 3 months later. At that time, the visual acuity had improved to 20/25 OD and 20/20 OS. The patient still described bilateral, relative scotomas and metamorphopsia. The yellowish-gray reaction in the macula was now replaced by a juxtafoveal cyst in each eye. The reddish, irregular, but well demarcated cystic thinning of the outer retina measured 50 μ OD and 25 μ OS. There was no associated exudative manifestation in the macula of either eye. A fluorescein angiogram revealed a minimal degree of retinal pigment epithelial "window defect" at the site of the cystic disturbance in the right eye. The patient was a bilateral emmetrope.

CASE 2

A 27-year-old white male medical student who sunbathed and exercised for 3 hours on March 29, 1986 in Teaneck, New Jersey, developed blurred vision in the right eye on March 30, 1986. There was also a slight but poorly characterized disturbance in the central vision of the left eye. The patient denied any direct sun gazing, the use of sunglasses, the wearing of a hat, the use of systemic or topical drugs, and a previous ocular or medical history. He also sustained a slight sunburn, which was unusual for this olive-skinned individual. An examination on April 9, 1986 revealed a visual acuity of 20/40 OD with eccentric fixation and 20/20 OS. A relative central scotoma with metamorphopsia was described in the right eye and a faint degree of juxtafoveal relative scotoma was noted in the left eye on Amsler grid testing. The anterior slit-lamp examination was normal in each eye. Biomicroscopic examination of the fundus with the Goldmann lens revealed a very faintly evident reddish juxtafoveal cyst in the right eye. There was also a perifoveal granular appearance to the retinal pigment epithelium in that eye. An indistinct appearance to the perifoveal region of the left eye was noted. A fluorescein angiogram was normal bilaterally. A follow-up examination on June 16, 1986 revealed a visual acuity of 20/20 OU with a persistence of a small relative scotoma and metamorphopsia OD, but no evident visual defect OS. The cystic macular lesion in the right eye was now clearly evident, but the perifoveal retinal pigment

epithelial disturbance had resolved. The foveal reflex in the left eye was now distinct or normal. The patient was a bilateral emmetrope.

CASE 3

A 14-year-old white female who sunbathed and exercised for 4 hours in Rye, New York on March 29, 1986 noted a central visual field defect on the following morning. The patient denied any direct sun gazing, the use of sunglasses, the wearing of a hat, the use of systemic or topical drugs, and a previous ocular or medical history. She also experienced an intense sunburn on that day. The patient was seen on April 2, 1986 by her local ophthalmologist who noted a visual acuity of 20/50 OD and 20/20 OS. A relative central scotoma and metamorphopsia was described on Amsler grid testing in the right eye. Only a very faint disturbance was described on Amsler grid testing in the left eye. A yellowish lesion was seen on ophthalmoscopy in the foveal area of the right eye. Only an indistinct foveal reflex was noted in the left eye. An examination on April 14, 1986 by an author (LAY) revealed a visual acuity of 20/40 OD with eccentric fixation and 20/20 OS. A relative central scotoma and metamorphopsia were still described in the patient's dominant eye, the right eye, on Amsler grid testing. The patient no longer noted a defect in the central vision of the left eye on Amsler grid testing. An anterior slit-lamp examination was unremarkable bilaterally. Biomicroscopic examination of the fundus of the right eye revealed a very subtle juxtafoveal, reddish cyst which was 25 μ in size. The surrounding retinal pigment epithelium was faintly greyish, but there was no definite exudative manifestation. The left eye was normal, and a fluorescein angiogram was normal bilaterally. A follow-up examination on June 26, 1986 revealed improvement of the visual acuity of the right eye to 20/25. There was a persistence of the relative central scotoma and metamorphopsia in the right eye, and the cystic-like juxtafoveal lesion was still present with a more distinct demarcation to its margins. The patient was essentially bilaterally emmetropic, since she used low myopic corneal contact lenses at the time of sun exposure.

CASE 4

An 18-year-old white man who sunbathed and exercised for 3½ hours in Merrick, New York, on March 29, 1986 noted a decrease in the central vision OS a few days later. He also experienced a severe sunburn. The patient denied any direct sun gazing, the use of sunglasses, the wearing of a hat, the use of systemic or topical drugs, and a previous medical history. He did have a history of a racquetball blow to the orbital framework of the left eye 18 months prior to the onset of his visual symptoms. He had been examined by two ophthalmologists following his injury, and no ocular pathology or visual dysfunction was detected on repeated examinations. Examination by a local ophthalmologist during the month of May of 1986, approximately 6 weeks after his history of sun exposure, was reported to reveal a diminished visual acuity to the level of 20/60, a relative scotoma and metamorphopsia, and a juxtafoveal cyst OS, which was the patient's dominant one. The

visual acuity was 20/20, and the clinical examination was reported to be normal OD at that time. On July 7, 1986 when the patient was examined by an author (LAY), the visual acuity had improved to 20/40− OS. A relative central scotoma with metamorphopsia was still described on Amsler grid testing. The anterior slit-lamp examination was unremarkable bilaterally. Biomicroscopic examination of the fundus revealed a juxtafoveal cyst in the left eye of approximately 75 μ in size. The right fundus was normal. A fluorescein angiogram was normal bilaterally. A follow-up examination on October 6, 1986 revealed a visual acuity of 20/20 OD, and an improvement in acuity to better than 20/30 OS. There was a persistence of the relative central scotoma and metamorphopsia, as well as the cystic-like juxtafoveal lesion which was now more distinct in appearance. The patient was a bilateral emmetrope.

The New York Fluorescein Angiography Club was surveyed in search of additional cases of solar retinopathy that had occurred during the 2-day period. Six other similar cases from five other retinal specialists were reported from this group. These cases were not included in this report because the authors did not personally examine these patients.

GEOPHYSICAL STUDIES

Since cases of solar retinopathy are seen at a rate of approximately one every 2 to 3 years on the Retinal Services of the Manhattan Eye, Ear and Throat Hospital, a series of geophysical inquiries were made in an attempt to explain the reported cases. Several governmental agencies and scientific institutions were consulted to determine the geophysical factors which were in effect during the 2-day period in the involved geographical areas. The consultants included the National Aeronautics and Space Administration (NASA), the Goddard Space Flight Center (GSFC), the National Oceanographic and Atmospheric Administration (NOAA), the Jet Propulsion Laboratory (JPL) and the General Electric Astrophysics Laboratory (GEAL).

The extraterrestrial and terrestrial solar radiation fluxes, in micro watts/m^2 per nanometer, solar zenith angle and alterations in meterologic or climatic conditions, such as changes in the presence of airborne pollutants, the humidity, status of the cloud cover, the surface altitude, the environmental reflectivity, the ground level visibility, and the atmospheric ozone protection levels, were analyzed. The National Climatic Data Center and the National Weather Service and John F. Kennedy International Airport provided meteorological data for the involved areas. On March 28, 1986 in Ann Arbor, Michigan, the site of case 1 and on March 29, 1986 in the Greater Metropolitan New York area, the location of cases 2 to 4, the skies were described as intensely bright, clear and

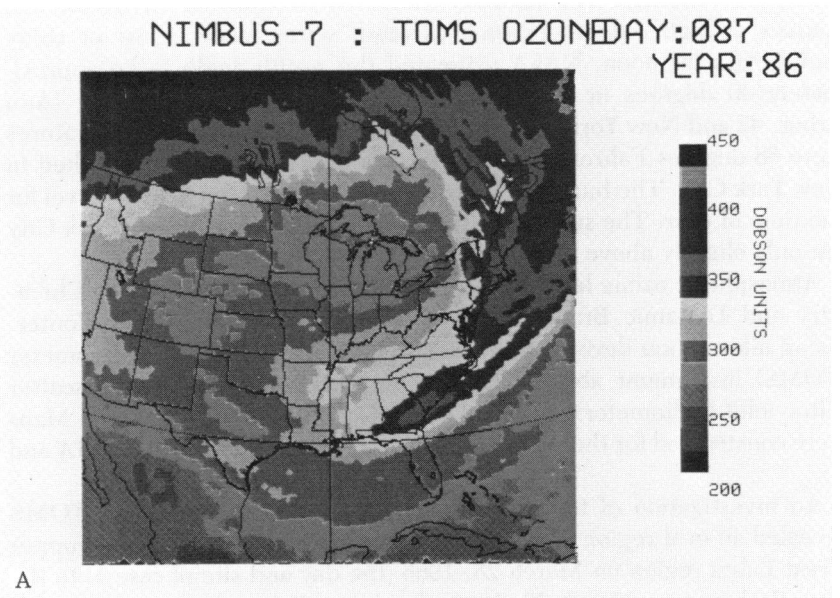
cloudless. The very sunny skies were associated with 15 to 20 miles of unlimited visibility. The patients described no unusual environmental sources of high reflectivity such as sand, water, snow, glass or shiny metal. At solar noon, NASA estimated the zenith angle to be approximately 40 degrees in each area, corresponding to the lattitude (Ann Arbor, 42 and New York City, 41). The unseasonably warm temperatures were 68 degrees Fahrenheit in Ann Arbor and 72 degrees Fahrenheit in New York City. The humidity was 30% in each locale, an average level for the time of year. The surface altitudes for Ann Arbor and New York City are only slightly above sea level.

Atmospheric ozone levels were obtained from the Atmospheric Chemistry and Dynamic Branch of NASA's Goddard Space Flight Center. Using information derived from the Total Ozone Mapping Spectrometer (TOMS) instrument aboard the Nimbus 7 Satellite (Solar Backscatter Ultraviolet Radiometer), National Color-Coded Total Ozone Level Maps were constructed for the 2-day period, March 28 and 29, 1986 (Fig 1A and B).

An investigation of the total ozone distribution measured by TOMS revealed an oval region of minimum ozone which moved from the upper Great Lakes region on March 28, 1986 (the day and site of case 1) to the New York area on March 29, 1986 (the day and site of cases 2 to 4) and, finally, to the Northeast states. Such features are components of the general total ozone minima associated with upper tropospheric ridges. Localized extreme minima form on the anticyclonic side of jet streams in response to the vertical motion field of jet streaks.

The spectral distribution and magnitude of sunlight incidence at ground level is strongly dependent on wavelength near the so-called atmospheric cut-off at about 295 to 300 nm. In addition, the solar radiation becomes increasingly diffuse at shorter wavelengths due to the spectral dependence of Rayleigh scattering. For midlatitudes the flux from the sky exceeds the direct flux from the sun at wavelengths shorter than about 310 nm. Sundararaman et al[36] have computed the direct, diffuse, and total ultraviolet flux under cloudless conditions for a range of solar zenith angles, geophysical conditions and wavelengths from 297.6 to 332.4 nm. The tabular output from this report has been used to estimate the increase in ultraviolet flux at the ground during the March 1986 ozone episode.

Total ozone varies with an annual cycle at all latitudes. For midlatitudes, the maximum also corresponds to the period of highest variability, with the day-to-day variations sometimes exceeding the amplitude in the annual cycle. These features are illustrated in Fig 2A and B, the annual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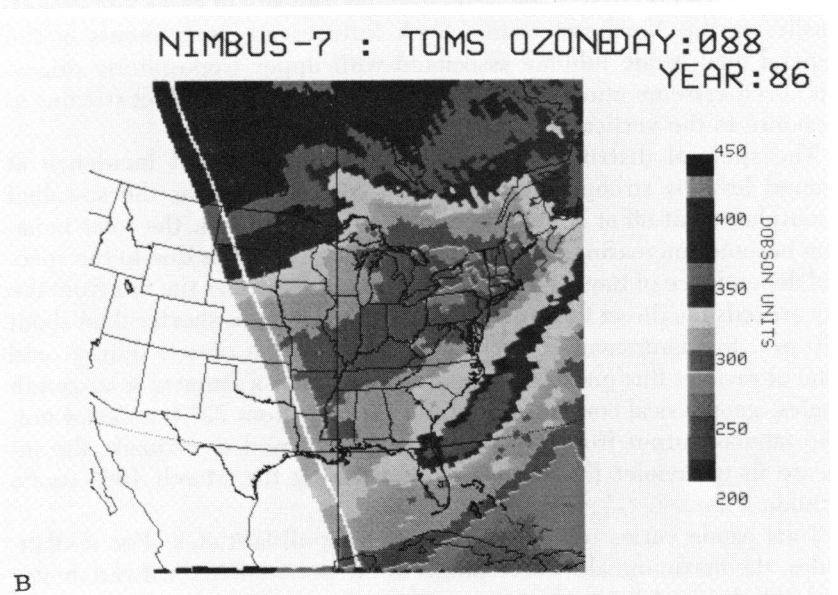
A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YEAR:86
450
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DOBSON UNITS

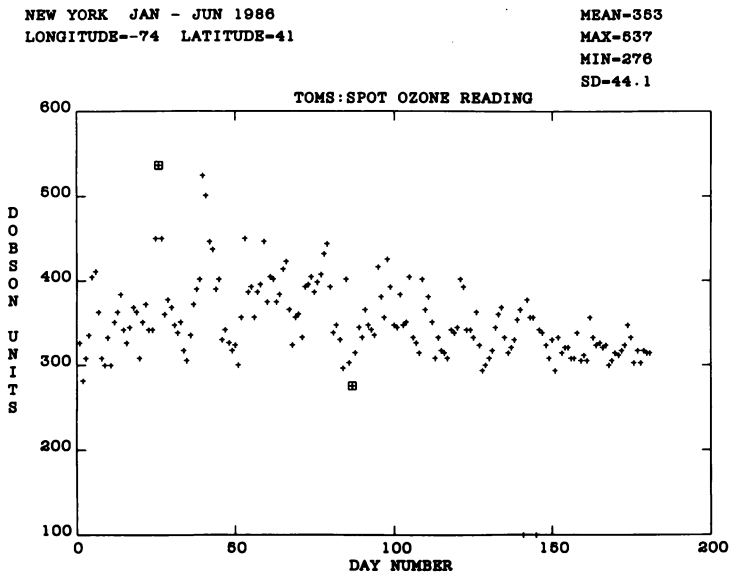
B

FIGURE 1
False color images for total ozone variations taken with Total Ozone Mapping Spectrometer (TOMS) aboard the Nimbus 7 Satellite. A: Mach 28, 1986. A low total ozone was present in the Western Great Lakes area, site of case 1. B: March 29, 1986. The low total ozone drifted to the New York area, site of cases 2 to 4. Low total ozone drifted to northeast states on the following day.

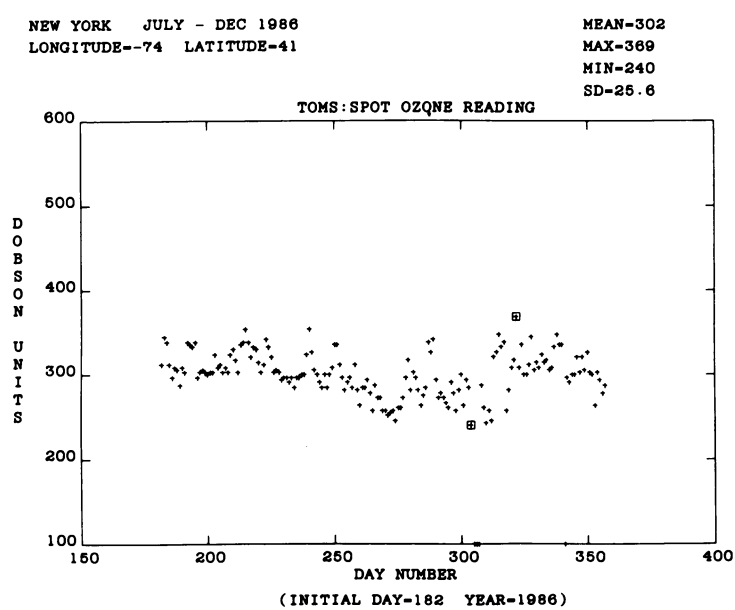
variation of total ozone over New York during 1986. The total ozone column is measured in Dobson units which are calculated from a theoretical compression of the column of ozone to form pure ozone. The thickness of this layer is measured at sea level with standard temperature and pressure. Each Dobson unit is equivalent to 0.001 cm pure ozone under these specified conditions.

For the present case the total ozone in the localized minimum was 293 DU at Ann Arbor, Michigan on the first day of the episode and 298 DU at New York on the second day of the episode. The behavior of total ozone during the month of March 1986 over Ann Arbor is shown in Fig 2C. The corresponding data for New York are presented in Fig 2D. At both locations the lowest value observed during the month was coincident with the passage of the low ozone center. The average total ozone at Ann Arbor for March 1986 was 363 DU. The comparable figure for New York was a mean of 352 DU. The passage of the minimum resulted in a full 70 DU (or 23%) decreased from the monthly mean at Ann Arbor and a 54 DU (or 15%) decrease at New York. Eight days earlier the ozone at each of the locations was at its monthly maximum; 438 DU at Ann Arbor, 444 DU at New York.

The ozone reduction from the monthly average will be assumed to represent the change in conditions during the solar retinopathy episode. The latitude of Ann Arbor is 42 N while that of New York is 41 N and the episode occurred 1 week after the spring equinox. The calculations for 40 N at equinox conditions will be assumed to apply. The change in total (direct and diffuse) solar flux at the ground (F) for nine wavelengths in the ultraviolet spectral region under clear sky conditions at local noon are listed in Table I. The columns labeled F_{max} and F_{ave} are the fluxes with the minimum observed total ozone at Ann Arbor (293 DU) during the event and with the monthly average total ozone at Ann Arbor (363 DU) during March 1986. The last column is the ratio of the maximum flux to average flux, a measure of the enhancement of radiation by the low total ozone. The radio depends strongly on wavelength, ranging from a maximum of 4.08 at 297.6 nm to a minimum of 1.01 at 332.4 nm. Any physiologic effects of a total ozone change are likely to occur at wavelengths below 310 nm where the flux changes are larger. Plots of the flux



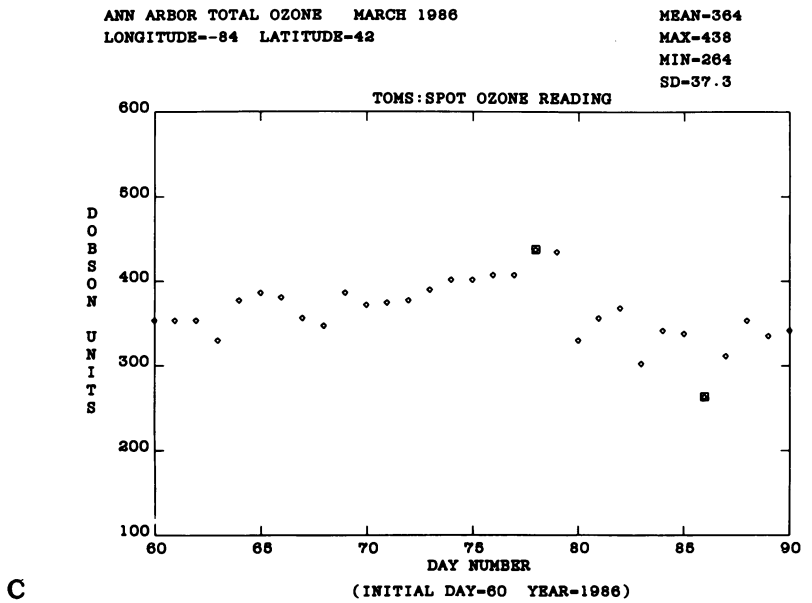
A



B

FIGURE 2

Total ozone distribution measured by TOMS aboard Nimbus 7 satellite. A: New York City area January–June 1986. B: New York City area July–December 1986. The minimum for the New York City area corresponds to the day of the reported cases of solar retinopathy.



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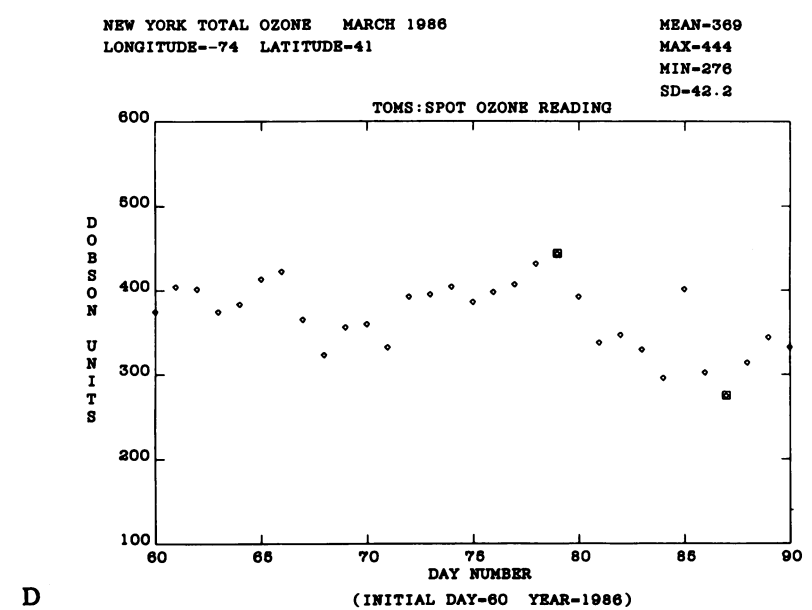


D

FIGURE 2 (Cont'd)
C: Ann Arbor March 1986. D: New York City area March 1986. March minima for Ann Arbor and New York City area correspond to reported cases of solar retinopathy.

TABLE I: SOLAR FLUX INCREASE DURING SOLAR RETINOPATHY REPORTED CASES

WAVELENGTH (nm)	ANN ARBOR (3/28/86)			NEW YORK AREA (3/29/86)		
	F_{max}	F_{ave}	F_{max}/F_{ave}	F_{max}	F_{ave}	F_{max}/F_{ave}
297.6	7.07 (2)*	1.73 (2)	4.08	6.39 (2)	2.16 (2)	2.96
300.4	7.00 (3)	2.98 (3)	2.35	6.59 (3)	3.41 (3)	1.93
305.4	5.36 (4)	3.52 (4)	1.52	5.20 (4)	3.76 (4)	1.38
308.8	9.50 (4)	6.98 (4)	1.36	9.29 (4)	7.33 (4)	1.27
311.4	1.46 (5)	1.18 (5)	1.23	1.44 (5)	1.22 (5)	1.18
317.6	2.81 (5)	2.56 (5)	1.10	2.79 (5)	2.60 (5)	1.07
325.4	4.27 (5)	4.15 (5)	1.03	4.26 (5)	4.17 (5)	1.02
329.1	5.42 (5)	5.33 (5)	1.02	5.41 (5)	5.35 (5)	1.01
332.4	5.59 (5)	5.53 (5)	1.01	5.58 (5)	5.54 (5)	1.01

	MARCH 1986 AVERAGE OZONE	MARCH 1986 MINIMUM OZONE
Ann Arbor	363 DU	293 DU
New York area	352 DU	298 DU

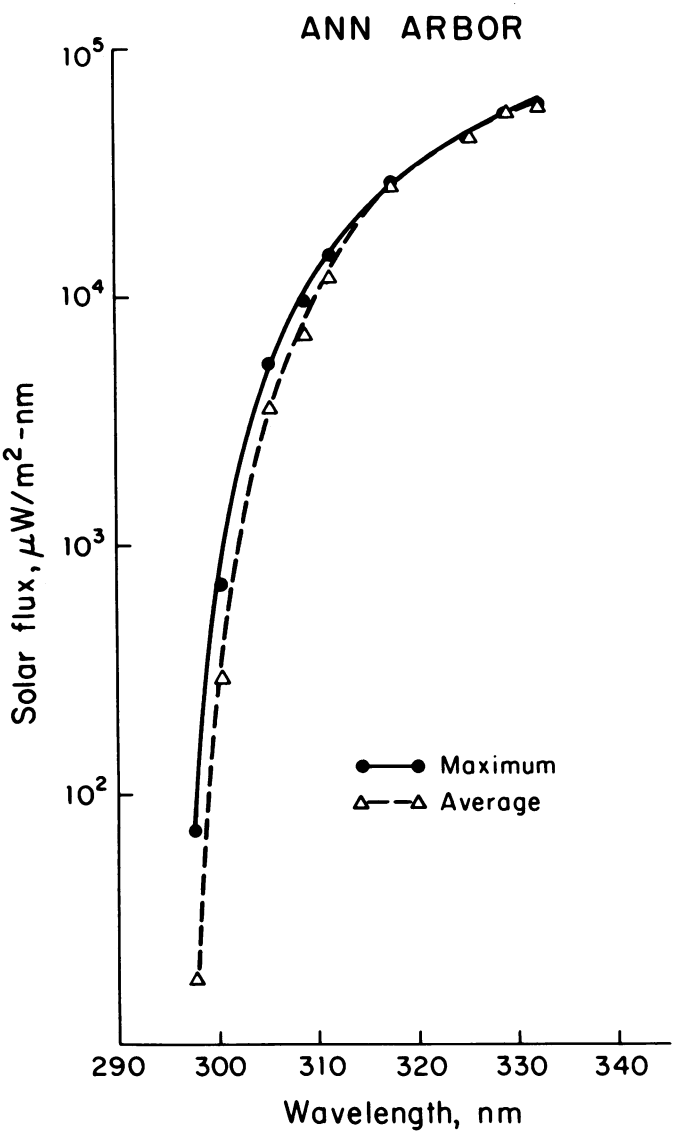
Lattitude, 40 degrees N; solar declination, 0 degrees; time, local noon; *( ), exponential power of 10.

spectral distribution of Ann Arbor and New York are shown in Fig 3A and B.

DISCUSSION

CLINICAL FEATURES OF SOLAR RETINOPATHY

Solar retinopathy which is also known as eclipse burn,[1] eclipse blindness,[19] eclipse retinopathy,[18] solar retinitis,[12,17,22,29,37] solar chorioretinal burn,[15] foveomacular retinitis,[20,25,28,38] photoretinitis,[10,39] and photomaculopathy,[40] generally occurs in patients who have viewed an eclipse, have gazed directly at the sun, or have been subjected to a form of accidental or experimental intense light. While the majority of cases of solar retinopathy have involved eclipse viewing, several papers in the ophthalmic literature have reported the maculopathy following direct sun viewing. These individual cases usually have a history of sun gazing because of presumed ritualistic, religious, sun-worshipping,[1,14,15,17] alleged malingering,[25,46] misconceived therapeutic measures to strengthen the eyes,[1,13] mental illness with elements of self-destruction or blindness, or use of hallucinogenic drugs such as LSD.[1,23,41-43] Military personnel assigned to survey the sky for enemy aircraft,[1,10,12,19,38] seamen,[1,3,12] and astronomers[1] are also at risk of developing this form of retinopathy. Solar retinopathy has also been described to occur in patients who have mini-


ANN ARBOR
10^5
10^4
10^3
10^2
Solar flux, $\mu W/m^2$-nm
Maximum
Average
290
300
310
320
330
340
Wavelength, nm

A

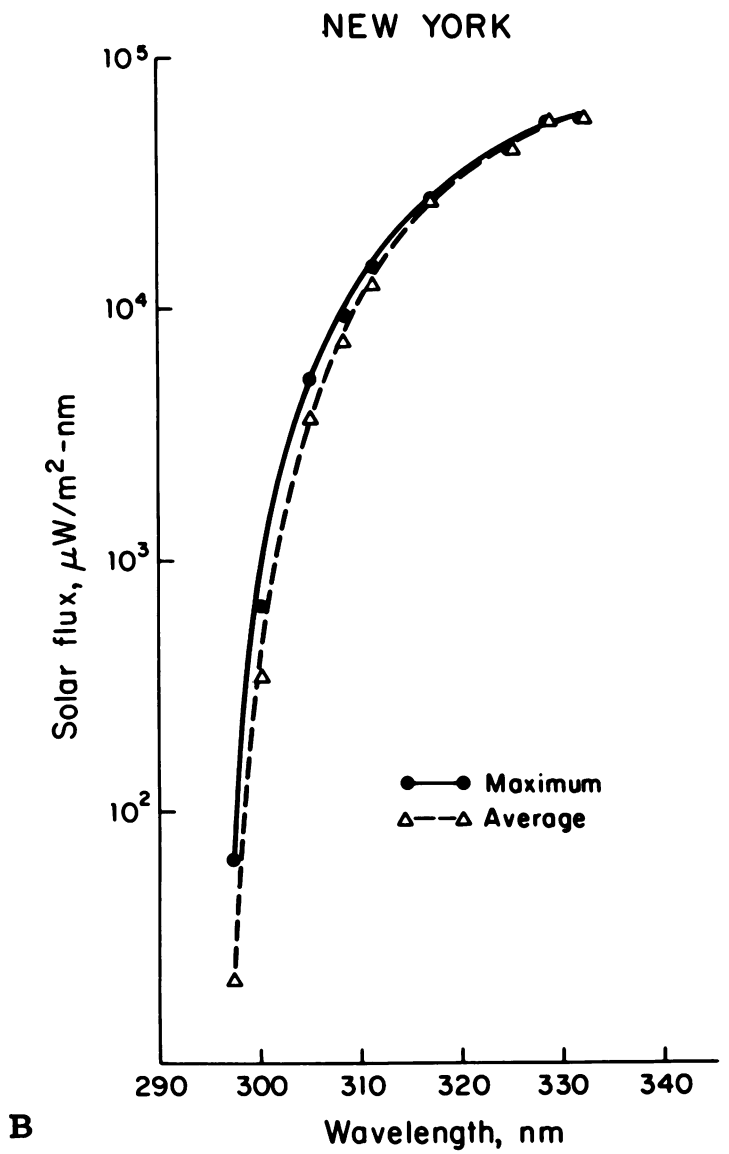


FIGURE 3
Plot of flux spectral distribution. A: Ann Arbor area. B: New York area.

mally viewed the sun[29,30] or who have been sun bathing, not sun gazing, as in this report.[1,13,14,21]

The typical patient with solar retinopathy is the young adult emmetrope or low hypermetrope.[1,9,13,23,26] Soon after gazing at the sun, these patients complain of reduced acuity, a central scotoma, chromatopsia, photophobia, and metamorphopsia shortly after sun exposure.[1,44,45] Most cases are bilateral, but asymmetric with a predisposition for right eye involvement, presumably because it is more likely to be the dominant eye.[1,13,24] Shortly after sun exposure, the visual acuity in patients with solar retinopathy is 20/30 to 20/100. The fundus examination depends on the severity of the photic damage. Very mild cases may reveal little or no visible changes in the macula on ophthalmoscopy. An indistinct foveal reflex or a slight greyish thickening to the retinal pigment epithelium (RPE) in the foveal area may be the only discernible manifestation evident to correlate to the patient's central vision symptoms. As the vision recovers, the macular manifestations essentially disappear. The foveal reflex becomes more distinct and the RPE normalizes. In moderate photic injuries a very minor degree of perifoveal or juxtafoveal RPE granularity or punctate atrophic disturbance may persist.

Examination of the macula in an acute phase of more severe cases of solar retinopathy, the so-called eclipse burn reveals a solitary or less commonly, multiple yellowish-grey spots at or near the foveola (Fig 4A). These lesions may be enveloped completely or incompletely by a faint grey annulus. By approximately 2 weeks after sungazing, the initial moderate to severe lesion is replaced by a 25 to 75 μ-sized, oval-shaped, reddish, sharply but irregular demarcated outer lamellar defect in the foveola, or more commonly in the juxtrafoveolar area. The excavated appearance, presumably from outer retinal shrinkage, can be seen clinically with the slit-lamp biomicroscope and the Goldmann lens, with high magnification and indirect illumination or more distinctly with the direct ophthalmoscope. It may be surrounded partially and irregularly by some greyish mottling to the pigment epithelium. The cust or apparent macular hole becomes clinically more evident as the acute manifestations resolve.

Fluorescein angiographic studies essentially reveal no late hyperfluorescence or leakage or abnormality in the retinal vasculature.[44-46] A few severe cases have revealed a minor degree of early hyperfluorescence or "window defect" secondary to atrophic changes at the level of the RPE.

The vision in most patients returns to the level of 20/20 to 20/30 within a period of 3 to 9 months. The cystic-like disturbance in the juxtafoveal lesion is generally more distinct at this time as the surrounding reaction at the levels of the RPE subsides (Fig 4B). A small central scotoma or

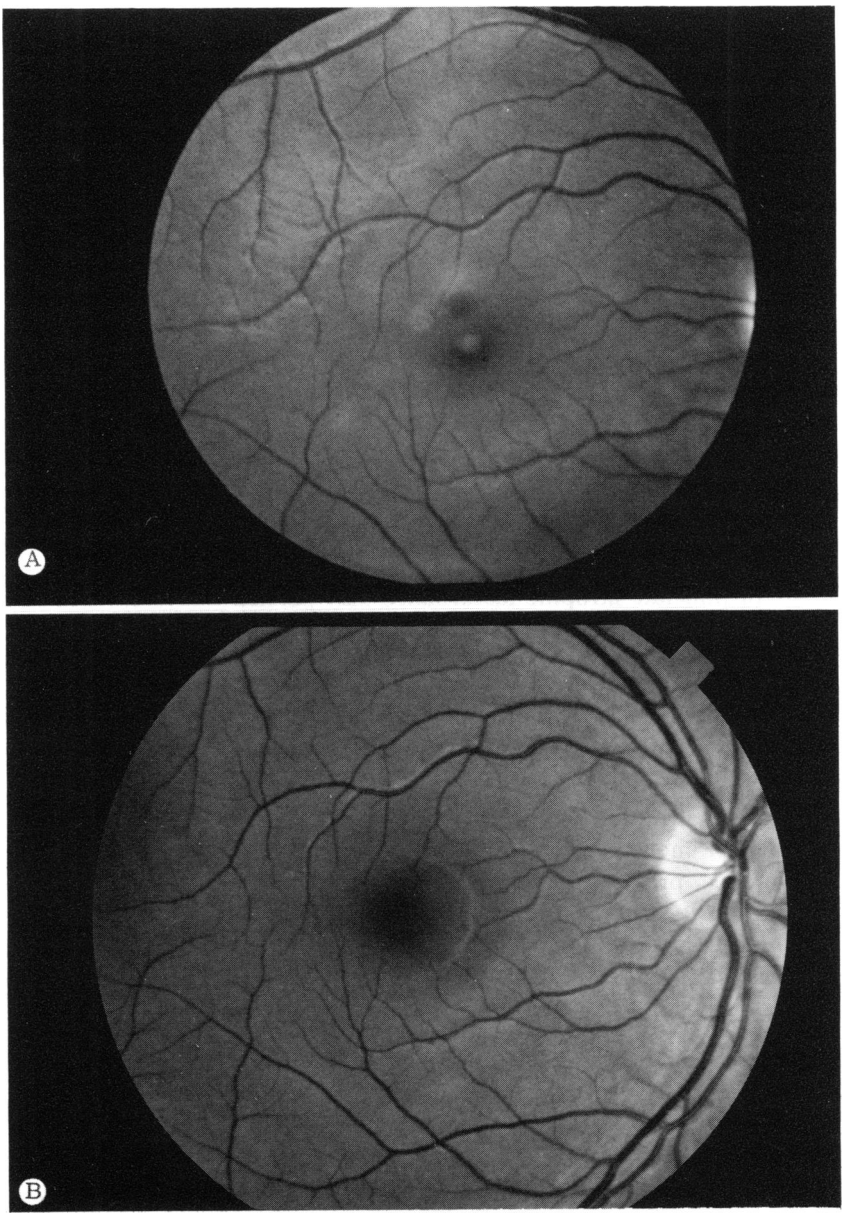


FIGURE 4
Clinical photos of solar retinopathy (case 1). A: Acute, yellowish foveal lesion. Visual acuity is 20/80−. B: Same eye 1 year later. There is a typical, foveal, small, reddish, well-delineated "cyst." Visual acuity is 20/25.

metamorphopsia may persist, and the foveolar lesion itself remains permanently, attesting to the antecedent sun exposure. The authors agree with Gass, who has said that the classical foveal lesion associated with severe photic injuries "remains permanently and is virtually pathognomonic of solar retinopathy."[44]

ANALYSIS OF RETINAL DAMAGE IN THE REPORTED CASES

There are likely to be many reasons for a given individual's susceptability to solar retinopathy or any other complex biological process. There are, however, two fundamental mechanisms which relate to the ocular damage, the *photobiological* reactions and the *geophysical* conditions. In order to analyze these factors in the reported cases, it is of value to review first the mechanisms and nature of adverse effects of solar radiation on the retina.

MECHANISMS OF RETINAL DAMAGE

Since the beginning of the 20th Century, an enormous amount of clinical and experimental research has been carried out in an attempt to understand the adverse effects of solar radiation and intense light on the retina.[31-38,40,47-93] At least three types of retinal damage from intense light have been described: *mechanical damage* from acoustic transients and shock waves in the retina created by mode-locked or Q-switched lasers; *thermal damage* from light absorption principally by the RPE and rise in the temperature of surrounding tissue; and *photochemical damage* from short wavelength light which does not produce an appreciable rise in temperature in the retina.[79,91] These effects in the eye caused by exposure to solar radiation are wavelength, intensity and time dependent. Mechanical damage depends on high irradiances and short exposure durations while thermal damage is independent of wavelength but related to the rate of delivery. Photochemical damage, by comparison, is associated with lower levels of power, longer exposure and extreme wavelength dependency.

Studies in the early 1900s supported the belief that retinal damage from high intensity light sources such as solar radiation were almost exclusively due to thermal reactions with denaturation of proteins by intense heat absorption.[48] It became evident that powerful light sources such as the sun and later xenon arc lamps and lasers could be focused through the ocular media onto the retinal surface producing tissue burns. Geeraets et al[51] studied threshold laser burns histopathologically and found the RPE to be the first and most severely affected tissue. They felt the lesions were

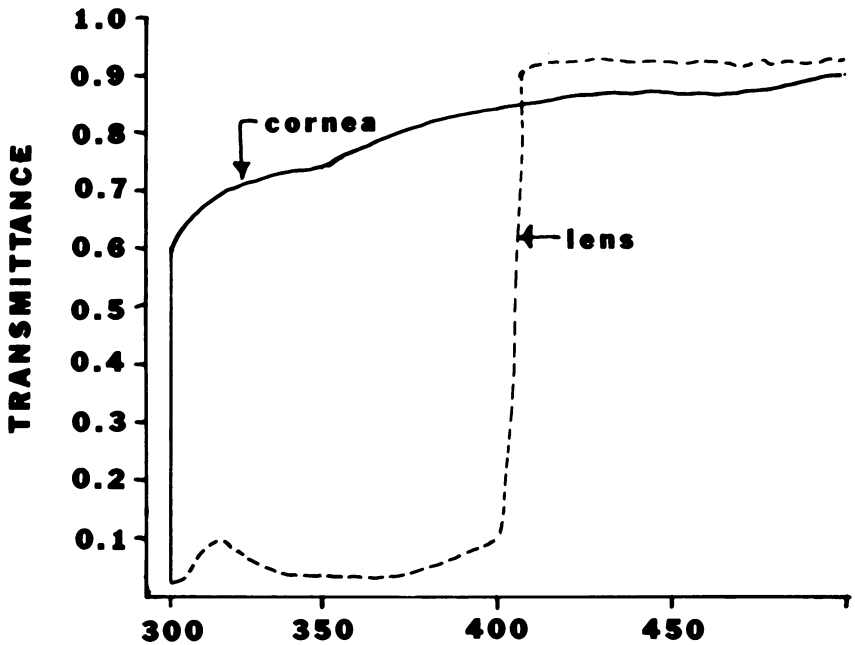


FIGURE 5
Transmittance of cornea and crystalline lens (adapted from Boettner EA, Wolter JA: Transmission of ocular media. *Invest Ophthalmol* 1962; 1:766-783).

most consistent with a thermal mechanism of injury. The focusing capability of the globe, especially in an emmetrope or mild hyperope would add to the concentration of energy at the foveola increasing the heat energy effect.

The thermal theory of injury in solar retinopathy generally persisted until the 1960s when several investigators reported light damage in animal studies from relatively low-energy light sources suggesting that these low energy light injuries could not be thermal.[53-56,74,75] The concept of photochemical damage to the retina was demonstrated first in rodents and primates and, subsequently, in humans.[33,35,53,55,70,71,74,75,82] Mainster and other investigators[67,70,71] showed that sungazing could only produce a temperature increase of less than 4°C which was far less than the 15° to 20°C temperature rise needed for photocoagulation. This was strong evidence that the retinal damage in solar retinopathy was likely to be a combination of thermal and photochemical reactions, or thermally-enhanced photochemical damage.

Three forms of photochemical reaction have been identified; the retinal damage by each type is dependent on characteristics of the tested species and on the physical parameters of intensity, duration, and wavelength.[34,68,69,75,76] Two types of photochemical damage are associated with relatively low levels of radiant energy. One is a visual pigment, rhodopsin-specific form of photochemical injury first noted in rats, other nocturnal animals,[34,65,75] and subsequently in primates.[88] A second mechanism for low energy level photochemical damage to the retina was described by Sperling and co-workers[61,68,74] Permanent destruction of blue sensitive cones and long-term inactivation of green sensitive cones were produced in monkeys.

The third mechanism of photochemical damage is produced by higher levels of energy which is still well below the power needed to produce a thermal effect. With this type of photochemical damage, there is an exponential rise in retinal sensitivity with a decrease in the wavelength.[32,34,54,92] Several investigators attempted to determine which wavelengths were the most efficient in producing this form of photochemical damage.[53,65,72,73,75,76,78,79] Ham and associates[72,73,76,78,79] examined the sensitivity of the retina to radiation damage as a function of wavelength with monochromatic lasers extending from 1064 to 441 nm. Exposure to the near infrared produced a typical photothermal reaction caused by a temperature rise of 25% above ambient. The 441 nm light produced a photochemical lesions with a temperature rise too low to induce denaturation. The spectral band width of 400 to 800 nm was more than five times more effective than the 700 to 1400 nm bandwidth in producing a retinal lesion in the monkey eye. In later experiments, this same research group found that retinal sensitivity to injury increased dramatically in the blue region of the visible spectrum. They concluded that solar retinopathy could be produced in the retina by short wavelength light (441 to 550 nm).[54,73]

In subsequent, more elaborate experiments, Ham and associates[76,78,79] studied the effects of shorter wavelength radiation in the near ultraviolet range (320 to 400 nm) or UV-A, noting both ophthalmoscopic and histopathologic evidence of damage to photoreceptors. They were concerned about adverse effects of these wavelengths in the aphakic eye and in the young, whose lenses do not effectively reduce the transmittance of UV-A radiation. They found that the retina was six times more sensitive to UV radiation (325 nm) than to short wavelength visible light (441 nm). The sensitivities were 5 J/cm at 325 nm and 30 J/cm at 441 nm. The histopathologic findings with the UV-A lesions differed significantly from the blue light damage. Excessive damage to the photoreceptors was the

principle finding, compared to primary RPE reaction seen with blue light.[72,77]

In humans, there was additional evidence in support of the role of photochemical effects in solar retinopathy. Clark et al[49] noted prolongation of dark adaptation times after 3 to 4 hours of working in the sun, not sun gazing. Hecht et al[50] showed similar prolongation of dark adaptation that persisted for as long as 10 days with sun exposure of 3 to 4 hours each day. They suggested that these prolonged and accumulative electrophysiologic disturbances were due to subthreshold levels of photochemical damage, which produced no visible change in the retina. Photochemical retinal damage in general has been shown to be cumulative or additive in its adverse effects.[52,53,92] In fact, divided doses of light have produced retinal damage more effectively than continuous exposure of the same total period and intensity.[53]

Postulated mechanisms of these photochemical injuries include (1) activation of destructive oxidizing reactions induced by toxic free radical formation,[53,57] (2) inhibition of intracellular enzymes or biochemical systems or other metabolic damage which might be important to the maintenance of ionic homeostasis, calcium storage, transport functions, or cell respiration,[53,88] (3) liberation of a toxic photoproduct secondary to chronic bleaching from light exposure,[53] (4) injury of molecules of deoxyribonucleic acid (DNA) transcription and protein synthesis,[34] and (5) direct destructive action of light on the mitochondria in different retinal layers.[34]

The experimental work of these investigators has established a photochemical basis for the retinal damage induced by solar retinopathy. Retinal irradiance by higher levels of energy by short wavelengths in the visible spectrum (blue light hazard) and by lower levels of UV-A or the near ultraviolet radiation (320 to 400 nm) is the postulated principle mechanism for the photochemical damage seen in acute solar retinopathy. This belief is consistent with the fact that photon energy increases as the wavelength decreases. The direct damage of visual photo pigments or sensitive cones by low levels of radiation are not likely to be significant photochemical mechanisms for the acute manifestations of solar retinopathy.[64,73,89]

Since repeated subthreshold exposure to blue light can produce cumulative retinal injury, it is conceivable that solar radiation may be associated with a long-term or chronic form of solar retinopathy. Some investigators have associated this potential damage with the pathogenesis of age-related macular degeneration.[88-92] The concept that long-term repetitive exposure to solar radiation is a contributing factor to the development

of macular degenerative manifestations was first proposed more than 65 years ago.[90] Mainster,[89] Young,[91] and Ham and Mueller[92] have recently analyzed this issue suggesting that chronic, excessive light exposure could accelerate the aging process in the macula, by depigmentation of the RPE and other mechanisms, predisposing to progressive degeneration. Although converging lines of clinical and experimental research lend support to this concept, there is virtually no long-term human experience to prove that reversible, repetitive phototoxic damage in early life tends to retinal degeneration in later life. The aging retina may be an inevitable biologic process which is the result of numerous, complicated biochemical, environmental, and immunogenetic mechanisms, one of which is phototoxicity.

PHOTOBIOLOGICAL ANALYSIS

Numerous photobiological factors affect the extent and severity of retinal damage in solar retinopathy. These include the nature and degree of exposure to the light source, certain ocular characteristics, and related systemic and possible individual susceptibilities.

Exposure

The photochemical damage is strongly dependent on the intensity, duration, and spectrum of the exposure. Each of the reported cases denied even casual glances at the sun during their 2 to 4 hours of exposure. Some minimal retinal irradiance from direct or reflected light, however, is likely. A severe sunburn was described by three of the four cases, confirming the intensity of the solar radiation.

Refractive State

The emmetrope and the low hyperemmetrope are at greater risk of solar retinopathy because of the ocular refraction of transmitted light which is focused sharply on the macula. Theoretically, a low hyperope would be at greatest risk because the chromatic abberations of the eye would place the blue end of the visible spectrum within the retina. When light is separated into its components by the optical system of the eye, the shorter blue rays come to a focus closer to the crystalline lens than the longer red rays. Blue light is +0.87 D anterior to yellow light focused on the retina. It may be more than coincidence that hyperopia is also a risk factor for age-related macular degeneration.

Pupillary Size

The amount of radiant energy incident on the retina depends on the transparency of the ocular media and the pupillary diameter. In a histo-

pathologic study of the human fovea after sun gazing, Tso and LaPiana[27] described the most severe reaction in a patient whose pupil had been dilated to 4 mm throughout the experiment. Clarke and Behrendt,[37] in a study on photochemical damage to the retina in unanesthetized monkeys, noted a paradoxical dilation of the pupil when exposed to intense light. In these two studies, pupillary dilatation increased the risk of retinal damage from radiant energy. Since the luminosity curve for the pupillary light reflex closely matches the luminosity curve for vision, individuals gazing at an eclipse may not be protected by a small pupil. The same is true for an increase in exposure to light outside of the visible spectrum, such as UV-A or UV-B. Any activity that stimulates a sympathetic discharge such as anxiety or exercise, or any drug that induces a mydriatic effect may also predispose an individual to solar retinopathy. The opposite is true for the gradual reduction in pupillary size noted with aging, the so-called senile miosis, with certain disease states such as diabetes mellitus and with the relatively miotic pupil in the very young by reducing the transmittance of radiant energy to the retina.

Ocular Media

In the mammalian eye, light between 400 and 1400 nm can penetrate to the retina. Based on the classical determinations by Boettner and Wolter[80] and Boettner,[81] the cornea naturally absorbs light which is shorter than 300 nm[87] (Fig 5). Recent work on the transmission of the cornea has indicated that the cornea can transmit UV-B light as short as 290 nm. The lens helps to protect the retina from ultraviolet light beginning at the age of 20 with a linear increase in its absorption until it peaks at approximately age 30. It essentially shields the retina from 300 to 400 nm (Fig 3). There is a slight increase in transmission of UV-B by the lens in the range of 310 to 320 nm.[80,81] The lens also protects the retina from the blue light hazard as it takes on a yellowish hue with age. In a sense, UV absorption by the lens and the associated cataract formation are adaptive measures by the eye to prevent macular photochemical damage. Thus, the young and the aphakic eyes are at greatest risk of solar retinopathy.

Pigmentation

Some investigators believe that melanin pigmentation plays a protective role against light damage in the retina.[33,90,91] It is true that light colored irides is a risk factor for age related macular damage. Yet, convincing evidence relating the hypopigmented state with solar retinopathy is lacking. Further research is needed to determine whether melanin is a protective factor in phototoxic retinal damage.

Dominant Eye

Unilateral cases of solar retinopathy are not uncommon.[2,13,24,26] The involved unilateral eye is generally the right eye, presumably the dominant one.[1,13] It is believed that patients tend to squint one eye, the nondominant eye, when sunviewing, to reduce their photophobia. Similarly, bilateral cases are very often asymmetric, with the right eye being more severely affected.

Systemic Factors

Certain systemic drugs such as tetracycline, psoralin, and the hematoporphines are known to be associated with photosensitization, increasing the retina's risk of photochemical damage. Analgesics, may play a role as a risk factor for solar retinopathy by reducing an individual's discomfort from sunviewing. The person viewing an eclipse is not subject to pain, increasing the duration of exposure to solar radiation. Noell and associates[53] and Friedman and Kuwabara[56] have noted that the threshold for retinal damage is lowered when an animal's body temperature is elevated. The same is true for an increased oxygen tension.[91] Another critical factor that may predispose to retinal damage from radiant energy is the nutritional state. Malnutrition or deficiencies in numerous agents such as vitamin A, vitamin C, vitamin E, or catalase may increase outer segment photoreceptor damage from excessive peroxidation. Additional research is needed to clarify the role of antioxidants in light damage. It would also be important to study the effects of dietary factors which control the concentration of xanthophyll in the retina. Increasing xanthophyll precursors in the diet could potentially reduce the risks of the blue light hazard.

Host Susceptibilities

Retinal photoreceptor vulnerability to light is less in albinism and is greater in retinitis pigmentosa.[85] There is an increased susceptibility to light damage with age and with strains of animals.[84] The normal diurnal cycle of light and dark seems to be another factor which reduces light damage susceptability.[86] The macula already compromised by a diseased state or age may be prone to light damage.[91,93] It is also inviting to speculate that selection and adaptation play a role in ameliorating the mechanisms of cellular damage from photochemical reactions as they appear to do in the skin. A simple protective selective factor would be a hereditary tendency for a prominent brow ridge which would shield the eye from solar radiation.

GEOPHYSICAL ANALYSIS

Several geophysical factors may also affect retinal damage in solar reti-

nopathy. These factors principally relate to an increase in solar radiation emission or to an increase in its transmission to the earth's surface.

Increased Solar Emission

The most significant factor affecting solar emission is the annual variance in the earth-sun distance. This accounts for a difference of up to 6% with the highest levels in the northern winter. Changes in solar emission during the sun's cycle are even less significant with essentially no variation in wavelengths longer than 220 nm. Therefore, a significant increase in extraterrestrial solar radiation is not a likely causative factor for solar retinopathy. Only the presence of environmental areas of high surface reflectivity (sand, watery snow, glass, metal) could increase the net solar irradiance by the addition of reflected light to the eye.

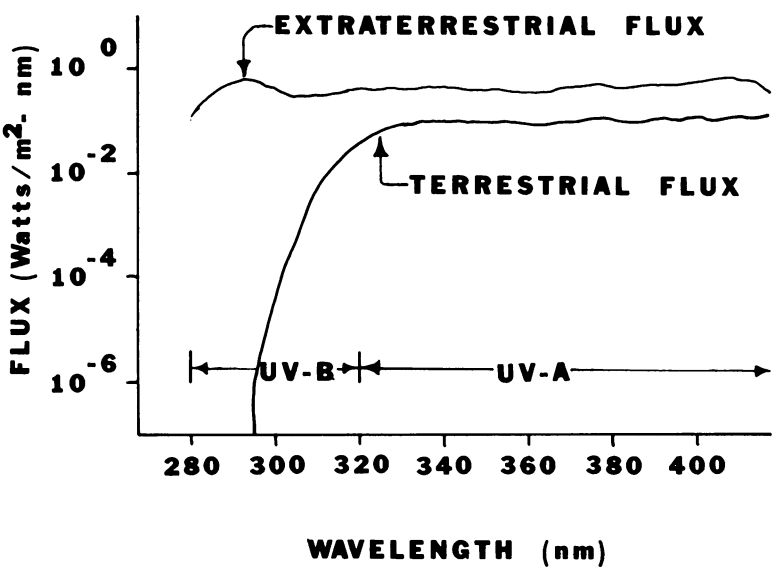
Increased Transmission

There are factors which may enhance or reduce solar radiation from reaching the eye. One of these is a high surface altitude. Perhaps the most important factor, however, is the earth's atmosphere. The numerous complicated processes which influence the absorption, scatter, and transmission of solar radiation by the atmosphere are incompletely understood. It is known, however, that the transmission of extraterrestrial flux to the earth's surface, declines as the solar zenith angle approaches 90 degrees, the horizon. The effect becomes most pronounced when the zenith angle is greater than 60 degrees, as the radiation must traverse a broader column of atmosphere. Essentially, the sun is most intense when directly overhead. It becomes yellow then orange later in the day as blue light and UV radiation are absorbed and scattered by an increasingly wide atmospheric column.

Attenuation of solar radiation also arises from scattering by atmospheric molecules, mainly O_2 and N_2. Some of the extraterrestrial solar radiation is actually back scattered into space. Clouds, dust, and moisture also play an important role in reducing the solar energy that reaches the retina. Thus, ideal geophysical conditions for producing cases of solar retinopathy occur on a warm day, at solar noon, at high altitudes, with a clear, cloudless, and a dust- and moisture-free blue sky.

These ideal geophysical conditions or risk factors for producing solar retinopathy have in the past related to solar radiation in the blue portion of the visible spectrum (blue light hazard) as well as the UV-A or near ultraviolet. The cases of solar retinopathy in this report introduce a new concept in the nature of photochemical damage to the eye in solar retinopathy, specifically the possible contributory role of UV-B light. In the geophysical analysis of the cases in this report, a significant drop in the

ozone layer was noted in the two involved geographic areas. On the first day, the ozone drop was recorded in the area of case 1. On the second day, the ozone drop had shifted to the site of cases 2 to 4. The flux of UV-B radiation at any given location is dominated by two factors: the total ozone content and the cloud cover. The reflectivity of clouds at UV-B wavelengths is similar to that at visible wavelengths so that an overcast sky will decrease UV-B dosages by a factor of two or more, depending on the cloud optical depth. The ozone minimum of March 28-29, 1986 was accompanied by clear skies thus permitting enhanced UV-B sunlight due to the reduced total ozone column to reach the ground. Latitudinal and seasonal variations in the atmosphere ozone affect the flux of solar UV radiation reaching the ground (Fig 6A). Absorption of UV radiation by the ozone layer is extremely wavelength-dependent, with a sharp decline in energy flux toward the shorter UV-B spectrum. This is the so-called "ozone cutoff" in which the flux on the earth's surface drops dramatically beginning in the region of 295 to 300 nm[82,93] (Fig 6B). This cutoff theoretically prevents any appreciable radiation with a wavelength of less than 290 nm from reaching the earth's surface. In their studies on the ocular effects of exposure in the ultraviolet radiation, Pitts et al[87] have found the action spectrum for the lens to begin at 295 nm extending to 325 nm. The



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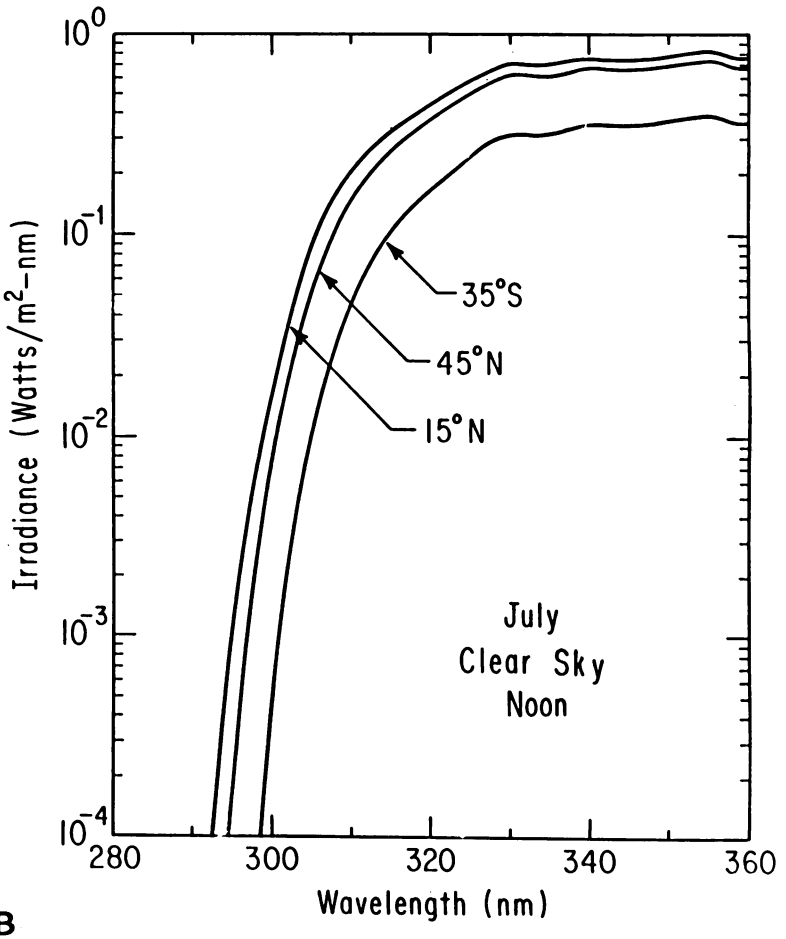

FIGURE 6
Effects of atmospheric ozone layer on ultraviolet radiation. A: Extraterrestrial and terrestrial ultraviolet radiation at various atmospheric ozone levels. B: Latitudinal variation in ultraviolet flux at earth's surface. (From Frederick JE: The ultraviolet radiation environment of the biosphere, in JG Titus (ed): *Effects of Changes in Stratospheric Ozone and Global Climate*. Vol I. Proceedings of International Conference on Health and Environmental Effects of Ozone Modification and Climate Control, 1986, pp 127-138. with permission.)

most effective range for producing lenticular damage was 295 to 315 nm with complete absorption of UV radiation by the cornea at 290 nm and below.[87] A very striking biophysical coincidence is manifested in the

ozone and corneal cutoffs of radiant energy. Both structures are opaque to wavelengths shorter than the 290 to 300 nm region. The ozone layer and the cornea provide nearly identical spectral shielding of the earth's surface and inner eye, respectively (Figs 5 and 6B).

Most people assume that very small amounts of UV-B radiation transmitted to the retina would not cause significant retinal damage, but this assumption is not based on experimental attempts to produce retinal lesions from this radiation in the intact monkey eye. Rather, the belief is based solely on the small percent transmittance of UV-B light by the ocular media. In unpublished experiments conducted by Pitts and Waxler for the United States Department of Human, Health and Human Services, moderate intensities of UV-B radiation produced significant damage to the retina of the phakic eye of the monkey. This retinal damage occurred even though less than 1% of the UV-B light penetrated to the retina. These investigators also suggest the higher photon energy characteristic of 300 nm radiation may be sufficient to produce retinal damage with a very low corneal dose, since the shorter UV-B photon has much greater energy than UV-A radiation. Their observations need to be reviewed and replicated before revising our basic concepts regarding UV-B exposure, transmissions by the ocular media and associated retinal damage. First, the transmittance of the ocular media in the 290 to 400 nm wavelength range needs to be accurately determined. It is also important to establish precisely the biological action spectrum for UV-B radiation since damage to the DNA molecule increases exponentially with wavelengths shorter than 320 nm.[82] This approaches the region where atmospheric ozone creates a major influence on solar UV-B flux reaching the earth's surface. These data are critical in understanding the adverse effects of solar radiation on the retina.

MULTIFACTORIAL PATHOGENESIS FOR SOLAR RETINOPATHY

The present study associates significant photobiologic and geophysical conditions as risk factors for solar retinopathy. It also introduces a new variable for consideration, an associated reduction in the atmospheric ozone layer and its consequences, an increase in the atmospheric transmission of UV-B radiation to the earth's surface and, possibly, low dose, high energy transmission through the ocular media to the retina. However, association does not necessarily indicate causation. As well, disorders of the macula, like any other biologically complex, multidimensional abnormality, are unlikely to be caused by a single factor. The particular geophysical disturbance in effect during the outbreak of these cases of

solar retinopathy is likely to represent one of several factors in the pathogenesis of solar retinopathy. There are also several other photobiologic and geophysical risk factors likely to be contributing to the pathogenesis of solar retinopathy in the cases in this report.

There is no question that the age of the involved patients (14 to 27 years) was significant, since the eye begins to develop lenticular changes which reduce the transmission of radiant energy and protect the fundus with age. The refractive state of the patients was also likely to be contributory. Three patients were emmetropic, and one was essentially emmetropic. Another predictable feature to the reported cases was the involvement of the dominant eye. In each of the three bilateral cases, the right eye, the dominant one, was more severely affected. The only unilateral case involved the left eye which was the patient's dominant eye. Exercise may have made minor contributions to the pathogenesis of the solar retinopathy with a possible increase in body temperature and pupillary dilation. Casual glances directly at the sun may have also played a role, but no patient admitted to even the minimal direct exposure known to be associated with solar retinopathy. Other known risk factors such as certain drugs that heighten phototoxic effects or dilate the pupil could not be implicated in the radiant energy damage to the retinas of these patients.

The geophysical factors that were likely to be related to the development of solar retinopathy in these reported cases included the markedly clear sky, exposure at solar noon, and the possible increase in UV-B radiation by the relatively low ozone layer. The authors postulate that a superimposition or cluster of unlikely events occurred to produce a very unusual phenomenon, solar retinopathy in a group of individuals who allegedly were not sungazing. It is likely, based on experimental evidence, that increased visible blue light on the region of 440 nm was the principle factor producing the photochemical retinal damage. Shorter wavelengths in the near UV-A were also likely contributors to the adverse effects on the retina. A more speculative but compelling concept, is a new variable in solar retinopathy, the possibility of UV-B damage from a small number of high energy photons reaching the retina because of a reduction in the atmospheric ozone layer.

Retinal damage by UV-B radiation is vastly under-researched. Most investigators believe that the cornea effectively filters out the highly energized UV-B photons. Additional research is needed to determine whether an increase in the UV-B mediated through the ozone drop could have played a significant role in the development of solar retinopathy in this reported series. If this exposure to UV-B proves to be significant enough to produce retinal damage, several important questions regarding

the ozone layer, UV-B transmission and solar retinopathy will need to be addressed. For example, why are there so few reports of solar retinopathy from other parts of the world where geophysical risk factors like those present in this series are more common? It is likely that inadequate knowledge or documentation may mask the true prevalence of the disorder in high risk developing countries such as those in the Caribbean. It is also true that mild photochemical injuries to the retina are extremely subtle with reference to the associated symptoms and clinical findings.[1,44,63] Furthermore, the visual recovery is relatively rapid and complete. Since most cases are mild, clinical reports are likely to involve only the most severe injuries.[89] Another possible explanation for the small number of reported cases from geophysically high risk areas may relate to individual variance in host susceptibility. Human selection or adaptation are known to exist for enzyme repair systems for a variety of environmental or acquired insults. The skin's adaptation to the sun is perhaps the most appropriate analogy.

A final consideration with respect to the ozone layer is its recorded drop of approximately 3% recorded by NASA scientists between 1978 and 1984. This observation is ominous for several reasons. A decay in this fragile blanket of gasses and the associated increased penetration of extraterrestrial UV radiation is a major personal health and environmental threat. Increased ultraviolet radiation is known (1) to increase the frequency of skin cancer, viral diseases and cataracts, (2) to suppress the body immune system, (3) to destroy plankton and algae which are fundamental elements in the marine food chain, (4) to suppress crops, and (5) to increase acid raid and smog. Most important of all, the same chemicals which are alleged to deplete the atmospheric ozone concentrations, chlorofluorocarbons, are major contributors to the impending Greenhouse effect which threatens to produce staggering health and global environmental and climatic changes which could significantly harm the earth's life support systems. This study adds the possibility of retinal damage to the devastating consequences of ozone depletion.

Modern epidemiological theory strongly emphasizes the concept of multifactorial etiology and multiplicity of response. Any of the potential risk factors may act independently of the others predisposing an individual to solar retinopathy. The influence of each contributing factor is likely to be subtle and complex. A direct and overriding connection between a given risk factor such as a reduced ozone layer and any other potential risk factor in solar retinopathy is not likely. The overall risk of solar retinopathy is not likely. The overall risk of solar retinopathy is understandably likely to be greater in individuals who possess two or more of the risk

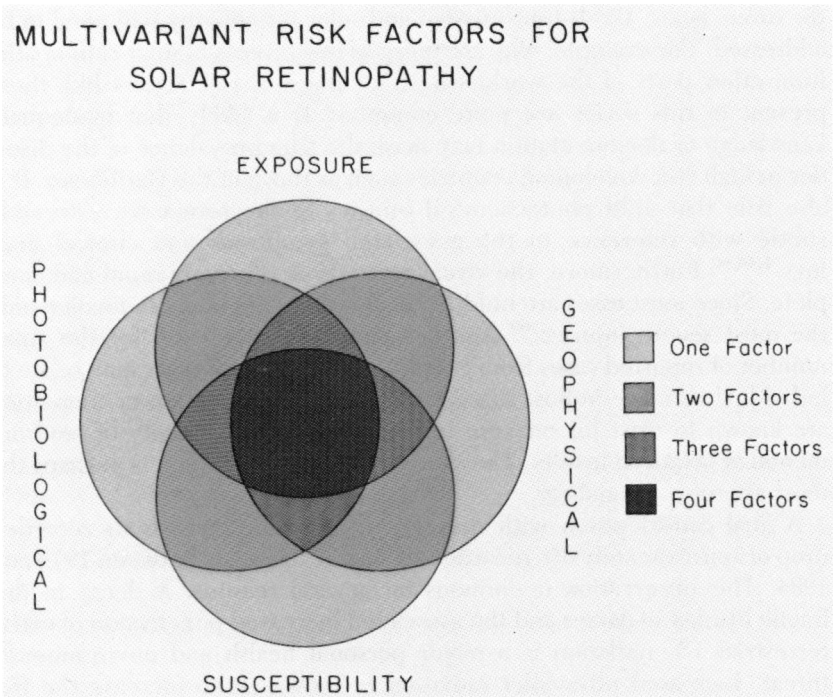

FIGURE 7
Multifactorial hypothesis for solar retinopathy.

factors than among those with only one or none (Fig 7). The multifactorial concept of disease also implies that a single risk factor such as a reduced ozone layer is neither a necessity nor a prerequisite for the development of solar retinopathy in a given patient. Consistent with a multiplicative model is the realization that a patient with solar retinopathy or any other complex medical disease may have none of its known risk factors.

In essence, a broad conceptual framework is needed to associate solar retinopathy with a reduced level of ozone or any other geophysical disturbance or disturbances. The independent and interactive influences of photobiological and geophysical factors associated with solar retinopathy must be identified for a complete understanding of its etiology and for a rational approach to its prevention. Meanwhile, this study does provide sufficient grounds to suggest that certain geophysical disturbances may play a role in predisposed patients in the development of solar retinopathy.

Since converging lines of clinical and experimental research conclude that the retina is vulnerable to radiation, particularly in the regions of the visible blue and ultraviolet, it is prudent for ophthalmologists to recommend to the general public the use of eye shading devices such as hats, visors, and in particular, protective lenses in a bright environment.[89,92-94] Hats and visors, like a prominant brow ridge, shield the cornea from solar radiation. They also influence the angle of radiation on the cornea, which leads considerable reflection of the incident radiant energy.

Another important way of protecting the eye from solar radiation is through the use of filtering lenses or sunglasses. Sophisticated, exquisitely designed psychophysical studies are needed to determine the longest wavelength protective filter that provides optimum safety without significantly affecting scotopic and photoptic vision.[89] The public should also be educated with respect to the purpose and nature of protective lenses. Several points in particular should be emphasized such as "darker is not necessarily better." A lens that filters out most of the visible light and transmits blue light and ultraviolet radiation may indeed be harmful. Such a lens not only transmits radiation which is photochemically most hazardous to the retina, it also inhibits the squint and pupillary light reflexes which normally reduce higher levels of exposure. The public should also be aware that the manufacturing and marketing of protective lenses are poorly controlled by regulatory agencies. Voluntary standards do exist for nonprescription sunglasses but there are no standards for prescription sunglasses. There is no way for a consumer to determine the spectral transmission of most protective lenses since they are seldom labeled and rarely verified by spectrophotometry. A variety of more stringent proposals are being considered by the American National Standards Institute (ANSI) and the Food and Drug Administration (FDA), but none has been enacted as yet.

In particular, very young children who are not aware of the dangers of intense light environments as well as aphakics or pseudophakics without UV filters in their implants and individuals with a family history of macular disease or existing macular degeneration should be urged to wear protective filters since they are at greatest risk of photochemical damage to the retina.[89,92,93] Until the ideal filter is established, it is intelligent to advise the public to use lenses which filter out wavelengths below 450 nm. This would eliminate the blue visible light and UV radiation which are potentially the most hazardous. It may also enhance image quality by reducing light scatter and chromatic aberrations and only minimally affect color balance.[92] This relatively simple and inexpensive safeguard of protective filters may not only reduce the risk of solar retinopathy, but may

also reduce the rate and frequency of cataract formation.[33,93] It may also reduce the chronic, cumulative photochemically mediated damage to visual photoreceptors by low levels of energy that may contribute to the pathogenesis of the aging macula, the leading cause of legal blindness in patients over the age of 60 years. The broader scope of solar retinopathy implied by this report must undoubtedly remain open to question until further evidence supports the concept that reduced ozone in the atmosphere predisposes to acute pathology and chronic photothermal damage related to the development of macular degeneration. The pathogenesis of acute solar retinopathy will likely be related to an inter-relationship between finely balanced components of complex photobiological and geophysical systems involving an individual's age, his ocular status, his degree of exposure, his intrinsic susceptibilities and a number of geophysical contingencies. The concept also offers a new approach to the prevention of solar retinopathy through identification of potential geophysical changes which may predispose certain individuals to the disorder and through measures that reduce exposure.

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DISCUSSION

DR THOMAS C. BURTON. An unusual occurrence of four cases of solar retinopathy in a 2-day period in late March of 1986 prompted the investigation into various risk factors, which might have potentiated this small epidemic. Other investigators have at least alluded to numerous related factors including young age, refractive state of the eye, individual susceptibility, ocular discomfort from bright light resulting in squinting, the sun's altitude, and atmospheric conditions.

The authors went to a remarkable extent to inquire of various governmental and aerospace agencies and laboratories to determine the climatic conditions, including the status of the ultraviolet protective barrier. They were able to establish that a 15% to 20% reduction in thicknes of the ozone layer and a substantial increase in the earth's irradiation with UV-B wavelengths concurred with the times when the reported cases were sunbathing. It seems to be of little consequence that all the individuals denied sungazing. Since they all had lesions pathognomonic of solar retinopathy, they can all be assumed to have been sungazing, a mistake in judgment that may require only 100 seconds to produce visual damage at noon on a clear day.

For a long period of time, it was assumed that solar retinopathy was thermally induced by longer visible wavelengths and the near infrared portion of the spectrum. It is now known that the temperature rise is insufficient to create a thermal burn, although a few degrees centigrade might enhance a photochemical injury.

Ham and associates believe that the shorter visible wavelengths around 441 nm are the most injurious, with the RPE cells principally affected and the photoreceptors secondarily affected. Lawwill has shown that the mitochondria of all layers of the retina from the retinal pigment epithelium to the nerve fiber layer are susceptible to damage by visible light. Again, Ham and co-workers showed that shorter wavelengths of 325 or 350 nm were six times more likely to damage the retina than visible light, and that photoreceptors seemed to be the primary site of injury.

The main question is whether the increased transmission through the atmosphere of wavelengths in the 300 to 325 nm range can be regarded as an additional risk factor in light damage to the retina.

The human lens provides a powerful barrier to the transmission of wavelengths shorter than 400 nm. With an absorption band centered at 360 nm, the lens permits an 8% transmission of 320 nm in children at age 5, and less than 0.1% by the age of 22. Whereas the sensitivity of the retina increases with shorter wavelengths, the number of such photons reaching the retina is drastically reduced by absorption in the lens. It seems most probable that solar retinopathy is caused by

short wavelengths in the visible spectrum, possibly with some thermal enhancement by longer wavelengths in the visible and near infrared spectrum.

Yet, there remain some disquieting elements. If the RPE is so easily damaged by visible wavelengths, why is there so little ophthalmoscopic or angiographic evidence of that in typical solar retinopathy. In addition, unpublished data suggests that less than 1% of incident UV-B light to the primate eye significantly damages the retina.

The authors are to be commended for an excellent review of the mechanisms of light damage to the retina, for helping us to think in more global terms in the causation and prevention of these peculiar injuries, and for contributing a challenging concept that small changes in flux of short wavelength, high energy photons, easily demonstrable with diminished thickness of the ozone layer, might contribute to retinal damage.

DR RICHARD L. LINDSTROM. I would like to make an anterior segment comment and also ask a question. The first one is, if the ozone layer which is like an artificial cornea was not there, did you check with your corneal colleagues to see if there was any keratitis. Were there patients, for example, that had keratitis from excess exposure to UVB? Second, an important question for the intraocular lens area. There is a Denver scientist who has studied eyes which have an ultraviolet absorbing lens implant in one eye and no ultraviolet absorbing lens in the other. He has shown that while you can't measure any change in total visual function, if they isolate the blue cones and do blue cone ERG they can show significant pathology, strictly in the blue cone ERG. Did you look at keratitis or blue cone ERG's?

DR J. DONALD M. GASS. First, I want to congratulate Doctor Yannuzzi and coworkers for their very interesting paper, and second, I wish to make a comment concerning something the previous discussor said. I do not believe that the changes seen in the macula after sun gazing are pathognomonic. There are at least two other instances where identical yellow foveal lesions occur and are followed within a matter of days or weeks thereafter, by a tiny lamellar facet in the fovea. The first is after blunt trauma to the eye. The second is in patients, usually older individuals who have a macular hole in one eye, and who present with acute visual complaints in the second eye. They have the little yellow lesion that looks almost identical to that in acute solar retinopathy. In a small percentage of these patients, spontaneous separation of the vitreous from the fovea will occur, and will pull a small plug out of the inner retinal surface, leaving a tiny reddish color foveolar pit. This pit is identical, with one exception, to that seen in the later stages of solar retinopathy. The exception is that biomicroscopically a small piece of the inner retina (an operculum) is visible on the back surface of the vitreous immediately in front of the foveolar pit.

DR HAROLD F. SPALTER. The scientists were so helpful to you, I was wondering if your observations have been transmitted to them at McMurdo Sound where there

is 24 hours a day of sunlight and a big window in the ozone layer. Is there, to your knowledge, any assistance being rendered, suggestions being made, or perhaps a prospective study being carried out to protect their eyes for they are, if your theory is correct, at maximum risk.

DR EDWARD JAEGER. Two questions for Doctor Yannuzzi. One, there seems to be a discrepancy between the number of sun worshippers and the number of cases reported. Perhaps we are missing this entity, but having had two daughters pass through the adolescent years of intense sun worshipping, there would appear to be no lack of exposure to solar rays, at least in this group. Are we under diagnosing this, clinically? Secondly, in the Philadelphia area a number of opticians have seized upon this subject and put on an advertising campaign in which, for a certain price, you can have your glasses "filter-treated," the ultraviolet filtered out. This can put us at risk because increasingly now I am being asked, "Doctor should I have my glasses treated?" I would estimate one call per day now. The expense ranges in the area of $20 to $25, so it is not inconsequential. Do you advise your patients to wear ultraviolet filtered lenses?

DR HUNTER L. LITTLE. I would like to congratulate Doctor Yannuzzi on a fascinating paper. It is most fitting that this paper be presented in San Diego. After completing my residency in Baltimore, I spent 2 years in the Navy stationed at the US Naval Hospital in San Diego where we saw similar cases as reported today. Doctor Melvin Kerr (who was one of our residents at the Naval Hospital) and I wrote a paper published in 1967 *Archives of Ophthalmology* on foveo-macular-retinitis as a result of our experience in having 16 cases admitted to the US Naval Hospital with this very same syndrome. We wrote the paper to distinguish this syndrome from central serous retinopathy since it had first been described as idiopathic flat detachment of the macula in a report written by a number of well known ophthalmologists serving in the Navy during WWII. These included Michael Hogan, David Herrington, Warren Wilson, and others. Their concern (as was ours in 1965) was the large number of young men with this syndrome.

The syndrome is characterized by a yellow spot in the fovea with visual loss and central scotoma both of greater degree than expected from the size of the yellow spot. The yellow spot disappears within 1 week leaving an apparent tiny macular hole. The clinical appearance and the absence of proliferation of the retinal pigment epithelium do not seem typical of a photocoagulation burn. Furthermore, all denied sun gazing. When I presented the material at the Wilmer meeting in 1966, Doctor Maumenee stated emphatically that the condition was solar retinopathy and that the naval and marine recruits had obviously gazed at the sun to obtain medical discharge.

The etiology of this syndrome, referred to as foveo-macular retinitis, remains undetermined; however, Doctor Yannuzzi rekindles interest in this perplexing problem. Indeed, if ultraviolet radiation is the causative factor, everyone subjected to prolonged exposure of sunlight should wear appropriate protection.

DR GEORGE L. SPAETH. This is a fascinating paper. It is a good idea, in my opinion, to believe what the patient is saying.

I have a question for Doctor Yannuzzi. In some patients with far-advanced glaucoma fundus photographs (simple ordinary fundus photography) causes a decrease in vision. The patients become symptomatic and they have worsening of their macular function. Have you looked at patients who already have macular disease to see if low light levels make them worse?

DR LAWRENCE A. YANNUZZI. To answer Doctor Lindstrom, our patients had no evidence of keratitis at the time of initial examination. David Sliney is currently interested in calculating the threshold for photokeratitis. He's aware that very low irradiances of ultraviolet radiation, particularly the shorter wavelengths, can cause erythema of the skin and photokeratitis. Curiously, individuals can readily be sunburned at a beach, but they seldom experience photokeratitis even though the calculated dose of direct UV-B irradiance necessary to elicit photokeratitis is less than that which causes erythema of the skin. His theory is that shielding by the brow ridge, corneal reflections, and partial lid closure greatly limit the exposure of the cornea and lens. Focalization of the corneal dose through the optical system of the eye to the macula, is the most likely explanation for the photochemical damage observed in the posterior segment of the eye.

Our patients unfortunately did not have electroretinal physiologic testing, but I would not be surprised if they did have blue cone specific abnormalities. This observation would be consistent with the experimental work in primates done by Harwerth and Sperling. With respect to UV protective lenses, it is important to keep in mind that there is really no standardized regulatory division of our government which carefully monitors their production and quality assurance. They are generally not labeled; their spectral transmittance is virtually unknown. Hopefully, the ANSI and the FDA will take a more active role in protecting the public in this regard.

I would most definitely agree with Doctor Gass in that macular trauma and the development of an idiopathic or so-called senile macular hole may indeed mimic the clinical appearance of solar retinopathy.

Yes, Doctor Spalter, we are trying our best to communicate our findings to the various scientific agencies which were so helpful in assisting us in the geophysical analysis of these cases. They are aware of the risks, and several physiologists and physicists are working in a multidisciplinary framework to study the adverse effects of solar radiation.

In response to Doctor Jaeger, yes, I do believe that we are underdiagnosing this condition. The symptoms can be very minimal and transient, and the manifestations virtually undetectable. This has been particularly evident in cases we have seen recently where the dominant eye has more significant visual change and macular derangement; whereas the nondominant eye is less affected symptomatically as well as clinically. Of major concern is the potential for a cumulative effect known to be associated with photochemical damage. As ophthalmologists, we must answer the question "does repetitive, subclinical damage ultimately

contribute to the degeneration we commonly see in the aging eye?"

With respect to your second question, I do not think it's a big price to pay for all of us to wear and to recommend filtered lenses. More work has to be done to determine just how much of the visible spectrum needs to be filtered out, and this is discussed more extensively in the manuscript. Certainly, there is a rationale for UV 450 protective filters in aphakes, in pseudophakes, in young people and in individuals with pre-existing macular disease or a history of macular disease, and in patients exposed to bright environments, including ophthalmologists.

Thank you, Doctor Little for your comments. In the preparation of our manuscript, I did reread your paper very carefully. It was indeed a very careful analysis of the problem and a comprehensive documentation of the manifestations. I have no doubt that your reference to foveomacular retinitis is indeed solar retinopathy as we know it today.

Finally I would like to thank Doctor Spaeth for his kind remarks. I am not aware of any studies which have documented reduced vision in glaucoma patients following fundus photographs. Light exposure during angiography represents a potential hazard to the retina relative to the spectral content of the light, the retinal irradiance, the exposure time, and the threshold for cellular damage. Undeniably, the majority of light exposure during fluorescein angiography is in the 460 to 500 nm or blue range where retinal damage is produced by low levels of irradiance. However, the potential hazard of fundus photography appears to be greater than that of fluorescein angiography because of its higher irradiance. The increased irradiance of fundus photography outweighs the wavelength dependency factor associated with fluorescein angiography. The recognition of this hazard has led retinal specialists and photographers to use faster color film which requires lower levels of irradiance.