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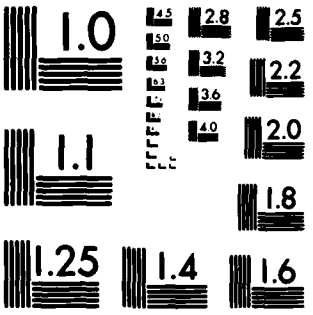

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W.T. HAM, Jr.

ANNUAL REPORT

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Annual Report

DADA17-72-C-2177 (1 Sep 81-15 Mar 82)
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August 1983

William T. Ham, Jr., Ph.D.

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1. Abstract

Repetitive exposures over a period of months to GaAs laser radiation under "worst" viewing conditions, i.e. parallel beam entering the eye, have disclosed no injurious effects. Both the MILES prototype laser and another laser operating at 830 nm were tested. The conclusion is that the MILES GaAs laser does not present an ocular hazard to the soldier in the field. Other experiments with more powerful GaAs lasers have shown that it requires 6 to 8 mW entering the monkey eye for periods of time ranging from 400s to 3000s to produce a minimal retinal lesion. This assumes that the eye remains fixed on the source. Argon/krypton 647 nm light is entirely equivalent to HeNe 633 nm light for retinal exposure times out to 1000s. Beyond 1000s, the 633 nm light requires significantly lower radiant exposures than 647 nm light to produce a lesion for reasons which are not understood. The radiant exposures for 40 μs pulses of 647 nm light at pulse repetition frequencies of 100 and 200 Hz required to produce a minimal lesion in the macaque retina have been determined for exposure times ranging from 1s to 3000s. A trained rhesus monkey has received 729 daily exposures of 1000s duration to the spectral band 330-420 nm at a corneal irradiance of 5 mW. cm^{-2} over a period of 3 years. Estimated radiant exposure to the anterior surface of the lens is greater than 2600 $J.cm^{-2}$. Both the exposed eye and the control eye appear normal to biomicroscopic examination. Another trained animal with pupils dilated to >8mm in diameter has received 131 similar daily exposures over a period of 8 months for an estimated exposure to the anterior surface of the lens of 472 $J.cm^{-2}$. Biomicroscopic examination discloses no anomalies in either the exposed or the control eye. Another trained monkey has replaced the one who died in June 1981. The retina of this animal has received 371 daily exposures of 1000s each to the spectral band 330-490 nm for an estimated radiant exposure of about 3000 $J.cm^{-2}$. Neither fluorescein angiography or funduscopic examination have detected any anomalies in the retinae and the lenses in both eyes appear normal. Exposures of another trained monkey with lens removed in exposed eye began in October 1981. The spectral bandwidth 330-420 nm produced 470 μW of power for a 3 cm diameter beam at the cornea. Calculations estimate a retinal exposure of about 1 $J.cm^{-2}$ for each daily exposure of 1000s. This animal began to show evidence of retinal depigmenta-

tion and edema 4 months after exposures began. By March 1983 after 316 daily exposures, there were large lesions in the superior and temporal macula and the exposure regime was stopped. The eyes will be processed for both histological and ultrastructural analysis. Evidently, the aphakic monkey retina is extremely sensitive to repetitive small doses of near UV radiation. Additional histologic data has been obtained on an aphakic monkey whose retina was exposed to 10 $J.cm^{-2}$ of 350 nm radiation. The overall histologic response to near UV in this aphakic eye was very similar to previous findings for blue light lesions except that direct damage to the neural retina, particularly cones, was more pronounced. Monkeys breathing oxygen during blue light exposure show a much lower threshold for retinal damage. Steroids injected intravenously one hour before exposure to blue light seem to afford some protection from retinal damage. Beta-carotene as a protective agent against oxygen and light damage is under investigation.

2. Investigation of Ocular Hazards of GaAs Radiation.

a. Repetitive exposures to Miles Prototype GaAs laser.

The trained animal selected for repetitive exposures to this laser had a long history of visual testing, beginning as far back as 1977 (1). Accurate baselines were available on visual acuity, spectral sensitivity and response time when the animal received 81 exposures, each 1000s in duration, during a 116 day interval in the fall of 1981. Exposure parameters were as follows: 30 nm bandwidth peaked at 910 nm, ellipsoidal beam (2 cm x 1.5 cm) at cornea, average power at cornea 880 μW, pupil diameter approximately 5 mm. Since the beam divergence of the MILES prototype laser was very small, it is estimated that spot size on the retina was less than 50 μm in diameter.

Baselines on visual acuity, spectral sensitivity and response time were kept on a daily basis, 5 days per week, during the exposures and for 3 months after exposures ceased. Since March 1982, similar baselines have been kept on a weekly basis (1 testing session per week) up to the present. No significant changes in visual acuity, spectral sensitivity or response time in either the control eye or the exposed eye have been noted since the eye was exposed in Sept. - Dec. 1981. These exposures were under "worst case" conditions while the animal was performing visual tests with Landolt rings. If extrapolation from rhesus monkey to man is assummed to be valid, this experiment provides convincing evidence that radiation from the MILES prototype laser is not an ocular hazard.

b. Other experiments with GaAs lasers.

Further evidence supporting the MILES data comes from an experiment in which another rhesus monkey was trained to press a lever for food when a visual stimulus appeared. One eye was irradiated with a GaAs laser while the unexposed eye served as control. This laser operated at 830 nm and was modulated at 22 MHz. A plano-convex lens provided a beam 2 cm in diameter at the cornea with an average power of 330 μW.

Over a six-month period, this animal underwent 102 exposures (1000 s daily, 5 days per week). Funduscopic examination during and after the exposures did not detect any differences between the foveas of the exposed and control eyes. Before this animal was sacrificed for histological examination, the unexposed or control eye was exposed to a more powerful GaAs laser operating at 830 nm with 22 MHz modulation (45 ns pulses). Average power at 22 MHz modulation was about 12 mW. Until this laser became available, we had been unable to produce a funduscopically visible lesion in the monkey retina with GaAs lasers.

The beam of radiation emitted by this laser was modified optically to produce a nearly parallel beam, 5 mm in diameter, that could be introduced into the eye of the anesthetized animal with pupil dilated to more than 8 mm. Power at the cornea was 8.0 mW. Ten 1500 s exposures were placed in the paramacular area of the control eye. These were spaced at 4 days, 2 days and 1 hour before sacrifice to provide a time scale for histological examination. Each of these 10 exposures produced a very small lesion (estimated to be less than 50 μm in diameter) that was funduscopically visible. Unfortunately, none of these lesions could be located in the tissue samples. While this was disappointing, it implies that the damage must have been minimal to escape histological detection. Histological examination of the foveas of the control and exposed eyes disclosed no essential differences. There was no histological evidence of damage to the fovea of the eye that received 102 exposures, each 1000 s in duration.

In an attempt to define a GaAs threshold for retinal damage, a limited number of exposures with this laser were made in other animals (cynomolgus monkeys) under anesthesia with dilated pupils. The criterion was the appearance of a funduscopically visible lesion at 24 hours postexposure. The maximum power for a nearly parallel cw beam, 5 mm in diameter, entering the eye was 8.45 mW. An exposure time of 400 s resulted in a minimal lesion observable at 24 hours after exposure. For 1000 and 3000 s exposures the power entering the eye was reduced to 7.2 and 6.1 mW respectively. The modulated beam,

22 MHz (45 ns pulses) required 900 s for a minimal lesion with 8.4 mW average power entering the eye. Exposures of 500, 700 and 800 s did not produce an observable lesion. For a 3000 s exposure, an average power of 7.5 mW was required to produce a lesion. There was some difference in retinal sensitivity between the cw and the modulated beam at the shorter exposure times but when these were extended to 3000 s the difference disappeared. These contrasting results are probably caused by experimental errors arising from the difficulties encountered when trying to detect very small minimal lesions.

The experiments outlined in a. and b. were performed under "worst" viewing conditions where the radiation was optically altered so that a major portion of the energy entered the eye, usually through an abnormally large pupil diameter. It requires from 6 to 8 mW entering the eye and focussed constantly on a very small area for exposure times ranging from 400 s to 3000 s to produce a minimal lesion in the macaque retina. It is difficult to imagine how a soldier could get such an exposure from the MILES laser system. Even if 8 mW entered the eye, the very small spot size on the fovea would never remain in the same location for an appreciable time.

References:

1. Clarke, A.M. Blue light exposure and long-term deficits in visual function. SPIE, 229, 51-54 (1980).

3. Investigation of Ocular Hazards from Krypton Red (647 nm) and HeNe (633 nm) Lasers.

Concern about the ocular safety of He-Ne scanning devices and the hazards of long-term chronic exposure prompted a review of the He-Ne data reported in 1976 (1). The large He-Ne laser is rf excited (≈ 1 MHz) rather than d.c. excited as is the argon/krypton laser. Operating in the TEM_{oo} cw mode, the previous radiant exposure of 5400 J. cm^{-2} for a 1000 s exposure was confirmed for the He-Ne 633 nm wavelength. A comparison of thresholds for He-Ne 633 nm argon/krypton 647 nm wavelengths was made, using 500 μm spot size and exposure times of 1,16,100,1000,3000 and 10,000 s. The data plotted in Figure 1 represent averages of 8 eyes in 4 monkeys; corneal power entering the eye in mW is plotted on the ordinate vs exposure time in s on the abscissa. Out to 1000 s there is no significant difference in thresholds for the two wavelengths but for 3000 and 10,000 s exposures there is a significant difference, so that for very long exposure times the 633 nm wavelength produces a minimal lesion at a significantly lower corneal power level, P_c, than the 647 nm wavelength. This cannot be explained by a difference of only 14 nm in wavelength. The 1 MHz ripple in the plasma of the He-Ne tube is the only difference between the two wavelengths and this may be another example of the enigma of modulated sources vs cw sources. In any event, these data show that the argon/krypton laser can be used as a substitute for He-Ne for exposures times out to 1000 s. This is important because the argon/krypton laser can be acoustically modulated at any frequency out to 20 MHz and we plan to make a definitive study of 647 nm vs 488 nm for several modulation frequencies.

Unfortunately, the argon/krypton laser broke down. It has been shipped to California for repairs. This has interrupted the study of threshold retinal injury as a function of modulation for the wavelengths 647 nm and 488 nm. Prior to breakdown, the radiant exposure for a minimal lesion was investigated in 4 monkeys (8 eyes) for 100 and 200 pulses per s at wavelength 647 nm, using the optically unaltered

laser beam that passed through holes in a rotating disc driven by an electric motor at 3000 rpm. The pulses were 40 μs in duration. With 2 holes placed 180° apart, the rotating disc produced 100 pulses per s; with 4 holes 90° apart 200 pulses per s were produced. In Table 1, the results are presented. Radiant exposures in J. cm^{-2} are calculated on the assumption that the laser beam produced 25 μm lesions to the 1/e levels of the Gaussian beam. The maximum irradiance at the retina, E_o in W. cm^{-2} is calculated from the formula

$$E_o = P_c T / 2\pi\sigma^2$$

where P_c is the power entering the eye, T is transmission through the ocular media (T=.93 for 647 nm) and σ is the parameter in the Gaussian distribution.

Table 1

Radiant Exposure in $J.cm^{-2}$ for a minimal lesion in monkey retina for 40 μs pulses at a frequency of 100 pulses per s of 647 nm light. Exposure times are 1,10,100,1000 and 3000 s.

Exposure time in s	Number of pulses	P_c in W	Rad. Exposure $J.cm^{-2}$
1	100	0.14	106
10	1,000	.080	606
100	10,000	.029	2,178
1,000	100,000	.014	10,607
3,000	300,000	.00675	15,342
Same as above for a frequency of 200 pulses per s			
1	200	.082	124
10	2,000	.0595	902
100	20,000	.041	6,213
1,000	200,000	.012	18,183
3,000	600,000	.00645	29,320

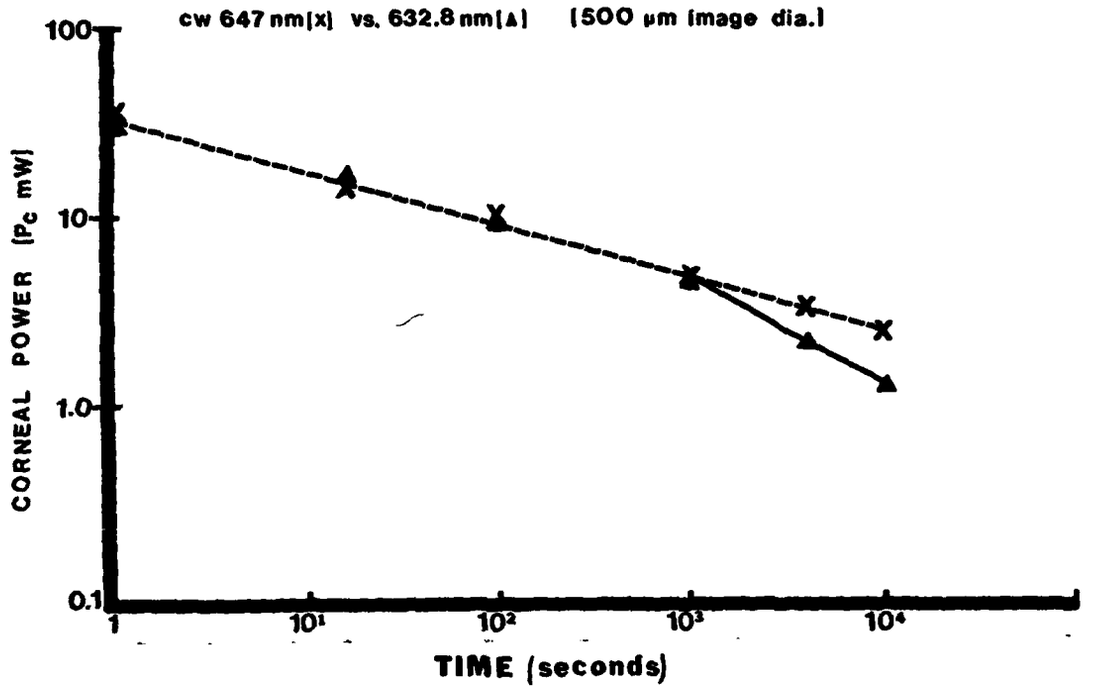

Figure 1

Corneal power entering the eye in mW needed to produce a minimal retinal lesion is plotted on the ordinate vs exposure time in seconds on the abcissa.

When the argon/krypton laser is repaired, future plans are to repeat the above experiments for the 488 nm line and then to extend our pulses per sec to 1000;10,000;100,000;1 MHz;10 MHz and 20 MHz for both wavelengths (647 nm vs 488 nm) using the acoustic modulator to produce the pulses. Pulse width using the modulator will vary from 1 ms pulses at a frequency of 1000 to 50 ns pulses for a frequency of 20 MHz. Such data should be interesting as a test of both wavelength and frequency modulation and may help to explain the differences between cw and modulated light threshold for thermal vs photochemical injury.

References:

1. Ham, W.T.Jr., et al. Retinal sensitivity to damage from short wavelength light. Nature 260, 153-155 (1976).

4. Long-term repetitive exposures of trained monkeys to near UV radiation and short wavelength light.

These experiments began as far back as 1979 and were designed to study the long-term effects of near UV on the lens and the effects of blue light on the retina at levels of irradiation corresponding roughly to the summer outdoor environment. At the present time, 4 trained animals are undergoing daily exposures of 1000 s, 5 days per week. The current status of each animal will be discussed in some detail.

a. Cataract.

This experiment was designed to test the hypothesis that small daily exposures to near UV radiation accelerate aging effects in the lens, leading eventually to senile cataract. Early lens changes were to be detected by fluorescence measurements in the irradiated and control eye as well as examination by the biomicroscope. The irradiated eye receives 5 $mW.cm^{-2}$ of near UV in the waveband 330-420 nm as provided by the 2500 W xenon lamp with quartz optics. Pupillary diameter during exposure is about 3 mm. Less than 1 % of this radiation

reaches the retina because of absorption in the ocular media. Exposures began on February 20, 1980 and have continued up to the present with few interruptions. As of March 7, 1983, this monkey had accumulated over 729 exposures with an estimated radiant exposure to the anterior surface of the lens of 2632 $J.cm^{-2}$. Both lenses and retinae have been examined periodically with the slit lamp and fundus camera. No differences between the control and exposed eye have been detected. Unfortunately, the instrument to measure fluorescence in the lens has not been developed to the stage where lens changes between control and exposed eye can be detected. Recent improvements in the integrating sphere and detector unit of this instrument are promising, so that it may still be possible to detect early changes.

The results to date of this experiment have led to the hypothesis that perhaps the iris protects the vulnerable equatorial region of the lens epithelium from near UV radiation. Under the condition of the exposure (cone of UV with small angle of divergence impinging on cornea) only scattered photons could reach the equatorial region.

b. Cataract.

To test the hypothesis that the iris protects the lens from near UV, another animal was trained and exposed under identical conditions except that both pupils were dilated with atropine, topically applied. Both pupillary diameters were greater than 8 mm during exposures that began on August 12, 1982. Up to March 7, 1983, over 131 exposures have accumulated for an estimated radiant exposure to the anterior surface of the lens of 472 $J.cm^{-2}$. As yet, there are no detectable differences between the two lenses by biomicroscopic examination.

c. Blue light effects on retina.

The animal that died on June 5, 1981 as noted in the last annual progress report was replaced on August 10, 1981 by another trained monkey. Exposure parameters are identical: 5 $mW.cm^{-2}$ corneal irradiance, spectral band 330-490 nm, provided by 2500 W xenon lamp with quartz optics and suitable filters, beam diameter on retina approximately

1.2 mm for an estimated radiant exposure of 8 $J.cm^{-2}$ per daily performance. In this experiment, 27.2% of the energy in the spectral band 330-490 nm reaches the retina as blue light. The lens absorbs most of the near UV and an appreciable proportion of the blue light. Thus, both tissues are at risk, the retina to blue light, the lens to a combination of blue light plus near UV. By March 7, 1983, 371 exposures were accumulated for an estimated 2968 $J.cm^{-2}$ to the retina on the assumption that the eye is fixed. In actuality, the animal is constantly moving his eyes, so that the 1.2 mm spot moves all over the macular area but probably the fovea gets the most exposure. Periodic examinations with fundus photography have detected no startling changes in the exposed retina or lens. Fluorescein angiography also was normal in both exposed and control eyes. There is a small but faint patch of depigmentation in the temporal macular and the animal has mild edema in the unexposed retina. Exposures will be continued until there is definite evidence of injury.

d. Repetitive near UV effects on retina of aphakic monkey.

Exposure of an aphakic monkey to the spectral band 330-420 nm began on October 27, 1981. The xenon lamp with quartz optics was adapted to give 470 μW of power to the exposed eye with a beam diameter at the cornea of 3 cm. The divergence of the beam was adjusted to produce a spot size on the retina of 1.2 mm, though this is only approximate since the aphakic eye has no accommodation. The pupil diameter of the aphakic eye is 6 mm. Calculations assuming a transmittance of 0.65 as calculated from Maher's data (1) predict a retinal irradiance of 1.07 $mW.cm^{-2}$. For a 1000 s exposure the estimated radiant exposure is 1.07 $J.cm^{-2}$. This is based, of course, on the eye remaining fixed on the source, an obviously incorrect assumption. Nevertheless, this experiment simulates roughly the magnitude of exposure to be expected on a bright sunny day. The experiment was stopped on March 8, 1983 after 316 exposures for an estimated 338 $J.cm^{-2}$ to the retina. Fluorescein angiography performed back in February 1982 had shown multiple focal areas of retinal pigment epithelium (RPE) depigmentation in the superior macula. Now funduscopic examination of

the exposed eye disclosed a large lesion in the superior paramacula where an edematous area had been observed previously and another large lesion in the temporal macula. There were numerous small depigmented areas in the superior macular, what looked like a retinal hole in the periphery at about ten o'clock, and a small retinal hemorrhage at about 5 o'clock in the periphery. The unexposed eye appeared perfectly normal. This animal is being sacrificed for histological and ultrastructural examination. Results will be reported in the next quarterly progress report.

This experiment has demonstrated that the retina of the rhesus monkey is extremely vulnerable to repetitive small exposures of near UV radiation. It is estimated that the retina of a human aphake would be exposed to about 10 $\mu W. cm^{-2}$ of near UV radiation on a clear sunny day between noon and 2 p.m. During this 2 hour period, a large area of the retina would get a radiant exposure of 72 m $J.cm^{-2}$. In 14 days, this would accumulate to $\simeq$ 1 $J.cm^{-2}$, assuming that radiant exposures were completely additive (no repair between exposures). This is well below what the aphakic monkey received in this experiment during a similar interval according to calculation but this calculation assumes that the animal was fixed on the near UV source, whereas the actual radiant exposure to any specific area must have been much lower. In the human case, the large area irradiated by the scattered near UV entering the eye would assure that some of this area was being irradiated continuously. There is good reason to believe that human aphakes need protection from near UV radiation.

References:

1. Maher, E.F. Transmission and absorption coefficients for ocular media of the rhesus monkey. Rep. SAM-TR-78-32, USAF School of Aerospace Medicine, Brooks AFB, TX 78235 (1978).

5. Effects of Near UV Radiation on the Monkey Retina.

The investigation of retinal sensitivity to near UV radiation in the aphakic monkey has been completed, including histological and

ultrastructural studies of the lesion. The data reported in the last annual progress report, pages 5-19, was published in the American Journal of Ophthalmology in the March 1982 issue. Reprints are enclosed.

To obtain more histological and ultrastructural data, another aphakic monkey was exposed to roughly twice the threshold radiant exposure at 350 nm, i.e. 10.3 $J.cm^{-2}$. Exposures were scheduled to provide lesions at 10,6 and 2 days and 1 hour before sacrifice. Retinal spot size was 1.2 mm, exposure durations were 1000 s, and all exposure sites were paramacula except one centered on the foveola at 1 hour before sacrifice. This latter lesion was characterized histologically at one hour by extensive cone damage as manifested by pyknosis and dense ellipsoids. The same was noted for the paramacular at one hour postexposure. Lesions in the paramacula at 2 days postexposure were conspicuous for dense cone ellipsoids. Dense cone cells were not found in the control samples. Numerous macrophages (and/or migrating RPE cells) were present in the subretinal space, especially at the level of photoreceptor inner segments. The RPE responded like a typical blue light lesion at 2 days postexposure. The normal apical distribution of melanin granules was disrupted by clumping and by rounding of RPE cells. There was mild inflammatory response in the choroid immediately below the choriocapillaris. Unfortunately, the 6 day tissue samples had a completely detached and missing neural retina. In the 10 day paramacular lesions, there was loss of cone photoreceptors, degeneration of photoreceptor outer segments, some debris and a few macrophages in the outer plexiform layer and even a few pyknotic nuclei in the inner nuclear layer. The RPE was single layered and depigmented with closely spaced nuclei. Presumably, proliferation and regeneration had preceded this stage. The overall, histological response to near UV radiation in this aphakic eye was very similar to previous findings for blue light lesions except that direct damage to the neural retina, especially cones, was more pronounced. Ultrastructural data on this eye is being processed.

6. Basic Mechanisms Leading to Photochemical Damage in the Retina During Blue Light Exposure.

Extended exposure (100-1000 s) of the macaque retina to short wavelength light (400-500 nm) at irradiances too low to raise the temperature by an appreciable amount, induces a photochemical type of lesion. The basic mechanisms leading to this type of damage are unknown but there is good reason to suspect that free radicals and excited species generated by the combination of light and oxygen play a role. The mammalian retina is unique among body tissues because it is the only tissue where light is focussed on a group of cells that is highly oxygenated. The choroid is structured so that a dense matrix of small blood vessels comprising a large surface area, the choriocapillaris, is situated immediately adjacent to the single layer of retinal pigment epithelium (RPE) cells and the outer segments (OS) of the photoreceptor cells.

The experiments reported here were begun in January 1983 and are in progress as of the 15th of March. They represent a first attempt to unravel some of the intricacies involved in photochemical damage to the retina.

a. Effects of oxygenation during blue light exposure.

Since oxygen is a notorious "bad actor" in radiation damage, the effects of a high oxygen tension in the arterial circulation on the threshold for the production of a blue light lesion was a primary objective of this research. The combination of light and oxygen can generate free radicals and sensitized or excited molecules, especially the toxic quartet of superoxide, hydrogen peroxide, hydroxyl radical and singlet oxygen (1,2). Protection against these toxic molecules is afforded by a number of substances, e.g. superoxide dismutase, catalase, glutathione peroxidase, vitamins E and C, steroids, carotenes, etc. (3,4). Future plans call for a study of the protective effect of these substances.

Monkeys under deep anesthesia are ventilated by one of several previously prepared tank ratios of oxygen/nitrogen . Blood samples

from the femoral artery are taken before and after 30 minutes of breathing mixture and analyzed for Po_2, CO_2, HCO_3, and pH. Rectal temperature is monitored continuously. A thermal blanket maintains the temperature of the animal at a reasonably constant level. Immediately following the 30 minute breathing spell, the retina is irradiated for 100 s at several different power levels. Funduscopic examination and photography at 24 and 48 hours postexposure establishes the threshold radiant exposure in $J.cm^{-2}$ by interpolation. Usually when 4 exposures are given at 4 different power levels, one or two of the exposures fails to appear as a funduscopically visible lesion. The retinal exposures are made by the long established technique in this laboratory of using the 2500 W xenon lamp with quartz optics and suitable filters to produce the bandwidth 435-445 nm peaked at 440 nm.

Enough progress has been made to demonstrate that oxygenation has a profound effect on the radiant exposure needed to produce a minimal blue light lesion. For example, one animal breathing 80% oxygen and 20% nitrogen with a Po_2 of 271 torr, required only 10 $J.cm^{-2}$ to produce a minimal lesion. This represents a lowering of the threshold by a factor of 3. This research has also shown that a monkey under deep anesthesia breathing normal air has a low Po_2 and therefore a higher threshold for minimal retinal damage than an unanesthetized animal breathing air under normal physiological conditions. Since all threshold data on monkeys in this laboratory were made while the animals were under deep anesthesia, a correction is needed to lower the threshold for animals breathing air under normal physiological conditions.

b. Methylprednisolone as a protective agent.

Steroids are thought to strengthen cell membranes. Methylprednisolone (125 mg) is being injected intravenously one hour preexposure and one hour postexposure to test its effect on the blue light threshold. Preliminary results indicate that this treatment at one hour before exposure increases the threshold by a factor of 2. Injection at one hour postexposure seems to have little effect.

c. Beta-carotene as a desensitizer of excited molecules.

Beta-carotene with 9 carbon double bonds in its chain is reported to be a potent desensitizer of excited or sensitized molecules, particularly singlet oxygen. Solatene, a preparation of β-carotene by Roche, is being fed to one monkey on a daily basis of 7.5 mg. Serum analysis for β-carotene and vitamin A is performed periodically by the Bio-Science Laboratories. The concentration of β-carotene has reached a level of 360 µg/100 ml, while the vitamin A level in the serum has remained within normal limits. It is planned to determine the blue light threshold under oxygenation in this animal.

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b. Ham, W.T.Jr. Ocular hazards of light sources: review of current knowledge. J. Occup. Med., 25, 101-103 (1983).

Abstracts: 1981-1983

a. Ham, W.T.Jr., Mueller, H.A., Ruffolo, J.J.Jr., Guerry, D., III & Guerry, R.K. Effects from repetitive exposures of rhesus eye to near

UV & blue light. (ARVO abst.) Invest. Ophthalmol. & Vis. Sci., 22, 198 (1982).

b. _____ et al. Basic mechanisms leading to photochemical injury of the mammalian retina. (ARVO abst.) Invest. Ophthalmol. & Vis. Sci., 24, 70 (1983).

In Press:

a. Ham, W.T.Jr. The photopathology and nature of the blue light and near UV retinal lesions produced by lasers and other optical sources. Chap. in "Laser Applications in Medicine and Biology" Vol. 4, edited by M.L. Wolbarsht, Plenum Press, N.Y. (in press).

b. Mainster, M.A., Ham, W.T., Jr. and Delori, F.C. Potential retinal hazards: instrument and environmental light sources. Ophthalmology (in press).

c. Ham, W.T.Jr., Mueller, H.A., Ruffolo, J.J.Jr., Millen, G.E., Cleary, S. F., Guerry, R.K. and Guerry, D. III. Basic mechanisms underlying the production of photochemical lesions in the mammalian retina. Current Eye Research (in press).

8. Additional Activities 1981-1983

October 13-15, 1981: Dr. Ham attended joint conference of American Academy of Industrial Hygiene and American Academy of Occupational Medicine at Nashville, Tennessee. Delivered invited paper entitled "Ocular hazards of light sources". Served on panel discussing hazards.

November 16-17, 1981: National Academy of Science--National Research Council, Washington, D.C. Dr. Ham attended a Symposium on "Nutrition, Pharmacology & Vision" sponsored by the Committee on Vision.

December 10, 1981: Dr. Ham reviewed a manuscript for the American Journal of Ophthalmology.

February 9-10, 1982: Dr. Ham and Mr. Mueller attended a meeting of the Committee of the American National Standards Institute (ANSI Z 311), held at U.S. Army Environmental Hygiene Agency, Aberdeen Proving Grounds, Aberdeen Md.

March 25-26, 1982: Dr. Ham served as ad hoc member of the Vision Research Program Committee (VRPC) of the National Eye Institute, National Institutes of Health, Bethesda, Md.

April 28, 1982: Dr. Ham delivered invited paper before the American Occupational Medicine Academy (AOMA) at Toronto, Canada. This paper entitled "Ocular hazards of light sources: review of current knowledge" was published in the Journal of Occupational Medicine 25, 101-103 (1983).

May 2-7, 1982: Dr. Ham, Dr. Guerry and Mr. Mueller attended the annual meeting of the Associated Research in Vision & Ophthalmology (ARVO) at Sarasota, Florida. Dr. Guerry gave a paper "Effects from repetitive exposure of rhesus eye to near UV and blue light". See under abstracts.

June 10-11, 1982: Dr. Ham attended a Symposium on "Epidemiology of Eye Diseases" held by the National Eye Institute, NIH, Bethesda, Md.

June 28-July 2, 1982: Dr. Ham & Mr. Mueller attended the Gordon Conference on "Lasers in Medicine and Biology" at Meredith, NH. Dr. Ham chaired a session on lasers in clinical medicine.

August 9-12, 1982: Dr. Ham attended a Symposium on Occupationally Induced Macular Degeneration sponsored by the National Institute of Occupational Safety & Health (NIOSH) at Cincinnati, Ohio. He gave a paper "Does long-term exposure to short wavelength light and/or near UV radiation accelerate retinal aging and senile macular degeneration?"

September 7-8, 1982: Dr. Ham & Mr. Mueller attended a seminar on laser standards and light damage held at the U.S. Army Environmental Hygiene Agency, Aberdeen, Md.

September 17, 1982: Dr. Ham attended a one day Workshop on Laser Treatment of Macular Disease instigated by Dr. Carl Kupfer at the National Eye Institute, NIH, Bethesda, Md. He discussed the physical aspects of argon and krypton laser irradiation of the retina.

October 4-8, 1982: Dr. Ham attended the V International Congress on Eye Research (ICER) at Eindhoven, The Netherlands. He gave a paper "The nature of retinal damage from short wavelength light and near UV radiation".

November 1-3, 1982: Dr. Guerry and Dr. Ham attended the annual meeting of the American Academy of Ophthalmology in San Francisco, CA. Dr. Ham is co-author of paper entitled "Potential retinal hazards: instrument and environmental light sources" delivered by Dr. Mainster and to be published in Ophthalmology (in press).

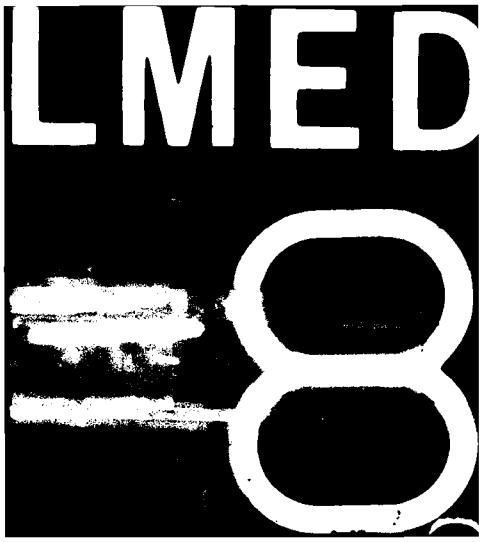
December 8, 1982: Dr. Ham attended the annual meeting of the American National Standards Institute (ANSI) Z 136 committee in New York, NY. He provided data on the ocular hazards of GaAs radiation in fiber optics communication systems.

February 17, 1983: Dr. Ham gave a seminar in the Department of Physiology and Biophysics at Virginia Commonwealth University, Richmond, VA. His subject was "Ocular hazards of infrared, visible and near UV radiation".

April 8-10, 1983: Dr. Ham and Mr. Mueller attended an International Symposium on Light and Oxygen Toxicity to the Eye, sponsored by The Department of Ophthalmology at the University of Maryland Medical Center, Baltimore, Md. Dr. Ham gave an invited paper "Basic mechanisms underlying the production of photochemical lesions in the mammalian retina" which has been accepted for publication in Current Eye Research.

April 26, 1983: Dr. Ham attended a postgraduate symposium on radiation hazards associated with high technology and fiber optics communicates systems, sponsored by the American Occupational Medicine Academy at Washington, D.C. and gave a paper on "Ocular hazards associated with high technology."

May 1-7, 1983: Dr. Ham & Mr. Mueller attended the annual meeting of the Associated Research in Vision and Ophthalmology (ARVO) and presented a paper "Basic mechanisms leading to photochemical injury of the mammalian retina" (see abstracts).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