AD-A171 385 BIOLOGICAL APPLICATIONS AND EFFECTS OF OPTICAL MASERS (U) VIRGINIA COMMONWEALTH UNIV RICHMOND W T HAM JUN 84 DAMD17-82-C-2083 1/1

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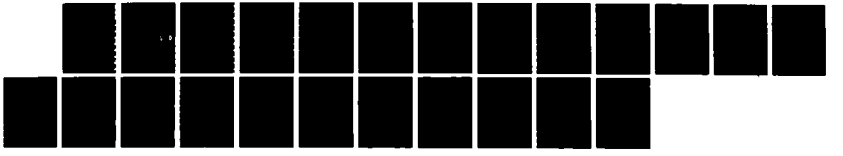

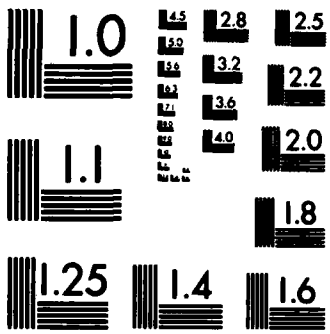

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LEVEL BIOLOGICAL APPLICATIONS AND EFFECTS OF OPTICAL MASERS ANNUAL REPORT JUNE 1984

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BIOLOGICAL APPLICATIONS AND EFFECTS OF OPTICAL MASERS

Annual Report

March 15, 1983 - March 15, 1984

June 1984

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

Supported by

U.S. ARMY MEDICAL RESEARCH AND DEVELOPMENT COMMAND
Fort Detrick, Frederick, Maryland 21701-5012

Contract No. DAMD17-82-C-2083

Virginia Commonwealth University
Richmond, Virginia 23219

SECURITY CLASSIFICATION OF THIS PAGE

REPORT DOCUMENTATION PAGE

Form Approved
OMB No 0704-0188
Exp Date Jun 30, 1986

1a. REPORT SECURITY CLASSIFICATION Unclassified	1b. RESTRICTIVE MARKINGS
2a. SECURITY CLASSIFICATION AUTHORITY	3. DISTRIBUTION/AVAILABILITY OF REPORT Approved for public release; distribution unlimited
2b. DECLASSIFICATION/DOWNGRADING SCHEDULE	
4. PERFORMING ORGANIZATION REPORT NUMBER(S)	5. MONITORING ORGANIZATION REPORT NUMBER(S)
6a. NAME OF PERFORMING ORGANIZATION Virginia Commonwealth University 6b. OFFICE SYMBOL (If applicable)	7a. NAME OF MONITORING ORGANIZATION
6c. ADDRESS (City, State, and ZIP Code) Richmond, Virginia 23219	7b. ADDRESS (City, State, and ZIP Code)
8a. NAME OF FUNDING/SPONSORING ORGANIZATION U.S. Army Medical Research and Development Command 8b. OFFICE SYMBOL (If applicable)	9. PROCUREMENT INSTRUMENT IDENTIFICATION NUMBER DAMD17-82-C-2083
8c. ADDRESS (City, State, and ZIP Code) Fort Detrick, Frederick, MD 21701-5012	10. SOURCE OF FUNDING NUMBERS PROGRAM ELEMENT NO. 62777A; PROJECT NO. 3E162. 777A878; TASK NO. BA; WORK UNIT ACCESSION NO 206

11. TITLE (Include Security Classification)
(U) Biological Applications and Effects of Optical Masers

12. PERSONAL AUTHOR(S)
William T. Ham, Jr., Ph.D.

13a. TYPE OF REPORT	13b. TIME COVERED	14. DATE OF REPORT (Year, Month, Day)	15. PAGE COUNT
Annual	FROM 3/15/83 TO 3/15/84	June 1984	

16. SUPPLEMENTARY NOTATION

17. COSATI CODES			18. SUBJECT TERMS (Continue on reverse if necessary and identify by block number)
FIELD	GROUP	SUB-GROUP	
06	18		
20	06		

19. ABSTRACT (Continue on reverse if necessary and identify by block number)

20. DISTRIBUTION/AVAILABILITY OF ABSTRACT ☐ UNCLASSIFIED/UNLIMITED ☒ SAME AS RPT ☐ DTIC USERS	21. ABSTRACT SECURITY CLASSIFICATION
22a. NAME OF RESPONSIBLE INDIVIDUAL Mrs. Virginia Miller	22b. TELEPHONE (Include Area Code) 301-663-7325 22c. OFFICE SYMBOL SGRD-RMS

DD FORM 1473, 84 MAR

83 APR edition may be used until exhausted
All other editions are obsolete

SECURITY CLASSIFICATION OF THIS PAGE

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1. Abstract

(2) The argon/krypton laser was back in operation in October 1983 after many vicissitudes during the fiscal year. Unfortunately, the Bragg cell that drives the acoustic modulator failed to function. The argon/krypton laser and acoustic modulator are operating satisfactory now with a Bragg cell on loan by Spectra-Physics while our Bragg cell has been sent to California for repairs. The research on threshold data as a function of wavelength and modulation frequency can now proceed. (3) Additional data on 40 μs pulses at a PRF of 400 Hz are presented in Table I. Figure 1 gives a log-log plot of threshold corneal power P_c vs exposure time in s for PRF's of 100, 200 and 400 Hz. P_c decreases with PRF for shorter exposure times but seems to converge for exposures greater than 1000 s. (4) A Workshop dealing with the possible long-term ocular effects of optical radiation on aging and macular degeneration is discussed and a final draft of the report of the Working Group assessing light effects on the retinal pigment epithelium (RPE) is appended to this report. A preliminary experiment on the additivity and reparation of repetitive exposures of light to the same retinal site is described. Repetitive 100 s exposures of blue light (440 nm ± 5 nm) at 24 hour intervals were found to be additive for 2 exposures at 50% of threshold, for 3 exposures at 40% of threshold, and for 4 exposures at 30% of threshold. Wavelengths at 490 and 520 nm were not additive even at 50% of threshold.

(5) Two macaque monkeys were used to study the histological consequences of retinal blue light lesions produced under high arterial blood oxygen tension. Analysis of the various photic lesions showed only moderate damage to the neural retina but very extensive damage to the RPE. This is the histopathological pattern of a typical blue light lesion shown in previous studies but more severe and appearing earlier after exposure. A report of this research is "in press" in Invest. Ophthalmol. & Vis. Sci. A preprint is enclosed. (6) The enhancement of retinal sensitivity to blue light damage when the animal is highly oxygenated (threshold lowered by a factor of 3 at arterial PO_2 of 270 mm of Hg) suggests but does not prove that O_2 free radicals and sensitized molecules ($O_2^{\cdot-}$, H_2O_2, $OH\cdot$, $^1O_2$) play an important role in photochemical damage to the retina. Beta-carotene provided protection from blue light damage plus oxygenation in the only animal tested so far. Results to date do not contradict the hypothesis that photodynamical action (probably singlet oxygen) is a contributing factor leading to photochemical lesions in the retina. A report describing these effects is published in Curr. Eye Res., reprint enclosed. (7) Two trained monkeys, one with normal pupil size, one with pupils dilated > 8 mm, have undergone 950 and 366 daily exposures respectively to the spectrum 330-420 nm ($5 mW \cdot cm^{-2}$ at cornea). As yet, no lenticular abnormalities have heen detected in either animal. Another trained animal with daily exposures to the spectrum 330-490 nm

developed a funduscopically visible lesion. This animal was recently sacrificed for histological analysis of the lesion. A trained aphakic monkey showed extensive retinal damage after 316 daily exposures to the spectrum 330-420 nm. The retinal irradiance during an exposure was approximately 1 $mW \cdot cm^{-2}$, a figure somewhat less than that to be expected on the retina of an aphakic human eye on a bright sunny day between noon and 2 p.m. This animal has been sacrificed. Histological analysis showed widespread RPE abnormalities in the superior and nasal paramacular fundus. This experiment demonstrates that the RPE in an aphakic eye can be damaged by small daily exposures to near UV radiation.

2. Argon/Krypton Acoustically Modulated Laser.

This has been a year of frustration in our attempts to investigate minimum radiant exposures for acoustically modulated wavelengths at 647nm and 488nm as provided by the argon/krypton laser. The program, as originally planned, called for a comparison of threshold data at these two wavelengths as a function of modulation frequencies 1,10 and 100 kHz and 1, 10 and 20 MHz. Our first problem was the argon/krypton laser; we were unable to make it oscillate. Spectra-Physics does not renovate old tubes but offered to sell us a new tube for $7,700 including shipping charges if we would ship the old laser to them. In an effort to save funds we opened negotiations with Phoenix Lasers, Ltd. who offered to renovate the old laser for a price ranging from $200 to $5,200 depending on the reason for the malfunction. The argon/krypton laser was shipped to them on July 6 1983. When it arrived in Palo Alto, California, Phoenix Lasers, Ltd. had gone out of business. We were able to arrange for the delivery of the laser to Spectra-Physics in Mountain View, California. They repaired and shipped it back to us in October 1983. But our troubles had only begun. The acoustic modulator, a complicated electro-optical device, was defective. For the next two months we tried a number of ways to correct the fault as suggested by the engineers at the New Jersey plant of Spectra-Physics. The major problem is that this type of acoustic modulator is obsolete and no longer manufactured. Finally, the engineer at the New

Jersey plant lent us his Bragg cell, the only one in his possession. When installed in our acoustic modulator it functioned perfectly. We have sent our Bragg cell to California for repairs. Currently, we are able to begin our research protocol with the Bragg cell on loan. We hope to complete the proposed program during the ensuing year.

3. Minimum Radiant Exposures for 40 μs Pulses of 647nm Light:

Data for pulse repetition frequencies (PRF) of 100 and 200 pulses per s and exposure durations ranging from 1 s to 3000 s have already been reported. Table I repeats those data for PRF's of 100 and 200 pulses per s and gives similar data for a PRF of 400 pulses per s.

Table I

Radiant Exposures H_o per pulse and H_o total in $J \cdot cm^{-2}$ for a minimal lesion in the monkey retina are given for 40 μs pulses at pulse repetition frequencies (PRF) of 100, 200, and 400 pulses per s for exposure durations ranging from 1 to 3000 s. E_o in $W \cdot cm^{-2}$ on the retina is calculated on the assumption that the laser beam produced 25 μm lesions at the I/e points of the Gaussian distribution, $E=E_o \exp(-r^2/2\sigma^2)$ according to the formula $E_o=P_c T \cdot (2\sigma^2)^{-1}$ where P_c in W is the power entering the cornea as measured, T is the transmission through the ocular media (0.93 for 647nm) and is the Gaussian parameter corresponding to a radius r of 12.5 μm.

Exposure Time s	Number Pulses	P_c W	E_o $W \cdot cm^{-2}$	H_o per pulse $mJ \cdot cm^{-2}$	H_o additive $J \cdot cm^{-2}$
1	1×10^2	.14	265.	10.6	1.06
1	2×10^2	.082	155.	6.22	1.24
1	4×10^2	.052	98.7	3.95	1.58
10	1×10^3	.080	152.	6.06	6.06
10	2×10^3	.059	113.	4.51	9.02
10	4×10^3	.039	74.7	2.99	11.9
10^2	1×10^4	.029	54.9	2.19	21.9
10^2	2×10^4	.031	58.7	2.35	47.0
10^2	4×10^4	.021	39.4	1.58	63.1
10^3	1×10^5	.014	26.5	1.06	106
10^3	2×10^5	.012	22.7	0.91	182
10^3	4×10^5	.010	19.0	0.76	305
3×10^3	3×10^5	.0067	12.8	0.51	153
3×10^3	6×10^5	.0065	12.2	0.49	293
3×10^3	12×10^5	.0054	10.0	0.41	494

In Figure 1 we have plotted the corneal power P_c entering the eye in W against exposure duration in s as shown in Table I. This is a log-log plot, The circles represent a PRF of 100 Hz, the x's 200 Hz and the inverted v's 400 Hz. It can be seen that P_c decreases with PRF for the shorter exposure durations but seems to converge as the exposure duration is extended beyond 1000 s. It seems logical to suppose that as the time between pulses decreases from 10 milliseconds to 2.5 milliseconds the additivity between pulses increases. Based on a thermal model this would mean temperature additivity as the time between pulses decreases. For high PRF's the pulse model should approach the CW condition. Spot size on the retina is an important factor. For very small spot sizes like those produced by lasers, as in this experiment, the time required to reach temperature equilibrium is very short $\simeq$ microseconds and the irradiances are high. Small spot size also means rapid dissipation of energy when the pulse is over, so that for low PRF's one would not expect temperature additivity.

4. Additivity and Reparation from Repetitive Exposures to Short Wavelength Light.

Interest is increasing in the possible effects of long-term chronic exposure to sunlight and man-made optical sources. For example, Dr. Ham attended a Workshop on Long-Term Visual Health and Optical Radiation sponsored by the Division of Risk Assessement, National Center for Devices and Radiological Health, Food and Drug Administration. This Workshop was co-sponsored by

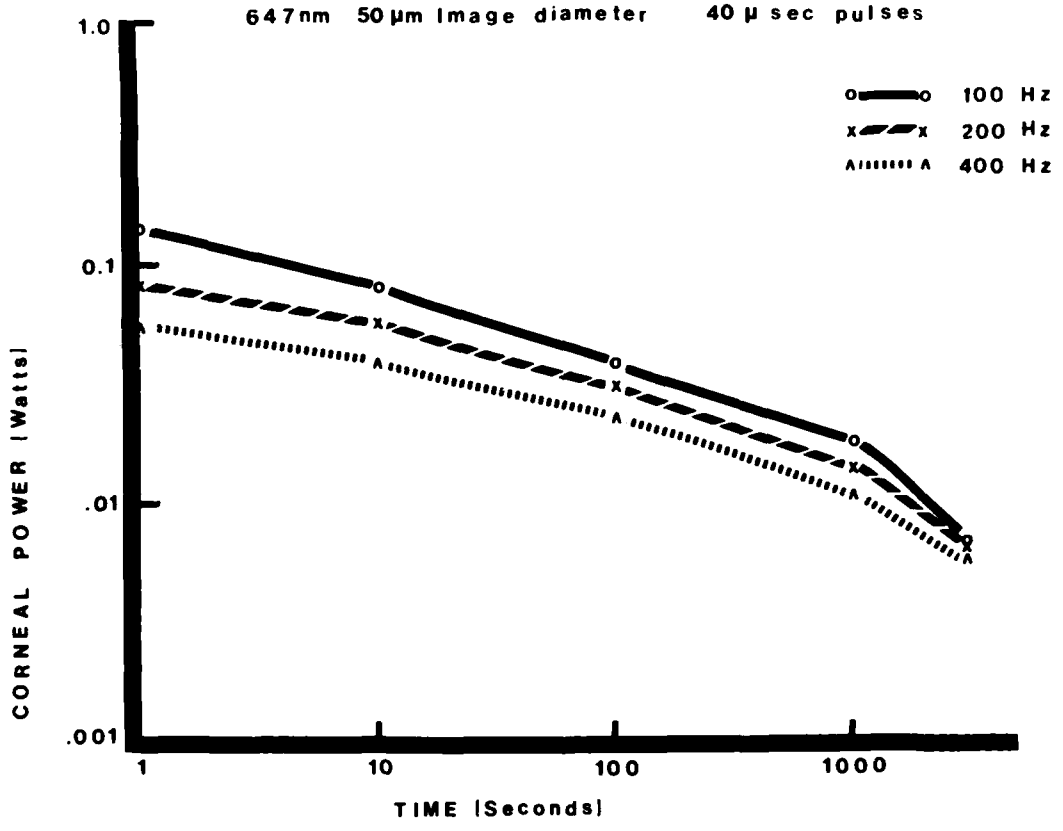
647nm 50 µm Image diameter 40 µ sec pulses
100 Hz
200 Hz
400 Hz
1.0
0.1
.01
.001
CORNEAL POWER (Watts)
1
10
100
1000
TIME (Seconds)

Figure 1

the Army Medical Research and Development Command, the Lawrence Berkeley Laboratory, Department of Energy and the Occupational Safety and Health Administration. The Committee on Vision, NAS-NRC was a participating organization. Leading questions posed by the Workshop concerned the effects of daily, chronic exposure to short wavelength light on aging and degeneration of the retina and lens leading to degenerative maculopathies and senile cataract. Dr. Ham chaired the Working Group assigned the task of assessing light damage to the RPE and its possible relationship to aging and macular degeneration of the retina. A preprint of the final draft submitted by the RPE Working Group is attached to this progress report. It is scheduled to be published by CRC Press in June 1985.

We have begun an investigation on the additivity and reparation of repetitive exposures of light to the same retinal site. Originally we intended to use repetitive exposures to the same retinal site at 24 hour intervals for light levels 50%, 40%, 30%, 20% and 10 % of threshold at 3 wavelengths of 440, 490, and 520 nm. We planned to use a 500 μm spot size and exposure times of 100 s. The protocol called for 15 exposures across the retina in 3 rows with 5 different exposure levels at each wavelength in a single eye. Each location would be carefully documented by fundus photography. The plan was to anesthetise the animal on a daily basis for 21 days including Saturdays and Sundays. We have completed the initial step of this protocol by exposing one

animal for a 5 day period using 20 nm filters peaked at 440, 490 and 520 nm as provided by the 2500 W xenon lamp with quartz optics. Five exposures at 50, 40, 30, 20 and 10 percent of a predetermined threshold were given at each of these wavelengths. After 5 daily exposures, 3 lesions were detectable at the 440 nm wavelength; that is 50% of threshold showed up as a lesion on the third day, 40% on the fourth day and 30% on the fifth day. No lesions appeared, even on the fifth day for the longer wavelengths 490 and 520 nm. This experiment demonstrates the sharp rise in retinal sensitivity to damage as the wavelength decreases toward the near ultraviolet. We estimate a factor of from 3-5 for retinal sensitivity at 440 nm as compared with 488 nm. We plan to redesign the protocol for this experiment at the longer wavelengths 490 and 520 nm. It probably will be necessary to use 90, 80, 70, 60, and 50 percent of threshold for these wavelengths.

5. Histological Analysis of Photochemical Lesions Produced Under Conditions of Elevated Blood oxygen.

This study was performed on 2 macaque monkeys using the 2500 W xenon optical system as radiation source for a narrow bandwidth, 440 $\pm$ 5 nm. Retinal image size was 1 mm and radiant exposures to the retina ranged from 36 to 11 $J \cdot cm^{-2}$. Oxygenation was accomplished under anesthesia by an endotracheal tube connected to a non-rebreathing apparatus equipped with separate inhalation and exhalation valves with attached gas bag that was kept slightly above atmospheric pressure with a needle valve

regulator. A previously prepared tank mixture with an 80/20 ratio of O_2/N_2 was used. Arterial (femoral) blood samples were taken before oxygenation began and after 30 minutes of breathing the 80/20 mixture. Blue light exposures were performed immediately after 30 minutes of oxygenation. Threshold data on each animal had been predetermined at least a week before the oxygenation studies.

When blood oxygenation is not experimentally elevated the threshold radiant exposure for a blue light lesion to be funduscopically visible at 2 days postexposure is about 30 $J \cdot cm^{-2}$. At a high blood PO_2 level (270 mm Hg) a radiant exposure of only 11 $J \cdot cm^{-2}$ gives a funduscopically visible lesion at 1 day post exposure. This large increase in retinal sensitivity to blue light damage is probably due to photodynamic action and may involve singlet oxygen though this remains to be proved. Analysis of the various photic lesions showed only moderate damage to the neural retina but a strong response was seen in the RPE. This is the histopathological pattern of a typical blue light lesion shown in previous studies but more severe. The effect of elevated blood O_2 is to increase retinal sensitivity to photic damage, to lower the damage threshold and the time of its appearance, and to increase the severity of damage at a given radiant exposure. Mild lesions observed at 23 and 57 days after exposure show remarkable recovery. A paper describing this research has been accepted for publication by Investigative

Ophthalmology and Visual Science. A preprint is enclosed with this annual progress report.

6. Basic Mechanisms Underlying the Production of Photochemical Lesions in the Mammalian Retina.

Dr. Ham delivered an invited paper with the above title at an International Symposium on Light and Oxygen Effects on the Eye sponsored by the Department of Ophthalmology, University of Maryland School of Medicine. This paper has been published in Current Eye Research. A reprint is enclosed in this annual progress report.

Briefly, we have pointed out that the mammalian retina is unique among body tissues because it is the only tissue where light is focused continuously on a group of cells that is highly oxygenated, i.e. the retinal pigment epithelium (RPE) and the photoreceptor cells that are among the most metabolically active cells in the body. Both light and oxygen can be individually toxic to cells. In combination toxicity should be enhanced. We have shown in the rhesus monkey that when the arterial blood oxygen is enhanced to a PO_2 of 270 mm Hg, the threshold for the blue light lesion is reduced by a factor of 3, from 30 $J \cdot cm^{-2}$ to 10 $J \cdot cm^{-2}$. This implies photodynamic action and probably involves singlet oxygen. However, this remains to be proved. From a practical standpoint the retina of a patient undergoing ophthalmic surgery, e.g. lens extraction, vitrectomy, etc., would be more sensitive to retinal light damage if breathing oxygen. Injections of methylprednisolone (125 mg i.v.) one hour before

exposure of the macaque retina to blue light seemed to provide some protection. However, more recent experiments have not substantiated this effect and we believe that much more research is needed before the role of steroids in light damage can be assessed. The carotenoid B-carotene has been shown to provide protection from blue light damage in one monkey. This is further evidence that singlet oxygen may be involved in the production of photochemical lesions by blue light.

7. Long-Term Repetitive Exposures of Trained Monkeys to Near UV Radiation and Short Wavelength Light.

These experiments began in 1979. They were designed to study the long-term effects on the lens and retina of daily exposures to near UV and short wavelength visible light similar to that from the sun at sea level. The 2500 W xenon lamp with quartz optics was equipped with suitable filters and mirrors to produce two spectral bandwidths, 330-420 nm corresponding approximately to near UV radiation from sunlight, and 330-490 nm corresponding roughly to the near UV plus short wavelength blue light found in sunlight. The lens absorbs a large proportion of the 330-420 nm spectrum; less than 1% of this radiation reaches the primate retina. On the other hand when the lens is removed by cataract surgery the retina is also exposed to near UV radiation. Approximately 27% of the light in the 330-490 nm spectrum reaches the retina in the normal eye, i.e. lens intact. Two experiments with trained monkeys were designed to study the effects of near UV on the lens. A third experiment was designed

to study the effects of blue light on the retina and the fourth experiment was designed to study the effects of near UV on the retina of an aphakic monkey. These experiments will be discussed separately.

a. Near UV effects on lens with normal and dilated pupil.

The animal with normal pupils received 971 daily exposures to 330-420 nm radiation as of 4/15/84. The irradiated eye receives 5mW·cm^{-2} at the cornea for 1000 s on a daily basis, 5 days per week. The latest examination by biomicroscope can detect no anomalies in either the exposed or the control eye. The pupils during irradiation have a diameter of 2-3 mm. We have hypothesized that perhaps the iris protects the vulnerable equatorial region of the lens from near UV photons. To test this hypothesis we are exposing another animal to the same spectrum under identical conditions except that the pupils are dilated to greater than 8 mm in diameter by the use of atropine. This animal has received 366 exposures as of 3/15/84. No anomalies have been detected in either eye by biomicroscopic examination. The retinae in both animals are normal as seen with the fundus camera. We plan to continue these exposure regimes with both animals.

b. Blue light effects on the retina.

The trained monkey exposed to the 330-490 nm spectrum began exposures in August 1981. The corneal irradiance was 5mW·cm^{-2} and the retinal irradiance was estimated to be 8mW·cm^{-2},

Periodic examinations disclosed no anomalies in either retinae or lenses up to 371 exposures when a small but very faint patch of depigmentation in the temporal macula of the exposed eye was noted by fundus camera examination. Fluorescein angiography was normal in both eyes. As exposures continued this patch of depigmentation became more prominent. After 580 exposures it appeared to have developed into a retinal lesion, half-moon in shape and about 400 μm x 100 μm in size. It was decided to sacrifice this animal for histological examination.

The animal's eyes were enucleated and he was sacrificed on April 20,1984. Dr. Ruffolo will report his histological findings in the near future. We believe that this experiment, while statistically unsatisfactory since it represents a single animal, does demonstrate that long-term, chronic exposure of the primate retina to blue light produces cumulative photochemical damage.

c. Near UV phototoxicity in the aphakic primate retina.

This animal had the lens removed in one eye several months before exposures began in October 1981 to the spectral band 330-420 nm. Exposures were 1000 s in duration on a daily basis, 5 days per week. The divergence of the beam of near UV radiation was adjusted to produce a 1.2 mm spot size on the retina, though this is only approximate since the exposed eye was unable to accomodate. Irradiance on the retina was estimated at $1 mW \cdot cm^{-2}$. Calculations based on the blue sky radiance at noon on a clear day estimate the retinal irradiance on the aphakic human retina

with a 2 mm diameter pupil to be between 2-3 $mW \cdot cm^{-2}$. After nearly 300 daily exposures fluorescein angiography showed multiple focal areas of retinal pigment epithelium (RPE) depigmentation in the fundus. After 316 exposures funduscopic examination showed two photic lesions and numerous small areas of RPE depigmentation near the macula. The animal was sacrificed in November 1983. Histological analysis showed widespread RPE abnormalities in the superior and nasal paramacular fundus. In the superior-temporal paramacular sample, containing the visible photic lesions, the neural retina was lost in specimen preparation. This was unfortunate since all of our observations to date on near UV retinal lesions have shown severe damage to the photoreceptors, especially the cones. We assume the same would have been true for this animal had the neural retina been available for observation.

The inferior temporal region of the fundus was essentially normal. This experiment roughly simulates daily exposures to bright sunlight and the results indicate that the RPE of an aphakic primate eye can be damaged by small daily exposures to near UV radiation.

8. Publications: 1983-1984

i. Mainster, M.A., Ham, W.T. Jr. and Delori, F.C. Potenial retinal hazards: instrument and environmental light sources. Ophthalmol. 90 , 927-932 (1983).

ii. Ham, W.T. Jr., Mueller, H.A., Ruffolo, J.J. Jr., Millen, J.E., Cleary, S.F., Guerry, R.K. and Guerry, D. III. Basic mechanisms underlying the production of photochemical lesions in the mammaliam retina. Curr. Eye Res. 3 , 165-174 (1984)

In Press:

i. 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.

ii. Ham, W.T. Jr. (Chairman), Allen, R.G., Feeney-Burns, L., Marmor, M.F., Parver, L.M., Proctor, P.H., Sliney, D.H. and Wolbarsht, M.L. The Retinal Pigment Epithelial Working Group on "The Involvement of the Retinal Pigment Epithelium in Light Damage," CRC Press.

iii. Ham, W.T. Jr., Mueller, H.A., Ruffolo, J.J. Jr., Guerry, R.K. and Clarke, A.M. Ocular effects of GaAs lasers and near infrared radiation. Applied Optics.

iiii. Ruffolo, J.J., Jr., et al., Oxygen Enhanced Retinal Photosensitivity.,Invest. Ophthalmol. & Vis. Sci.

Abstracts: 1983-84
Ruffolo, J.J. Jr., Mueller, H.A., Ham, W.T. Jr., Guerry, D. III and Guerry, R.K. Retinal responce to chronic exposure of an aphakic eye to a 330-420 nm spectral bandwidth. Invest. Ophthalmol. & Vis. Sci. 25 , 89 (March 1984).

9. Additional Activities:

April 4-6, 1983: Dr. Ruffolo visited our laboratory and prepared specimens for histological and ultrastructural analysis from 2 monkeys after exposure to 440 nm light under high arterial O_2 tension.

April 8-10, 1983: Dr. Ham and Mr. Mueller attended a 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." Published in Current Eye Research.

April 26, 1983: Dr. Ham attended a postgraduate symposium on radiation hazards associated with high technology and fiber optics communication 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 and Harold 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"

July 26, 1983: Dr. Ham attended an ad hoc panel discussion on "Safety guidelines for blue-green and ultraviolet lasers" sponsored by the Naval Medical Research and Development Command at Bethesda, MD.

Sept. 6-9, 1983: Dr. Ruffolo visited our laboratory for conferences on research progress and the design of future experiments.

Sept. 10, 1983: Dr. Ham attended a Symposium "Current Trends in Ophthalmology-10L Update" presented by the Richmond Eye and Ear Hospital in Richmond, VA. Dr. Ham gave an invited lecture on "Ultraviolet effects on the eye and protective measures."

Sept. 25-27, 1983: Dr. Ham attended a Workshop on Long-Term Visual Health and Optical Radiation sponsored by the Division of Risk Assessment, National Center for Devices and Radiological Health of the Food & Drug Administration. He chaired the Retinal Pigment Epithelial Working Group who were assigned the task of investigating light damage to the RPE and its possible relationship to aging and macular degeneration of the retina. A paper evolving from this Working Group is scheduled to be published in the CRC Press in June 1985.

Oct. 6-8, 1983: Dr. Ham attended a Symposium on "Free Radicals in Molecular Biology and Aging" sponsored by the American Aging Association (AGE) in Washington, D.C.

Nov. 9-11, 1983: Dr. Ruffolo visited our laboratory and prepared specimens for LM and EM from a trained aphakic monkey sacrificed on Nov. 9, 1983 after 316 daily exposures to the spectral band 330-420 nm. This study was presented at ARVO, Sarasota, Florida in May 1984.

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