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Action Spectrum and Correlation between Irradiation and Temperature Rise in the Retina

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Abstract- This study investigates the action spectrum and correlation between irradiation and temperature rise in the retina due to infrared and visible electromagnetic radiation. The research highlights how variations in transmittance (from 0.794 to 0.702) and incident power on the cornea influence retinal irradiance and temperature distribution. The findings reveal that irradiance increases with wavelength and exposure time, with a sharp rise observed at 1064 nm in the near-infrared spectrum. Retinal temperature exhibits a complex relationship with exposure time, initially increasing but decreasing for prolonged durations, suggesting photochemical and photothermal interactions. The study also incorporates the Stiles-Crawford effect (SCE I) to model temperature distribution, demonstrating its impact on retinal thermal response. Data from rhesus monkey retinas further validate the observed trends, showing consistent irradiance patterns under varying transmittance conditions. The results underscore the importance of wavelength, exposure duration, and beam characteristics in assessing retinal damage risks from optical radiation.

Index Terms **-** Retinal irradiation, thermal damage, action spectrum, infrared radiation, Stiles-Crawford effect, photothermal injury.

I. INTRODUCTION

Electromagnetic spectrum consisting of radiofrequency, microwave, infrared, visible, ultraviolet, x-rays and gamma radiations causes retinal damage mainly in three ways: photochemicallly, photothermally, and photo mechanically [1]. Photothermal damage of retina is due to wavelength, power density, and pulse width ranging from microsecond to second. The sensitivity of retina to photothermal damage was determined by Ham et. al. [1] at eight different wavelengths from 441.6 nm to 1064 nm in the rhesus monkey [2]. We report that for different beam size, the temperature distribution in the retina and the irradiation due to different wavelengths and exposure time.

II. RADIATION EFFECT ON THE EYE

The energy of an infrared photon is much lower than that of a visible or ultraviolet photon. The tissues most sensitive in the eye are the cornea and aqueous humour, as the infrared radiation raises the overall temperature of the anterior eye. The lens absorbs only a small proportion of infrared, the overall exposure level would need to be high, or the result of smaller repeated doses. Long wavelength infrared rays also reach the retina and can cause permanent damage to the delicate photoreceptors.

To understand these three mechanisms, we consider wave-particle duality of light first described by Einstein in 1905. Light is composed of a continuous spectrum of different radiant wavelengths [3].

As the light is quanta of energy i.e.; photon, to more particulate it is necessary to consider the properties of light [4-7]. Infrared radiation effect on the eye and affected tissues.

A. For near IR (IR-A) 780-1400 nm

From sources Alexandrite (710-800 nm) Gallium Arsenide (850-950 nm) Neodymium-Yag (1064 nm) Helium-Neon (1150 nm) Iodine (1315 nm) and sun, furnaces, lamps, glass blowing, the affected tissues of human eye are Retina (pigment epithelium), Iris (lens, cataract) Blanched retinal lesion or oedema proceeding to vitreous haemorrhage. The symptoms are cherry red flash or glow, dull after image scotoma may be accompanied by pain rapid onset of scotoma without light flash (Shock if Q-switched ND-Yag laser) - pain. This may cause deep retinal coagulation, may involve choroid, possible lens or vitreous involvement Lesions elongated if caused by CW laser.

B. For Far IR (IR-B) 1400-3000 nm

From sources Erbium 1540nm, Holmium 2060 nm (pulsed lasers) and sunlight, furnaces, lamps, the affected tissues are cornea (opacity) lens (cataract). The symptoms are Pain Blepharospasm Visual loss.

C. For Far IR (IR-C) 3 µm-100 µm

From the sources Deuterium (pulsed), Fluoride 3.8-4 µm, Carbon dioxide 10.6 µm and Furnaces, lamps, the affected tissues are Cornea Conjunctiva Skin. The

symptoms are White flash, intense pain Blepharospasm Corneal opacity Perforation [8].

III.THERMAL DAMAGE TO THE EYE

A photon can be absorbed by a molecule when the energy of the photon is equal to the energy difference between the molecule's current energy level and an allowed higher energy level. The vibration and rotational quantum states of the molecule predominates in the near infrared [9]. Therefore, a rise in temperature occurs due to the vibrational energy gained by the molecule dissipated through the collision with another molecule. Thermal lesions occur when the irradiance of the radiation is high enough to raise the temperature more than 10°C above the ambient temperature in the retina [10].

Thermal effects generally arise from the exposure time and the irradiance. The amount of energy required to produce thermal effect increases for long exposure time [10].

Thermal damage to the cornea is the loss of transparency, opacification, haze, exfoliation, debris. Thermal damage to the aqueous humour is Flare and that of iris is Swelling, cell death, miosis, and hyperaemia/inflammation. Thermal damage to Lens is anterior opacities, sutures more visible and that of Vitreous Humour is Haze.

Thermal damage to the retina is Oedema, burns, depigmentation [8].

IV.THERMAL EFFECT TO THE RETINA

Any infrared rays that are transmitted through the ocular media to the retina are absorbed by the pigment epithelium of the retina. Injury occurs in the neural layers through indirect heating. The effect of the infrared on the retina and choroid is to cause a rise in temperature, which causes enzymes to denature; in general, temperatures more than 10°above ambient body temperature will produce permanent thermal damage. Many physical factors affect whether damage is done, including pupil size, the optical quality of the retinal IR image, exposure duration, size of the source and of the retinal image, location on the retina, the type and spectral distribution of the source and rate of delivery of the energy. Obviously, exposure duration is a major factor. As the exposure time increases, the radiant power entering the eye necessary to produce retinal

structural damage decreases until, at a certain level, duration becomes irrelevant and the damage appears to be determined by the irradiance reaching the retina alone. Heating effects to the retinal pigment epithelium can occur from infrared exposure durations as low as microseconds. Even shorter durations of laser infrared exposure (picoseconds) cause a different type of damage that of acoustic or shockwave effects to the melanin pigment. Many studies have attempted to establish threshold exposure values for the human retina. In addition to the physical factors, such as exposure duration and irradiance levels, eyes vary in their degree of retinal pigmentation, which undoubtedly has an effect on the degree and extent of damage by infrared radiation.

Studies have shown that the longer wavelength infrared radiation requires higher irradiance at the cornea to produce a retinal burn. This is pertinent since at lower wavelengths, much more infrared passes through to the retina [11-13].

V.IRRADIATON AND TEMPERATURE RISE TO THE RETINA

Retinal damage primarily depends on wavelength, power density, exposure time and pulse width ranging from micro second to second. Thus, in the retinal pigment epithelium (RPE) a high energy level produced causing a thermal denaturation in both the RPE and neural retina. Thermal damage depends on energy absorption to produce temperature which can damage the neural tissue and RPE.

The irradiance of the retina can be calculated from the equation

$$H = H_o e^{-\frac{r^2}{2\sigma^2}} \qquad (1)$$

Where H =irradiance at radius r from the Gaussian peak.

σ = Calculated from the beam profile by measuring r at the $\frac{1}{e^2}$ points.

H_o = maximum irradiance of the retina calculated from the equation below as

$$H_o = \frac{P_c T}{2\pi\sigma^2}$$

$$H = \frac{P_c T}{2\pi\sigma^2} e^{-\frac{r^2}{2\sigma^2}} \qquad (2)$$

For different wavelengths and exposure time, the maximum retinal temperature above the ambient temperature is given in the table. Incorporating the Stiles Crawford effect of the first kind [14] as given by the relation

$$\eta = \eta_o e^{-\rho r^2} \qquad (3)$$

The above relation can be modified (in the presence of SCE I) as

$$H = \frac{P_c T}{2\pi\sigma^2} e^{-\rho r^2} e^{-\frac{r^2}{2\sigma^2}}$$

$$H = \frac{P_c T}{2\pi\sigma^2} \boldsymbol{e}^{-\boldsymbol{r}^2\left(\boldsymbol{\rho} + \frac{\mathbf{1}}{\mathbf{2}\boldsymbol{\sigma}^2}\right)}$$

Where $\rho = \frac{0.115}{mm^2}$ =directionality parameter [15]

Letting K_1 the maximum temperature above ambient on the retina during irradiation in ^{0}C determined from the mathematical model of Clarke *et al*., 1969 [10] in the absence of directionality and K_2 the maximum temperature above ambient on the retina during irradiation in ^{0}C in presence of SCE directionality, we may get the relation

$$K_2 = K_1\, e^{-\rho r^2} \qquad (4)$$

So, the maximum temperature above ambient on the retina during irradiation in ^{0}C will follow a SCE I distribution.

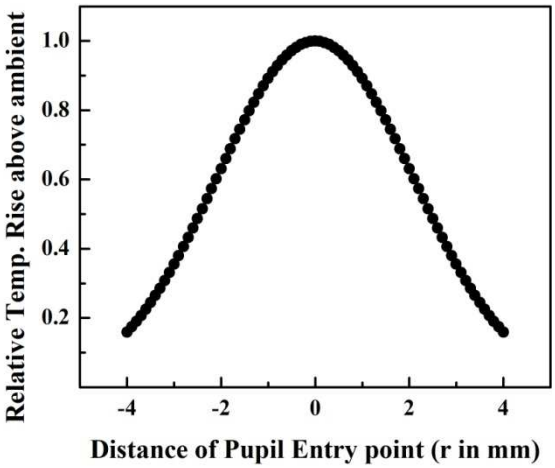

Fig 1. Relative Temperature rise plotted against pupil entry point from the centre

Table 1: H_0 in J/cm^2 vs wavelength for different exposure time

Exposure time (s)	H_0 in J/cm^2							
	Wavelength 441.6 nm	Wavelength 457.9 nm	Wavelength 488nm	Wavelength 514.5 nm	Wavelength 580nm	Wavelength 610nm	Wavelength 632.8 nm	Wavelength 1064 nm
1	0.91	5.1	9.4	14.5	26.1	22	29.9	56.1
16	6.6	51.2	97.6	165	184	200	243	600
100	20	52	77	220	760	810	840	3250
1000	30	60	150	320	4000	5800	5400	24000

Table 2: Temperature in ^{0}C vs wavelength for different exposure time

Exposure time(s)	K in ^{0}C							
	Wavelength 441.6nm	Wavelength 457.9nm	Wavelength 488nm	Wavelength 514.5nm	Wavelength 580nm	Wavelength 610nm	Wavelength 632.8nm	Wavelength 1064nm
1	2	10	17	25	43	36	49	55
16	1	6	11	18	19	20	25	37
100	0.4	1	1	4	12.5	13	14	32
1000	0.05	0.1	0.1	1	6.6	9.5	9	23

Table 3: H_0 in J/cm^2 vs. exposure time for different wavelength

Wave length in nm	H0 in J/cm2			
	1sec exposure	16sec exposure	100sec exposure	1000sec exposure
441.6	0.91	6.6	20	30
457.9	5.1	51.2	52	60
488	9.4	97.6	77	150
514.5	14.5	165	220	320
580	26.1	184	760	4000
610	22	200	810	5800
632.8	29.9	243	840	5400
1064	56.1	600	3250	24000

Table 4: Temperature in ^{0}C vs exposure time for different wavelength

Wave length in nm	K in ^{0}C			
	1sec exposure	16sec exposure	100sec exposure	1000sec exposure
441.6	2	1	0.4	0.05
457.9	10	6	1	0.1
488	17	11	1	0.1
514.5	25	18	4	1
580	43	19	12.5	6.6
610	36	20	13	9.5
632.8	49	25	14	9
1064	55	37	32	23

Table 5: H_0 calculation for 500 beam size for 1sec exposure

Wave length in nm	Transmittance T	P_c in mW	H_0 in W/m^2
441.6	0.45	2.0	0.229299363
457.9	0.69	7.3	1.283312102
488	0.83	11	2.32611465
514.5	0.87	16.3	3.612993631
580	0.91	28.2	6.538089172
610	0.92	23.5	5.508280255
632.8	0.93	31.5	7.463694268
1064	0.76	14.5	2.807643312

Table 6: For 16sec exposure

Wavelength in nm	Transmittance T	P_c in mW	H_0 in W/cm^2
441.6	0.45	0.9	0.103184713
457.9	0.69	4.6	0.80866242
488	0.83	7.1	1.501401274
514.5	0.87	11.6	2.571210191
580	0.91	12.4	2.874904459
610	0.92	13.3	3.117452229
632.8	0.93	16	3.791082803
1064	0.76	97	18.78216561

Table 7: For 100sec exposure

Wave length in nm	Transmittance T	P_c in mW	H_0 in W/cm^2
441.6	0.45	0.67	0.076815287
457.9	0.69	0.74	0.130089172
488	0.83	0.90	0.190318471
514.5	0.87	2.5	0.554140127
580	0.91	8.2	1.901146497
610	0.92	8.6	2.015796178
632.8	0.93	8.9	2.108789809
1064	0.76	84	16.26496815

Table 8: For 1000 sec exposure

Wave length in nm	Transmittance T	P_c In mW	H_0 in W/cm^2
441.6	0.45	0.062	0.00710828
457.9	0.69	0.082	0.014415287
488	0.83	0.17	0.035949045
514.5	0.87	0.36	0.079796178
580	0.91	4.3	0.996942675
610	0.92	6.2	1.453248408
632.8	0.93	29	6.87133758
1064	0.76	62	12.005 09554

Table-9: Exposure of the rhesus monkey retina to a SSS (300--1400 nm) for time intervals of l, 10, 100, and1000s. Criterion was a funduscopically minimally visible lesion at 48 h post exposure." Beam diameter on retina, 159 μm. Power, P_e, incident on cornea is in mW. Retinal radiant exposure, H_0, in J/cm^2. Retinal temperature in °C above ambient estimated from model of White *et al.,* 1971. Transmittance through monkey ocular media is 0.702.

Exposure time(s)	P_c(mW)	Transmittance(T)	H_0 in W/cm^2
1	33.1	0.702	5.854249194
1	27.9	0.702	4.934548415
10	19.5	0.702	3.448877924
10	15.9	0.702	2.812162
100	2.17	0.702	0.38379821
100	1.83	0.702	0.323663928
1000	0.27	0.702	0.047753694
1000	0.19	0.702	0.033604452

Table 10: Exposure of the rhesus monkey retina to the visible component of the SSS at sea level, 400-800 nm, for time intervals of 1, 10, 100, 500 s. Biological criterion was a minimal retinal lesion as funduscopically detectable at 48 h post exposure. Beam diameter on retina, 159 μm. Power, P, incident on cornea in mW, retinal radiant exposure H_0, in J/cm^2. Retinal temperature in °C above ambient estimated from model of White et al., 1971. Transmittance through monkey ocular media, 0.794

Exposure time(s)	P_c(mW)	Transmittance(T)	H_0 in W/cm^2
1	18.66	0.794	3.732830249
1	16.74	0.794	3.348744822
10	12.09	0.794	2.418537927
10	10.31	0.794	2.062458728
100	0.96	0.794	0.192042714
100	0.88	0.794	0.176039154
500	0.21	0.794	0.042009344
500	0.15	0.794	0.030006674

VI. RESULTS AND DISCUSSION

Table 1 and 2 the irradiation increases with increase in wavelength and exposure time but in near infrared i.e.; at wavelength 1064 nm it increases very sharply. At wavelength 632.8nm and 100s exposure time, the irradiance remains at a constant stage and then increases sharply. This constant stage shows that at visible spectrum the irradiation doesn't change with this exposure time.

From table 2 and 4 shows that the temperature in the retina increases with increased exposure time for different wavelengths. When the exposure time increases i.e. for 1000s exposure time the temperature of retina rises slowly with increase in wavelength. But for a particular wavelength with increase in exposure time the temperature of retina decreases. From the graph at 1sec exposure time and wavelength from 441.6 nm to 580 nm, the temperature of retina increases but for 610 nm it suddenly decreases. This may be due to the photochemical reason and then increases sharply causing photothermal injury.

From table 5, for 500μm beam size and for 1sec exposure time, the irradiance increases with increase in wavelength but at 610nm decreases and then 632.8nm it increases and after that it decreases for near infrared i.e; for 1064 nm wavelength at 1,16,100,1000sec exposure time it increases very sharply.

For (300-1400 nm) and different exposure time and power incident on the cornea and a particular transmittance 0.702 mw, the irradiance decreases with increase in exposure time but after 100sec it decreases linearly.

After changing the wavelength for (400-800 nm), transmittance 0.794 mw the irradiance for different exposure time is same.

VII. CONCLUSION

This study demonstrates a clear correlation between irradiation and temperature rise in the retina, emphasizing the critical roles of wavelength, exposure time, and transmittance in photothermal damage. The findings reveal that irradiance increases significantly with longer wavelengths, particularly in the near-infrared spectrum (e.g., 1064 nm), while retinal temperature exhibits a nonlinear dependence on exposure duration. The incorporation of the Stiles-Crawford effect (SCE I) further refines the understanding of thermal distribution in retinal tissues. Notably, changes in transmittance (e.g., from 0.794 to 0.702) yield

consistent irradiance patterns for varying corneal power inputs, suggesting predictable thermal responses under different optical conditions. These results underscore the importance of controlling exposure parameters to mitigate retinal damage, particularly in applications involving lasers or intense light sources. Future research should explore additional biological variables, such as pigmentation differences, to enhance predictive models for retinal safety.

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