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A New Factor (time constant) to Specify Class I and Class II Photochemical Damage

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***Abstract*:** Photochemical injury to the retina involves at the central part (mainly in outer layers) of the retina. There are two classes of photochemical damage i.e. Class I and Class II. Class I damage is generally occurred in photoreceptors while Class II is in Retinal Pigment Epithelium (RPE). Occasionally, the Class II damage is found in photoreceptors also. Factors which play crucial role in the determination of the class of damage include animal species/ model, exposed retinal area, concentration of visual pigment. Here we include the relationship between the irradiance and the time constant (the time at which the retinal damage increases or decreases exponentially) and how the irradiance of the retina (a crucial component for photochemical damage) is governed by the directionality of cone photoreceptors which is the site for Class I damage. The relationship between irradiance (E) and the rate of change of time constant (t) with respect to E ($\frac{dt}{dE}$) has also been studied to distinguish between class I & II.

***Index Terms*:** Photochemical Damage, Irradiance, Time Constant, Exposure time, Action Spectrum, Cone Directionality, Interference Pattern, Pupil Entrance Point.

I. INTRODUCTION

All we know that light helps us to show anything but it may be harmful for us .In extensive research it is clear that there are three types of damage occur in the eye(retina) namely photomechanical damage, photo thermal damage, and photochemical damage. Light energy must be absorbed in order to cause pathological changes. The portion of energy absorbed in any tissue depends on the transparency of the tissue for the incident, whereas the transparency depends on the wavelength of the light.

Light is converted into neural signal through a series of delicate process [1]. Here we only concentrate on the photochemical damage. Photochemical damage involves cellular damage by supercharged molecules e.g. sunburn of the skin is a familiar human photochemical damage. In photochemical damage the absorption of light leads to the production of toxic substances with a negligible rise in temperature .Photochemical damage has only recently been recognized and has been demonstrated to occur with excessively high light levels (e.g. Ham et al [2]. It has also been reported after long lasting exposures to ambient light (e.g. Levels (e.g. Noel et al [3].

In literature survey it is clear that the photochemical damage seems to have two distinctly action spectra [1,4]. (i.e. visual pigment and UV action spectrum). Again, it concerns the relation between exposure time for threshold damage and irradiance level [1,5].

Photochemical damage distinguished in to two broad categories one is class I and other is class II. Class I damage was first observed by Noell and he notified that in response to long duration exposures (>8h) to constant green light (490-580μm) light, this damage occur. The class I damage is also known as Noell damage [6]. This damage is reported by various workers in Reme, 2005 in mice [7], Harwerth and Sperling 1975 [8] in macaque, Penn 1985 in fish [9], Chicken Machida, 1994 in Chicken [10] etc. However, none of the studies in species other than rats measured an action spectrum, thus the evidence for Noell damage is circumstantial.

Whereas class II damage was first observed by Ham and he showed this type of damage in very high light levels .This damage is mainly in RPE (Retinal Pigment Epithelium). This damage has been studied in monkeys (Ham et. al. 1976) [2], rabbits (Hoppler et al. 1988) [11], rats (Busch et al. 1999) [12] and squirrels (Collier et al. 1989) [13]. In this type the damage is generally confined to the pigment epithelium. These experiments were mainly done on anesthetized primates and only small parts of retina were irradiated.

In this paper here we describe a theoretical description based on time constant for clarify the photochemical damage i.e. the distinction between class I and class II by considering the different et al made by different scientists. And also we give an equation which gives the relationship between class I and class II damage.

II. DIFTERENT FACTORS FOR THE DETERMINATION OF THE CLASS OF DAMAGE

There are so many crucial factors which determine the class of damage. Here we will discuss four different suggestions for this factor, there of which have been proposed previously. As a fourth, we introduce a new suggestion which may lead to clarify the class of damage field into consideration. The only exception is occurred in rabbits with relatively large field but the type is class II [21,22]. It is still unclear that why field size would influence vulnerability, action spectrum and cellular site of damage. It is also here we mention that the use of anaesthetics is unlikely to be a crucial factor in determining class. And deep anaesthesia might slower down the vulnerability to damage [23].

Animal Model

Class I and Class II damage were found in rodents and primates respectively. The action spectra are available for these types of animal species. Nocturnal animals are more vulnerable to light damage as compare to diurnal animals [1]. This is true for environmental light level but at the retinal level this is less evident [14]. It may be clear that all experimental aspects show that class I damage can also be attributed to monkeys [15-17] and pigeons [18], at retinal irradiance level similar to rats. Class II damage was found in nocturnal owl monkeys [19] and primarily nocturnal rabbits.

Thus, animal model seems not the most likely factor determining class of damage.

Size of exposed retinal field

Ham and Mueller [20] suggested that the size of irradiated field could be the determinant factor for determining the class of damage. For class I, Large fields would only need low levels of light whereas for class II, small fields would need high levels of light [1]. So, class I and class II damage are completely separated by taking the Concentration of visual pigment.

Usually the visual pigment cannot mediate damage when all pigment is bleached [23]. In this condition another pigment, or a mixture of pigments, with an absorption spectrum peaking in the UV, should induce the damage. The change in mechanism and spectrum should occur in the intensity domain where bleaching in known to exhaust the store of visual pigment because of the concentration of visual pigment. From the literature, it may be assume that the visual pigment is much more effective in inducing damage then the other pigment(s). This is concluded from the action spectrum found in rats at irradiances about a factor 10 higher than the half bleach irradiance [3]. Though

the concentration of visual pigment is important but the repetitive exposures are more damaging than one exposure of the same irradiant dose. When visual pigment is repetitively allowed to regenerate, it has a higher mean concentration.

Value of Time constant

Time constant is defined as the time at which the retinal damage increases or decreases exponentially. We may derive the relationship between irradiance and exposure time at threshold by assuming that the concentration of a toxic agents ("C" in units of molarity, M) is determined by the balance between production and decay. (''Production'' is proportional to retinal irradiance E in mW. Cm^{-2} and damage Vulnerability h, depending on the action spectrum.)

(And ''decay'' is proportional to ''C'')

So, from the above we will get,

$$\frac{dC}{dt} = hE - \frac{C}{\tau} \qquad (1)$$

The differential equation has a solution.

$$C = hE\,\tau\,[1 - \exp(-t/\tau)] \qquad (2)$$

Hence, $$t = \tau \ln \left\{\frac{E}{E-(C/h\tau)}\right\} \qquad (3)$$

For threshold damage,

C is considered to have a constant value.

For Class II – When the concentration of the damage inducing pigments does not depend on E, a fit with class II threshold data can be obtained by substituting.

$$\tau = 3.5 \times 10^5 S.$$

And optimizing the parameter ($C/h\tau$)

$$t = 3.5 \times 10^5 \ln \left(\frac{E}{E-1.04}\right) \qquad (4)$$

The value of the time constant is conspicuously similar to the repair, time constant of UV- induced damage to the cornea suggested to be 1.7×10^4s.

So when damage is mediated by a pigment with light dependent concentration, then the vulnerability of tissue (h) depends in irradiance.

The relation between t and E for class II damage asymptotes at about 1 mW cm^{-2}, which is about 50 times the irradiance of half bleach.

So we have to consider the bleach products as constant.

For class I: The above situation is different, because the damage mediating pigments are photo labile and concentration of these pigments show large changes within the irradiance.

Let P_R be the fraction of unbleached pigment relative to fully regenerated condition, and R_{λ}. be the absorption spectrum of unbleached pigment.

hE (in $M.S^{-1}$) is then the weighted integral of spectral retinal irradiance (E_{λ}).

The absorption of spectrum of unbleached pigment = $P_R . E_{\lambda}$ For white light effects of self screening can be neglected.

$$hE = \eta_R \int E_{\lambda} P_R R_{\lambda} . d\lambda \qquad (5)$$

Where η_R is the efficiency to induce damage after absorption of one photon by the visual pigment.

For rhodopsin the steady state bleach ratio is,

$$P_R = \frac{E_0}{E+E_0} \qquad (6)$$

Where, $E_0 = 0.02 mW\ cm^{-2}$

For a fixed spectral composition of the irradiant source the equation (5) can be written as;

$$hE = \eta'_R \frac{E.E_0}{E+E_0} \qquad (7)$$

Where, η'_R (in $M.cm^2\ S^{-1}\ .mW^{-1}$) is the efficiency of rhodopsin to cause damage with the light source used.

Combining equation (2) & (7) we will get,

$$C = \eta'_R \frac{E.E_0}{E+E_0} \tau\ [1\text{-}\exp(-t/\tau)]$$

$$\text{Or, } 1\text{-}\exp(-t/\tau) = \frac{C(E+E_0)}{\tau\ \eta'_R.E.E_0} \qquad (8)$$

Hence taking ln on both sides we get,

$$t = \tau \ln\{\frac{C(E+E_0)}{\tau\ \eta'_R.E.E_0}\}$$

$$\text{If } \frac{C}{\tau\ \eta'_R.E_0} = \mu$$

Then we will get,

$$t = \tau \ln\{\frac{E}{(1-\mu).E_0 - \mu E_0}\} \qquad (9)$$

A fit with class I threshold data was obtained by putting, the unknown E_0 & τ & $\mu = 0.08$. Thus

$$t = 3.5 \times 10^5 \ln\{\frac{E}{0.92E - 0.0016}\} \qquad (10)$$

This is the equation for time constant for class II.

The relation for both class of damage would obtain by adding equation (4) & (10).

Equation (4) is,

$$t = 3.5 \times 10^5 \ln(\frac{E}{E - 1.04})$$

& Equation (10) is,

$$t = 3.5 \times 10^5 \ln\{\frac{E}{0.92E - 0.0016}\}$$

$$So,\ t+t = 3.5 \times 10^5 \ln(\frac{E}{E-1.04}) + 3.5 \times 10^5 \ln\{\frac{E}{0.92E-0.0016}\}$$

$$Or,\ 2t = 3.5 \times 10^5[\ln(\frac{E}{E-1.04}) + \ln\{\frac{E}{0.92E-0.0016}\}]$$

$$Or,\ t = 1.75 \times 105\ [\ln \frac{E^2}{(E-1.04)(0.92E-0.0016)}\ ] \qquad (11)$$

This is equation for relationship between time constant and irradiance of both classes. The different value of 't' are given in table.

III. DERIVATION OF $\frac{dt}{dE}$ FOR CLASS I PHOTOCHEMICAL DAMAGE.

We know for class I,

$$t = 3.5x10^5 \ln(\frac{E}{0.92E - 0.0016})$$

Differentiating both sides we will get,

$$dt = 3.5 \times 10^5\ d\ [\ln f(x)]$$

$$= 3.5x10^5 \frac{1}{f(x)}\ d\ f(x)$$

$$= 3.5 \times 10^5 (\frac{0.92E-0.0016}{E})\ d\ (\frac{E}{0.92E.0.0016})$$

$$= 3.5 \times 10^5 \left(\frac{0.92E-0.0016}{E}\right) d\{E\,(0.92E- \quad 0.0016)^{-1}\}$$

$$=3.5x10^5\left(\frac{0.92E-0.0016}{E}\right) d\,(0.92E^2+0.0016)$$

$$=3.5x10^5\left(\frac{0.92E-0.0016}{E}\right) d\,(1.84E+0.0016)$$

$$= 3.5x10^5 \left(\frac{(0.92E-0.0016)(1.84E+0.0016}{E}\right)$$

$$= 3.5\ X10^5 \text{ x } [0.92\ X1.84E2 +0.92X0.0016E\text{-}1.84x0.0016E– (0.0016)^2]$$

$$\frac{dt}{dE}= 3.5 \text{ x } 10^5[1.693E\text{-}00.0015]$$

IV.DERIVATION OF $\frac{dt}{dE}$ FOR CLASS II PHOTOCHEMICAL DAMAGE.

We know for Class II,

$$t = 3.5 \text{ x } 10^5 \ln \{ \frac{E}{E-1.04}\}$$

Differentiating both sides we will get,

$$\text{Or, } dt \quad =3.5 \text{ x } 10^5\left(\frac{E-1.04}{E}\right) d\left(\frac{E}{E-1.04}\right)$$

$$= 3.5 \text{ x } 10^5 \left(\frac{E-1.04}{E}\right) d\,\{E\,(E-1.04)^1\}$$

$$= 3.5 \text{ x } 10^5 \left(\frac{E-1.04}{E}\right) d\,(E^2-1.04)$$

$$= 3.5 \text{ x } 10^5 \left(\frac{E-1.04}{E}\right) d\,(2E-1.04)$$

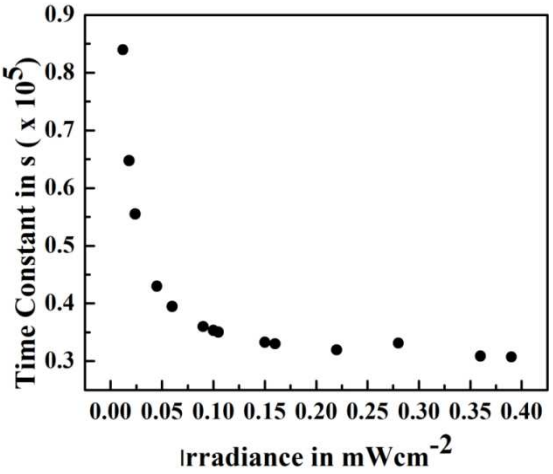
$$= 3.5 \text{ x } 10^5 \left(\frac{(E-1.04)(2E-1.04)}{E}\right)$$

$$=3.5x10 \quad \frac{E\ X\ 2E+X\ 1.04-1.04\ X\ 2E-1.04\ X\ 1.04}{E}$$

$$\frac{dt}{dE} = 3.5 \text{ x } 10^5\ [2E\text{-}1.08]$$

V.RESULTS

The graphs plotted for class I damage between E and t and that for class II are different. The ratio of maximum retinal irradiance to the minimum is same as the ratio of maximum exposure time to the minimum exposure time for class I photochemical damage. But the ratio of exposure time is three times the ratio of irradiance for class II damage. That is, class I damage is three times more sensitive to exposure time than class II damage.



VII. CONCLUSION

Photochemical injury to the retina involves at the central part (mainly in outer layers) of the retina. There are two classes of photochemical damage i.e. Class I and Class II.Class I damage is generally occurred in photoreceptors while Class II is in Retinal Pigment Epithelium(RPE).Occasionally the Class II damage is found in photoreceptors also.

Factors which play crucial role in the determination of the class of damage include animal species/ model, exposed retinal area, concentration of visual pigment. Here we include the relationship between the irradiance and the time constant (the time at which the retinal damage increases or decreases exponentially) and how the irradiance of the retina (a crucial component for photochemical damage) is governed by the directionality of cone photoreceptors which is the site for Class I damage.

The relationship between irradiance (E) and the rate of change of time constant (t) with respect to E($\frac{dt}{dE}$) has also been studied to distinguish between class I & II.

The ratio of maximum retinal irradiance to the minimum is same as the ratio of maximum exposure time to the minimum exposure time for class I photochemical damage. But the ratio of exposure time is three times the ratio of irradiance for class II damage. That is, class I damage is three times more sensitive to exposure time than class II damage. Though the time constant decreases with increase of retinal irradiance, the time constant per unit irradiance increases with increase of irradiance, for both classes of damages. An interference pattern generated on the retina by two coherent beams alters the

retinal irradiance according to the contrast present for class I PCD. When the contrast is unity, the retinal irradiance (or PCD) reaches the peak irrespective of the position of beams' entry. But when the contrast is zero, the retinal irradiance and thus PCD depends on the position of beam entry, maximum for axial entry and 24% of the maximum for most peripheral entry. Irradiance (or PCD) mimics cone directionality. When the contrast lies between 1 to 0, the irradiance and thus the PCD is controlled both by the cone directionality and contrast of the interference pattern.

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