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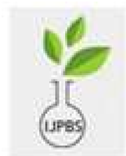

International Journal of Pharma and Bio Sciences

ISSN 0975-6299

ATTENUATION OF RETINAL DAMAGE AFTER EXPOSURE TO WHITE LIGHT BY TAURINE ASSESSED BY ELECTRORETINOGRAM AND COMET ASSAY

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ABSTRACT

The purpose of this study is to investigate the protective effect of taurine against photochemical damage in rabbit retina. Twenty seven rabbits were divided into three groups: control group (A), exposed to light without (B) and with taurine (C). Groups B and C were divided into four subgroups according to the time of exposure to white light (3, 6, 12 and 24 hr). Group C treated with taurine (4 g taurine /100 g diet) for 2 weeks before exposure to white light (2500± 100 lux). Retinal function was assessed by electroretinogram (ERG). The comet assay also was used to examine qualitative and quantitative aspects of DNA. Statistically significant reduction in a- and b - waves was apparent ($p < 0.05$) in group (B) that exposed to light and reach maximum reduction after 24 hr of exposure, the percentage difference was 81 % and 83 % for a-and b- waves respectively. The reduction of a-and b- wave amplitude after light exposure was attenuated by taurine intake. Also, the extent of DNA damage proportionally increased with increasing time of exposure to white light that appear in the elevated mean percentage tail DNA and tail moment that reached 6.16±0.4% and 14.2% respectively at 24 hr of exposure to white light. Taurine effect was appeared clearly by decreasing this percentage to 3.3±0.2% and 5.2% for the same group. Our results revealing the importance of taurine that played neuroprotective role in retina after exposed to white light.

KEYWORDS: Taurine, White light, Rabbits, ERG and Comet assay.

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INTRODUCTION

Excessive light exposure leads to photoreceptor degeneration in many animals [1,2] , and can be a risk factor for onset and progression of age-related macular degeneration and possibly some forms of retinitis pigmentosa[3]. Photoreceptor cell death is an irreversible injury and can cause night blindness, constriction of the visual field, and finally the loss of central vision. Photoreceptor cell death has been reported to be induced by various factors[4], such as calcium levels, nitric oxide (NO), reactive oxygen species, mitochondria, and so on. In particular, the retina consumes significant amounts of oxygen in the human body and easily produces reactive oxygen species such as superoxide (O_2•−) and hydrogen peroxide (H_2O_2). The oxidation capacity of these radicals is weak, but they react to metals within a living body and light and immediately change to hydroxyradicals (•OH). Hydroxyradicals are the most reactive radicals that can injure DNA and the cell membrane. Thus, oxidative stress disrupts the normal balance of antioxidation and oxidation response in the human body, and is involved in the progression of many diseases, including retinal diseases. Oxidative stress can contribute to neuronal toxicity and has been implicated in neuronal cell death[5]. Many researchers have investigated the efficacy of antioxidants such as ascorbate[6,7], dimethylthiourea[8], thioredoxin[9] , phenyl-N-tertbutylnitrone[10,11], and 4-hydroxy-2,2,6,6-tetramethylpiperidine-N-oxyl[12] against light-induced retinal damage. Thus, oxidative stress is likely to be involved in the pathogenesis of light-induced retinal damage.

Taurine or 2-aminoethanesulfonic acid (fig 1) is a derivative of cysteine, an amino acid which contains a sulfhydryl group. It is one of the few known naturally occurring sulfonic acids[13]. Taurine is abundant in the retina, especially in photoreceptor cells and Mϋller cells[14, 15]. It not only acts as a neuromodulator inhibitor, Ca modulator and osmoregulator, but also interferes with the metabolism of lipid synthesis and stabilizes the membrane system[16]. It is documented that taurine can inhibit lipid peroxidation, thereby protecting the retina from oxidative damage[17,18]. Some studies have also indicate an anti-apoptotic effect of taurine[19,20].

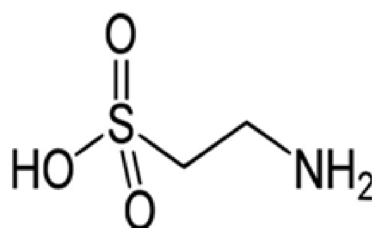

Figure1.
General structure of taurine

The electroretinogram (ERG) recorded at the corneal surface provides a useful tool to measure outer retinal activity [21]. ERG recordings have found application to the animal retina, where the ERG has been used to characterize retinal function following the manipulation of gene expression in photoreceptors and other retinal neurons [22, 23]. Because the ERG can be recorded noninvasively, the response also provides a useful means to monitor the severity and progression of retinal

degeneration[24, 25] as well as to evaluate the effects of experimental treatment strategies in animal models of hereditary retinal disease [26]. Most of the associated lesions after exposure to white light can give rise to DNA strand breaks. The comet assay (also termed the single-cell gel electrophoresis assay) is a rapid and sensitive method used for the detection of DNA strand breaks in retina[27]. It can therefore provide valuable information regarding the molecular mechanisms that counteract light insult.

The present study was undertaken to evaluate the protective efficacy of taurine against damage induced by white light (2500±100 lux) in albino rabbits using ERG and comet assay.

MATERIALS AND METHODS

i) Animals and chemicals

New Zealand white rabbits of either sex, weighing 2-2.5 Kg were selected from the animal house facility at the Research Institute of Ophthalmology, Giza, Egypt. The research protocol was approved by the local ethical committee that applies the ARVO (the association for research in vision and ophthalmology) statements for using animals in ophthalmic and vision research. All chemicals were purchased from Sigma Chemical Co (USA).

ii) Exposure to light

After dark adaptation for 24 h, the pupils were dilated with 1% cyclopentolate hydrochloride eye drops 30 min before exposure to light. Non-anesthetized rabbits were exposed to intense light 2500± 100 lux of white fluorescent light (Toshiba, Japan) that was calibrated at the Photometry Department, National Institute of standards, Giza, Egypt. The time of exposure was 3, 6, 12, and 24 hours beginning at 8:00 AM in cages with reflective interiors. The temperature during the exposure to light was maintained at 25±1.5 °C.

iii) Treatment with taurine

27 rabbits were divided into three groups control group (A), exposed to light without (B) and with taurine (C). Groups B and C were divided into four subgroups according to the time of exposure to white light (3, 6, 12 and 24 hr). Group C treated with taurine (4 g taurine /100 g diet)[28] for 2 weeks before exposure to white light.

iv) Electroretinogram

Retinal function was assessed by electroretinogram (ERG), animal were kept in total darkness for at least 60 min. All subsequent ERG procedure was performed under dim red light. Rabbits were anesthetized by injection with 0.1 ml/Kg Xyla-ject as muscle relaxant firstly. After 15 min, 50 mg/ Kg ketamine hydrochloride was administrated intramuscularly. The supplementary doses of an anesthetic were given as needed, the rabbit's body temperatures were maintained at 37°C and the eye was dilated with one drop of 1% isopto-Atropine. ERG was recorded by using three Ag-AgCl electrodes. The active electrode was a wick electrode placed at the corneal periphery. The other two electrodes were placed on the skin of the lower eyelid and on the ear, as reference and earthed electrodes, respectively[29]. White flash was used in this work with fixed intensity (4 lux) and duration (0.2 s). The resulted electrophysiological signals were pre-amplified using CEPTU preamplifier (Bioscience, UK). The pre-amplified signals were delivered to a computer system attached to a digital oscilloscope (Velleman Co., Taiwan), to be recorded and analyzed by the provided software.

v) Comet assay

One gram of crushed samples were transferred to 1 ml ice-cold PBS (phosphate buffer saline, pH 7.9). This suspension was stirred for 5 min and filtered. Cell suspension (100 µl) was mixed with 600 µl of low-melting agarose (0.8% in PBS), where 100 µl of this mixture was spread on the slides. The coated slides were immersed in lyses buffer (0.045 M TBE, tris borate EDTA pH 8.4, containing 2.5% SDS) for 15 min. The slides were then placed in electrophoresis chamber containing the same TBE buffer, but devoid of SDS. The electrophoresis conditions were 2 V/cm for 2 min and 100

mA. Finally, the slides were stained with ethidium bromide (EtBr) 20µg/ml at 4°C. The observation was the samples still humid and the DNA fragment migration patterns of 100 cells for each dose level were evaluated with a fluorescence microscope (With excitation filter 420-490 nm [issue 510 nm]). The comets tail lengths were measured from the middle of the nucleus to the end of the tail with 40x increase for the count and measure the size of the comet. For visualization of DNA damage, observations are made of EtBr-stained DNA using a 40x objective on a fluorescent microscope. Although any image analysis system may be suitable for the quantitation of single cell gel electrophoresis (SCGE) data, we use a Komet 5 image analysis software developed by Kinetic Imaging, Ltd. (Liverpool, UK) linked to a charge-coupled device (CCD) camera to assess the quantitative and qualitative extent of DNA damage in the cells by measuring the length of DNA migration and the percentage of migrated DNA. Finally, the program calculates tail moment. Generally, 50 to 100 randomly selected cells are analyzed per sample[30].

vi)Statistical Evaluation

Data were represented as the mean ± SD. For comparison between multiple groups the analysis of variance (ANOVA) procedure was used, where a commercially available software package (SPSS-11, for windows) was used and the significance level was set at $p < 0.05$.

RESULTS

Figure (2) shows the typical ERG results for control rabbits as well as for those animals exposed to 3,6,12 and 24 hours of white light (2500±100 lux) and those rabbits treated with taurine. For the control rabbits, the a-wave amplitude which corresponds to photoreceptor function was on the average of 1371±50 µv while the average b-wave which reflects the electrical activity in the inner retina subsequent to photoreceptor stimulation was 2031±90 µv.

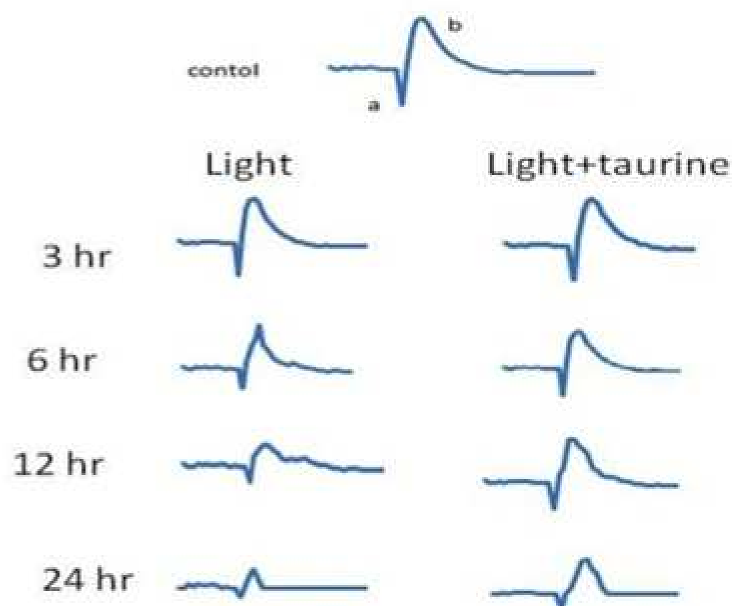

Figure 2.
Dark adapted ERGs records for control rabbits and those exposed to light for 3, 6, 12 and 24 hr with or without taurine.

Statistically significant reduction in a- and b - waves was apparent in figures (3, 4). For exposure times equal to or exceeding 6 hours the percentage difference was 26% and 30 % for a and b-waves respectively. Increasing the exposure time to 12 hours in resulted was associated with a percentage difference of 61 % and 62 % for a-and b-waves respectively, longer exposure (24 hours) led to more severe effect, where the percentage difference was 81 % and 83 % for a-and b-waves respectively. However fig (3,4) importantly, the amplitudes of a- and b-waves after light exposure remained significantly larger in the rabbits who had given taurine compared with those control after 6 hours of exposure. There were no difference in the components of ERG between normal rabbits and rabbits treated with taurine except lower a-wave amplitude, the percentage difference of a-wave were 11%. The percentage difference was 23% and 44% for a-wave and 10% and 19% for b-wave after light exposure to 12 and 24 hours respectively thus reduction of a-and b-wave amplitude after light exposure was attenuate by taurine intake.

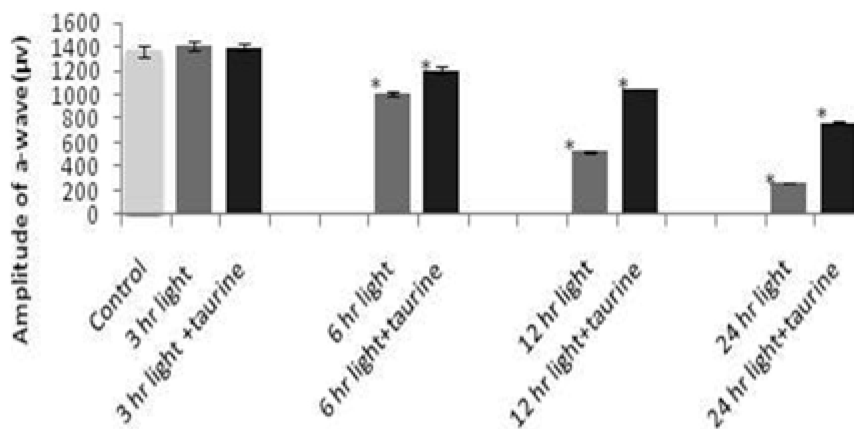

Figure 3.
Different light exposure time and amplitude of a-wave (μv) in rabbits treated with or without taurine. *Statistically significant

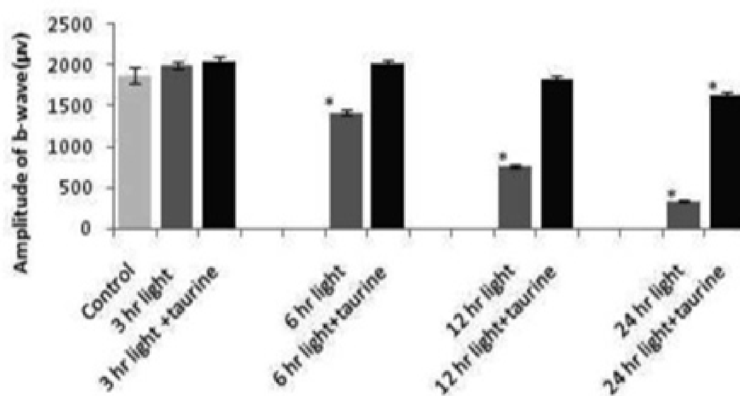

Figure4.
Different light exposure time (hr) and amplitude of b-wave (μv) in rabbits treated with or without taurine. *Statistically significant

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When cells processed for the comet assay were examined by fluorescence microscopy, fluorescent structures corresponding to the ethidium bromide–stained nuclear DNA of the cells was revealed. In undamaged (control) cells the DNA was tightly compressed and maintained the circular disposition of the normal nucleus (fig. 5A). After exposure of the rabbits to white light, the profile of the nuclear DNA was altered with the appearance of a fluorescent streak extending from the nucleus (fig. 5 panel 1-4). Also appear of some repair and less damage of cells after taurine supplementation (fig. 5 panel 5-8). Cells containing damaged DNA have the appearance of a comet with a bright head and tail. In contrast, undamaged DNA (in unaffected cells) remains tightly coiled and appears after the electrophoresis as an intact nucleus with no tail, thus allowing the cells' differentiation from those that have incurred DNA damage.

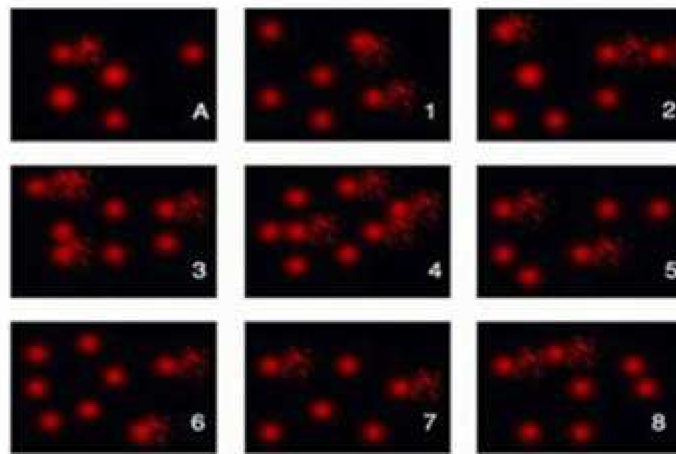

Figure 5.
Retinal cells were processed by alkaline comet assay. Group A were the rabbits control group,Group B (1-4) were the rabbits exposed to white light for 3,6,12 and 24 hr and Group C (5-8) were the rabbits exposed to white light after taurine treatment

The mean percentage of tail DNA (fig 6), reflecting the proportion of DNA that has migrated from the head, is then calculated as an average for the 50-100 cells selected for measurement. The mean percentage of tail DNA in control is 1.74±0.3% then significant increased ($p<0.05$) with time of exposure to white light and reached the maximum percentage of 6.16±0.4% at 24 hr of exposure to white light. After taurine treatment, the mean percentage of tail DNA is significant decrease for all groups compared with those exposed to light for all periods except at 3 hr of exposure to white light with or without taurine indicate no significant difference in mean percentage of tail DNA. In addition, a parameter mean tail moment whose magnitude reflects the frequency of DNA strand breaks per nucleus was calculated and illustrated as a histogram in fig (7). The mean tail moment for control group was 3.27±0.2. Its values was increased by a maximum percentage of about 14.2% for the longest period of exposure to white light which is 24 hr. Taurine effect was appeared clearly by decrease this percentage to 5.2% for the same group. Figures (8) and (9) illustrated the percentage tailed cells and tail length for control and all groups exposed for white light with or without taurine. The results of these two parameters indicate the same phenomena for mean tail moment in which taurine decrease the effect of white light but not reaching the control limit

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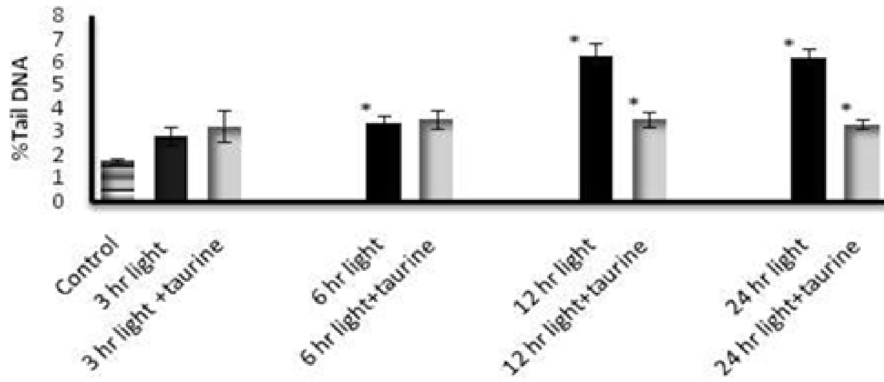

Figure 6.
Mean percentage of tail DNA to control rabbits and groups exposed to white light with or without taurine for all periods 3,6,12 and 24 hr.*Statistically significant

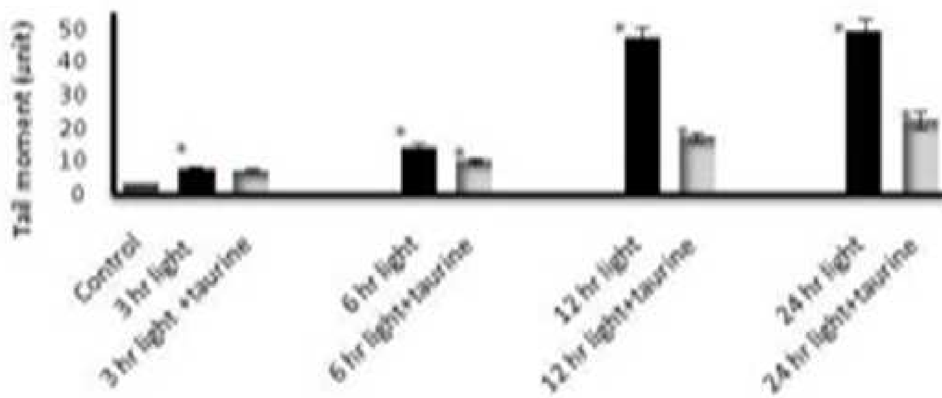

Figure 7.
Tail moment (unit) whose magnitude reflects the frequency of DNA strand breaks per nucleus for control and exposed to light with or without taurine. *Statistically significant

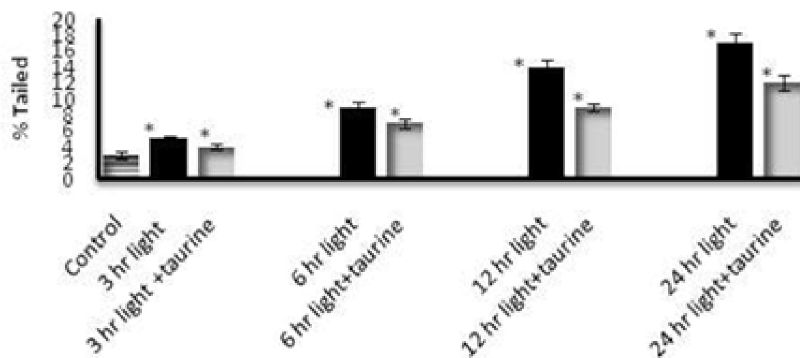

Figure 8.
Percentage tailed cells for control group, exposed to white light group, and group treated with taurine before exposed to white light. *Statistically significant

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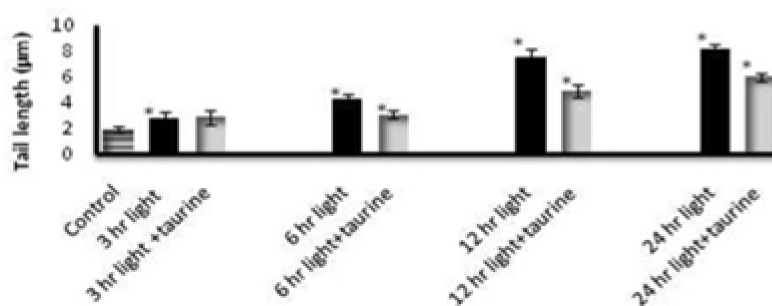

Figure 9.
Tail length for control and exposed to white light with or without taurine to all periods (3,6,12 and 24 hr).*Statistically significant

DISCUSSION

Light has both beneficial and detrimental effect on cellular macromolecule[31]. In mammalian cells light mediates synthesis of vitamin D[32]. In plants light is responsible for photosynthesis and mediates many other cellular effects via generation of hydrogen peroxide[33]. In bacteria white light induces repair of UV-induced DNA damage by photoreactivation. Light-induced retinal cell death is caused by an increase in intracellular calcium levels, nitric oxide (NO), rhodopsin mutation, and free radicals, including reactive oxygen species[4]. Excessive light exposure induces many reactive oxygen species, including free radicals, and their production can be overcome by a retinal defensive mechanism, such as superoxide dismutase (SOD)[34]. Free radicals damage DNA, RNA, Proteins, and enzymes, lead to the formation of tumors and cause cancers, cardiovascular diseases, Nervous disorders, premature ageing, and pulmonary disorders[35].

Taurine is present at high levels in the retina of many vertebrate[36].This amino acid is known to possess neuroprotective and neurotrophic properties in the central nervous system during development and regeneration[37-40]. Mammals synthesise taurine from sulfur precursors, but the ability of different species to do so varies greatly[39]. Dietary sources of taurine are thus necessary for those animals that cannot synthesise sufficient taurine, for example, the cat and man. Dietary taurine is absorbed via the digestive system and then is transported by the Na^+- dependent taurine transporter into the retina through the blood–retinal barrier[41]. The role of taurine that may be played in the retina is the promotion of retinal cell differentiation during rod photoreceptor development[42]. In the present study we investigate the role of taurine to minimize light effects. The ERG findings indicated that visual function after light exposure was impaired, consistent with previous reports[43]. The reduction in a-wave amplitude after light exposure, which reflects photoreceptor cell damage, was consistent with the finding of photoreceptor cell loss through comet assay .The reduction in b-wave amplitude, which reflects the neural transduction from the photoreceptor cells, most likely corresponded with the degeneration of photoreceptor cells. These changes were attenuated by the intake of taurine. Therefore, these data demonstrated that taurine has a protective effect in the photoreceptor cells against light induced retinal degeneration. By means of the comet assay, the present study has elucidated some of the molecular changes subsequent to white light .In the comet assay a damaged cell takes on the appearance of a comet, with head and tail regions. A variety of geometric and densitometric parameters are provided by the image analysis software, which allows an estimation of the amount of

DNA in the head and tail regions and the extent of migration into the tail region. Because the tail length and density reflect the number of single-strand breaks in the DNA, the percentage of DNA in the tail provides a quantitative measure of the damaged DNA. As seen in the dose–response experiments of the present study, the extent of DNA damage increased proportionately with increasing time of exposure to white light. Also elevated mean tail moment, indicative of DNA damage. After treatment with taurine all comet assay parameters were decreased that means cells are able to repair sublethal DNA damage so that most cells show decreasing tail moments that indicating minimizing the introduction of strand breaks arising from DNA repair by the cells. Possible function of taurine in retina are protection of photoreceptor-based on the shielding effects of taurine on rod outer segment, regulation of Ca^{2+} transport-based on the modulatory effects of taurine on Ca^{2+} fluxes in the presence and absence of ATP (adenosine triphosphate) and regulation of signal transduction-based on the inhibitory effects of taurine on retina phosphorylation[44,45].

CONCLUSION

Our results revealed the importance of minimizing the use of white light. This study shows that white light at intensity of 2500 lux produces detectable DNA damage in retinal cells and taurine played neuroprotective role in retina after exposed to white light. This damage can be detected by means of ERG. Also suggest the possible use of comet assay to evaluate the role of antioxidants in preventing retinal damage.

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