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Research Article

Retinal Light Damage Through Prolonged Visible Light Exposure

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Abstract

Computers and laptops are increasingly used at work and at home. As a consequence, the exposure of short wavelength blue light, which is emitted by LED displays, increases too. By its action, light is well-known to enact damage on the neurosensory retina, especially on the underlying structures. In particular, this happens through photothermal, photomechanical, and photochemical mechanisms. This paper will point out what kind of medical, respectively pathological, changes within the eye happens and how much of visible light the eye can withstand before damage occurs.

Keywords: *Blue Light; Light-Emitting-Diodes (LED); Computer; Damage; Eye*

Introduction

The ability of light to cause damage to the retina has been researched for hundreds of years. If light penetrates the eye it potentially can damage the retina, especially the photoreceptors and the retina pigment epithelium (RPE) [1]. Furthermore, this can lead to the loss of vision, which may regress with recovery, however, just slightly depending on the length of exposure. Furthermore, there are remarkable similarities between light damage and changes found in advanced atrophic macula degeneration. Organisciak., *et al*. [2] conducted a clinical study on adult albino rodents and found out that light-induced-retinal-damage (LIRD) lead to a reshape of the neurons within the retina. Sparrow, Nakanishi and Parish [3] explored blue light-induced damage to the retinal pigment epithelium (RPE). They debated that this leads to blood-retinal barrier dysfunction and sub-retinal decreases in the apical-to-basal transport by the amino acids Leucine and Chloride. Particularly, there was a quantifiable and significantly reduced growth of the RPE and endothelial cells after a nineteen hours blue-wavelength light exposure. Behar-Cohen., *et al*. [4] argued that the eye is exposed to artificial light sources, especially in daily life, around 2650 billion MWh/year, which represents almost nineteen percent of the worldwide electricity production. Yet, the impending risks of these new light sources have to be explored. They give some comprehensive and objective analysis to avoid potential risks.

This brings up the questions, light is dangerous or can light threaten the sight just by working on a computer? Furthermore, a detailed clarification and definition of visible light and possible pathological changes are reviewed from different points of view.

LED Technology

A Light-Emitting-Diode (LED) is principally monochromatic which means that it has a small wavelength spectrum compared with a phosphor emission spectrum [4]. Visible light is seen by the adult human eye in the range of 380 - 780 nanometers. Like all radiations, light carries energy, i.e. the shorter the wavelength is, the more energetic the light is. Figure 1 shows the spectral power distribution of LED and phosphorous and will highlight the difference especially in the lower wavelength range.

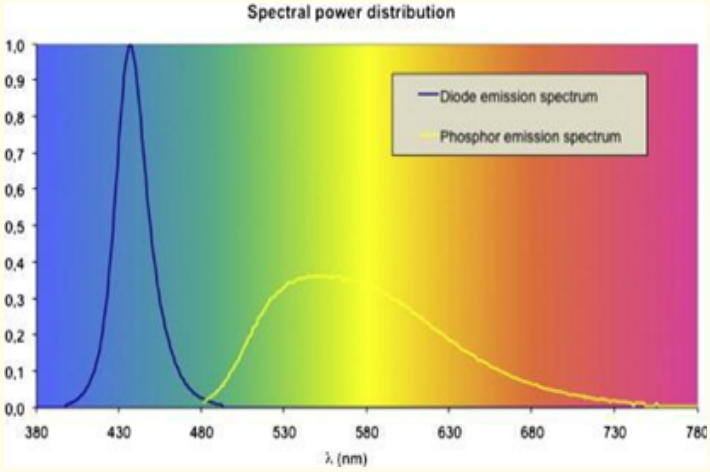

Figure 1: *Spectral representations of using LEDs and with yellow secondary emission from phosphor, emitting with light. (www.sciencedirect.com, Behar-Cohen et al. (2011)) [4].*

The fact that white light ensures the subjective clearest sight and image, each product (mobile phone, laptop, PC) is equipped with LCD displays emitting light within this short wavelength spectrum. Behar-Cohen., *et al*. [4] contended within their article, that short wavelength light is also called cold white, which ranges around 5500 Kelvin, correlated color temperature (CCT). This CCT is used to define the color´s shade, whether it is associated as hot or cold light. On the other hand, warm light presents a yellow orange tint and have the correlated color temperature below 3500K.

Behar-Cohen., *et al.* [4] also mentioned that one of the key issues that LED significantly penetrates the lighting market is due to the fact that this is an efficient high power illumination method. Generally, three existing methods to generate light from a light emitting diode are in place. The first is combining a diode emitting at a short wavelength with a phosphor emitting at a larger wavelength, which Figure 1 points out. The second is using a diode emitting in the near ultraviolet coupled with one or several phosphors. The third method uses at least three diodes emitting at different visible wave lengths by combining them, and consequently producing white light.

How does light reach the retina

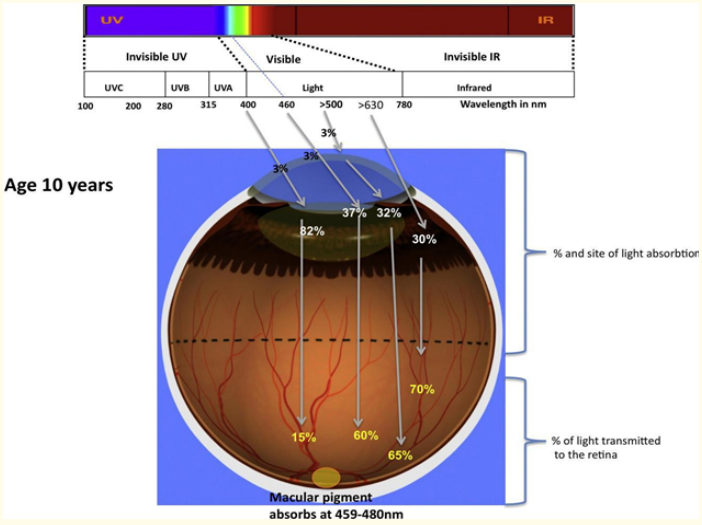

Figure 2: *Interaction of visible light (www.sciencedirect.com, Behar-Cohen et al. (2011)) [4].*

The entire sunlight spectrum or the artificial light radiations are received by the eye [4]. However, there are some different eye tissues the light has to penetrate until it reaches the retina. The first layer, the cornea, which is seen on the top of figure 2, absorbs all the UVB and UVC radiations and about 30 to 40% of UVA which lies between 320 and 360 nanometers. Prolonged exposure of UVB and UVA may cause reversible lesions of the corneal epithelium. On the other hand, UVC radiations can induce deeper lesions within the layers of the cornea which can lead to opacity and neovascularization in which new blood vessels grow into the cornea. As mentioned above, visible light ranges from 380 to 780 nm, which is also partly absorbed by the retina.

The next layer, which is adjacent to the lens, is the iris. It contains pigments and melanin which absorb all the visible light wavelength and it is round in shape. The diameter can change dependent on the light from 1 to 7 mm, decreasing with age. For instance, it will constrict when light is penetrating the eye. The next light absorption will be within the lens. It will absorb longer wavelength until UVA. The absorption of visible light changes with age. Until 25 years only around 20% of light is transmitted. With increasing age the cells of the lens change, hence, it will absorb most of the blue light. After passing these areas the light reaches the retina.

Retinal Light Damage: Types of Damage

An interaction with blue light impinging to the retina or accumulating in the retina can induce damage to the RPE (Retinal Pigment Epithelium) cells, the photoreceptors and to other cells [4]. The authors also found out that short wavelength light is the most dangerous one.

Corresponding to Youssef, Sheibani and Albert [1], the mechanisms which cause damage to the retina include, photothermal, photomechanical and photochemical reactions. Photomechanical damage contains irrelevant correlation to visible light exposure; hence this mechanism will not be further discussed.

Organisciak., *et al*. [2] pointed out that the effect of prolonged light exposure due to varying times of day, diet, region of the retina, prior light rearing history and generic background. They used different animal models to provide insights into understanding light damage. This different species analysis should give more insights into the fact that different humans in diverse climates and places have different pathological reactions to light. However, their data also indicated that damage or cell death mechanisms were not easily extrapolated between different species. Hence, there is a need for additional comparative light damage studies in a variety of animal models, compared in different regions.

One example of light damage is photothermal damage which occurs due to the energy from light to the retinal tissue [1]. The wavelength lies at the upper end of the visible spectrum which leads to an intensification of kinetic energy, furthermore, to a collision of the molecules which finally lead to an increase of the temperature within the retina. Furthermore, they referred that irreversible thermal damage in the retina typically occurs by a raised temperature of at least 10 degree Celsius. However, their study showed that a single five minutes exposure to light did not induce any significant damage to the retina. On the other hand, a repeated (three, four times) five minutes exposure was followed by one hour dark recovery time which consequently lead to a significant retinal damage.

Behar-Cohen., *et al*. [4] found out that the Correlated Color Temperature (CCT) of blue light is closer to the white light which belongs to the cold light. As mentioned above, the associated CCT ranges are around 5500K, luminance (cd/m^2), and the comparatively standard daylight ranges around 6500K, indeed much higher than the LCD emitted blue light. Obviously, the LCD or LED technology does not lead to a photothermal damage within the eye. This kind of light evokes another problem called photochemical damage.

Photochemical damage is the most common damage, concerning light damage to the eye, which happens through mechanisms by which light exposure causes retinal damage [1]. In particular, a lower wavelength blue light which has a higher energy associated with long duration exposure generates free radicals within the retina. Free radicals are atoms with an unpaired number of electrons [5]. Their danger comes from the fact that they react with specific DNA components or the cell membrane which can cause cell dysfunction or cell death. This leads to an absorption of radiant energy which causes excitation of electrons. Furthermore, the tissue within the retina is vulnerable to free radicals and the mechanism to reduce them. Consequently, this can cause extensive damage to the retinal photoreceptors which are responsible to convert the light into chemical impulses.

Behar-Cohen., *et al*. [4] explained this process by the fact that radicals and reactive oxygen species (ROS) are formed which lead to a photodynamic effect. The photochemical damage occurs when light is absorbed by a chromophores, responsible for the color, which undergoes a chemical transformation and interact with another molecules. These chromophores are then, consequently, producing free radicals and oxygen, known as photosensitizers. The fact that the photoreceptors and the RPE are closely adjacent to the choroidal blood supply, which is highly oxygenated, can potentially increase the risk of photochemical damage. In addition to the photothermal and photochemical damage, a prolonged sunlight exposure can lead to an eye disease called age-related macular degeneration (AMD).

Organisciak., *et al*. [2] pointed out the hypothesis that all complete photoreceptor degenerations devolve into extensive remodeling triggered by light-induced retinal damage (LIRD) in adult albino rodents. A hypothesis emergent from data analysis confirms, that this LIRD model closely mimics late-stage atrophic age related macular degenerations (AMD), which is one of the leading causes for vision loss. The macula is the part of the retina which is mainly responsible for the color vision and for the visual function, hence, this kind of damage can have devastating and irreversible outcomes. This typically damaging phase occurs in three phases. In Phase 1, the stress on photoreceptors is evident by photoreceptor outer segment detachment. This will lead to delocalization and changes in synaptic and photoreceptor architecture which cause the photoreceptors to die. In Phase 2, the outer nuclear layer is progressively dismantled via photoreceptor death and active removal of debris, which emerged after the first phase. At the end of Phase 2, another layer, the Müller cells, collapses and leads to a scar similar appearance. The third and last phase causes remodeling of all types of the remaining neurons. The LIRD retina is the first known retinal disease model where mature neurons transform and leave the nervous system and migrate into other tissues. This predicts the disruption of the blood-retina interface and causes an atrophic AMD, which definitely can lead to full vision loss.

In contrast, Behar-Cohens., *et al*. [4] research showed that no consensus regarding any causal links between sunlight exposure and AMD has emerged from epidemiologic studies. The sub-clinical findings occurred after oxidative stress associated with aging processes in the retina. However, their study showed correlation between sunlight and 5-year incidence of early related macular changes. People with blond or red hair, and with prolonged leisure time spent outdoors, which leads to an increased light exposure, were more likely to develop early AMD changes. For instance, a ten-year incidence study confirmed these findings that the exposure to the summer sun for more than 5 hours a day led to a higher risk of developing increased retinal pigment abnormalities and early AMD changes as compared to those exposed for less than 2 hours a day during the same period [6]. This relative exposure to UV and blue light showed associations between exposure and AMD signs. Patients with advanced AMD degenerations had significantly higher exposure to blue or visible light over the preceding 20 years suggesting that blue-light exposure could be related to the development of AMD, however, particularly in the more advanced ages. On the other hand, long-term studies with the correlated LED emitted blue light are not available yet, but these studies showed a significant risk for the future. A possible risk to enhance the cell damage through light exposure is Vitamin A.

Mihai and Washington (2014) found out that accumulation of Vitamin A dimers, such as A2E, in the human eye might be responsible for the formation of RPE debris, which indicates an early retinal degeneration. Their work demonstrates that cell death, Vitamin A can induce, is relatively low without light exposure. Conversely, it has been shown to induce greater cell death upon light exposure. Furthermore, their studies showed that this A2E induced cell death within the retina can lead to further degenerations, such as the previous discussed age related macular degeneration (AMD).

Results

Organisciak., *et al.* [2] debated that the onset of LIRD and the accompanying remodeling of the neurons is rapid with instantaneous signs of metabolic stress within the retina, respectively in the photoreceptors, the RPE, the choriocapillaris and the Müller cells. In particular, the degenerative process started within 15 days and the extensive remodeling within 60 days.

The study of Youssef., *et al*. [1] revealed no conclusive associations of increased sun or blue light exposure and the subsequently development of AMD. However, there still remains an essential belief that oxidative stress caused from photothermal or photochemical exposure contributes to many of the seen changes in age-related macular degeneration [7-9].

Conclusion

Prolonged light exposure has definitively an aggressive and devastating effect the neural retina. Furthermore, this pathological remodeling process, which was discussed above, showed significant similarities, respectively progressions to atrophic age related macular degeneration (AMD), which augurs poor vision outcomes [2]. Due to the improvement of the technology laptops and tablets are used for almost all tasks privately and jobwise. The dissemination all over the world including Third World countries and the prolonged usage of computers instead of printed sheets will definitely be a critical ocular health issue within the next upcoming years.

The knowledge about the mechanisms of light damage to the eye has grown extensively within the last years. However, there are various remaining questions and possibilities how to reduce the effects of these potentially toxic exposures. For instance, the potential of surgical techniques, such as vitreoretinal, which means within the eye, Indocyanine administration, to overcome or to minimize such diseases [1]. Undoubtedly, surgical or supplemental medical interventions would definitely decrease ocular diseases and preserve sight. Unfortunately, medical prophylaxis is still within the research phase concerning this issue, hence, special filter glasses which block blue wavelength light, as for instance Rodenstock's Solitaire® Protect Balance glasses, are still one of the best options to prevent such light induced diseases.

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