REVIEW ARTICLE

# Blue Light Hazard from Photopolymerization Unit: Literature Review

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## ABSTRACT

**Aim:** The aim of this article is to highlight the potential hazards from intense blue light emitting from the photopolymerization units used for curing of dental materials. The article also aims to explain the mechanism of retinal cell damage from intense blue light radiation.

**Material and method:** Articles related to photochemical damage caused by the short wavelength radiation were included in the review. The articles were collected from the electronic data base and hard copies of journal articles. The articles were searched with search criteria of blue light hazard, photochemical damage from blue light, hazard from photopolymerization units and hazards from short wavelength radiation. A narrative review was prepared to explain the mechanism of damage caused by high intensity blue light radiation.

**Conclusion:** It was concluded that blue light intense radiation is capable of causing potential damage to photoreceptor cells of the retina leading to age related macular damage of the eye.


**Keywords:** Blue light, light cure, photpolymerization unit.

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## INTRODUCTION

Retinal photoreceptor is adapted to function over a wide range of light conditions. It is evident from research data that prolonged exposure to intense visible light can lead to photoreceptor cell damage. In nocturnal animals the damage to retinal cells can be induced only by 2-3 times above normal room lighting.[1,2] Intense blue light can be harmful if directly exposed to bare eyes. It has been reported that blue light can cause macular degeneration of the light sensitive retina of the eye. Blue light exposure for prolonged periods can cause ocular damage leading to development of age related macular damage.[4,3]

The short wavelength blue light interferes with the visual cycle of the photosensitive cells. Lamps of high intensity are used in dentistry to cure certain advanced restorative dental materials. The dental team should be aware of the direct and indirect effect of the irradiation emitted from the photopolymerization unit. The eyes of the lamp operators are at risk from the back reflection of the blue light. The absorbed radiation may cause phototoxic and photoallergic reaction.[5]

Intense blue light are now commonly used as decorative lights. High intensity blue light are used in the dental treatment to cure certain dental materials. The light cure restorative dental materials were introduced in dentistry a few decades back. Presently the science of dental materials has been revolutionized by the advent of light cure materials. The activator in the light cure resins and cements is camphorqunione. This compound is activated by high intensity blue light. The light cure materials have gained popularity as the operators control over working and setting time is greatly increased. Moreover the strength of the light cure material is superior to self-cure material.

Light-cure material is used maximum by Orthodontist, Endodontist and Pedodontist. That is why these specialists have the maximum exposure to intense light of photopolymerization unit. Not only the dentist but also the attending staff and patients also get exposed to the blue light radiation. There is some concern about the Ultra violet radiation emitted from the photopolymerization units while curing. Mostly the radiation is in the visible spectrum and

is of blue color but because the blue light is shortwave length some amount of ultraviolet radiation is inevitably produced.[6,7]

The ultraviolet light produced by curing lamps is minimal and has not been found to be harmful for eyes. Light cure units with high power lamps showed potential to cause blue light mediated ocular damage. The damage is maximum after cumulative viewing of light at distance of 30 cm or less.[8]

The spectral output from visible light curing unit extends into the ultraviolet and infra-red spectrum. This spectral region is often referred as the retinal hazard region. Photochemical damage of the retina can occur from intense radiation whereas Infrared radiation can cause cataract. On an average orthodontist bonds upto 20 attachments per day with an acceptable curing time of 20 sec per tooth. With this calculation orthodontist spends 28 hrs per year holding the light cure gun.[9,10]

**Effect on circadian rhythm and melatonin**

Blue light has been shown to affect the circadian rhythm and daily sleep wake cycle. Light can elicit acute physiological and alerting responses in humans, the magnitude of which depends on the timing, intensity, and duration of light exposure. The alerting response of light as well as its effects on thermoregulation and heart rate is also wavelength dependent. Exposure to 2 h of monochromatic light at 460 nm in the late evening induces a significantly greater melatonin suppression.[11]

Melotonin in humans is strongly suppressed by blue light. Blue monochromatic (446-477nm) light has also been shown to be more effective than longer-wavelength light for enhancing alertness. Disturbed circadian rhythms and sleep loss have been described as risk factors for astronauts and NASA ground control workers, as well as civilians. Such disturbances can result in impaired alertness and diminished performance.[12]

The photostimulation of retina by light results in transmittance of stimulus to the hypothalamic suprachaismatic nuclei through the retinohypothalmic tract.[13] The suprachaismatic nuclei sends stimulus to the pineal gland and penial gland responds by regulating the secretion of the melotonin hormone (Fig 1).[14,15]

Rats lacking rods and cones regulate their circadian rhythms by a new photoreceptor system. Small subsets of retinal ganglion cells are directly photosensitive and utilize an opsin/vitamin A-based photopigment called melanopsin. Melanopsin is maximally sensitive in the blue part of the spectrum. Photosensitive retinal ganglion cells mediate a broad range of physiological responses to light, ranging from

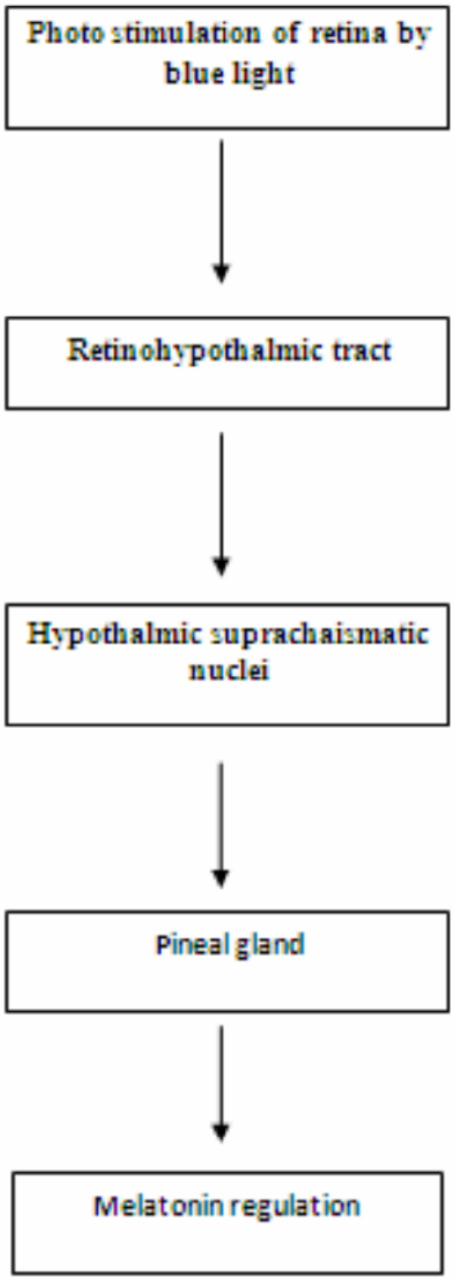


Figure 1: Regulation of Melatonin from Blue light exposure

the regulation of circadian rhythms to pupil constriction.[16,17]

**Eye aversion response:** Eye has a natural aversion response to bright light. This aversion response limits the exposure to less than 0.25s. Blue light emitted by the LED units do not evoke this protective response. Clinicians must remember that person having got a cataract surgery is more susceptible to retinal damage from exposure to high intensity light. People taking antimalarial drugs, chlorpromazine, St Johns Wort, dimethylchlorotetracycline and 8- methoxypsoralen should avoid direct exposure to bright light as even short exposure to high intensity light can be potentially

damaging.[18,19]

**Eye protection:** Dentist normally wear some kind of eye protection while curing with blue light .Although the dentist use a filter to protect themselves from direct exposure to blue light but the attenders are largely unprotected. Simply using a tinted glass as filter can reduce photochemical injury. Some protection from the blue light takes place naturally as the lens acquires a slight yellow discoloration with age. To reduce the harmful effect of short wavelength the artificial lens placed in the eye is of yellowish tinge rather than clear.

The intensity of the photo-polymerization units should be specified by the manufacturers. Generally the manufacturer provides an attached filter to light cure unit. These filters are paddle shaped and are not able to stop the scattered radiation. All the photopolymerization units have a compact fiberoptic stem. The fiber optic stem are able to transfer the light from the source to the object with minimal loss of energy and scattering (Fig. 2).

Operators sometimes wear special goggles with side shields. These goggles are of colored glasses. Such glasses provide better protection from scattered blue light but

Figure 2: Curing light with fiberoptic stem and paddle shaped filter.

the disadvantage is that a restorative dentist may not be able match the shades of composite while doing esthetic procedures.

**Photochemical damage**: Photochemical damage has been widely studied, because it can cause retinal damage within the intensity range of natural light. Photochemical lesions are primarily located in the outer layers at the central region of the retina. Several factors can modify the susceptibility of the retina to photochemical damage.

There are two types of photochemical damage .class I damage is mediated by visual pigments and lesion primarily occurs in the photoreceptors. Class II damage is generally confined to the retinal pigment epithelium. Blue light has got the maximum potential for damage because of which a new concept of blue light hazard is emerging.[20]

Retina is the photosensitive layer of the eye and is mainly responsible for vision. The retina is very delicate and can be damaged from intense radiation. The bleached rhodopsin can be regenerated in two ways. In the first method the bleached rhodopsin is regenerated metabolically in the visual cycle. Second method is the photochemical method in which photo-reversal of bleaching takes place by intense blue light. The later method is faster and that is why the photon catching capacity of the retina is increased. The increased number of photons received by the retina makes it susceptible to damage in the intense blue light.[21]

**Effect on mitochondria and cytochrome oxidase:** Age related maculopathy is mediated by exposure to light. The mitochondria of the retinal pigment epitheilial cell are damaged by the intense exposure to short wavelength blue light. These damaged mitochondria release reactive oxygen species. The reactive oxygen species play a significant role in age related maculopathy of the retina.[22]

Recent evidence has shown that blue light is thirty times more efficient than yellow light in causing dysfunction of the blood retinal barrier. The most efficient wavelength was 418 nm which corresponds with absorption spectrum of cytochrome oxidase.[23] The blue light exposure to retinal cells decreases cytochrome oxidase activity. In the inner segments of photoreceptors and in the outer plexiform layer of the retina the cytochrome activity can reduce to almost half of its normal activity. This inhibition of the cytochrome activity is reversible but overexposure may lead to irreversible damage.[24,25]

The absorption of blue light by cytochrome oxidase in rat retina inhibits this enzyme, and may reduce the retinal oxidative metabolism. Irreversible inhibition of the oxidative metabolism may decrease the activity of the Na/K-ATPase, which redistributes ions and increases intracellular osmotic pressure and causes cellular edema. Severe retinal edema may be the cause of retinal degeneration.[26]

Retinal ganglion cell axons are laden with mitochondria. Mitochondria have enzymes like cytochrome oxidase and flavin oxidase which absorb light radiation particularly in the blue region of the spectrum. This absorbed light leads to production of reactive oxygen species. The reactive oxygen species so produced pose a risk for the survival of the ganglion cells. The risk is enhanced manifolds if the energy status of the cell is compromised because of conditions like glaucoma or in Leber's Hereditary Optic Neuropathy.[27-29]

**Apoptosis of ganglion cells**: The cells of retinal epithelium can be necrosed by intense blue light whereas moderate

blue light can induce programmed cell death or apoptosis.[30] Blue light exposure can induce damages to human RPE (retinal pigment epithelium) cells in vitro, which include apoptosis, apoptotic necrosis and necrosis. These changes are caused by triggering the mitochondrial permeability transition, which results in decrease of delta Psi(m) and release of cytochrome C. delta Psi(m) can be used as a earlier parameter of bluelight-inducedapoptosis.[31] Blue light exposure over threshold can induce damage to human RPE cells, probably by triggering the mitochondrial permeability transition, which results in the decrease of delta Psi(m) and the release of cytochrome C.[32]

DNA: Intense exposure to blue light has the potential of damaging the DNA of the retinal pigment cells.[33-35] When A2E laden retinal cells are exposed to blue light marked propensity for apoptosis is seen in these cells. A2E generates singlet oxygen when irradiated with blue light .Singlet oxygen reacts with carbon double bonds of A2E epioxides and singlet oxygen react with DNA to cause oxidative damage.[36]

Retinal pigment epithelial cells produce lipofuscin. Blue light irradiation destabilizeslysosomal membrane in lipofuscin loaded retinal pigment epithelium cells. This results in significantly reduced viability of these cells .The accumulation of lipofuscin thus speed up the progress of the age related macular degeneration.[37] Human eyes have chromophores and visual pigments which absorb light energy. Lifelong exposure to light gives rise to additional light absorbing molecules. With decreasing wavelength the number of absorbing molecules increases which in turn increases the photochemical damage.[38]

**Lipofuscin accumulation in retinal pigment epithelium:** Retinal pigment epithelium cells are responsible for nourishing the retinal visual cells. This layer is firmly attached to the underlying choroid and overlying retina. The retinal pigment epithelial cell removes the spent disks from the outer segment of the photoreceptors. The disks are degraded quickly in younger individuals but in older individuals the degradation is not complete. This incomplete degradation leads to accumulation of lipofuscin. Lipofuscin is a yellowish pigment made of free-radical damaged protein and fat. Lipofuscin accumulation leads to formation of Drusen in the Bruch's membrane.[39-41]

Lipofuscin is aphotoinducible generator of oxygen species and is predominantly present withen the lysosomes. When illuminated with visible light lipofuscin is capable of causing lipid peroxidation, enzyme inactivation and protein oxidation. These effects are pH dependent are reduced by antioxidants, superperoxide dismutase and 1,4-diazabicyclo(2,2,2)-octane confirming the role of both superoxide anion and singlet oxygen. Lipofuscin probably compromises cell funtion by causing loss of lysosomal integrity.[42]

Lysosomal glycosaminoglycan catabolism of RPE cells is also strongly inhibited by A2-E and Lysosomal pH is increased by A2-E. The quaternary amine character of the A2-E apparently causes a perturbation of the acidic intralysosomal microenvironment, resulting in diminished hydrolase action and consequent accumulation of undegraded material.[43]

Lipofuscin represents a mixture of various biomolecuels which includes upto ten types of fluorophores. A major fluorophore of lipofuscin has been identified as the Schiff-base reaction product *N*-retinylidene-*N*-retinylethanolamine (A2-E.A2-E has been shown to impair lysosomaldegradative functions of retinal pigment epithelial cells in vitro by elevating the intralysosomal pH.[44-47] Analysis of extracts of human RPE has revealed that the major hydrophobic fluorophore of RPE lipofuscin is A2E.[45,47]

## CONCLUSION

Intense blue light has marked potential for damaging the light sensitive cells of the retina. This threat has been recently highlighted in the literature as the blue light hazard .Blue light hazard from the photpoymerization unit is a significant cause of concern for dentist their patients and attenders. The dentist should take all possible measures to cut down exposure to reduce the hazard from high intensity radiation. The office staff should be educated and motivated to ensure personal safety as well as the safety of the patients.

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