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High-Energy Blue Light and Eye Health: Consequences of Increasing Exposure

The light the human eye responds to is a narrow band of electromagnetic radiation between 390 to 700 nanometers and within this band, high-energy blue light makes up some of the highest energy—between 400 to 500 nanometers. Unlike UV-light exposure, which is almost exclusively from the sun, high-energy blue light is emitted from a multitude of sources including sunlight, digital devices (e.g., computers/laptops, smartphones, television screens) and artificial indoor lighting, specifically compact fluorescent and LED bulbs. Therefore, compared to UV-light, daily exposure to high-energy from both outdoor and indoor sources is significantly greater.

Absorption of almost all ambient UV-light occurs primarily in the cornea and crystalline lens and results of long-term exposure can manifest within the outer layers of the eye as cataracts. High-energy blue light, however, penetrates deeper into the eye and has the potential to damage retinal structures through photochemical and photo-oxidative reactions in the retinal pigment layer. Therefore, UV-light from the sun may be less of a causative factor for conditions associated with retinal damage such as age-related macular degeneration (AMD) and there are several studies to suggest this.

Short-term effects of high-energy blue light manifest as “eye fatigue” and are characterized by symptoms including blurry vision, dry eyes and headaches. In a mouse model, it was shown that damage from high-energy blue light can occur within 3 hours of exposure with significant photoreceptor loss after 3 weeks.

The mechanisms by which high-energy blue light damages the eye are multifactorial but primarily mediated through photo-oxidative reactions and the generation of reactive oxygen species (ROS). Due to its high metabolic rate and exposure to high-energy blue light, the retinal pigment epithelial cells (RPE) are a primary target of photo-oxidative damage. Damage to RPE is a vicious cycle of oxidative stress and inflammation: oxidative stress triggers an inflammatory response and, in turn, inflammation enhances the production of reactive oxygen species (ROS). Without adequate protection, increased oxidative stress inactivates a major proteolytic pathway called the ubiquitin-proteasome pathway (UPP). The UPP functions to degrade unneeded or damaged proteins in all cells and plays a major role in regulatory mechanism central to cellular processing that includes inflammation, immune and stress responses, and antigen processing. A fully functioning UPP is required for cells to cope with various stress, including oxidation. However, extensive oxidative insults, as seen in RPE exposed against high-energy blue light exposure

Protective role of Lutein and Zeaxanthin Isomers

By Lynda M. Doyle, MS Human Nutrition, Vice President of Global Marketing, OmniActive Health Technologies

Absorption of almost all ambient UV-light occurs primarily in the cornea and crystalline lens and results of long-term exposure can manifest within the outer layers of the eye as cataracts. High-energy blue light, however, penetrates deeper into the eye and has the potential to damage retinal structures through photochemical and photo-oxidative reactions in the retinal pigment layer. Therefore, UV-light from the sun may be less of a causative factor for conditions associated with retinal damage such as age-related macular degeneration (AMD) and there are several studies to suggest this. Short-term effects of high-energy blue light manifest as “eye fatigue” and are characterized by symptoms including blurry vision, dry eyes and headaches. In a mouse model, it was shown that damage from high-energy blue light can occur within 3 hours of exposure with significant photoreceptor loss after 3 weeks. The mechanisms by which high-energy blue light damages the eye are multifactorial but primarily mediated through photo-oxidative reactions and the generation of reactive oxygen species (ROS). Due to its high metabolic rate and exposure to high-energy blue light, the retinal pigment epithelial cells (RPE) are a primary target of photo-oxidative damage. Damage to RPE is a vicious cycle of oxidative stress and inflammation: oxidative stress triggers an inflammatory response and, in turn, inflammation enhances the production of reactive oxygen species (ROS). Without adequate protection, increased oxidative stress inactivates a major proteolytic pathway called the ubiquitin-proteasome pathway (UPP). The UPP functions to degrade unneeded or damaged proteins in all cells and plays a major role in regulatory mechanism central to cellular processing that includes inflammation, immune and stress responses, and antigen processing. A fully functioning UPP is required for cells to cope with various stress, including oxidation. However, extensive oxidative insults, as seen in RPE exposed against high-energy blue light exposure

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to high-energy blue light, can impair UPP, resulting in the accumulation of damaged proteins, dysregulated cell processing and increased inflammation. Since the RPE is a major source of pro-inflammatory mediators and a primary target of photo-oxidative impairment of UPP, the formation of ROS from high-energy blue light may contribute to inflammation and eye-related issues, like AMD. Several nutrients play critical roles in protecting the retina from photo-oxidative damage and perhaps none are more important than the macular carotenoids: lutein and the zeaxanthin isomers.

**The Macular Carotenoids: Preferential Protectors Against High-Energy Blue Light**

Lutein and the two zeaxanthin isomers, RR-zeaxanthin (3R,3'R-zeaxanthin) and RS (meso)-zeaxanthin (3R,3'S-RS-zeaxanthin), are the only three carotenoids found in the eye, specifically in the macula—the area of the retina responsible for highest visual performance and susceptible to the greatest amount of photo-oxidative damage. The location of their respective areas of deposition is highly specific: lutein is preferentially deposited in the peripheral macula, RR-zeaxanthin in the mid-peripheral macula and RS (meso)-zeaxanthin in the center of the macula. Increased dietary intake of lutein and zeaxanthin is associated with increased macular pigment density (MPOD – the thickness or density of the protective layer of carotenoids in the macula) in healthy adults. Epidemiological studies have reported an inverse association between lutein and zeaxanthin and the risk of developing ocular diseases such as AMD and cataracts.

Each of these carotenoids are important in protecting the retina and enhancing visual performance by acting as high-energy blue light filters, quenching ROS and inhibiting lipid peroxidation of cell membranes generated from photo-oxidation. Ensuring optimal levels of all three carotenoids is critical to protecting the eye against high-energy blue light. Lutein and the zeaxanthin isomers absorb different wavelengths of light and together absorb a broader spectrum of high-energy blue light, which offers greater protection of retinal tissue. Lutein and zeaxanthin isomer absorb different wavelengths of light and together absorb a broader spectrum of high-energy blue light, which offers greater protection of retinal tissue. The specialized locations and functions of each macular carotenoid suggests that the best way to support eye health and visual performance is to consume all three macular carotenoids through diet or supplementation. Given that the average US dietary intake of lutein and zeaxanthin is far below levels shown in research to be beneficial (less than 2 mg lutein and 0.5 mg zeaxanthin), supplementation may be a more viable approach to maintain optimal levels of the macular carotenoids and protect the eyes against high-energy blue light.

**Lutein & Zeaxanthin - Macular Carotenoids to Protect Against High-Energy Blue Light**

While lutein and zeaxanthin isomers absorb a broad spectrum of high-energy blue light, which offers stronger protection of retinal tissue, lutein and zeaxanthin isomer absorb different wavelengths of light and together absorb a broader spectrum of high-energy blue light, which offers greater protection of retinal tissue.

**References**


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