STM JOURNALS

International Journal of
Photochemistry and
Photochemical Research

Volume 3, Issue 1, 2025
January–June
DOI Journal: 10.37591/IJPPR

https://journals.stmjournals.com/ijppr

Review IJPPR

Emerging Trends in Photobiology: Exploring the Photophysics and Photobiology of the Eye

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Abstract

The eye consists of three main segments: the cornea, lens, and retina. The primary function of the anterior ocular tissue, the cornea and lens, is to transmit and focus light onto the retina without distortion. They also filter UV light (under 400 nm), preventing it from reaching the retina. While light reaching the retina is mostly used for vision, it can also have various effects on the eye's constituents, both beneficial and harmful. This article examines how light interacts with the eye, the protective mechanisms involved, and the potential role of light in aging, disease, and other biological processes beyond vision, such as mood, hormonal secretion, and the growth and maintenance of rods and cones. The retina, being sensitive to light, can be affected by prolonged exposure to both visible and UV light, leading to oxidative stress and potential damage. Protective mechanisms, such as antioxidants and carotenoids, play a crucial role in mitigating these risks. Additionally, light influences circadian rhythms and can impact the release of hormones like melatonin, further affecting the body's internal processes. Understanding the interplay between light exposure, eye health, and systemic biology is vital for the development of interventions to prevent vision loss and preserve overall well-being.

Keywords: Oxidative stress, protective mechanisms, carotenoids, circadian rhythms, and hormonal regulation

INTRODUCTION

General Characteristics

The anterior segment of the eye comprises the cornea, which provides most of the focusing power, and the lens. During fetal development, the lens is initially supplied with blood, but as it matures, it becomes isolated from the blood supply, receiving nourishment from the aqueous humor. The lens's primary function is focusing at distances up to 10 feet (accommodation). The retina is held in place by the pressure from the vitreous humor, a viscous, clear solution. The retina has several layers, including visual pigments, rods, and cones, with the photoreceptors growing toward the back of the retina and being phagocytosed by the retinal pigment epithelium. In front of the retina are neural cells that process visual signals. Light must pass through these layers before reaching the photoreceptors.

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Received Date: February 07, 2025
Accepted Date: February 11, 2025
Published Date: February 12, 2025

Citation: Ben Okri. Emerging Trends in Photobiology: Exploring the Photophysics and Photobiology of the Eye. International Journal of Photochemistry and Photochemical Research, Volume 3, Issue 1. 2025; 3(1): 31–37p.

The macula is the central 5.5 mm region of the retina, and the fovea, the central 1.5 mm area, is where sharp focus occurs. The fovea contains the highest concentration of cones and lacks the outer retinal layers to reduce scattering of light. Distortions in focus are minimized in the fovea by the presence of blue-absorbing chromophores, lutein and zeaxanthin, which reduce chromatic aberration by absorbing blue light, which is refracted more by the lens than longer wavelengths.

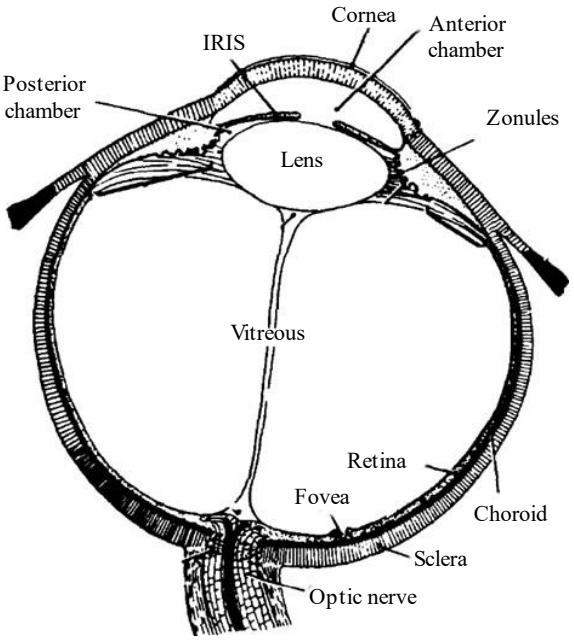

Figure 1. Cross-sectional diagram of the human eye.

Six key factors influence photochemical reactions in the aging and disease processes of the eye: (1) light intensity, (2) the wavelengths absorbed by each tissue and their resulting photochemistry, (3) oxygen levels in tissues, (4) the presence of quenchers that may interrupt photochemical processes, (5) the location of photo-oxidative damage, and (6) the presence of exogenous sensitizers Figure 1 [1-3].

Light Intensity and Photochemical Processes

Light can cause tissue damage through three primary mechanisms: thermal, photochemical, and thermal-acoustical processes. Thermal damage is most prominent at longer wavelengths, whereas shorter wavelengths primarily cause damage through photochemical reactions. High-intensity, short-pulse light, such as that from lasers, can result in an optical breakdown, leading to mechanical damage among other effects. However, photochemical processes are the most significant under normal, ambient light conditions in the eye.

Transmission Characteristics of the Eye

The cornea absorbs all light below approximately 295 nm, preventing it from reaching the lens. The absorption is primarily due to nucleic acids and proteins in the cornea. The lens absorbs light in the range of 295-400 nm, with the primary absorbers being protein-bound tryptophan and 3-hydroxykynurenine (a tryptophan metabolite), substances mostly present in the lenses of primates. As the lens ages, its protein undergoes structural modifications, causing yellowing. This yellowing increases the lens's ability to absorb light between 300 and 400 nm, extending to around 500 nm, which could contribute to photochemical damage. In the retina, several compounds can absorb light and potentially cause photo-oxidative damage. These include melanin, visual pigments like rhodopsin, cytochrome c, and lipofuscin, which forms as a result of aging. Although melanin may protect against photochemical damage, it can be cytotoxic at higher light intensities. Recent studies suggest that protoporphyrin in the retinal blood may enhance ocular damage susceptibility [4].

Oxygen Levels in the Eye

Oxygen plays a critical role in photochemical processes. It is essential for many photochemical reactions, and low oxygen levels can slow or even negate these reactions. Oxygen enters the eye primarily through the cornea, where it is well-oxygenated. As it moves toward the lens, the oxygen concentration decreases. The retina, supplied by blood, is generally well-oxygenated, especially in its center, which has a high metabolic rate.

Antioxidants and Protective Mechanisms

Several antioxidants present in the eye, including glutathione, ascorbic acid, and vitamin E, act as quenchers for oxidative reactions. Research shows that increasing ascorbate levels can offer protection to both the lens and retina. Supplementation with ascorbate has been shown to protect lens proteins from aggregation and cross-linking induced by UV light. Additionally, dietary ascorbate can help protect retinal components like rhodopsin and photoreceptors in animal models.

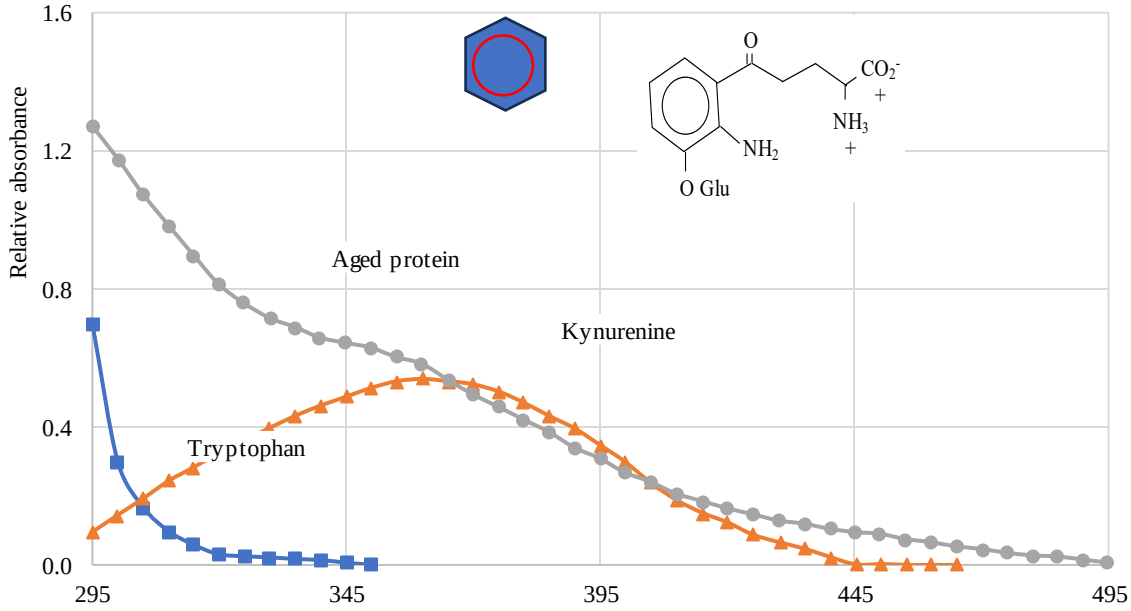

Figure 2. Absorbance spectra showing the degradation of tryptophan and accumulation of kynurenine in aged protein.

Research also demonstrates that enhanced intake of vitamin E (α-tocopherol) can protect the lens from light-induced damage. Protective effects have been observed with thiol-containing radioprotectors against photosensitized damage in the lens and retina. Moreover, specific protective mechanisms exist in different parts of the eye. For instance, the carotenoids lutein and zeaxanthin in the retina not only filter harmful blue light, which is known to cause significant retinal damage, but may also function as quenchers of singlet oxygen, further helping to mitigate light-induced damage Figure 2.

Photophysics and Photochemistry

Tryptophan: Tryptophan (Trp) plays a significant role in the photochemistry of the human lens, particularly in the young lens and the anterior cortex of aging lenses. The photochemistry of Trp is complex, generally involving the expulsion of an electron, leading to the formation of a carbon-centered radical after deprotonation. This process is influenced by various factors, such as the wavelength of light, the presence of functional groups, and the solvent's hydrophilicity. Shorter wavelengths and aprotic solvents tend to enhance photoionization. The presence of nearby functional groups can alter Trp's photodecomposition. For instance, when an amino group is present near the indole ring in Trp-containing peptides, it can cause deamination and the attachment of aliphatic portions to the indole. Such reactions can result in the modification of protein residues, which may ultimately impair the protein's function [5-6]. The photolysis of proteins typically leads to a series of complex reactions, including molecular and macromolecular changes such as cross-linking, yellowing, and charge changes. One key step in this process is the conversion of Trp to N-formyl-kynurenine (NFK), which has its own complex photochemistry. NFK absorbs light at longer wavelengths than Trp (320 nm vs. 280-290 nm), making the lens more vulnerable to damage.

O-Glucoside of 3-Hydroxykynurenine: 3-Hydroxykynurenine (3-HKG) is a major absorber of light in the young lens, and its absorption raises concerns about the potential for photochemical damage. Studies of the fluorescence decay from young and old lenses show that the fluorescence of the young lens has a monoexponential decay with a lifetime of 70 ps, similar to that of isolated 3-HKG. In contrast, older lenses exhibit longer-lived fluorescence, likely due to the increasingly rigid environment within the lens. These findings are consistent with studies on simpler compounds, suggesting that proton abstraction in the excited state is an important deactivation pathway. However, no long-lived states or singlet oxygen production were observed in the intact lens, supporting the idea that 3-HKG functions primarily to protect the retina from radiation in the 295-400 nm range while minimizing damage to the lens.

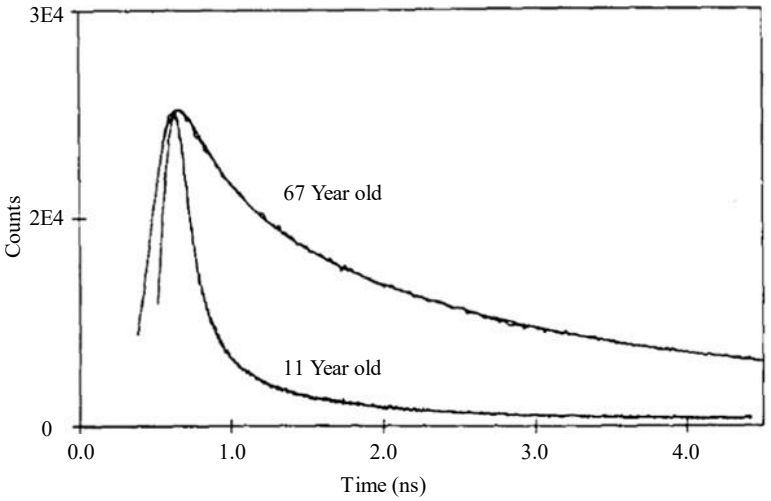

Figure 3. The inset displays the chemical structure of kynurenine.

Aged Human Lens Protein: Aged human lens proteins, particularly yellow proteins, have been extensively studied for their steady-state fluorescence properties. These proteins exhibit a complex emission spectrum in the 420-440 nm range, which increases with age and becomes more intense toward the nucleus of the lens. Studies on sliced human lenses have shown a decrease in fluorescence in the nucleus of older lenses, with new compounds forming that have distinct fluorescence characteristics. Figure 3. In contrast to young lenses, the fluorescence decay of older lenses is more complex, fitting into at least three components with lifetimes of 0.07, 0.53, and 1.7 ns. This suggests that the primary fluorescing species in older lenses are the yellow compounds attached to proteins, not 3-HKG.

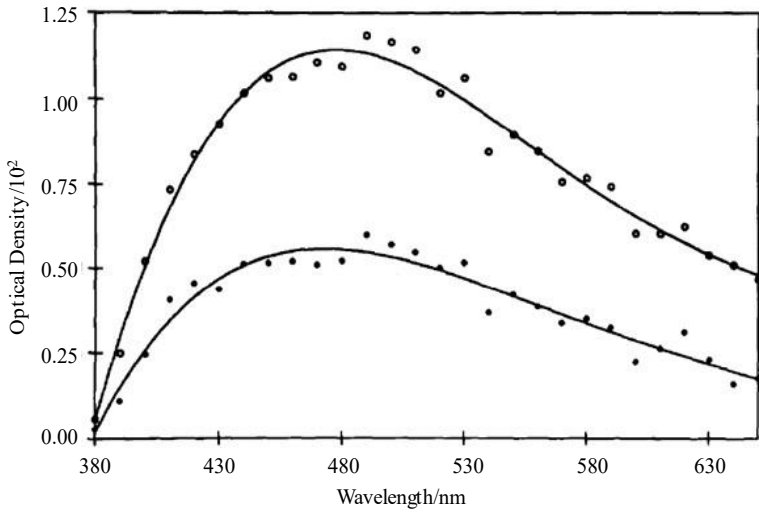

Figure 4. Spectral variation of optical density as a function of wavelength.

Additionally, older yellow lens proteins exhibit long-lived transient states, such as triplet states or radicals, which may arise from the newly formed fluorescent species. Time-resolved studies show that excitation of yellow lens proteins at 355 nm produces two distinct transient species with lifetimes of 24 ps and 400 ps. Figure 4 The absorption spectra of these transients at 3.5 ps and 42 ps after excitation show a peak around 490 nm, with the spectrum at 42 ps being broader and shifted slightly toward the blue. These long-lived transient states may contribute to the progression of lens damage [5].

Deleterious Effects of Light on the Retina

Studies have shown that light can cause two types of damage to the retina: damage due to prolonged exposure to low levels of light and damage from short bursts of intense light. Low-level light damage primarily affects the rod outer segments, while intense light can damage the retinal pigment epithelium (PE), photoreceptors, and occasionally other retinal neurons. The action spectrum for low-light damage resembles the absorption spectrum of rhodopsin, suggesting that it begins damaging the retina at lower light levels. Rhodopsin, while primarily involved in light transduction, has a quantum yield for this process of less than one [6]. Intense light damage, often referred to as the "blue light effect," shares an action spectrum with the UV-visible spectrum of melanin, though bleached rhodopsin (retinal) and cytochrome c have also been implicated as mediators. Retinal has a known quantum yield of 0.096 for the production of singlet oxygen, which can be damaging.

In the aging retina, lipofuscin, which accumulates in the PE, adds a new chromophore that could potentially contribute to photochemical events, even if its direct role is not fully understood. This complex mixture of components has a UV-visible spectrum similar to aging components found in the human lens, with absorptions extending to 500 nm. Accumulation of lipofuscin impairs PE cell function, and it may even be extruded from the PE cells and deposited in Bruch's membrane, causing thickening and impairing PE and photoreceptor metabolism.

Increased Retinal Sensitivity in Aphakic Monkeys

In aphakic monkeys (monkeys that have had their lens removed), the retina is approximately six times more sensitive to light below 400 nm, suggesting that without the lens, the retina becomes more sensitive to UV light. Extensive damage occurs to both rods and cones, as well as to the PE, under these conditions. To address this, cataract patients now typically receive plastic lens implants that have absorptive properties similar to those of the natural lens. A study found that patients with clear plastic lenses implanted after cataract surgery showed a significant decrease in blue cone sensitivity after five years, further highlighting the role of light sensitivity and its effects on retinal health [7-10].

Photophysics and Photochemistry of Retinal Pigments

While much research on retinal photophysics has focused on the mechanism of transduction—where light absorption causes conformational changes in proteins that initiate electrical signals to the brain—there has been less focus on the broader photochemical impacts of light on retinal health. Steady-state fluorescence studies on fractionated melanin and lipofuscin extracts indicate that these pigments consist of complex mixtures that emit fluorescence in the visible range. The fluorescence intensity generally increases with age. Gated fluorescence experiments also reveal a mix of decay times and spectra. In young human PE cells, melanin shows similar spectral and decay properties to bovine melanin, but as the eye ages, the amount of shorter-lived components increases, and the emission spectrum shifts toward longer wavelengths [11].

Age-Related Accumulation of Fluorophores

As the retina ages, lipofuscin accumulates and exhibits spectral characteristics that shift toward longer wavelengths, likely at the expense of shorter wavelengths. This age-related shift may be indicative of a causal relationship, where the components emitting at shorter wavelengths could be precursors to those emitting at longer wavelengths.

Table 1. Absorbance and decay rates for each transient.

	D_1	K_1	D_2[a]	K_2
Protein	1	5.2×10^4	0.94	3.7×10^3
Plus DTT	1	9.0×10^4	0.46	5.5×10^3
Nitrogen[b]	1	5.6×10^4	0.80	2.7×10^3
Air[b]	1	1.1×10^5	0.59	3.5×10^3

Age-Related Macular Degeneration (AMD)

Age-related macular degeneration (AMD) is a leading cause of blindness, involving progressive damage to the outer retina, Bruch's membrane, and the choroid. While AMD develops slowly over time, there is some evidence linking light exposure to its onset. For example, the disease tends to occur in the region of the retina that receives the most light. Additionally, there is an inverse relationship between iris melanin content and the occurrence of AMD, suggesting that increased melanin in the iris might offer some protective effect. Furthermore, individuals with cataracts, which cause lens opacity, may experience. Tale 1. a reduction in UV exposure to the retina, which could affect the disease's development [12-15].

CONCLUSIONS

The eye is continually exposed to photo-oxidative stress due to constant light exposure, which contributes to various degenerative processes over time. Unlike the skin, which serves as a protective barrier, the eye remains transparent, making it particularly vulnerable to light-induced damage. This unique characteristic provides an exceptional opportunity to study the interaction between light and complex biological systems. By employing advanced spectroscopic techniques, researchers can gain valuable insights into both normal and pathological ocular processes. In addition to aging and the development of diseases like macular degeneration, these techniques can help unravel other biological processes in the eye, offering potential avenues for targeted therapies and interventions. Moreover, these studies could lead to the development of novel protective strategies, improving ocular health and longevity.

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