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Anthocyanins in eye health: Multi-target protective mechanisms, bioavailability challenges, and translational perspectives

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ABSTRACT

The increasing prevalence of digital screen exposure and population aging poses rising threats to ocular health, driving the search for natural multi-target protective agents. Anthocyanins—water-soluble flavonoids abundant in dark-colored fruits and vegetables—have attracted considerable interest for their potential to prevent eye diseases. This review systematically evaluates the multi-dimensional protective mechanisms of anthocyanins, including antioxidant and anti-inflammatory activities, improvement of retinal microcirculation, promotion of rhodopsin regeneration and dark adaptation, and neuroprotection of retinal ganglion cells and photoreceptors. The extremely low oral bioavailability of intact anthocyanins is critically discussed, and the role of gut microbiota in converting anthocyanins into bioactive phenolic acid metabolites (e.g., protocatechuic, ferulic, and hippuric acids) is highlighted. The review also addresses structure-activity relationships, food matrix effects, and bioavailability-enhancing strategies such as nanoencapsulation, acylation/methylation, and synergistic combinations with other phytochemicals. Finally, future research directions and translational perspectives for developing anthocyanin-based functional foods and precision nutrition strategies are proposed, providing a comprehensive theoretical basis for leveraging anthocyanins in eye health management.

1. Introduction

The eye is the key sensory organ to perceive light signals, which is very important for the survival and activity of humans and animals. In today's society, the widespread use of electronic screens has raised concerns about ocular health in adolescents, including asthenopia, dry eye, and retinal light damage (Yeh et al., 2025). At the same time, with the aging of the population, eye diseases such as age-related macular degeneration and diabetic retinopathy have become important challenges in the field of global public health. The pathogenesis of these diseases involves oxidative stress, inflammation, vascular abnormalities, and neurodegeneration. Prolonged electronic device use elevates ocular reactive oxygen species (ROS) levels, leading to oxidative stress, inflammatory pathway activation, and consequently, the onset of eye diseases (Choo et al., 2022). Therefore, the search for natural active ingredients with multi-target protective effects has become a research hotspot, and daily supplementation of substances with anti-inflammatory and anti-oxidative stress functions has also received increasing attention (Zhang et al., 2023).

Anthocyanins are a group of water-soluble flavonoid compounds that are widely present in plants with bright red, purple, and blue colors. They are commonly found in dark fruits and vegetables such as blueberry, lycium ruthenicum, purple potato, and blackcurrant. In recent years, a large number of *in vitro* and *in vivo* studies and clinical trials have shown that anthocyanins have multifaceted protective effects on eye health. It not only has significant antioxidant and anti-inflammatory activities but also plays a role in retinal protection, visual function improvement, and delaying the progression of eye diseases through a variety of molecular mechanisms. Long-term supplementation of anthocyanins can effectively prolong tear film break-up time, relieve dry

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eye and asthenopia (Fan et al., 2023), inhibit retinal ROS production and inflammatory response, improve microcirculation, regulate eye muscle coordination, and thus reduce retinal damage and asthenopia.

This article reviews the role of anthocyanins in eye protection, focuses on the related molecular mechanisms, and briefly discusses the application potential and future research directions of anthocyanins in order to provide reference for the in-depth exploration and utilization of anthocyanins in the field of eye health.

2. The eye-protective mechanism of anthocyanins

2.1. Antioxidant and anti-inflammatory effects: resist oxidative damage and chronic inflammation in the eye

Oxidative stress and chronic inflammation underpin many eye diseases. Excessive ROS accumulation induces lipid peroxidation, protein damage, and DNA oxidation in ocular tissues, which in turn activates inflammatory signaling cascades, leading to cellular dysfunction, apoptosis, and ultimately the onset of retinopathy (Liu et al., 2012). The retina, particularly retinal pigment epithelium (RPE) cells, is vulnerable to ROS accumulation from aging or light exposure (including UV and visible light), leading to retinopathy (Liu et al., 2012). Anthocyanins, via their phenolic hydroxyl groups, scavenge free radicals, inhibit lipid peroxidation, and upregulate endogenous antioxidant enzymes, such as superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPX), thereby reducing ROS and malondialdehyde (MDA) levels in the retina after blue light irradiation (Li, Li, et al., 2024). Mechanistically, anthocyanins mitigate oxidative and inflammatory damage in RPE cells by suppressing mitogen-activated protein kinase (MAPK) phosphorylation (JNK1/2, p38) (Silván et al., 2016) and activating the nuclear factor erythroid 2-related factor 2/heme oxygenase-1 (Nrf2/HO-1) signaling pathway (Milbury et al., 2007; Song et al., 2016). Among tested anthocyanins, cyanidin-3-O-glucoside (C3G) exhibits the strongest ROS scavenging activity (57.5% ± 4.2% at 40 μg mL^{-1}) (Wang, Zhao, et al., 2015). It should be noted that the concentrations used in the cited *in vitro* studies (e.g., 40 μg mL^{-1}) are substantially higher than the plasma concentrations of intact anthocyanins achievable through oral consumption in humans (Cmax ≈ 100–200 nM). Therefore, the mechanistic pathways identified under these conditions should be interpreted as evidence of potential molecular actions rather than direct proof of their operation at physiological concentrations. The relevance of these mechanisms *in vivo* may be more appropriately attributed to the combined effects of anthocyanin-derived phenolic acid metabolites, which reach markedly higher circulating levels. Long-term intake of bilberry or blueberry extracts alleviates asthenopia and dry eye, while blueberry anthocyanins (e.g., malvigenin glycosides) dose-dependently protect RPE cells from H_2O_2-induced damage via MAPK regulation. These findings collectively demonstrate that anthocyanins preserve retinal structure and function by reducing oxidative stress, offering protection against various eye diseases.

Anthocyanins can also alleviate inflammatory eye diseases by inhibiting the expression of inflammatory factors and regulating related signaling pathways. *Lycium ruthenicum* anthocyanins can reduce the expression of nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB) pathway-related proteins, interleukin-6 (IL-6), tumor necrosis factor-α (TNF-α), interleukin-1β (IL-1β), and other pro-inflammatory factors and chemokines induced by blue light irradiation and increase the level of anti-inflammatory factor interleukin-10 (IL-10) (Ye et al., 2025). Thus, the inflammatory response is inhibited (Li, Li, et al., 2024). In the model of RPE cell injury induced by 4-hydroxyhexenal (HHE), the NLR family pyrin domain containing 3 (NLRP3) inflammasome is activated, the expression of related genes (NLRP3, IL-1β, IL-18, and caspase-1) is up-regulated, and the release of pro-inflammatory cytokines (IL-1β and IL-18) is increased (Wang, Zhao, et al., 2015). However, C3G pretreatment can significantly improve the above inflammatory indicators by inhibiting the c-Jun N-terminal kinase–c-Jun/activator protein-1 (JNK-c-Jun/AP-1) pathway, reducing the level of inflammation, and protecting RPE cells (Jin et al., 2018). These studies suggest that anthocyanins derived from dark-colored fruits and vegetables, through their potent antioxidant and anti-inflammatory activities, provide fundamental protection to ocular tissues by inhibiting key inflammatory pathways and reducing retinal inflammation (Table 1).

2.2. Improve retinal microcirculation and vascular function

The retina is one of the most metabolically active tissues in the human body. It has high demands for oxygen and energy and is rich in polyunsaturated fatty acids. Due to long-term exposure to light and an oxygen-rich environment, the retina is prone to generating ROS. ROS triggers lipid peroxidation, protein damage, and DNA oxidation, which in turn initiate an inflammatory cascade. Ultimately, these events lead to cellular dysfunction and apoptosis (Cosker et al., 2020; Zhu et al., 2023).

Retinal microcirculation is a network of small vessels that supplies oxygen and nutrients to the retina. This network is essential for meeting the high metabolic demands of the retina and for maintaining normal visual function. Diseases such as diabetic retinopathy and hypertensive retinopathy can cause retinal microcirculation disorders. These disorders manifest as pathological changes, including capillary leakage, occlusion, and neovascularization. Among these, oxidative stress and inflammation induced by a high-glucose environment are key factors that damage retinal vascular endothelial cells (Chong et al., 2024).

Studies have shown that anthocyanins significantly improve vascular function. They enhance vascular elasticity and inhibit abnormal vascular proliferation, thereby maintaining retinal microcirculation homeostasis. Primarily through antioxidant and anti-inflammatory effects, anthocyanins protect vascular endothelial cells from damage induced by high glucose and oxidized low-density lipoprotein. For example, blueberry-derived anthocyanins reduce levels of ROS and MDA. They also increase the activities of CAT and SOD. These effects enhance cell viability and attenuate high glucose (HG)-induced damage to human retinal capillary endothelial cells (HRCECs), thus protecting retinal capillary structure.

Furthermore, anthocyanins maintain the activity of endothelial nitric oxide synthase (eNOS). They promote the release of physiological nitric oxide (NO), which dilates blood vessels and enhances vascular elasticity. Consequently, retinal microcirculation is improved, and asthenopia is relieved (Ghosh & Konishi, 2007). Clinical studies have shown that after ingestion of blackcurrant or blueberry anthocyanins, blood flow velocity in the ophthalmic artery, retina, and choroid increases in healthy individuals or subjects with mild asthenopia. Laser Doppler flowmetry has also confirmed that retinal and choroidal blood flow significantly increases (Ohguro et al., 2012).

At the same time, anthocyanins contribute to the autonomic regulation of retinal microcirculation. Retinal vessels have the ability to dynamically adjust their diameters according to local metabolic demands (Wang et al., 2022). Anthocyanins can enhance this autoregulatory mechanism by improving endothelial function and neurovascular coupling, thereby maintaining blood flow stability during blood pressure or intraocular pressure fluctuations (Cremonini et al., 2019; Kang et al., 2018). Studies have also found that anthocyanins can improve the blood perfusion of the optic nerve head and regulate the diurnal fluctuation of intraocular pressure, suggesting their potential benefits in patients with normal-tension glaucoma.

In addition, anthocyanins can up-regulate the expression of tight junction proteins, such as zonula occludens-1 (ZO-1) and occludin, and inhibit the excessive production of vascular endothelial growth factor (VEGF), thereby enhancing the function of the blood-retinal outer barrier and reducing vascular leakage. In diabetic retinopathy models, anthocyanins can inhibit VEGF expression and protein kinase B (Akt) signaling pathway activation and reduce retinal vascular leakage and pathological neovascularization (Li et al., 2020; Liu et al., 2020).

Table 1
Studies on the anti-inflammatory and anti-oxidative mechanisms of anthocyanins in protecting the eyes.

Source of Anthocyanins	Animal/Cell Type	Disease/Injury Model	Experiment Results	References
Lycium Ruthenicum	Human retinal pigment epithelial (ARPE-19) cells	Blue light-induced retinal injury model	Anthocyanins can enhance the expression of antioxidant enzymes, inhibit inflammatory response and apoptosis through the Nrf2 pathway, and protect the retina from blue light damage	Li, Li, et al. (2024)
Blueberry	ARPE-19 cells	UVB irradiation induced oxidative damage model	Anthocyanins attenuate UVB-induced ROS generation and MAPK phosphorylation to protect RPE cells	Silván et al. (2016)
Blueberry	Rat	Diabetic retinopathy was induced by STZ	Anthocyanins reduce retinal oxidative stress and inflammation caused by diabetes through Nrf2/HO-1 pathway	Song et al. (2016)
Black Fruit Solanum	Rat	Light-induced retinal injury model	Anthocyanins can reduce photoreceptor apoptosis, anti-inflammatory and anti-oxidation, and play a protective role through the Nrf2/Sirt3/SOD2 pathway	Ye et al. (2025)
Bilberry	Rabbit	A model of visible-light-induced retinal degeneration	Anthocyanins can enhance the activity of antioxidant enzymes, inhibit inflammatory factors, reduce photoreceptor apoptosis, and have a protective effect on retina	Wang, Zhang, et al. (2015)
Blueberry	ARPE-19 cells	H2O2 -induced oxidative damage model	Anthocyanins protect RPE cells from oxidative stress by activating ERK/p38 pathway and enhancing antioxidant enzyme activity	Huang, Wu, et al. (2018)
Blueberry	ARPE-19 cells	Blue light and A2E induced ER stress model	Anthocyanins enhance RPE barrier function and protect retinal health by inhibiting endoplasmic reticulum stress and activating Nrf2 pathway	Peng et al. (2022)
Blueberry	Mouse, ARPE-19 cells	High glucose-induced injury model	Anthocyanins can reduce the levels of inflammatory factors and apoptosis of retinal cells induced by high glucose by activating GLP-1R/Akt signaling pathway, which has hypoglycemic and retinal protective effects	Li, Du, et al. (2025)
Black Bean Seed Coat	Human lens epithelial (HLE-B3) cells	H2O2 -induced oxidative stress model	Anthocyanins can protect lens epithelial cells from oxidative damage by inhibiting the expression of apoptosis-related proteins, which has the potential to prevent cataract	Mok et al. (2014)
Grape	Mouse	Uv-induced cataract model	Anthocyanins can inhibit the development of UV-induced cataract by regulating Nrf2 and XIAP pathways	Liu et al. (2024)
Blueberry	ARPE-19 cells	High glucose and H2O2 induced injury model	Anthocyanins can protect HRCECs and inhibit the expression of VEGF, which is expected to be used for the prevention and treatment of diabetic retinopathy	Huang, Wu, et al. (2018)
Black Fruit Solanum	Rat	Naio3-induced dry AMD model	Anthocyanins can reduce oxidative damage, up-regulate the expression of crystallin, and protect the retinal nervous system	Xing et al. (2021)
Bilberry	ARPE-19 cells	A2E photooxidation and membrane permeability model	It can protect RPE cells by quenching singlet oxygen, inhibiting A2E photooxidation and membrane damage	Jang et al. (2005)

Blueberry anthocyanins can also improve insulin signal transduction and reduce the apoptosis of retinal cells induced by high glucose by activating the glucagon-like peptide-1 receptor/protein kinase B (GLP-1R/Akt) signaling pathway, showing the potential to prevent and treat diabetic retinopathy.

In conclusion, anthocyanins improve microvascular endothelial

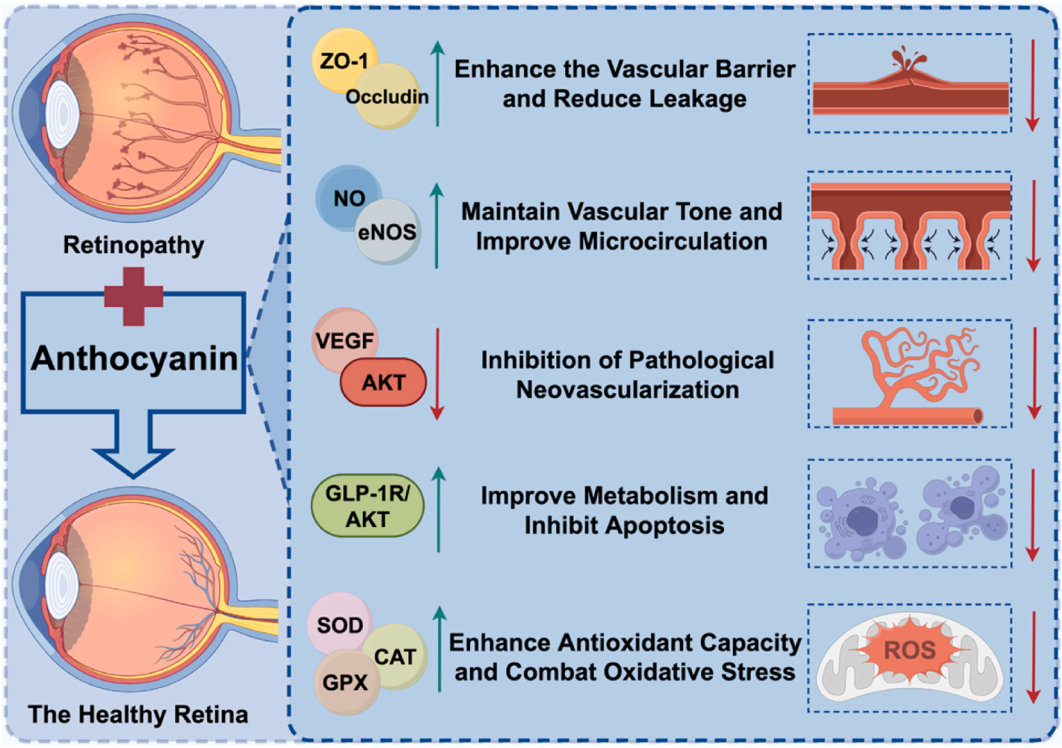


Fig. 1. Specific mechanisms by which anthocyanins improve retinal microcirculation and vascular function.

function through their anti-inflammatory and antioxidative effects, thereby increasing retinal blood flow and protecting microvascular structures (Fig. 1). These results suggest that anthocyanins play an important role in maintaining eye health and preventing vascular-related eye diseases. For people at risk of asthenopia, myopia, glaucoma, dry eye, floaters and diabetic retinopathy, increasing the intake of foods rich in anthocyanins may help to prevent or delay the progression of related diseases.

2.3. Promote regeneration of rhodopsin and improve visual function

Dark adaptation is an important indicator of retinal function, as it depends on the regeneration rate of the photoreceptor pigment rhodopsin (RHO) in rod cells (Hofmann & Lamb, 2023). RHO, composed of opsin and 11-cis retinal, is the key visual pigment for sensing low light in rod cells, and its regeneration ability directly affects dark vision and contrast sensitivity. Upon light exposure, RHO decomposes into all-trans retinal and opsin, after which it requires the involvement of retinal pigment epithelial cells to complete the regeneration cycle (Smylla et al., 2022; Tu et al., 2024). Factors such as light damage and oxidative stress can destabilize RHO and lead to visual function decline.

Many studies have found that anthocyanins from certain sources can promote RHO regeneration and improve visual function. It has been speculated that cyanidin and delphinidin aglycones, which have some structural characteristics similar to retinol, might influence rhodopsin regeneration; however, no direct molecular evidence (e.g., binding affinity, docking, or crystallographic data) supports an interaction with opsin or visual cycle enzymes. Currently, the exact mechanism remains unknown. Nonetheless, numerous human intervention trials have provided functional evidence: short-term intake of anthocyanin-rich blueberry or blackcurrant extracts significantly improves the speed of dark adaptation threshold recovery in healthy subjects and individuals with asthenopia (Kalt et al., 2014; Nakaishi et al., 2000). These effects may be indirect, potentially involving reduction of oxidative stress, improvement of retinal microcirculation, or upregulation of rhodopsin gene expression. *Aronia melanocarpa* anthocyanins (AMA) intervention reduces the attenuation of electroretinogram (ERG) a-wave and b-wave amplitudes in light-damaged rats, indicating protection of retinal electrophysiological function, and significantly increases retinal expression of RHO and the rod inhibitory protein (SAG) (Ye et al., 2025). Further analysis showed that C3G, the major functional component of AMA, has a strong binding affinity for RHO and SAG, suggesting that AMA may directly stabilize or promote the synthesis and function of these visual proteins. Additionally, the regulation and stabilization of retinal microcirculation by anthocyanins indirectly provide a favorable microenvironment for RHO regeneration.

In terms of improving functional vision, a randomized controlled trial in healthy adults found that the intake of a compound supplement containing anthocyanin, astaxanthin, and lutein for 6 weeks could significantly improve the decline in accommodative function caused by the operation of a visual display terminal and reduce the subjective symptoms of "eye focus difficulty" and "near object difficulty" (Kizawa et al., 2021). Another study showed that supplementation of black currant anthocyanins could improve the visual motion perception of aging mice and improve their ability to track low-contrast motion patterns (optokinetic response), which may be related to the up-regulation of brain-derived neurotrophic factor (BDNF), epidermal growth factor (EGF), and other neurotrophic factor gene expression in the retina and primary visual cortex (V1) (Sugita et al., 2025). These studies suggest that anthocyanins not only protect visual pigments and promote rhodopsin regeneration at the molecular level but also improve dynamic vision, contrast sensitivity, and visual function.

2.4. Protect retinal nerve cells and photoreceptors

The retina not only acts as a light-sensitive tissue but also constitutes a sophisticated neural network. The progressive loss of retinal ganglion cells (RGCs) and photoreceptor cells (rods and cones) is the common pathological end point of blinding eye diseases such as glaucoma and retinitis pigmentosa (Yang et al., 2023). Therefore, it is important to protect these neurons from apoptosis and degeneration.

Anthocyanins have shown multi-target neuroprotective potential and exert clear protective effects against retinal nerve cell death induced by various damaging factors. For example, factors such as high intraocular pressure can cause glutamate accumulation outside retinal cells, over-activating N-methyl-D-aspartate (NMDA) receptors. This leads to calcium overload and excitotoxicity, ultimately resulting in RGC apoptosis (Li, Du, et al., 2025). Studies have shown that bilberry anthocyanins can antagonize NMDA receptors and reduce glutamate-induced damage to RGCs and RPE cells (Wu et al., 2024). The underlying mechanisms include upregulating the anti-apoptotic protein B-cell lymphoma 2 (Bcl-2), downregulating the pro-apoptotic protein Bcl-2-associated X protein (Bax), and inhibiting cysteine-aspartic protease 3 (caspase-3) activation, thereby blocking the mitochondrial apoptotic pathway (Matsunaga et al., 2009).

In addition, purple rice-derived anthocyanins significantly inhibited tunicamycin-induced endoplasmic reticulum (ER) stress, reduced caspase-3 activation in RGCs, and suppressed tunicamycin-induced RGC degeneration in a mouse *in vivo* model (Tanaka et al., 2013). The same extract also effectively scavenged 2,2-diphenyl-1-picrylhydrazyl (DPPH) radicals, superoxide anions, and hydroxyl radicals, reduced ROS levels in 661W photoreceptor cells after light exposure, and subsequently inhibited light-induced photoreceptor degeneration in both *in vitro* and *in vivo* models (Paik et al., 2012). Collectively, these findings indicate that anthocyanins protect retinal neurons through multiple mechanisms, including NMDA receptor antagonism, modulation of mitochondrial and ER stress-related apoptotic pathways, and reduction of oxidative stress.

In other injury models, long-term administration of black bean seed coat anthocyanins (4 weeks) can significantly preserve the thickness of the outer nuclear layer of photoreceptors, improve the electroretinogram (ERG) response, and protect retinal neurons from structural and functional damage in the rat N-methyl-N-nitrosourea (MNU)-induced retinal degeneration model (Hwang et al., 2012). Different anthocyanin monomers isolated from blueberry, blackberry, and strawberry have protective effects on RPE cells against visible light damage, among which C3G shows the strongest antioxidant, anti-angiogenic, and anti-aging activities (Wang et al., 2016, 2019).

In addition to direct anti-apoptosis and anti-oxidation effects, anthocyanins can also up-regulate the expression of neurotrophic factors such as BDNF to provide nutritional support for RGCs. Under oxidative stress conditions, anthocyanins can help to remove damaged organelles and protein aggregates by regulating autophagic flow, such as enhancing the microtubule-associated protein 1 light chain 3-II (LC3-II) level and reducing sequestosome-1 (p62) accumulation, and maintain neuronal homeostasis (Guo et al., 2024; Li et al., 2022).

Taken together, this evidence suggests that anthocyanins provide a broad protective network for retinal neurons and photoreceptors by inhibiting apoptotic pathways, scavenging free radicals, alleviating endoplasmic reticulum stress, regulating autophagy, and enhancing neurotrophic support.

3. Application potential and prospect of anthocyanins

3.1. Prevention of age-related eye diseases: from cataract to macular degenerationImprove retinal microcirculation and vascular function

Age-related eye diseases, including cataract and age-related macular degeneration (AMD), are the leading causes of vision loss in the elderly. Its pathogenesis is closely related to long-term accumulated oxidative damage, protein denaturation, chronic inflammation, and metabolic disorders (Cvekl & Vijg, 2024; Guymer & Campbell, 2023). Based on the multiple mechanisms described above, anthocyanins show

comprehensive potential in preventing and delaying age-related eye diseases such as AMD and cataracts.

A cataract is an opacification caused by oxidative degeneration and aggregation of lens proteins (Mishra et al., 2023). Anthocyanins and their metabolites are able to cross the blood-aqueous barrier into intraocular tissues and protect lens epithelial cells from ultraviolet or hydrogen peroxide-induced oxidative damage, thereby contributing to the maintenance of lens transparency (Sesso et al., 2024; Song et al., 2020). *In vitro* experiments have shown that anthocyanins have anti-apoptotic protective effects on human lens epithelial cells (HLE-B3) under oxidative stress. For example, black bean seed coat anthocyanins can protect HLE-B3 cells from H_2O_2-induced oxidative stress and apoptosis by down-regulating pro-apoptotic proteins (Bax, caspase-3) and up-regulating anti-apoptotic protein Bcl-2 (Mok et al., 2014). In a porcine lens organ culture model, it also maintained lens transparency, suggesting its potential to prevent cataracts. Animal experiments have further confirmed that dietary supplementation of grape powder (GP) containing anthocyanins and other polyphenols can improve the reduction–oxidation reaction (REDOX) homeostasis of the lens and inhibit the formation of UV-induced cataracts in mice by regulating Nrf2 and X-linked inhibitor of apoptosis protein (XIAP) pathways (Liu et al., 2024).

AMD is the leading cause of blindness in the elderly population in developed countries, which is characterized by the decline of RPE cell function, lipofuscin accumulation, and drusen formation, leading to geographic atrophy (dry AMD) or choroidal neovascularization (wet AMD) (Blasiak et al., 2023; James, 2024). Anthocyanins have shown great value in the prevention and treatment of AMD through multi-target intervention. A number of studies have confirmed that anthocyanins can comprehensively protect RPE cells from blue light, visible light, H_2O_2, N-retinylidene-N-retinylethanolamine (A_2E) photooxidation, and other damage through multiple pathways such as anti-oxidation, anti-inflammation, anti-apoptosis, and anti-angiogenesis and directly combat the core pathological links of AMD, thereby slowing the progression of AMD from the upstream (Guymer & Campbell, 2023). Epidemiological data suggest that diets rich in anthocyanins are associated with a reduced risk of AMD (Klusek et al., 2025), and long-term supplementation of anthocyanins has also been found to alleviate AMD-like pathological changes in animal models (Li, Li, et al., 2024).

Dry AMD currently lacks effective therapies, and its core pathological processes include oxidative stress, inflammation, RPE dysfunction, and photoreceptor cell death (Girgis & Lee, 2023). Studies have shown that anthocyanins extracted from *Solanum nigrum* fruit can improve the dry AMD model of rats induced by sodium iodate and reduce the ERG b-wave amplitude and retinal structure disorder (Huang, Yan, et al., 2018; Zuo et al., 2025). Proteomics analysis further revealed that its mechanism of action was closely related to the significant up-regulation of the expression of a variety of lens proteins with neuroprotective effects, such as αA-, βB2-, βA3-, αB-, and γS-crystallin (Xing et al., 2021).

Moreover, anthocyanins inhibited the photooxidation of A_2E. A_2E is the main component of lipofuscin in RPE cells, which accumulates in lysosomes and is difficult to degrade. Its photooxidation damages cells and leads to photoreceptor degeneration in patients with dry AMD (Yang et al., 2025; Arunkumar & Bernstein, 2023). Studies have shown that a variety of anthocyanins can inhibit the photooxidation of A_2E by quenching singlet oxygen and enhance the resistance of RPE cells to A_2E penetration, thereby protecting RPE cells and delaying the progression of dry AMD (Jang et al., 2005).

For wet AMD, blueberry anthocyanin extract (BAE) and its monomer components can reduce the expression of VEGF in RPE cells, suggesting its potential to inhibit choroidal neovascularization (Foss et al., 2022). Studies have also found that C3G can protect RPE cells from the damage of 4-hydroxynonenal (HNE), the end product of lipid peroxidation, and inhibit cell senescence and angiogenesis, which is of great significance for delaying the progression of AMD (Yang et al., 2023)

3.2. Structure-activity relationship, bioavailability and synergism of anthocyanins

The biological activities of anthocyanins are closely related to their core C6-C3-C6 flavonoid skeleton and various structural modifications. Supplementary Fig. 1 (Zang et al., 2022) shows the chemical structures of six common anthocyanins and their aglycones, whose key structural features determine specific activities such as antioxidant, anti-inflammatory, and anti-cancer effects. Studies have shown that the eye protection effect of anthocyanins is closely linked to their chemical structure (Cheng et al., 2023). In general, a higher degree of hydroxylation confers greater antioxidant capacity. For example, C3G exhibits strong antioxidant, anti-angiogenic, and anti-aging activities due to its ortho-dihydroxyl structure on the B-ring (Chen et al., 2025). However, pelargonidin has the lowest hydroxylation degree and relatively weak antioxidant capacity, but it possesses special anticancer potential (Sood et al., 2024).

Beyond general antioxidant capacity, different anthocyanins show distinct and often complementary mechanisms in ocular protection, leading to unequal efficacy. For instance, delphinidin-3-O-rutinoside (D3R) is particularly effective in enhancing cell viability under oxidative stress. In terms of anti-inflammatory pathways, C3G excels at suppressing the JNK-c-Jun/AP-1 axis, thereby alleviating ocular inflammation. For photoreceptor recovery, C3G and its analog cyanidin-3-O-rutinoside (C3R) effectively promote rhodopsin regeneration, which is critical for dark adaptation and night vision. Regarding vascular regulation, both C3G and malvidin-3-O-glucoside (Mv-3-glu) significantly downregulate VEGF expression, contributing to retinal vascular homeostasis. Several anthocyanins, including petunidin, have been found to inhibit the photooxidation of lipofuscin (A_2E) in retinal pigment epithelial cells, thus protecting against light-induced damage. Among these, C3G demonstrates the broadest spectrum of protective effects, covering antioxidant, anti-inflammatory, rhodopsin regeneration, and vascular protection. In contrast, pelargonidin-3-O-glucoside (Pg-3-glu) shows notable anti-aging activity but is less comprehensive than C3G in other aspects. Collectively, although all anthocyanins are generally beneficial to eye health, they support the visual system through multi-target synergistic effects. Their distinct advantages enable precision nutritional interventions targeting specific ocular conditions (Khoo et al., 2019; Li, Du, et al., 2025; Parmenter et al., 2023).

The effect of whole fruit is much better than that of purified extracts, a phenomenon referred to as the "food matrix effect," which arises from the complex network of protection, sustained release, and synergy provided by the intact food matrix (Lem et al., 2022). In addition, oligomeric anthocyanins and complete anthocyanin-rich extracts have also shown promising effects, suggesting that other components in the food matrix may produce synergistic effects (Ghosh & Konishi, 2007; Rapisarda et al., 2022). Although most studies are still at the preclinical stage, randomized controlled trials have preliminarily confirmed the clinical benefits of anthocyanin-rich supplements in relieving digital eye fatigue and improving dry eye symptoms (Mattioli et al., 2020; Nuszkiewicz et al., 2025).

Bioavailability is the key factor restricting the application of anthocyanins. Despite their well-documented multi-target ocular protective potential, anthocyanins face critical bottlenecks of low oral bioavailability and poor chemical stability, which substantially constrain their translational application as functional food ingredients. As shown in Fig. 2, the prototype of anthocyanins has an extremely low absorption rate (<1%) in the human body. The chemical instability and low bioavailability of anthocyanins represent core obstacles limiting their health benefits. Numerous studies have demonstrated that anthocyanins are highly susceptible to external environmental factors, including light, oxygen, temperature, and pH variations, which lead to molecular degradation and substantially diminish their actual health effects (Saini et al., 2024). During gastrointestinal digestion, anthocyanins undergo

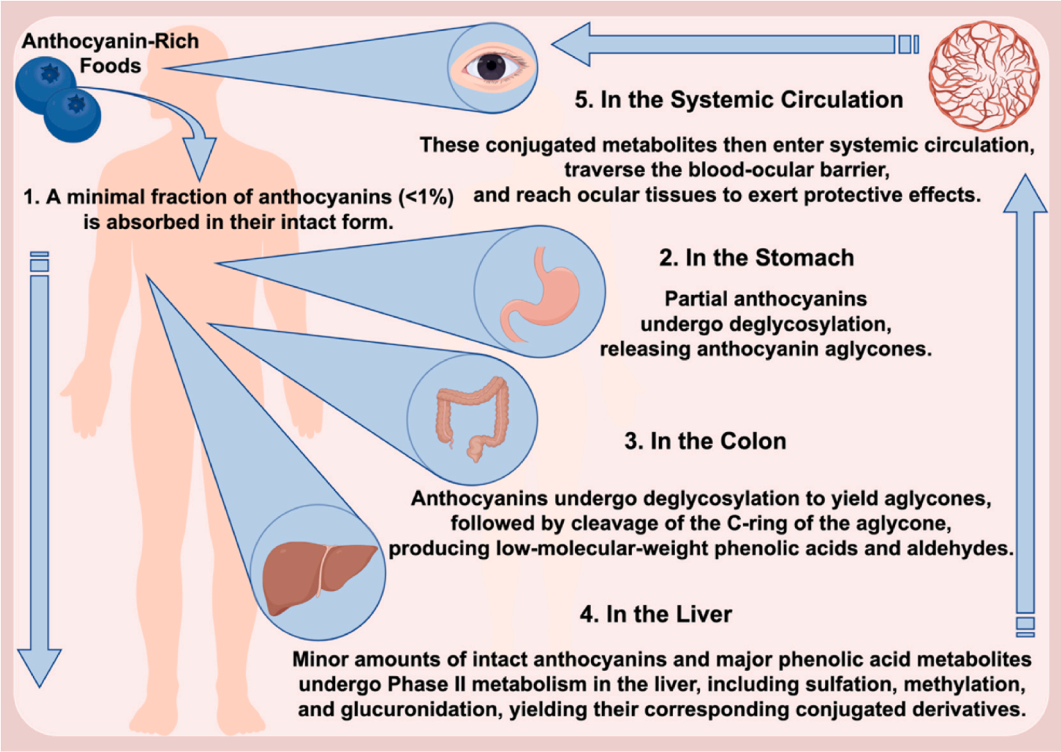


Fig. 2. Major metabolic pathways of anthocyanins in humans.

deglycosylation, methylation, sulfation, and glucuronidation (Ohara et al., 2024; Wattanathorn et al., 2023), followed by biphasic metabolism in the liver to form various metabolites that enter the circulation and eventually reach the eye to exert protective effects.

Although prototypical anthocyanins have limited absorption, their metabolites may still be biologically active. Among the different structural forms, acylated anthocyanins exhibit high stability in the gastrointestinal tract but have very low absorption, relying mainly on colonic metabolism to produce specific phenolic acids and exert long-term effects (Parekh et al., 2024). In contrast, methylated anthocyanins possess relatively higher absorption rates due to their strong lipophilicity and better membrane permeability (Gutiérrez-;Quequezana et al., 2018). Therefore, designing stable and highly bioavailable anthocyanin derivatives using acylation and methylation strategies may represent a key approach to improving anthocyanin bioavailability (Alonso et al., 2018; Wang et al., 2025; Olmedilla).

To address these bioavailability bottlenecks, multiple strategies have been developed. Among these, nanoencapsulation technology stands out as one of the most promising approaches. By self-assembling nanostructures using materials such as citrus pectin and lysozyme, nanoencapsulation effectively protects anthocyanins from gastrointestinal degradation (Rosales et al., 2024). A 2024 study utilizing radiolabeled anthocyanins demonstrated that nanoencapsulation significantly altered the absorption kinetics of C3G, increasing both blood bioavailability and tissue uptake. Beyond nanodelivery systems, techniques including acylation, copigmentation, and the preparation of nanogels, nanoemulsions, and liposomes have also been confirmed to markedly improve the stability and bioavailability of anthocyanins (Zhang et al., 2025). Furthermore, the food matrix effect plays a crucial role in preserving anthocyanin integrity. Studies have shown that anthocyanin retention during intestinal digestion is considerably higher in whole fruit (44%) than in purified extracts (12%), indicating that the intact food matrix substantially enhances anthocyanin bioaccessibility through complex protection, sustained release, and synergistic networks (Kumkum et al., 2026). Collectively, how to improve bioavailability and targeting by modification of dosage forms (such as liposomes and nanocarriers) and compounding with other components remains the focus of future research.

In terms of synergistic effects, studies have shown that anthocyanins may produce stronger antioxidant and anti-inflammatory effects when combined with other phytochemicals, such as lutein, zeaxanthin, vitamin C, quercetin, resveratrol, etc. (Mrowicka et al., 2022; Widomska et al., 2023; Plevin & Galletly, 2020). For example, C3G combined with zeaxanthin has a synergistic protective effect against UVB-induced damage to RPE cells (Silván et al., 2016). Vitamin C can maintain an acidic environment, protect the stability of anthocyanins, and regenerate oxidized anthocyanins to form an antioxidant cycle (Rodrigo et al., 2022). In addition, the combination with other bioactive substances that also have anti-inflammatory and anti-oxidative stress effects can jointly regulate signaling pathways through multi-target effects, resulting in additive or enhanced effects (Massimo et al., 2021; Zhang et al., 2023). For example, a combined application with curcumin and taurine can jointly regulate inflammatory pathways and inhibit inflammatory factors, and the effect is better than that of single components (Kono et al., 2014; Silván et al., 2016). Therefore, the development of scientifically formulated compound supplements may be an effective strategy to improve the eye protection effect.

3.3. Gut Microbiota–Anthocyanin interactions: metabolic conversion and ocular implications

Gut microbiota are a core determinant of anthocyanin oral bioavailability. The vast majority of anthocyanin prototypes are absorbed at extremely low rates in the human body. After oral intake, they enter the colon and undergo extensive interactions with the gut microbiota, which convert them into low-molecular-weight phenolic acid metabolites with higher biological activity. The pharmacokinetic profiles of these metabolites and their specific links to ocular structures form the theoretical basis for the efficacy of orally administered anthocyanins.

Specific microbial communities in the colon catalyze the hydrolysis of anthocyanin glycosides via extracellular β-glucosidases, releasing aglycones that are further cleaved at the C-ring to produce various phenolic acids. *In vitro* and animal studies have identified several key bacterial genera closely associated with anthocyanin biotransformation, including *Bifidobacterium* and *Lactobacillus*. These beneficial bacteria not only mediate anthocyanin metabolism but are also reciprocally promoted by anthocyanins, forming a bidirectional interaction network (Zeng et al., 2026). *Bacteroides fragilis* and *Eubacterium ramulus* have also been found to participate in the fermentative metabolism of pigmented rice anthocyanins, producing p-coumaric acid, salicylic acid, and other phenolic acids. Recent research further revealed that blueberry and blackberry anthocyanins ameliorate metabolic syndrome by modulating *Prevotella histicola*, suggesting that specific bacterial strains may play a key role in mediating the health benefits of anthocyanins (Du et al., 2025). Moreover, *Akkermansia muciniphila* and *Lactobacillus reuteri* are significantly enriched upon intervention with multi-acylated anthocyanins, indicating that structurally modified anthocyanins exert selective regulation of gut microbiota.

The phenolic acid metabolites generated from anthocyanins by gut microbiota are diverse, mainly including protocatechuic acid, syringic acid, gallic acid, p-coumaric acid, vanillic acid, ferulic acid, and hippuric acid (Iqbal et al., 2023). A human pharmacokinetic study using ^{13}C-labeled C3G systematically identified 17 metabolites in serum, including intact C3G, its degradation products protocatechuic acid and phloroglucinaldehyde, 13 metabolites of protocatechuic acid, and one metabolite of phloroglucinaldehyde. The maximum plasma concentrations of these phenolic acid metabolites ranged from 10 to 2000 nM, with time to peak between 2 and 30 h and elimination half-lives up to 0.5–96 h. The most abundant metabolites were hippuric acid and ferulic acid, with serum peak times of approximately 16 and 8 h, respectively (de Ferrars et al., 2014). Notably, the plasma concentrations of small phenolic acids such as protocatechuic, syringic, and gallic acids can reach up to 100 times those of the parent anthocyanins and are capable of crossing the blood–brain barrier to exert systemic effects (Zeng et al., 2026). Another study reported that the maximum plasma concentration of intact anthocyanins is only 14–592 nmol/L (after ingestion of 68–1300 mg, peaking at 4–5 h), contrasting sharply with the much higher levels of their microbial metabolites, further confirming the central role of microbial metabolism in the *in vivo* activity of anthocyanins (Iqbal et al., 2023).

Whether the phenolic acid metabolites of anthocyanins can reach ocular tissues and exert protective effects depends on their ability to cross the blood–retinal barrier (BRB). *In vitro* BRB permeability studies have shown significant differences among various classes of polyphenols: phenolic acids > non-anthocyanin flavonoids > intact anthocyanins. Glycosylation and methylation improve the BRB permeability of anthocyanins, whereas C-4 carbonylation and structural instability severely impair it. Hydrophilic phenolic acids exhibit better BRB penetration than their hydrophobic counterparts (Liu et al., 2017). This indicates that the phenolic acid metabolites generated from anthocyanins by gut microbiota—especially hydrophilic phenolic acids—have an intrinsic advantage in trans-BRB delivery.

Multiple studies have further confirmed that phenolic acid metabolites can act directly on retinal cells. For instance, p-coumaric acid and related phenolic compounds significantly inhibit VEGF-induced proliferation, migration, tube formation, and endothelial permeability in human retinal microvascular endothelial cells. Molecular docking simulations revealed the high binding affinity of these compounds to vascular endothelial growth factor receptor 2 (VEGFR2) (Kim et al., 2024). The retina, as a tissue with high oxygen consumption, is particularly vulnerable to oxidative damage. Phenolic acid metabolites can cross the BRB, enter the retinal microenvironment, directly scavenge reactive oxygen species, suppress inflammatory signaling pathways (e.g., NF-κB and Nrf2), and restore redox homeostasis, thereby protecting against various retinopathies. Animal studies have also demonstrated that after oral administration of blackcurrant anthocyanins, intact anthocyanins can cross the blood-aqueous barrier and the BRB to distribute into ocular tissues, suggesting that the delivery of intact anthocyanins and the penetration of their phenolic acid metabolites are parallel and complementary processes. In summary, the high concentrations of phenolic acid metabolites generated by gut microbiota, owing to their smaller molecular size and greater hydrophilicity, can effectively penetrate the BRB and distribute within retinal tissues, contributing to ocular protection through antioxidant and anti-inflammatory mechanisms.

4. Conclusions

In summary, existing evidence confirms that anthocyanins exert important protective effects on ocular health through multiple mechanisms, as illustrated in Fig. 3: (1) Antioxidant and anti-inflammatory effects—Anthocyanins scavenge reactive oxygen species, activate endogenous antioxidant pathways (e.g., Nrf2), and inhibit inflammatory signaling (e.g., NF-κB), thereby reducing oxidative damage and chronic inflammation in ocular tissues. However, it should be noted that most *in vitro* mechanistic studies used concentrations substantially higher than those achievable in human plasma; the *in vivo* relevance of these pathways is likely mediated by phenolic acid metabolites derived from gut microbial conversion. (2) Improvement of retinal microcirculation and vascular function—Anthocyanins enhance vascular elasticity, protect endothelial cells via eNOS/NO regulation, inhibit pathological angiogenesis, and maintain blood-retinal barrier integrity, offering benefits for diabetic retinopathy and other vascular-related eye diseases. (3) Promotion of rhodopsin regeneration and visual function—Human intervention trials have demonstrated that short-term intake of anthocyanin-rich extracts accelerates dark adaptation and alleviates asthenopia. However, the molecular mechanism remains unclear; the previously hypothesized “retinoid-like” interaction lacks direct evidence and should be interpreted with caution. (4) Neuroprotection of retinal neurons and photoreceptors—Anthocyanins protect against glutamate-induced excitotoxicity, endoplasmic reticulum stress, and mitochondrial apoptosis pathways, thereby preserving retinal ganglion cells and photoreceptors.

Beyond these mechanisms, the low oral bioavailability of intact anthocyanins (<1%) remains a critical bottleneck. The gut microbiota play a central role in converting anthocyanins into phenolic acid metabolites, which reach much higher plasma concentrations (10–2000 nM) and can cross the blood-retinal barrier. Therefore, future research should focus on the following: (1) conducting large-scale, long-term randomized controlled trials to clarify effective doses and clinical efficacy in different eye diseases; (2) elucidating structure-activity relationships and the ocular bioavailability of specific metabolites; (3) developing bioavailability-enhancing strategies such as nanoencapsulation, acylation/methylation, food matrix optimization, and synergistic combinations with other nutraceuticals (e.g., lutein, zeaxanthin).

In conclusion, anthocyanins represent a class of natural, safe, and multi-target bioactive compounds with significant potential for maintaining eye health and preventing chronic ocular diseases. Incorporating anthocyanin-rich foods or well-designed supplements into daily diets may offer a feasible strategy to address global eye health challenges, provided that bioavailability limitations are adequately addressed.

CRediT authorship contribution statement

Dongdong Zhao: Writing – review & editing, Supervision, Methodology, Conceptualization. **Yifan Rao:** Writing – review & editing, Writing – original draft, Software, Formal analysis, Data curation. **Jinlong Tian:** Validation, Formal analysis, Data curation. **Zhihuan Zang:** Resources, Project administration. **Rodrigo Quintana:** Resources, Conceptualization. **Baoru Yang:** Visualization, Conceptualization. **Yadong Li:** Visualization, Project administration. **Yuxi Lang:**

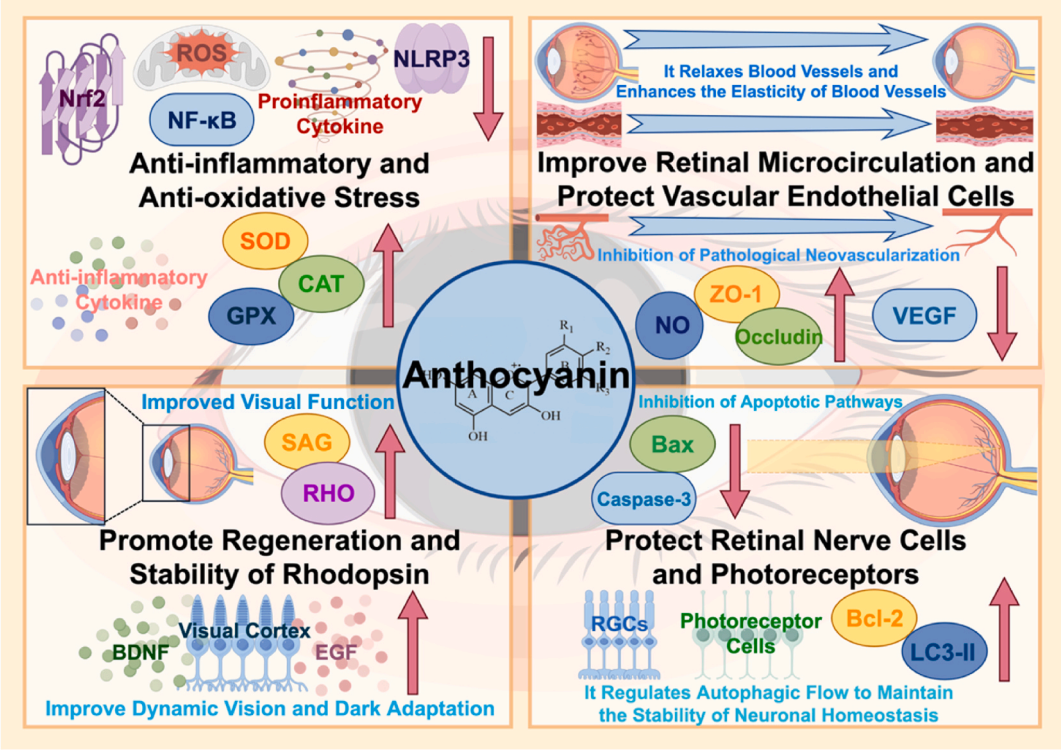


Fig. 3. Core mechanisms of eye protection by anthocyanins.

Supervision, Investigation. **Ying Zhang:** Project administration, Formal analysis. **Bin Li:** Project administration, Formal analysis, Conceptualization.

Figures statement

Figs. 1–3 were created by Yifan Rao (co-first author) using Figdraw (www.figdraw.com). These figures are original and have not been published elsewhere.

Search strategy and selection criteria

A systematic literature search was performed in PubMed, Web of Science, and Scopus from inception to December 2025. The search strategy combined terms related to anthocyanins (e.g., "anthocyanin," "cyanidin," "delphinidin," "malvidin," "pelargonidin," "petunidin," "peonidin," and "anthocyanin-rich extract") and terms related to eye health (e.g., "eye," "ocular," "retina," "retinal pigment epithelium," "RPE," "age-related macular degeneration," "AMD," "diabetic retinopathy," "glaucoma," "cataract," "asthenopia," "digital eye strain," "rhodopsin," "dark adaptation," "microcirculation," and "blood-retinal barrier"). Only peer-reviewed original research articles and reviews written in English were included. Studies were considered eligible if they investigated the protective effects of anthocyanins (monomers or extracts) on ocular health, regardless of study model (*in vitro*, *in vivo*, or clinical). Exclusion criteria included conference abstracts, patents, dissertations, studies not involving anthocyanins or eye health, and articles lacking sufficient mechanistic data. After title/abstract screening and full-text assessment, a total of 97 articles were finally included and cited in this review. The literature search was updated in March 2026 to incorporate recently published key findings (e.g., Kumkum et al., 2026; Zeng et al., 2026).

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.fbio.2026.109322.

Data availability

No data was used for the research described in the article.

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