Review Article

Vitamins and ocular health: a perspective on nutritional influence in vision disorders

Pragati Garg [1] , Shrinkhal Shrinkhal [1] , Ruchi Shukla[1] , Aparajita Shukla [1] , Swarastra P Singh [1] and Shourriya Garg [2]

[1] *Department of Ophthalmology, All India Institute of Medical Sciences, Raebareli Uttar Pradesh, India*
[2] *Jhalawar Medical College, Rajasthan, India*

ABSTRACT

Background: Vitamins are essential for ocular health, and their deficiencies are associated with xerophthalmia, age-related macular degeneration (AMD), cataracts, glaucoma, diabetic retinopathy (DR), dry eye disease (DED), keratoconus, and optic neuropathies. Beyond preventing nutritional deficiency disorders, vitamins contribute to phototransduction, antioxidant defense, immune regulation, and maintenance of retinal, corneal, and optic nerve function. This narrative review summarizes current evidence on the role of individual vitamins in ocular health, highlights clinical findings, and identifies research gaps.

Methods: A targeted search of PubMed/MEDLINE and Google Scholar was conducted for English-language studies published primarily between 2000 and 2025, supplemented by earlier landmark studies relevant to established nutritional interventions. Keywords included "vitamins", "ocular health", "eye disease", "antioxidants", "retina", "cornea", and "nutritional deficiency". Relevant clinical trials, observational studies, reviews, and guidelines were included. Non-English-language studies and unrelated nutritional research were excluded.

Results: Vitamin A is crucial for phototransduction and epithelial integrity; deficiency leads to night blindness, xerophthalmia, and corneal disease. B-complex vitamins, particularly B1, B2, B6, B12, and folate, are associated with neuro-ophthalmic function, retinal vascular health, and homocysteine metabolism. Vitamin C, a potent antioxidant, may protect against oxidative damage in the lens and retina and promotes corneal wound healing. Vitamin D has been investigated in DED, glaucoma, DR, keratoconus, and AMD because of its proposed anti-inflammatory, antioxidant, neuroprotective, and anti-angiogenic properties; however, evidence supporting supplementation remains condition-specific and generally limited. Vitamin E has antioxidant functions in retinal tissues and forms part of the combined AREDS/AREDS2 formulations, although vitamin E supplementation alone has not demonstrated a substantial reduction in incident AMD. Additional evidence supports the complementary role of lutein, zeaxanthin, omega-3 fatty acids, and zinc in maintaining retinal integrity and ocular surface health. Deficiencies, malabsorption syndromes, aging, and poor dietary habits contribute to ocular morbidities linked with hypovitaminosis.

Conclusions: Adequate intake of vitamins and selected micronutrients is essential for ocular surface maintenance, retinal function, optic nerve health, and protection against oxidative stress-related diseases. Nutritional assessment and correction of confirmed deficiencies are important in at-risk populations. However, evidence supporting supplementation to prevent or delay ophthalmic disease is condition- and nutrient-specific, with the clearest clinical benefit established for AREDS2 supplementation in appropriately selected patients with AMD. Further large-scale randomized controlled trials are warranted to establish evidence-based therapeutic guidelines and optimal supplementation strategies.

KEYWORDS

vitamin, B vitamin, riboflavin, vitamin B 6, pyridoxamine, vitamin B 12, hydroxocobalamin, vitamin D, vitamin E, vitamin A, ascorbic acid, folic acid, omega 3 fatty acid, flavonoid, eye, cataracts, dry eye disease, age-related macular degeneration, optic neuropathy

Correspondences: Pragati Garg, Department of Ophthalmology, All India Institute of Medical Sciences, Raebareli Uttar Pradesh, India. Emails: ophthal_pragati@aiimsrbl.edu.in, drpragati89@gmail.com. ORCID iD: https://orcid.org/0000-0002-6811-3779.

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INTRODUCTION

Approximately 250 million people worldwide suffer from varying degrees of vision loss. Leading causes include eye conditions such as cataracts, age-related macular degeneration (AMD), glaucoma, and diabetic retinopathy (DR). As these conditions disproportionately affect older adults, with an ageing population the number of affected individuals is predicted to increase exponentially [1].

Maintenance of ocular health is influenced by adequate nutritional intake, particularly vitamins. Given the high metabolic demands of the retina, vitamins serve as important co-factors in phototransduction, antioxidant defense, immune regulation, and neuronal conduction [2]. Vitamin deficiencies, often underrecognized, can lead to a spectrum of ocular manifestations; from dry eye disease (DED) and night blindness to optic neuropathy and irreversible vision loss. The role of nutritional supplements, particularly those containing antioxidants, in promoting ocular health is a subject of ongoing investigation. There is significant research interest in the role of dietary antioxidants and the potential therapeutic benefits of antioxidant vitamin and mineral supplements as a simple and cost-effective strategy for disease prevention and/or control [3]. While numerous products are marketed for vision enhancement, the literature review reveals nuanced findings regarding their effectiveness for the average consumer. Antioxidant supplementation with lutein may slow AMD progression [4]. However, the evidence for other ocular conditions varies. For glaucoma, some studies suggest a benefit while others find no effect, although antioxidant-rich fruits and vegetables may offer protection [5–7].

Xerophthalmia remains the classic manifestation of vitamin A deficiency, with studies reporting high prevalence in at-risk populations [8]. Nutritional optic neuropathy is associated with vitamin B12 deficiency with moderate association to vitamin D3 deficiency. Such deficiencies are uncommon but often coexist with poor diet, chronic alcoholism, and malabsorptive disorders. Neurotrophic vitamins (B1, B6, and B12) serve as essential cofactors in enzymatic processes that support optic nerve function, while folate deficiency has also been implicated in visual system dysfunction and optic atrophy [9]. In DR multiple vitamin deficiencies often coexist, most notably involving vitamins D3, B12, and E [10–12]. Glaucoma is increasingly being linked to deficiencies in vitamins D3 and B12, with modest associations to vitamins E and A [5]. Observational studies have reported associations between lower serum vitamin E levels and AMD severity [13]; however, randomized trials of vitamin E alone have not demonstrated a substantial reduction in incident AMD [14]. Folate deficiency has been associated with 75% and 89% higher risks of incident early AMD and any AMD, respectively, over 10 years [15]. This supports the view that a spectrum of micronutrient deficiencies may contribute synergistically to disease onset and progression in ocular pathology. Table 1 summarizes evidence linking vitamin deficiencies to ocular diseases. The strength of association varies by nutrient and disease type, with certain deficiencies acting synergistically to influence disease onset and progression [3, 16, 17].

Loskutova et al. [6] systematically reviewed 33 intervention trials and found that certain nutrients, especially flavonoids and forskolin, may improve ocular blood flow and reduce intraocular pressure in glaucoma. While early findings suggest potential neuroprotective benefits, the evidence remains inconclusive, warranting further high-quality research [6]. In DR, antioxidant supplements might provide a minor benefit, if any, but only as an adjunct to strict glycemic control [18]. Evidence regarding antioxidant supplementation in retinopathy of prematurity [19, 20] and retinitis pigmentosa [21, 22] is nutrient- and outcome-specific and remains inconclusive; therefore, no general supplementation recommendation can be made.

Vitamins and other micronutrients play diverse and important roles in maintaining ocular health and preventing disease [23]. Vitamin A is essential for visual phototransduction and maintaining the integrity of the cornea and conjunctiva, while deficiencies can lead to night blindness and xerophthalmia. B-complex vitamins, particularly B12, are crucial for neuro-ophthalmic function, with deficiency linked to optic neuropathy. Vitamin C, a potent antioxidant, may protect the lens and supports corneal wound healing. Vitamin D status has been investigated in relation to DED, keratoconus, AMD, and other ophthalmic conditions. However, evidence supporting supplementation is limited and condition-specific [16, 24, 25] (Tables 2 and 3).

Adequate nutrition, particularly vitamins, is increasingly recognized as important for ocular health. A review highlighted the potential role of various micronutrients and nutraceutical products in treating ocular surface diseases. These include omega-3 fatty acids; vitamins A, B12, C, and D; selenium; curcumin; and flavonoids [26].

The modern ophthalmologist must be well-versed in identifying and managing vitamin-related ocular diseases, especially in high-risk populations like strict vegetarians, the elderly, diabetics, and individuals with chronic systemic illnesses [3, 9–11]. This narrative review aims to summarize the current understanding of the impact of individual vitamins on eye health, highlight clinical evidence, and identify gaps in knowledge for future research.

METHODS

A targeted search of PubMed/MEDLINE and Google Scholar was conducted for English-language studies published primarily between 2000 and 2025, supplemented by earlier landmark studies relevant to established nutritional interventions. Keywords included “vitamins,” “ocular health,” “eye disease,” “antioxidants,” “retina,” “cornea,” and “nutritional deficiency.” Relevant clinical trials, observational studies, reviews, and guidelines were included, whereas non-English-language publications and unrelated nutritional research were excluded.

Table 1. Summary of key vitamin deficiency associations with major ocular diseases

Ocular Disease	Key Vitamin Deficiency Associations	Notes
Xerophthalmia	Vitamin A	Classic manifestation of vitamin A deficiency; high prevalence in at-risk populations [8].
Nutritional Optic Neuropathy	Vitamins B12, B1, B6, B9, and D3	Strongly associated with vitamin B12 deficiency and moderately associated with vitamin D3 deficiency; commonly occurs in the setting of poor diet, alcoholism, or malabsorption. Neurotrophic vitamins support normal optic nerve function [3].
Diabetic Retinopathy	Vitamins D3, B12, and E	Multiple deficiencies often coexist; notable involvement of D3, B12, E [10–12].
Glaucoma	Vitamins D3, B12, E, and A	Increasingly linked to D3 and B12; modest association with E and A [5, 24, 27, 28].
Age-related Macular Degeneration (AMD)	Vitamins E and B9	Lower serum vitamin E levels have been associated with greater AMD severity in an observational study [13]. Folate deficiency was associated with 75% and 89% higher risks of incident early AMD and any AMD, respectively, over 10 years [29].

Table 2. Vitamin A in eye health, functions, deficiency manifestations, and clinical relevance

Aspect	Details
Role	Visual phototransduction through the regeneration of rhodopsin [30].
	Epithelial integrity of the conjunctiva and cornea [8, 26].
Deficiency Manifestations	Night blindness [8, 30].
	Xerophthalmia [8].
	Bitot's spots [8].
	Keratomalacia [8].
	Corneal ulceration and perforation [3, 8].
Clinical Implications	Vitamin A supplementation remains a cornerstone in pediatric ophthalmic public health programs across developing countries to reduce childhood blindness [1, 8]. Topical retinoids may aid in ocular surface healing in keratinizing disorders [3, 26].

RESULTS and DISCUSSION

Vitamin A: The Retinoid in Retinal Function

Vitamin A is a vital fat-soluble nutrient that plays a crucial role in maintaining several bodily functions, including support of normal vision and eye health, especially in low light conditions through its role in rhodopsin regeneration. It also contributes to modulating immune function and inflammation, promoting reproductive health and fertility by regulating gene expression and cellular differentiation, and supporting the growth and development of vital organs. Further, vitamin A helps regulate energy metabolism and glucose synthesis plus maintain healthy skin and mucous membranes through its role in keratinocyte differentiation and epithelial integrity. As such, it is an essential nutrient for overall health and well-being [30]. Vitamin A plays an essential role in visual phototransduction through regeneration of rhodopsin and in maintaining epithelial integrity of the conjunctiva and cornea [3, 8, 26, 30]. Vitamin A deficiency can lead to night blindness, xerophthalmia, Bitot's spots, keratomalacia, and corneal ulceration and perforation [3, 8, 30] (Table 2).

Vitamin A supplementation continues to serve as a cornerstone of pediatric ophthalmic public health initiatives in developing countries, playing a vital role in reducing the burden of childhood blindness. Further underscoring the importance of vitamin A in both preventive and clinical ophthalmology, and adding to its systemic benefits, topical retinoids show therapeutic value in promoting ocular surface healing in keratinizing disorders [3, 26, 30].

B-Complex Vitamins: Essential Neuro-Ophthalmic Cofactors

Vitamin B-complex refers to a group of water-soluble vitamins that play a vital role in cellular metabolism and neurological function. In the context of ocular health, various B vitamins show protective and therapeutic potential against several eye disorders [3, 9, 11, 31].

Vitamin B1 (Thiamine): Thiamine is essential for energy metabolism and neural function. Deficiency has been associated with optic neuropathy, including Wernicke's encephalopathy-related vision changes. Emerging evidence also links chronic thiamine deficiency to an increased risk of developing glaucomatous optic neuropathy due to mitochondrial dysfunction in retinal ganglion cells [2, 9, 31].

Vitamin B2 (Riboflavin): Riboflavin is an essential cofactor in redox reactions and acts as an antioxidant. It plays a role in protecting the lens from oxidative stress. Riboflavin deficiency has been linked to photophobia, blurred vision, and cataract. B2 is also used in corneal collagen cross-linking therapy to enhance stromal strength in keratoconus [3, 7, 17, 32].

Vitamin B3 (Niacin): Niacin has been investigated for its neuroprotective role in glaucoma. Studies suggest that nicotinamide, a form of B3, helps maintain mitochondrial function in retinal ganglion cells and may prevent glaucomatous damage. However, very high doses are linked to macular edema and cystoid maculopathy [2, 5, 6, 16].

Vitamin B5 (Pantothenic Acid): Though less directly studied in ocular disease, pantothenic acid is involved in coenzyme A synthesis and cellular repair mechanisms. It has shown benefits in DED as part of ocular surface healing agents, particularly in combination with hyaluronic acid [3, 16, 26].

Vitamin B6 (Pyridoxine): Pyridoxine is necessary for neurotransmitter metabolism. Its deficiency has been associated with microvascular retinopathy, optic neuritis, and visual dysfunction in alcohol dependence and malnutrition. Excess supplementation may lead to peripheral neuropathy [3, 9, 11, 31].

Vitamin B9 (Folic Acid) and B12 (Cobalamin): These are important in homocysteine metabolism. Elevated homocysteine levels are implicated in retinal vascular occlusion, non-arteritic anterior ischemic optic neuropathy, and open-angle glaucoma [11, 33, 31].

Vitamin B12 (Cobalamin): It is crucial to note that assessing the status of B vitamins can be complex. Research in elderly subjects, for instance, shows a strong correlation between Vitamin C intake and its biochemical status, whereas the same reliability is not observed with all B vitamins. Specifically, one study found a poor correlation between riboflavin (B2) intake and biochemical riboflavin status in elderly individuals [34]. This highlights that dietary intake alone may not always be a dependable indicator of an individual's actual Vitamin B status, and underscores the importance of biochemical assessments [31, 35]. Emerging evidence indicates a potential association between high-dose vitamin B12 intake and an increased risk of glaucoma. Analysis of data from the National Health and Nutrition Examination Survey (NHANES) 2005–2008 revealed a significant positive correlation between vitamin B12 intake and prevalence of glaucoma, suggesting the need for further investigation into this possible relationship [27].

Vitamin C: Collagen Synthesis and Lens Protection

Vitamin C is a potent antioxidant present in high concentrations in the aqueous humor and tear film. It delays cataract formation by preventing oxidative damage to lens proteins. Vitamin C enhances corneal wound healing and supports trabecular meshwork health, indirectly reducing intraocular pressure [7, 16, 17, 26, 36].

Cataract development has been linked to systemic diseases and risk factors like cardiovascular disease, diabetes, and hypertension. Antioxidant vitamins may protect the lens and retina, either directly or by promoting overall bodily health. The lens is vulnerable to oxidative damage, with free radicals affecting proteins and lipids. While animal studies suggest a protective role for vitamin C against cataracts, human epidemiological studies provide inconsistent support for nutrition's role in cataract development [16]. Vitamin C contributes biologically to collagen synthesis and epithelial repair. However, direct clinical evidence that vitamin C supplementation improves healing after refractive surgery or reduces recurrent corneal erosion is limited. Further controlled studies are required before condition-specific supplementation can be recommended [37–39].

Vitamin D3: The Multi-System Protector with Expanding Ophthalmic Roles

Vitamin D3 (cholecalciferol), once primarily associated with bone health, is now recognized for its role in a wide range of ocular conditions owing to its anti-inflammatory, antioxidant, anti-angiogenic, and neuroprotective properties. One notable association is that with DED, a common condition characterized by ocular dryness, discomfort, redness, and irritation. Studies show that patients with DED exhibit significantly lower vitamin D3 levels, where low serum 25-hydroxyvitamin D levels correlate with tear film instability, compared to individuals without the condition. The anti-inflammatory effects of activated vitamin D are mediated through inhibition of T-helper and cytotoxic T-cell activation, as well as suppression of inflammatory mediator production. Small clinical studies suggest that correction of vitamin D deficiency may improve selected signs or symptoms of DED in vitamin-D-deficient patients. However, the heterogeneity of available studies does not support routine vitamin D supplementation for all patients with DED. [24, 40–43] (Table 3).

Vitamin D3 deficiency in keratoconus has been linked to corneal collagen breakdown and increased matrix metalloproteinase activity, both of which contribute to disease progression. A small prospective study of adolescents with keratoconus and vitamin D insufficiency suggested that supplementation might be associated with greater corneal stability. However, the absence of a randomized untreated control group and the limited sample size prevent conclusions about causality or routine clinical use [25].

In glaucoma, vitamin D3 deficiency has been associated with retinal ganglion cell apoptosis and trabecular meshwork fibrosis, both contributors to disease pathogenesis. Experimental models suggest that vitamin D may influence intraocular pressure and aqueous outflow. Clinical studies have reported heterogeneous associations between vitamin D status and glaucoma, and there is insufficient evidence to recommend vitamin D supplementation as a glaucoma treatment. While clinical

studies have identified an association between vitamin D3 status and primary open-angle glaucoma, findings remain heterogeneous, indicating the need for further high-quality research to establish definitive clinical correlations [24].

In DR and diabetic macular edema, vitamin D3 may exert a protective effect by inhibiting the ROS–TXNIP–NLRP3 (reactive oxygen species/TRX-interacting protein/NOD-like receptor family pyrin domain-containing 3) inflammatory pathway, thereby reducing microvascular damage. Vitamin D3 deficiency has been associated with elevated vascular endothelial growth factor (VEGF) levels, disruption of the blood–retinal barrier, and the development or progression of macular edema. In addition to its role in bone metabolism, vitamin D functions as a potent antioxidant that reduces free radical formation, may exert significant anti-inflammatory effects, and modulates both autophagy and apoptosis. Experimental evidence suggests that vitamin D may modulate inflammatory and oxidative pathways involved in DR. A clinical trial in vitamin-D-deficient patients with DME also investigated correction of deficiency as an adjunct to bevacizumab, with possible delayed anatomical and visual benefits. Nevertheless, current evidence is insufficient to conclude that vitamin D supplementation independently prevents or slows DR progression [44–46].

AMD is a leading cause of vision impairment, particularly in industrialized nations. Experimental and observational studies have proposed that vitamin D may influence inflammatory and oxidative pathways involving the RPE. However, clinical evidence that vitamin D supplementation prevents AMD or slows its progression remains insufficient. The landmark Age-Related Eye Disease Study (AREDS) demonstrated that a defined combination of antioxidant vitamins and zinc reduced progression to advanced AMD in appropriately selected high-risk patients [47]. The formulation does not prevent AMD onset and is not indicated for individuals without qualifying AMD. The review also notes that vitamin supplementation may modestly delay cataract progression, although it does not prevent it. Importantly, nutritional supplementation should be viewed as an adjunct to, rather than a replacement for, a healthy lifestyle and balanced diet [17].

The AREDS and AREDS2 trials have been instrumental in establishing the role of nutritional supplementation in lowering the risk of progression to advanced AMD. The original AREDS formulation, which included defined doses of vitamin C, vitamin E, zinc, copper, and beta-carotene, was shown to significantly reduce the likelihood of developing advanced AMD. These findings provided an evidence base for targeted micronutrient therapy in high-risk individuals [36].

In AREDS2, lutein and zeaxanthin were evaluated as alternatives to beta-carotene and are included in the current AREDS2 formulation. Omega-3 supplementation provided no additional benefit for reducing progression to advanced AMD, and vitamin D was not included in the AREDS2 formulation [48]. A balanced diet rich in these essential nutrients is fundamental for maintaining ocular health and reducing the risk of age-related conditions [14, 49–51].

Vitamin D3 deficiency has been associated with an increased risk of posterior subcapsular cataract formation, particularly among individuals with diabetes. The proposed pathogenic mechanism involves disruption of calcium homeostasis within the lens epithelium, which may impair lens transparency and promote opacification. Lower serum vitamin D levels have been associated with nuclear and posterior subcapsular cataracts in some observational studies. However, these findings do not establish causality or demonstrate that vitamin D supplementation prevents cataract formation [52].

In experimental retinoblastoma models, vitamin D analogues, including calcitriol and 1,25-dihydroxy-16-ene-23-yne vitamin D3, attenuated tumor growth predominantly through the induction of apoptosis, with little or no effect on tumor cell proliferation. The study showed increased apoptotic cell death and upregulation of p53 and p21 in xenograft retinoblastoma tumors treated with vitamin D analogues. However, these findings are limited to preclinical models, and no clinical evidence regarding therapeutic efficacy, reduction of chemotherapy-related toxicity, or combination treatment with agents like cisplatin was evaluated in that study. Therefore, further clinical investigations are required to determine the potential therapeutic role of vitamin D analogues in retinoblastoma management [53].

Vitamin E and Photoreceptor Protection

As a lipid-soluble antioxidant, vitamin E may protect photoreceptors and the RPE from lipid peroxidation. However, randomized evidence has not demonstrated a substantial effect of vitamin E supplementation alone on incident AMD [14]. Its established clinical role in AMD is as one component of the combined AREDS/AREDS2 formulations. Vitamin E, comprising both tocopherols and tocotrienols, is a potent antioxidant that plays a crucial role in protecting cells from oxidative damage by neutralizing free radicals. While tocopherols, the primary forms of vitamin E, are well-known for their antioxidant properties and bioavailability, tocotrienols have garnered increasing attention for their unique biological activities, including neuroprotective, anti-cancer, and cholesterol-lowering effects. Research indicates that vitamin E, including both tocopherols and tocotrienols, offers significant benefits in protecting against radiation damage, regulating cholesterol, and supporting cardiovascular and neurological health. Given the limitations of existing glaucoma treatments, the potential role of vitamin E merits further investigation [28]. Sources of Vitamin E include almonds, sunflower oil, and spinach [7, 16, 17, 36].

Emerging Nutritional Molecules

AREDS and AREDS2 formulations reduce progression to advanced AMD in appropriately selected patients with intermediate AMD or advanced AMD in one eye; they do not prevent AMD onset. In AREDS2, omega-3 supplementation provided no additional benefit, while lutein and zeaxanthin became preferred substitutes for beta-carotene, particularly for current and former smokers. Further research is required to determine optimal dosing strategies, clarify the specific role of meso-zeaxanthin, and explore potential benefits in other ocular conditions such as retinopathy of prematurity (ROP), DR, and cataracts [50].

Table 3. Investigated associations and potential effects of vitamin D in ocular conditions

Condition	Effect/Benefit
Dry eye disease	Correction of documented deficiency may improve selected DED outcomes, but evidence remains limited and heterogeneous [24].
Keratoconus	A small uncontrolled study suggested possible stabilisation in vitamin-D-insufficient adolescents; randomized controlled evidence is lacking [25].
Glaucoma	Experimental effects on intraocular pressure and aqueous outflow have been reported; clinical therapeutic benefit has not been established [24].
Diabetic retinopathy with macular edema	Experimental anti-inflammatory and anti-angiogenic mechanisms have been reported. Limited clinical evidence has evaluated correction of vitamin D deficiency as an adjunct to anti-VEGF treatment [44].
Age-related macular degeneration	Mechanistic and observational associations have been reported; supplementation has not been shown to prevent or treat AMD [24].
Cataracts	Lower serum vitamin D levels have been associated with some cataract subtypes; a preventive effect of supplementation has not been established [52].

Macular pigments—lutein, zeaxanthin, and meso-zeaxanthin—are concentrated in the retina and play a protective role against AMD. Supplementation with lutein and zeaxanthin can increase macular pigment optical density and may modestly improve selected measures of visual function. However, evidence that these carotenoids independently slow AMD progression remains limited [50, 51]. Further studies are needed to determine optimal dosing and explore their role in ROP, DR, and cataracts.

These pigments are used in pre- and postoperative care for phototoxicity protection. Lutein and zeaxanthin effectively neutralize ROS but become oxidized in the process and require regeneration by other antioxidants like vitamin E and vitamin C. Without a proper antioxidant cycle, these compounds can act as prooxidants, potentially harming the retina. Understanding the specific oxidative mechanisms in each eye compartment is vital for targeted protection. Dietary antioxidants, appropriate sunglasses, and advanced ocular imaging may help prevent retinal damage, and future therapies, including gene therapy, may benefit from precise monitoring of antioxidant effects [49].

Omega-3 fatty acids are shown to improve meibomian gland function and reduce evaporative dry eye by enhancing tear film stability and improving the quality of meibomian gland secretions. High-dose docosahexaenoic acid (DHA) supplementation has shown promising therapeutic potential in managing DED associated with meibomian gland dysfunction. However, clinical evidence remains mixed, as some studies, such as the Dry Eye Assessment and Management (DREAM) trial, report no significant benefit. These findings emphasize the need for further well-designed, large-scale studies to better define the role of these fatty acids in the management of DED [54]. Other studies report improvements in meibomian gland dysfunction scores and tear break-up time with high-dose omega-3 supplementation, particularly formulations rich in DHA, but their small sample sizes and lack of dietary control limit generalizability of the findings. To establish definitive conclusions regarding efficacy and safety, as well as to determine optimal dosage and duration of supplementation, longer-term, multicenter randomized controlled trials are warranted [55].

Omega-3 fatty acids are shown to inhibit pro-inflammatory cytokines in DR, thereby reducing retinal inflammation and oxidative stress. Preclinical evidence suggests that omega-3 fatty acids may modulate inflammatory and oxidative pathways involved in DR. However, clinical efficacy in preventing or slowing DR progression has not been established [56]. While preclinical and observational studies support their potential benefits, dedicated clinical trials specifically targeting patients with DR are needed to support efficacy, establish optimal dosing, and assess long-term safety [56]. Some small clinical trials have reported improvement in selected DED outcomes with omega-3 supplementation, whereas the larger DREAM trial found no significant benefit over placebo. Therefore, the clinical efficacy of omega-3 supplementation for DED remains uncertain [36, 54, 55].

Zinc has several potential roles in AMD through its effects on antioxidant defence, autophagy, and retinal cellular homeostasis [57]. Separately, dysregulated iron homeostasis and retinal iron accumulation have been implicated in oxidative retinal injury and AMD pathogenesis [58]. However, although zinc protoporphyrin is a marker of iron-restricted erythropoiesis, a causal role for elevated zinc protoporphyrin in AMD has not been established. Clinically, zinc forms part of the combined AREDS/AREDS2 formulations used in appropriately selected patients with AMD [47].

Zinc also plays a crucial role in regulating both innate and adaptive immunity, influencing T cells, B cells, natural killer cells, and macrophages [59, 60]. Age-related zinc deficiency contributes to “immunosenescence” and “inflamm-aging”, characterized

by increased pro-inflammatory cytokine production. Zinc supplementation is shown to reduce systemic and retinal inflammation, partly by enhancing T-regulatory cell function and shifting immune balance away from T-helper (Th)1 and Th17-mediated autoimmunity. In models of uveitis and autoimmune retinal disease, zinc promotes resolution by inducing tolerogenic dendritic cells and upregulating FoxP3 in T-regulatory cells. In the aging retina, zinc influences the function of resident immune cells like microglia and macrophages, which are essential players in oxidative damage and AMD progression. Zinc also affects macrophage zinc transporter expression, which impacts ROS generation and phagocytic capacity. Further, zinc may modulate retinal inflammation indirectly through effects on the gut microbiome. Together, these findings support potential therapeutic roles of zinc in preventing or mitigating retinal inflammatory disorders, particularly in the elderly [61].

Nutritional history and targeted laboratory assessment should be considered when deficiency is clinically suspected, particularly in patients with unexplained bilateral visual loss, possible nutritional optic neuropathy, restricted diets, alcohol misuse, malabsorption or other risk factors for hypovitaminosis. Evidence is currently insufficient to recommend routine nutritional screening for all patients with DED, keratoconus or glaucoma [3, 16, 26, 36, 61].

This narrative review has several strengths. It summarizes evidence on the role of vitamins and micronutrients in ocular health across a range of ocular conditions, including ocular surface diseases, retinal disorders, optic neuropathies, and age-related degenerative conditions. Additionally, the review integrates findings from high-quality systematic reviews, randomized controlled trials, and large observational studies, thereby enhancing the reliability of the summarized evidence. However, several limitations must be acknowledged. As a narrative review, the methodology is inherently subject to selection bias, as the literature search was not fully systematic or meta-analytically structured. Heterogeneity in study designs, populations, supplementation dosages, and outcome measures across included studies limits direct comparability and precludes quantitative synthesis. Despite growing evidence supporting the role of vitamins in ocular health, several important research gaps remain. Future studies should focus on large-scale, multicenter randomized controlled trials to establish definitive causal relationships between specific vitamin deficiencies and ocular disease progression. Standardization of biochemical markers for vitamin status and uniform diagnostic criteria for ocular outcomes are essential to improve comparability across studies. Clinical implications should be interpreted according to the underlying level of evidence. Correction of a confirmed vitamin deficiency is distinct from supplementation in vitamin-replete individuals. Except for established indications—such as treatment of vitamin A deficiency and use of the AREDS2 formulation in appropriately selected patients with AMD—current evidence does not support routine vitamin testing or supplementation for all patients with DED, glaucoma, DR, keratoconus or cataract. Observational associations and experimental mechanisms should not be interpreted as evidence of therapeutic efficacy.

CONCLUSIONS

This review emphasizes the important role of vitamins and micronutrients in preserving ocular health. While vitamin A and B-complex deficiencies manifest with well-established ocular symptoms, emerging evidence suggests that vitamins D3, C, and E may play a role in DED, glaucoma, DR, keratoconus, and AMD. Although consensus exists on the benefits of antioxidant-rich diets and select supplementation, especially in conditions like AMD, evidence remains variable for other disorders, pointing to ongoing debate and the need for individualized care. Key gaps include a lack of large-scale, randomized trials assessing long-term outcomes of supplementation, inconsistent biomarker correlations, and limited data on emerging nutrients. Future research should focus on defining optimal dosing, understanding nutrient interactions, and determining which high-risk populations may benefit from targeted nutritional assessment and intervention.

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