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Beyond Vision: The Role of Clinical Nutrition in Preventive Ophthalmology and Long-Term Eye Health

A Narrative Review

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Abstract

The eye is a metabolically active organ whose normal structure and function depend on an adequate supply of energy, essential fatty acids, vitamins, minerals and bioactive compounds. Nutrition is therefore biologically relevant to ocular health across the life course. However, the strength of clinical evidence varies substantially by nutrient and eye condition. This narrative review examines the relationship between dietary patterns, selected micronutrients and common ophthalmic disorders, with emphasis on age-related macular degeneration (AMD), diabetic retinopathy, dry eye disease, cataract and glaucoma. It considers the roles of vitamin A, vitamins C and E, zinc, copper, omega-3 long-chain polyunsaturated fatty acids, lutein and zeaxanthin, while distinguishing mechanistic plausibility and observational associations from demonstrated clinical benefit. The strongest intervention evidence is condition-specific: the Age-Related Eye Disease Study (AREDS) and AREDS2 formulations reduce progression to advanced AMD in appropriately selected people with intermediate AMD or advanced AMD in one eye, but do not prevent AMD onset in the general population. Evidence for routine supplementation to prevent cataract, glaucoma or diabetic retinopathy is insufficient, and trials of omega-3 supplementation for dry eye have produced mixed results. A dietary pattern rich in vegetables, fruits, legumes, whole grains, nuts and appropriate sources of unsaturated fats is a reasonable foundation for general and cardiometabolic health, with potential relevance to ocular health. Nutrition care should complement – not replace – eye examinations, risk-factor management and ophthalmic treatment. Collaborative practice between ophthalmologists and qualified nutrition professionals can support individualized dietary assessment, correction of deficiencies, safe supplement decisions and realistic prevention messaging. This review highlights evidence gaps and proposes an integrated, non-promotional framework for nutrition-informed preventive eye care.

Keywords*— *Ocular health; clinical nutrition; preventive ophthalmology; age-related macular degeneration; diabetic retinopathy; dry eye disease; lutein; zeaxanthin; omega-3 fatty acids; dietary patterns.

I. INTRODUCTION

Vision contributes to mobility, learning, independence and quality of life. Ocular tissues are exposed to light and oxygen and contain highly specialized structures with substantial metabolic requirements. The retina has a high rate of oxygen consumption; photoreceptor outer segments undergo continuous renewal; and the crystalline lens must maintain transparency over decades. These characteristics create biological reasons to investigate the contribution of nutrition to eye health.

Nutrition may influence ocular health through several pathways: provision of substrates for tissue maintenance; support of phototransduction and epithelial integrity; antioxidant and redox functions; modulation of inflammatory signalling; vascular and metabolic health; and effects on systemic disorders that cause ocular complications. The relationship is not uniform. A nutrient may be essential for normal vision when deficient but offer no additional benefit when intake is already adequate. A biomarker or plausible mechanism does not, by itself, establish that supplementation prevents disease.

The public frequently encounters claims that particular foods or supplements can "improve eyesight," reverse refractive error or prevent all eye diseases. Such claims overstate the evidence. Myopia, for example, is primarily related to ocular growth and environmental factors, while cataract, glaucoma, AMD and diabetic retinopathy have distinct pathophysiology. No single diet or supplement prevents all these conditions. The clinical task is to identify where nutrition has a demonstrated role, where associations remain uncertain, and where standard ophthalmic care is indispensable.

This narrative review synthesizes selected landmark trials, systematic reviews, professional guidance and mechanistic literature. It is intended for clinicians and health professionals working at the interface of nutrition and eye care. It does not report new patient data, and it should not be interpreted as a clinical practice guideline.

II. REVIEW APPROACH AND SCOPE

A narrative rather than systematic review format was selected to integrate evidence across multiple ocular conditions and nutritional domains. The literature base informing this paper includes landmark randomized controlled trials, major ophthalmic research programmes, peer-reviewed reviews and guidance from recognized eye-health and evidence-review organizations. Priority was given to human clinical outcomes and randomized evidence where available. Mechanistic findings and observational associations are described as supportive or hypothesis-generating rather than proof of causation.

The review focuses on nutrition-related aspects of adult ocular health, while acknowledging selected life-course considerations. It does not attempt a formal risk-of-bias assessment, meta-analysis or exhaustive search of every nutrient. Consequently, it should be updated before journal submission to meet the target journal's reporting and reference requirements. The term "preventive ophthalmology" is used here to describe risk reduction, early detection and coordinated care—not the substitution of dietary measures for diagnosis or treatment.

III. BIOLOGICAL BASIS: WHY NUTRITION MATTERS TO THE EYE

The retina is rich in polyunsaturated fatty acids and is exposed to light-driven oxidative processes. Photoreceptor membranes contain high concentrations of docosahexaenoic acid (DHA), an omega-3 fatty acid that contributes to membrane structure and function. The retinal pigment epithelium (RPE) supports photoreceptors, recycles visual-cycle components and participates in the removal of cellular waste. Oxidative stress, inflammation, mitochondrial dysfunction and impaired cellular clearance are implicated in several retinal disorders, including AMD and diabetic retinal disease.

The macula contains the xanthophyll carotenoids lutein and zeaxanthin, with meso-zeaxanthin also present. These pigments contribute to macular pigment optical density and absorb some short-wavelength light; they also have antioxidant properties in experimental systems. The lens, meanwhile, relies on tightly regulated proteins, glutathione and antioxidant systems to preserve transparency. Vitamin C is present in the aqueous humour at concentrations exceeding those in plasma, while vitamin E protects lipid membranes from oxidative damage. Zinc is involved in numerous enzymes and retinal functions, and vitamin A derivatives are essential to the visual cycle.

These mechanisms explain why severe deficiency of certain nutrients can impair vision. Vitamin A deficiency, for example, can cause impaired dark adaptation and xerophthalmia. Nevertheless, biological necessity should not be confused with a recommendation for high-dose supplementation in well-nourished people. The dose-response relationship may plateau, excessive intake can be harmful, and the effect of a nutrient depends on baseline status, disease stage, formulation, adherence and interactions with other nutrients. [18,19]

IV. KEY NUTRIENTS AND DIETARY SOURCES

4.1 Vitamin A and provitamin A carotenoids

Vitamin A is required for the formation of retinal, a component of rhodopsin and other visual pigments. It also supports epithelial differentiation and immune function. Preformed vitamin A is found in foods such as eggs, dairy products and liver; provitamin A carotenoids are present in orange, yellow and dark-green vegetables. Inadequate vitamin A intake can lead to night blindness and, in severe cases, xerophthalmia and corneal damage. This is a well-established deficiency disease, particularly relevant in populations experiencing food insecurity or malabsorption.

The public-health response to vitamin A deficiency is different from the use of high-dose supplements for nonspecific eye complaints. Excessive preformed vitamin A can be toxic and is of particular concern during pregnancy. Nutrition professionals should assess dietary intake, clinical context and possible malabsorption before recommending supplementation. Food-based adequacy and medically supervised treatment of deficiency are preferable to indiscriminate megadosing. [18,19]

4.2 Lutein, zeaxanthin and meso-zeaxanthin

Lutein and zeaxanthin are dietary xanthophylls found in leafy green vegetables, corn, peas, egg yolk and some other colourful foods. They are selectively concentrated in the macula. Their potential functions include filtering some blue light and quenching reactive oxygen species. Observational studies have associated higher dietary intake or circulating concentrations of these carotenoids with more favourable retinal outcomes, although confounding and reverse causation cannot be excluded. [13,18]

The clinical significance of these compounds is most clearly established within the AREDS2 supplement formulation for people with specified stages of AMD. This does not mean that lutein or zeaxanthin supplements are proven to prevent AMD in healthy individuals, nor that they correct refractive error or restore lost vision. A varied diet containing leafy greens and other vegetables is a sensible way to obtain these carotenoids, while supplement decisions in AMD should be based on ophthalmic staging and clinician advice.

4.3 Vitamins C and E

Vitamin C is a water-soluble antioxidant and cofactor in collagen synthesis. Vitamin E is a lipid-soluble antioxidant that helps protect cell membranes. Fruits and vegetables, including citrus fruits, berries, guava, peppers and broccoli, are sources of vitamin C. Nuts, seeds and vegetable oils provide vitamin E. Their antioxidant functions have prompted investigation in cataract and retinal disease.

The original AREDS trial demonstrated benefit from a specific combination of antioxidants and zinc in reducing progression to advanced AMD among participants at increased risk. It did not establish that taking isolated vitamins C or E prevents AMD in the general population. High-dose supplementation may interact with medications or increase adverse-event risk in some individuals. Dietary adequacy should remain the default approach unless a specific clinical indication exists.

4.4 Zinc and copper

Zinc is present in high concentrations in the retina and is involved in enzyme activity, protein synthesis and normal immune function. Dietary sources include meat, seafood, dairy, legumes, nuts and seeds. Phytate in some plant foods can reduce zinc bioavailability, which may be relevant in diets heavily reliant on unrefined cereals and legumes without appropriate preparation or dietary variety. Copper is required for normal physiological function and is included in the AREDS formulation to reduce the risk of copper deficiency associated with high-dose zinc.

The zinc dose used in AREDS-based formulations is pharmacological rather than a routine dietary requirement. Such formulations should not be self-prescribed for general "eye strengthening." The indication depends on AMD stage, and the potential for gastrointestinal effects, interactions and altered mineral status should be considered.

4.5 Omega-3 long-chain polyunsaturated fatty acids

DHA is a structural component of retinal photoreceptor membranes. EPA and DHA also participate in lipid mediator pathways involved in inflammatory resolution. Oily fish are important dietary sources; plant foods such as flaxseed, chia seeds and walnuts provide alpha-linolenic acid (ALA), which is converted to EPA and DHA inefficiently and variably. [18]

The biological rationale for omega-3 intake is strong, but clinical results differ by condition and outcome. In the AREDS2 trial, adding EPA and DHA did not significantly reduce progression to advanced AMD. In dry eye disease, randomized trials and systematic reviews have reported inconsistent effects. It is therefore appropriate to encourage nutritionally balanced sources of unsaturated fats as part of a healthy dietary pattern, but not to promise that fish-oil supplements will treat dry eye or prevent AMD.

4.6 Other micronutrients and bioactive compounds

Selenium, riboflavin, vitamin D, B vitamins, polyphenols and other plant compounds have been investigated in relation to ocular health. Some have plausible roles in antioxidant defence, cellular metabolism, vascular function or inflammation. However, evidence is heterogeneous and often observational, based on small studies, or derived from experimental models. There is currently no basis for recommending broad-spectrum high-dose "eye supplements" to prevent glaucoma, cataract or diabetic retinopathy in the general population. Deficiencies should be identified and treated for established nutritional and medical reasons, not on the assumption that more is necessarily better.

V. NUTRITION AND MAJOR EYE CONDITIONS

5.1 Age-related macular degeneration

AMD is a multifactorial disease affecting the macula and is a major cause of central visual impairment in older adults. Age, genetic susceptibility and smoking are important risk factors. Diet has been investigated as a modifiable influence, with studies examining dietary patterns, carotenoids, antioxidant nutrients and fish intake. Observational research has associated Mediterranean-style dietary patterns and higher intake of selected nutrients with lower risk or slower progression, but such associations do not prove that an individual food prevents AMD.

The AREDS trial established that a defined antioxidant-and-zinc formulation reduced the risk of progression to advanced AMD in people with intermediate AMD or advanced AMD in one eye. AREDS2 evaluated lutein and zeaxanthin, omega-3 fatty acids and modifications to the original formula. Adding omega-3 fatty acids did not produce an overall additional benefit. The AREDS2 formulation, which replaces beta-carotene with lutein and zeaxanthin, is preferred over beta-carotene-containing formulations, particularly because beta-carotene supplementation increased lung cancer risk in current and former smokers in the trial programme. Long-term follow-up further supported replacing beta-carotene with lutein and zeaxanthin. [1,2,3]

These results must be applied to the population studied. AREDS-type supplements are not intended to prevent AMD in people without disease or to treat early AMD indiscriminately. They do not restore vision already lost. Ophthalmologists should determine disease stage and eligibility; nutrition professionals can support dietary quality, smoking cessation counselling referral, weight and cardiometabolic risk management, and safe adherence to a prescribed formulation. [1,2,4]

5.2 Diabetic retinopathy and diabetes-related eye disease

Diabetic retinopathy is a microvascular complication of diabetes and can progress from non-proliferative changes to sight-threatening disease. Duration of diabetes, chronic hyperglycaemia, hypertension, dyslipidaemia and kidney disease are important risk factors. Nutrition influences several of these systemic determinants. Medical nutrition therapy that supports individualized glycaemic management, blood-pressure control, lipid management and healthy body composition is therefore relevant to reducing overall diabetes complication risk. [16,17]

However, it would be inaccurate to claim that a particular vitamin or supplement prevents or reverses diabetic retinopathy. Observational studies have explored associations between dietary patterns, antioxidants and retinal outcomes, but intervention evidence is not sufficient to replace established diabetes care. Glycaemic targets and pharmacotherapy should be individualized by the treating clinician. Regular retinal screening, timely ophthalmic referral and treatment of sight-threatening retinopathy remain essential.

A nutritionist can contribute by assessing meal patterns, carbohydrate quality and distribution, fibre intake, medication-food interactions, food access and adherence barriers. Dietary strategies should be culturally appropriate and sustainable. For example, a pattern emphasizing vegetables, pulses, whole grains in suitable portions, nuts and unsaturated fats may support cardiometabolic goals. It should not be

represented as a stand-alone treatment for retinal disease.

5.3 Dry eye disease

Dry eye disease is a multifactorial condition characterized by loss of tear-film homeostasis, with symptoms and signs involving tear-film instability, hyperosmolarity, inflammation and ocular-surface damage. Risk factors include age, hormonal changes, medications, contact lens use, environmental exposure, screen-related reduced blink frequency and systemic conditions. Nutritional status may influence ocular-surface function, but it is only one component of a complex disorder.

Omega-3 fatty acids have been studied because of their role in lipid mediators and inflammation. Nevertheless, trial findings are mixed. The DREAM randomized clinical trial did not find a significant advantage of high-dose fish-derived omega-3 supplementation over placebo for dry eye symptom and sign outcomes. Other studies have reported benefits in selected settings, but differences in formulation, dose, population and outcome measures limit generalization. Current evidence does not justify presenting omega-3 supplements as a reliably effective treatment for all patients with dry eye. [10]

Adequate hydration and a balanced diet are reasonable general-health measures, but they should not be described as substitutes for lubricating drops, lid hygiene, environmental modification or prescription treatment when indicated. Persistent symptoms, pain, photophobia or visual change warrant ophthalmic assessment. A nutrition professional should review dietary restrictions, overall fat quality and possible deficiencies without promising a cure.

5.4 Cataract

Cataract involves loss of transparency of the crystalline lens, commonly associated with ageing, but also influenced by diabetes, smoking, ultraviolet exposure, corticosteroid use and other factors. Oxidative damage is one proposed contributor to lens protein modification and aggregation. Nutrients with antioxidant functions have therefore been investigated in observational studies and supplementation trials.

Although some observational studies associate higher fruit and vegetable intake or certain nutrient patterns with lower cataract risk, randomized evidence has not shown that AREDS/AREDS2 supplementation prevents cataract or reduces the need for cataract surgery. Evidence does not support recommending high-dose antioxidant supplements as a cataract-prevention treatment. Smoking cessation, ultraviolet-protective eyewear, diabetes management and regular eye examinations are more defensible preventive messages. Surgery remains the established treatment when cataract causes functionally significant visual impairment.

5.5 Glaucoma

Glaucoma describes a group of optic neuropathies characterized by progressive retinal ganglion cell and optic nerve damage. Intraocular pressure is a major modifiable risk factor, although disease can occur at statistically normal pressure. Age, family history and other factors influence risk. Nutrients and dietary components have been investigated for possible effects on ocular blood flow, oxidative stress and neuronal resilience, but the evidence is not sufficient to recommend routine antioxidant supplementation as glaucoma prevention or treatment.

The National Center for Complementary and Integrative Health notes that current data do not support vitamin A, C and E supplementation for glaucoma. Dietary advice should therefore focus on overall health and comorbidity management rather than disease-specific supplement claims. Intraocular pressure assessment, optic nerve evaluation, visual-field testing and prescribed pressure-lowering treatment remain central to care. Patients should not discontinue eye drops or delay follow-up because they have started a supplement. [5]

VI. DIETARY PATTERNS AND PRACTICAL FOOD-BASED GUIDANCE

A whole-diet approach is more defensible than focusing on isolated "superfoods." Mediterranean-style dietary patterns, characterized by vegetables, fruits, legumes, whole grains, nuts, fish and unsaturated oils, have been associated with favourable cardiometabolic outcomes and have been investigated in relation to AMD. The observational nature of much of this evidence means that residual confounding is possible. Nonetheless, this pattern aligns with broader recommendations for cardiovascular and metabolic health, conditions that share risk factors with several ocular disorders.

Practical guidance can be adapted to local food cultures. In an Indian dietary context, dark-green leafy vegetables, carrots, pumpkin, sweet potato, seasonal fruits, citrus, guava, tomatoes, pulses, beans, nuts and seeds can contribute a range of micronutrients and carotenoids. Eggs, dairy, fish and other animal foods may provide additional nutrients depending on dietary preference. A vegetarian pattern can be nutritionally adequate when planned to include diverse protein sources, appropriate fats and micronutrient-rich foods. For people with diabetes, carbohydrate portions and meal composition should be individualized rather than applying a universal "eye diet."

Food preparation and accessibility matter. A recommendation that is unaffordable, culturally unfamiliar or difficult to sustain is unlikely to be effective. Nutrition counselling should consider food security, cooking practices, allergies, gastrointestinal tolerance, religious or ethical dietary patterns and comorbidities. The aim is dietary adequacy and long-term adherence, not restrictive eating or the purchase of branded products.

VII. LIFESTYLE AND PREVENTIVE EYE-CARE BEHAVIOURS

Nutrition is one part of a broader prevention strategy. Tobacco cessation is particularly important because smoking is a major modifiable risk factor for AMD and has broader vascular and ocular effects. People with diabetes should receive recommended retinal screening and maintain coordinated management of glucose, blood pressure and lipids. Adults should seek age- and risk-appropriate eye examinations, especially when they have diabetes, a family history of glaucoma, visual symptoms or known retinal disease.

Outdoor ultraviolet exposure is relevant to ocular surface and lens health; appropriate sunglasses and brimmed hats can reduce exposure. Screen use may contribute to digital eye strain and symptoms of dryness, particularly when prolonged viewing reduces blink frequency. Practical measures include regular visual breaks, conscious blinking, suitable screen distance and lighting, and assessment of persistent symptoms. These strategies may improve comfort but should not be promoted as proven methods to prevent retinal degeneration.

Adequate sleep, physical activity and management of obesity and cardiovascular risk factors support general health. Their direct effect on specific eye outcomes varies, but they are important components of integrated preventive care. Sudden visual loss, new flashes or floaters, a curtain-like shadow, severe eye pain or acute redness with visual change require urgent medical assessment rather than dietary intervention.

VIII. COLLABORATIVE MODEL: OPHTHALMOLOGIST AND NUTRITION PROFESSIONAL

An integrated eye-care model can improve the consistency and safety of nutrition-related advice. The ophthalmologist establishes the diagnosis, stages disease, identifies urgent pathology, orders relevant investigations and determines ophthalmic treatment. The registered dietitian or appropriately qualified nutrition professional assesses dietary intake, nutritional risk, food access, comorbidities, supplement use and readiness for behaviour change. Both professionals should communicate clearly about the limits of evidence and the intended role of nutrition.

A practical workflow may include: (1) ophthalmic assessment and diagnosis; (2) identification of relevant systemic conditions and nutritional risk; (3) review of dietary pattern, medications and supplements; (4) individualized food-based counselling; (5) clinician-led decisions about condition-specific supplementation; (6) follow-up that tracks adherence, tolerability and relevant clinical outcomes. For AMD, this includes confirming whether the patient meets criteria for an AREDS2-type formulation. For diabetes, it includes integrating medical nutrition therapy with glycaemic and cardiovascular risk management. For dry eye, it includes avoiding unsupported promises about omega-3 supplementation while supporting the ophthalmologist's treatment plan.

Communication should avoid implying that nutrition can reverse structural eye damage or replace prescribed medication, injections, laser treatment or surgery. Brand-associated educational programmes should disclose commercial interests, avoid product-led claims and ensure that supplement recommendations are independent, evidence-based and appropriately supervised.

IX. SAFETY, LIMITATIONS AND EVIDENCE GAPS

Nutritional interventions are not risk-free. High-dose vitamin A can cause toxicity and is unsafe in excess during pregnancy. Beta-carotene-containing high-dose supplements are inappropriate for current and former smokers in the context of AMD supplementation because of the lung-cancer signal observed in trials. High-dose zinc can cause gastrointestinal effects and interfere with copper status. Vitamin E and omega-3 supplements may be relevant to bleeding risk or medication interactions in some patients. Supplement quality, dose accuracy, contamination and interactions also require consideration.

The evidence base has limitations. Dietary intake is often measured by food-frequency questionnaires, which are subject to recall error. People with healthier diets may also exercise more, smoke less and have better access to healthcare, creating confounding. Nutrient status may differ from reported intake, and genetic factors can modify disease risk. Clinical trials may enroll selected populations and use formulations that cannot be extrapolated to ordinary foods or commercial supplements. Ocular outcomes also differ: symptom scores, visual acuity, retinal imaging changes and progression to late disease are not interchangeable.

Further research should prioritize adequately powered randomized trials with clinically meaningful outcomes, standardized diagnostic criteria and transparent reporting of baseline nutritional status. Studies should distinguish prevention of disease onset from slowing progression in established disease. Research in diverse populations, including South Asian populations with high burdens of diabetes and cardiometabolic disease, is particularly important. Implementation studies could evaluate whether collaborative nutrition-ophthalmology pathways improve dietary quality, screening attendance, adherence and patient understanding without increasing unnecessary supplement use.

X. IMPLICATIONS FOR PREVENTIVE EYE-CARE SERVICES AND HEALTH COMMUNICATION

Eye-care brands, clinics and public-health programmes can contribute by making reliable nutrition information accessible. Educational content should explain what is known, what remains uncertain and when professional assessment is needed. For example, it is appropriate to state that AREDS2 supplementation can slow progression in selected patients with intermediate AMD; it is not appropriate to claim that a generic eye vitamin prevents blindness or improves vision in everyone.

A responsible service model could include nutrition screening for people with diabetes or diagnosed AMD, referral pathways to qualified nutrition professionals, culturally adapted dietary resources and periodic review of supplement lists. Any commercial relationship involving supplements should be disclosed. Patient-facing material should use absolute risk and eligibility information where possible, distinguish food from supplements and avoid fear-based marketing.

The role of a nutrition professional is not to diagnose eye disease or prescribe ophthalmic treatment. It is to translate nutrition science into safe, practical care and to collaborate with ophthalmologists so that dietary advice is aligned with the person's diagnosis, medication, nutritional status and preferences.

XI. CONCLUSION

Nutrition is biologically important to ocular function, but its clinical contribution is condition-specific and should be communicated with precision. Adequate vitamin A is essential for normal vision, and selected nutrients have plausible antioxidant, structural and inflammatory roles in ocular tissues. The strongest supplement evidence concerns the AREDS/AREDS2 formulation for slowing progression to advanced AMD in people who meet defined ophthalmic criteria. Evidence does not support routine high-dose supplementation to prevent cataract, glaucoma or diabetic retinopathy, and omega-3 supplementation has not shown consistent benefit for dry eye disease. [15,20]

A varied, nutrient-dense dietary pattern supports general and cardiometabolic health and may contribute to long-term eye health, but it cannot replace screening, diagnosis or evidence-based ophthalmic treatment. The most credible model is collaborative: ophthalmologists provide disease-

specific assessment and treatment, while nutrition professionals support dietary adequacy, risk-factor management and safe, individualized counselling. Preventive eye care should be grounded in evidence, proportionate claims and continuity of care.

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CONFLICTS OF INTEREST

The author is associated with NutriFork by Shivani K. Mehra. This review is intended as an educational academic synthesis and does not endorse a commercial supplement or eye-care product. Any additional financial or institutional relationships should be declared to the target journal before submission.

AUTHOR CONTRIBUTION

Shivani Kalhan Mehra conceptualized the review, synthesized the nutrition literature and drafted the manuscript. Ophthalmology-specialist review and verification should be obtained before submission to a clinical ophthalmology journal.

ETHICS APPROVAL

Not applicable; this is a narrative review and contains no human participant data.

DATA AVAILABILITY

Not applicable.

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