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A REVIEW OF FOOD PATTERNS AND EYE HEALTH

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# HOW DIET AFFECTS MYOPIA IN CHILDREN AND ADOLESCENTS: A REVIEW OF FOOD PATTERNS AND EYE HEALTH

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## ABSTRACT

**Background:** Myopia is an increasingly important pediatric public health problem, with prevalence rising rapidly among children and adolescents worldwide. Although genetic predisposition, near work, and reduced outdoor exposure are recognized contributors, diet has emerged as a potentially modifiable factor that may influence ocular growth and refractive development.

**Methods:** A narrative review was conducted using recent peer-reviewed literature on myopia in children and adolescents, focusing on dietary patterns, nutrient-specific associations, and biological mechanisms linking nutrition with axial elongation and refractive error.

**Results:** Available evidence suggests that higher intake of omega-3 polyunsaturated fatty acids may be associated with shorter axial length and lower myopia risk, whereas higher saturated fat intake may be linked to less favorable refractive outcomes. Pro-inflammatory dietary patterns have also been associated with higher myopia prevalence in some adolescent groups. However, the evidence remains inconsistent. Recent meta-analytic data did not confirm significant overall associations between total macronutrient intake and myopia, and most existing studies are observational. Findings regarding vitamins and carotenoids are similarly mixed, with no clear consensus for most micronutrients.

**Conclusions:** Diet may contribute to myopia development and progression as part of a broader lifestyle and environmental framework, but current evidence does not support definitive dietary recommendations for myopia control. Nutritional quality should be considered a potentially relevant component of pediatric eye health, alongside established preventive and clinical myopia management strategies. Further longitudinal and interventional studies are needed to clarify causality and clinical relevance.

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## KEYWORDS

Myopia, Children, Adolescents, Diet, Nutrition, Ocular Health

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## Introduction

Myopia, or nearsightedness, is one of the most rapidly increasing visual disorders in children and adolescents and is now recognized as a major public health issue rather than a simple refractive inconvenience [12; 11]. The condition results from a mismatch between the optical power of the eye and its axial length, causing distant images to focus in front of the retina and appear blurred [11; 7]. Its importance extends beyond refractive error itself, because progressive myopia is associated with an increased lifetime risk of retinal detachment, glaucoma, cataract, and myopic maculopathy [11; 12].

The rise of pediatric myopia has been especially pronounced in urbanized regions, particularly in East and Southeast Asia, although similar upward trends have also been reported globally [5; 11]. Because this increase has occurred too rapidly to be explained by genetics alone, contemporary myopia research has focused increasingly on modifiable environmental and behavioral factors operating during childhood, when the eye is still growing and therefore more susceptible to cumulative influences [5; 12]. Reduced outdoor time, prolonged near work, intensive educational demands, and increased screen exposure are among the best-established contributors to myopia onset and progression, while sleep patterns and other lifestyle factors remain less consistently supported [5; 7; 4]. Current management therefore relies on a combination of behavioral prevention, optical strategies, and pharmacological interventions such as low-dose atropine [11; 4].

Within this broader framework, diet has emerged as an increasingly relevant but still underexplored factor in pediatric myopia research [12; 2]. Historically, nutritional influences received much less attention than light exposure or near work because myopia was primarily interpreted through a visual-behavioral lens [2; 5]. However, this perspective has become less satisfactory as evidence has accumulated that ocular growth

is also influenced by systemic physiology, including metabolism, inflammation, oxidative stress, vascular function, and tissue remodeling, all of which may be affected by dietary quality [12; 20]. The retina, choroid, and sclera are metabolically active tissues that depend on adequate nutrient supply and are sensitive to inflammatory and oxidative processes, making a diet–myopia relationship biologically plausible [12; 6]. Omega-3 fatty acids, carotenoids, vitamins, and other nutritional factors are known to support ocular physiology, whereas diets characterized by higher saturated fat intake or stronger inflammatory potential may plausibly contribute to conditions favoring axial elongation [21; 20].

Recent empirical findings have made this issue increasingly difficult to ignore [21; 20]. In the Hong Kong Children Eye Study, higher dietary intake of omega-3 polyunsaturated fatty acids was associated with shorter axial length and less myopic refraction, whereas higher saturated fat intake was associated with greater myopia risk and longer axial length in children aged 6 to 8 years [21]. These findings are notable because they were based on a large pediatric sample and adjusted for important confounders, including outdoor time, near work, body mass index, and parental myopia [21]. At the same time, the broader literature remains inconsistent. A 2024 systematic review and meta-analysis found no significant pooled associations between total carbohydrate, protein, or fat intake and myopia risk in individuals younger than 18 years, suggesting that broad macronutrient categories may be too crude to capture relevant nutritional effects [10]. By contrast, a 2025 meta-analysis reported that higher carbohydrate, cholesterol, and sodium intake were positively associated with myopia in adolescents, whereas protein intake appeared protective, although the evidence was limited and largely observational [19]. Similarly, an NHANES-based study found that higher serum cis- $\beta$ -carotene was associated with increased odds of myopia and high myopia, whereas vitamins A, C, D, and E, lutein, and zeaxanthin were not significantly associated after adjustment [16]. Research on dietary inflammatory load has also suggested that more pro-inflammatory diets may be associated with higher myopia prevalence in some adolescent groups [20].

Taken together, these findings suggest that the relationship between nutrition and myopia may depend less on total macronutrient intake and more on food quality, nutrient subtype, inflammatory profile, and broader lifestyle context [10; 20]. Diet is unlikely to act in isolation, since children with lower-quality diets may also spend less time outdoors, engage in lower physical activity, experience different sleep routines, and live in more academically pressurized or urbanized environments [5; 12]. For that reason, dietary influences should be interpreted as part of a broader behavioral and environmental ecology rather than as an isolated exposure [5; 20]. At the same time, dietary considerations must be placed in the context of existing evidence-based myopia management, which currently relies on interventions with stronger clinical support than nutrition alone [11; 4]. Diet should therefore be viewed not as a replacement for established treatment strategies, but as a potentially complementary and modifiable factor that may strengthen broader prevention efforts if future evidence becomes more robust [12; 7].

The aim of the present narrative review is to examine how diet may affect myopia in children and adolescents by synthesizing current evidence on dietary patterns, nutrient-specific associations, and biological mechanisms relevant to ocular growth [21; 20]. It also seeks to evaluate the strength of the available evidence, identify major contradictions and limitations, and consider how nutrition may fit within a multifactorial model of pediatric eye health and myopia prevention [10; 4].

### **Methodology**

This article was conducted as a narrative review designed to synthesize and critically interpret the available literature on how diet may influence myopia in children and adolescents, rather than as a systematic review or meta-analysis [12; 11]. A narrative approach was considered appropriate because the evidence base is heterogeneous and includes observational studies, cohort analyses, cross-sectional surveys, review articles, and emerging mechanistic literature rather than a large body of directly comparable intervention trials [12; 10].

The review was guided by the central question of whether dietary patterns, specific nutrients, and nutrition-related biological processes may influence myopia onset, prevalence, axial elongation, or refractive progression in children and adolescents [21; 20]. Priority was given to peer-reviewed literature focusing on pediatric populations and on exposures directly or indirectly related to diet, including dietary patterns, macronutrient composition, fatty acid intake, dietary inflammatory potential, and micronutrient biomarkers [16; 19]. Broader ocular nutrition studies were also considered when they provided mechanistic insight relevant to refractive development, such as oxidative stress, choroidal circulation, retinal metabolism, or scleral remodeling [6; 12].

The evidence was organized into five main thematic areas: the epidemiological context of pediatric myopia, biological mechanisms linking diet and ocular growth, nutrient-specific associations, dietary patterns, and the role of nutrition within the broader framework of myopia prevention and management [21; 20; 12]. Greater interpretive weight was given to studies using pediatric populations, clearly defined refractive outcomes, and statistical adjustment for major confounders such as age, sex, body mass index, outdoor time, near work, parental myopia, and educational level [21; 20].

As a narrative review, this article does not claim the exhaustive reproducibility of a formal systematic review and should be interpreted within that methodological boundary [12; 11]. The synthesis adopts a cautious perspective because much of the available evidence remains observational, heterogeneous in dietary assessment and outcome definitions, and difficult to separate from overlapping behavioral and socioeconomic influences [10; 19; 20].

### **Biological Mechanisms Linking Diet and Myopia**

Understanding how diet may influence myopia requires moving beyond simple association and considering the biological systems that regulate ocular growth during childhood and adolescence [12; 11]. Myopia develops primarily through excessive axial elongation, a process involving coordinated signaling among the retina, retinal pigment epithelium, choroid, and sclera [8]. From a nutritional perspective, the central question is whether diet can alter the metabolic, inflammatory, and vascular conditions that shape this growth process [12; 9]. Although no single mechanism has been conclusively confirmed in humans, several biologically plausible pathways have emerged from recent reviews and observational studies [12; 10].

#### **Inflammation as a Possible Mediator**

One of the most widely discussed mechanisms linking diet and myopia is inflammation [20; 18]. Recent reviews suggest that myopia is increasingly understood not only as a refractive disorder but also as a condition involving inflammatory and immune-related processes, particularly when progression is associated with tissue remodeling and vascular dysfunction [21; 18]. Inflammatory signaling may contribute to scleral extracellular matrix degradation through molecules such as matrix metalloproteinase-2 and pro-inflammatory cytokines, thereby facilitating scleral remodeling and axial elongation [21; 18].

This pathway is relevant to diet because dietary patterns differ in inflammatory potential [20; 12]. In an NHANES-based study, higher Dietary Inflammatory Index scores were associated with increased myopia prevalence in older adolescents and in certain educational subgroups [20]. Although this evidence is cross-sectional and cannot establish causality, it supports the hypothesis that pro-inflammatory diets may create a biological environment more favorable to myopic change [20; 21]. At present, inflammation is best interpreted as a plausible mediating pathway rather than a confirmed mechanism [12; 10].

#### **Choroidal Blood Flow, Oxygenation, and Scleral Hypoxia**

Another important concept is the role of the choroid and scleral hypoxia [17; 8]. The choroid is a highly vascular structure responsible for oxygen and nutrient delivery to the outer retina and sclera, and growing evidence suggests that changes in choroidal thickness and blood flow may contribute to myopia onset and progression [8; 22]. A 2025 study in myopic children found that choriocapillary flow deficits increased with greater axial length and more myopic refractive status, supporting the relevance of altered choroidal blood flow in pediatric myopia [22].

This vascular perspective is relevant to diet because nutrition can influence perfusion, endothelial function, lipid composition, and inflammatory tone [21; 6]. The Hong Kong Children Eye Study proposed that omega-3 polyunsaturated fatty acids may protect against myopia partly by improving choroidal circulation, thereby reducing scleral hypoxia [21]. This aligns with broader mechanistic reviews suggesting that reduced choroidal blood flow may promote scleral ischemia and hypoxia, which in turn may trigger structural changes and ocular elongation [8; 17]. Hypoxia-related signaling may also alter scleral fibroblast differentiation and collagen production, thereby weakening the biomechanical resistance of the sclera and favoring axial elongation [17; 14]. From a nutritional standpoint, this pathway provides a plausible explanation for why diet could influence myopia risk indirectly through vascular and metabolic effects rather than through direct optical mechanisms [21; 12].

#### **Glucose Metabolism and the High-Glycemic Diet Hypothesis**

A further mechanism discussed in the literature is the relationship between high-glycemic-load diets, glucose metabolism, and myopia [2; 15]. This hypothesis proposes that diets rich in rapidly absorbed carbohydrates may promote hyperinsulinemia and insulin resistance, which in turn could alter growth-related

signaling in the sclera and choroid [2]. Specifically, increased insulin-related signaling has been proposed as a driver of scleral tissue change and ocular elongation during childhood growth [15; 2].

Although this framework remains partly hypothesis-driven, it remains one of the more coherent nutritional models of myopia development [2; 12]. Its plausibility is supported indirectly by recent findings. In the Hong Kong Children Eye Study, higher saturated fat intake was associated with less favorable refractive outcomes, and saturated fat was discussed in relation to insulin resistance and excessive axial elongation [21]. In addition, a 2025 Mendelian randomization study reported that higher HbA1c was associated with increased myopia risk, whereas higher adiponectin appeared protective [13]. These findings do not prove that sugar intake directly causes myopia, but they do support continued investigation of glucose–insulin biology as part of the diet–myopia relationship [13; 19].

### **Specific Nutrients and Myopia**

Research on nutrition and myopia has increasingly shifted from broad questions of diet quality toward specific nutrients and nutrient classes that may influence ocular growth through inflammatory, vascular, or metabolic pathways [12; 19; 10; 20].

#### **Omega-3 Polyunsaturated Fatty Acids**

Among the nutrients studied so far, omega-3 polyunsaturated fatty acids (PUFAs) have some of the strongest support in relation to pediatric myopia [21; 12]. They are biologically relevant because they contribute to membrane structure, retinal function, anti-inflammatory signaling, and vascular regulation [6; 9]. The strongest current pediatric evidence comes from the Hong Kong Children Eye Study, which found that higher dietary omega-3 intake in children aged 6 to 8 years was associated with shorter axial length, less myopic refraction, and lower myopia risk after adjustment for major confounders [21]. Children in the lowest quartile of omega-3 intake had longer axial length and more myopic refractive status than those in the highest quartile, suggesting a potentially protective association during a critical developmental period [21; 12].

Mechanistically, omega-3 fatty acids may support ocular development by improving choroidal blood circulation, reducing inflammatory signaling, and limiting scleral hypoxia [21; 17]. However, caution remains necessary. A recent systematic review of randomized controlled trials concluded that although polyunsaturated fatty acids and other nutraceuticals have plausible mechanisms, consistent clinically meaningful slowing of myopia progression has not yet been demonstrated [9]. Thus, omega-3 fatty acids can currently be regarded as one of the most promising dietary candidates, but not as a proven stand-alone intervention [21; 9].

#### **Saturated Fat**

If omega-3 fatty acids represent a potentially protective nutrient class, saturated fats have emerged as a possible risk factor [21; 20]. In the Hong Kong Children Eye Study, higher saturated fat intake was associated with longer axial length and less favorable refractive outcomes, suggesting that fat quality may matter more than total fat intake alone [21; 10].

This association is biologically plausible because diets high in saturated fats are linked with adverse metabolic and inflammatory profiles, including insulin resistance and endothelial dysfunction, both of which may affect ocular vascular regulation and scleral remodeling [21; 13]. Given the growing emphasis on choroidal blood flow, tissue hypoxia, and extracellular matrix remodeling in myopia progression, it is reasonable to hypothesize that saturated-fat-heavy diets may contribute to these processes [17; 8]. However, saturated fat should not be interpreted in isolation, since children with higher saturated fat intake may also consume more ultra-processed foods and fewer fruits, vegetables, and fish [20; 6]. At present, the evidence supports concern, but not a simple causal claim [21; 10].

#### **Carbohydrates and Glycemic Load**

The role of carbohydrates, particularly high-glycemic-load carbohydrates, remains one of the most debated topics in the nutrition–myopia literature [3; 19]. The core hypothesis is that diets rich in refined, rapidly absorbed carbohydrates may promote hyperinsulinemia and altered growth-related signaling, thereby influencing scleral and choroidal development [2; 15]. This model has long been discussed because it offers a plausible link between modern dietary patterns and the rising prevalence of juvenile myopia [2].

Recent evidence provides some support, but not full confirmation. A 2025 systematic review and meta-analysis reported a positive association between higher carbohydrate intake and myopia in adolescents, although the studies were few, mostly observational, and highly heterogeneous [19]. By contrast, a 2024 meta-analysis found no significant pooled association between total carbohydrate intake and myopia risk, spherical equivalent, or axial length [10]. These results suggest that total carbohydrate quantity may be less informative than carbohydrate quality, glycemic index, degree of processing, or related metabolic status [10; 2]. This

interpretation is further supported by evidence that higher HbA1c may be associated with increased myopia risk, whereas adiponectin may be protective, and by the overlap between high refined-carbohydrate diets and pro-inflammatory dietary patterns [13; 20]. Thus, the most plausible interpretation is that carbohydrate-related risk is tied more closely to high-glycemic, highly processed dietary exposure than to carbohydrates as a general category [3; 20].

#### Vitamins, Micronutrients, and Carotenoids

Evidence for vitamins and micronutrients in pediatric myopia remains inconsistent [6; 16]. In an NHANES-based study of American adolescents, serum vitamins A, C, D, and E were not significantly associated with myopia after adjustment, suggesting that nutrients widely regarded as beneficial for eye health are not necessarily clearly related to refractive status in pediatric populations [16; 12]. Vitamin D deserves particular caution because lower vitamin D status may reflect reduced outdoor exposure rather than a direct nutritional effect, since sunlight is both a major determinant of vitamin D levels and a well-established protective factor against myopia onset [16; 5].

Carotenoid evidence is also mixed. In the same NHANES-based analysis, lutein and zeaxanthin were not significantly associated with myopia, while higher serum cis- $\beta$ -carotene was associated with greater odds of myopia and high myopia [16]. Because the mechanism remains unclear and the finding comes from a single biomarker-based observational analysis, it would be premature to draw direct dietary recommendations from it [16; 9]. Likewise, randomized trials of carotenoid supplementation have not shown consistent clinically meaningful slowing of myopia progression, even though modest benefits in visual function or macular pigment have been reported [9; 16].

#### Dietary Patterns and Myopia

Dietary patterns may be more informative than individual nutrients because children do not consume nutrients in isolation; they eat combinations of foods shaped by family habits, school environments, food availability, and socioeconomic context [12; 5]. For that reason, the relationship between diet and myopia should be interpreted at the level of overall eating behavior rather than single nutrients alone [20; 21]. This is particularly relevant in pediatric populations, where dietary quality is closely intertwined with physical activity, outdoor exposure, sleep, and screen habits [12; 5]. Such an approach may also help explain why broad macronutrient meta-analyses sometimes show null findings while more pattern-oriented studies still detect meaningful associations [10; 19].

Western-style and pro-inflammatory dietary patterns appear most consistently on the risk-associated side of the literature. The Western dietary pattern is generally characterized by higher intake of refined carbohydrates, added sugars, saturated fats, processed meat, fast food, and ultra-processed products, together with lower intake of fish, legumes, fruits, and vegetables [2; 6]. Although not all pediatric studies explicitly label this pattern as “Western,” many dietary exposures associated with greater myopia risk—such as saturated fat, inflammatory dietary load, and possibly high-glycemic intake—fit within this broader framework [21; 20]. This pattern is biologically plausible in myopia because it tends to promote inflammation, metabolic dysregulation, and vascular stress, all of which have been proposed as contributors to scleral remodeling and axial elongation [20; 13].

A particularly important finding concerns the **pro-inflammatory diet**. Using NHANES data, Ye and colleagues reported that higher Dietary Inflammatory Index scores were associated with greater myopia prevalence in adolescents, especially in those aged 16 to 18 years and in specific educational subgroups [20]. This shifts attention away from single nutrients toward the inflammatory character of the diet as a whole [20; 18]. The association is biologically plausible because inflammatory cytokines, immune signaling, and extracellular matrix degradation have all been implicated in axial elongation [21; 18]. At the same time, these findings remain observational, and pro-inflammatory dietary patterns are likely to coexist with other risk-related behaviors such as lower physical activity, poorer sleep, and greater academic strain [5; 20]. Even so, the available evidence suggests that inflammatory dietary profiles deserve serious consideration in pediatric myopia research [20; 12].

On the more favorable side of the literature are **nutrient-dense whole-food patterns**, which emphasize vegetables, fruits, legumes, nuts, fish, and minimally processed foods while reducing heavily processed products and poorer-quality fats [1; 6]. Direct pediatric evidence for a clearly defined Mediterranean-style or whole-food diet in myopia remains limited, but the biological rationale is strong because these patterns provide omega-3 fatty acids, fiber, antioxidants, carotenoids, and anti-inflammatory micronutrients while lowering dietary inflammatory burden [21; 20]. Broader ocular nutrition literature has already suggested that Mediterranean-type patterns are beneficial

for eye health because of their nutrient density and anti-inflammatory properties [1; 6]. However, this should not be overstated in the context of myopia. At present, such diets can be described as **theoretically favorable**, but not yet as directly proven anti-myopia diets [10; 9; 12; 21].

The **modern processed-food environment** provides another useful way to interpret dietary patterns. Childhood diet is increasingly shaped by convenience, school schedules, marketing, and food affordability, all of which encourage greater reliance on ultra-processed foods [5; 12]. These foods often combine several features already implicated in the myopia literature, including high saturated fat, refined carbohydrates, excess sodium, low fiber, and reduced micronutrient density [19; 20]. Although it would be too strong to claim that processed foods or sugary products directly cause myopia, frequent exposure to such patterns may contribute to the same inflammatory and metabolic pathways already discussed in this review [13; 15; 20]. This reinforces the importance of looking beyond individual nutrients and considering the broader nutritional environment in which children's visual development occurs [12; 5].

Perhaps the most important conclusion from the dietary-pattern literature is that diet should be understood as part of a **lifestyle cluster**, not as an isolated pediatric exposure [12; 5]. Children with healthier diets may also spend more time outdoors, be more physically active, sleep better, and have more structured daily routines, whereas children with lower-quality diets may be more likely to live highly indoor, sedentary, and screen-dense lives [5; 12]. This creates both an opportunity and a challenge for interpretation: dietary patterns may matter partly because they reflect biological food exposure, and partly because they are markers of broader health behavior [10; 20]. For that reason, any relationship between diet and myopia is likely to be multifactorial and context-dependent rather than a simple one-factor effect [12; 5].

Overall, the dietary-pattern evidence supports a cautious but meaningful conclusion. Patterns characterized by higher inflammatory load, greater reliance on processed foods, poorer fat quality, and stronger metabolic disruption appear more often on the risk-associated side of the literature, whereas nutrient-dense, less inflammatory patterns appear more compatible with biological protection, even if direct anti-myopia evidence remains limited [20; 6]. The current literature does not justify claiming that any specific dietary pattern can prevent or treat myopia in a clinically established way [10; 9]. Nevertheless, dietary patterns should now be considered part of the broader modifiable environment of pediatric eye health and a legitimate area for further research, particularly alongside established measures such as increased outdoor time and evidence-based myopia control strategies [11; 4].

### **Diet in the Context of Other Lifestyle and Social Determinants of Myopia**

Diet is unlikely to influence myopia in isolation. In children and adolescents, eating patterns are embedded within a broader network of behaviors and social conditions that also affect ocular development, including outdoor time, near work, screen exposure, educational pressure, and family environment [5; 12]. For this reason, nutrition should be interpreted as one component of a multifactorial lifestyle system rather than as a stand-alone risk factor [5; 4].

#### **Outdoor Time and Indoor Lifestyle Patterns**

Among modifiable lifestyle factors, time spent outdoors remains one of the most consistently supported protective influences against myopia onset in children [7; 5]. Greater outdoor exposure is associated with lower risk of developing myopia, even if its effect on slowing progression in already myopic children appears more limited [7; 11]. Proposed mechanisms include exposure to brighter natural light, retinal dopamine signaling, and other light-dependent influences on ocular growth [5; 4].

This matters for diet because children who spend more time outdoors may also have healthier routines overall, including more regular meals and less sedentary snacking linked to prolonged indoor screen use [5; 12]. Conversely, predominantly indoor lifestyles may combine reduced daylight, increased near work, and food environments dominated by convenience foods or highly processed snacks [5; 20]. Thus, when poorer dietary patterns are observed in myopic children, part of the association may reflect clustering with low outdoor exposure rather than a fully independent dietary effect [21; 5].

#### **Near Work, Screen Exposure, and Educational Routines**

Another major determinant of pediatric myopia is near work, including reading, studying, and digital device use [5; 7]. Reviews consistently identify prolonged near work and intensive educational activity as important contributors to myopia risk, while screen exposure often combines accommodative demand with reduced outdoor time and more sedentary indoor behavior [5; 4; 11].

The relationship between near work and diet is behaviorally important [12; 20]. Children who spend long periods on screens or homework may be more likely to consume sugary drinks, salty snacks, or fast food

during sedentary routines, thereby combining visual stress with lower-quality dietary exposures [20; 2]. This may help explain why dietary inflammatory load and processed-food intake sometimes appear associated with myopia: such foods are often consumed in the same contexts as intensive indoor near work [20; 5]. In this sense, some apparent dietary risk may represent a broader indoor educational lifestyle pattern rather than a purely nutritional effect [12; 10]. This also suggests that preventive strategies may be more effective if they target both diet and daily visual habits together [4; 11].

#### Educational Pressure, Family Context, and Urbanization

Educational intensity is one of the most frequently cited environmental drivers of myopia, especially in settings where children spend long hours on reading, tutoring, and other near-work tasks [5; 11]. In highly competitive academic environments, children may experience sustained indoor study, reduced outdoor time, elevated stress, and irregular eating behavior, all of which may contribute to a more myopiagenic lifestyle context [5; 20]. Educational pressure may therefore act as a social amplifier of both visual and nutritional risk [5; 12].

Family habits and socioeconomic context further shape both diet and myopia-related behaviors [5; 12]. Parents influence meal quality, screen limits, opportunities for outdoor play, and access to preventive eye care, making the family environment a major mediator of risk [5; 4]. Socioeconomic status may affect myopia through multiple pathways: some higher-education households increase near-work pressure, while lower-resource households may face poorer food environments, less access to recreational space, or lower health literacy regarding eye care and nutrition [5; 12]. This complexity is one reason why dietary findings in myopia must be interpreted cautiously [10; 20].

Urbanization adds another layer by concentrating many of the same risk-related factors, including more indoor education, less unstructured outdoor time, more processed food exposure, and easier access to digital entertainment [5; 11; 12]. In this sense, diet becomes a socially embedded exposure rather than merely an individual choice [5].

#### Integrated Lifestyle Model

Taken together, the evidence suggests that diet operates within an integrated lifestyle model of myopia [5; 12]. Lower dietary quality may coincide with reduced outdoor time, more screen-based near work, and educational or urban pressures that collectively create conditions favorable to myopia onset and progression [5; 20]. By contrast, healthier routines may combine nutrient-dense food patterns with more daylight exposure and better-regulated daily structure, potentially creating a more protective developmental environment for the eye [12; 4].

This perspective helps explain why diet may matter even when evidence for individual nutrients remains inconsistent, because dietary patterns may exert influence through interaction with other lifestyle behaviors [10; 5]. It also suggests that future prevention strategies may be most effective when they combine nutritional guidance with established recommendations on outdoor exposure, screen hygiene, and evidence-based myopia management rather than treating each factor separately [4; 11].

### Clinical and Public Health Implications

The evidence reviewed here suggests that diet should not be ignored in pediatric myopia care, but neither should it be overstated. At present, nutrition is best understood as a **potentially relevant adjunctive factor** within a broader, multifactorial model of myopia prevention and management rather than as a stand-alone treatment target [12; 11]. This is clinically important because current myopia management already includes interventions with substantially stronger evidence, such as increased outdoor time, low-dose atropine, orthokeratology, and defocus-based optical strategies, and dietary advice should be positioned alongside—not in place of—those approaches [11; 4].

#### Implications for Clinical Assessment and Counseling

One practical implication is that clinicians caring for children at risk of myopia may benefit from taking a broader lifestyle history that includes not only family history, near work, outdoor time, and screen exposure, but also general dietary pattern and food quality [12; 5]. This does not require detailed nutritional analysis at every visit, but it does justify including diet in a wider risk-profile discussion, particularly in children with rapidly progressing myopia or unfavorable lifestyle routines [12; 21]. A brief assessment of habitual intake—such as frequency of fish consumption, reliance on ultra-processed foods, and regularity of meals—may help identify children whose diet reflects a broader high-risk lifestyle pattern [21; 20].

Based on current evidence, clinicians can reasonably encourage **balanced, nutrient-dense dietary patterns** as part of general pediatric eye-health counseling, even though such advice cannot yet be presented

as a proven myopia-control therapy [1; 12]. In practical terms, this means encouraging regular intake of fish and other omega-3 sources, fruits, vegetables, legumes, and minimally processed foods, while discouraging heavy reliance on foods high in saturated fat, sodium, and refined carbohydrates [1; 21]. The strongest nutrient-specific clinical signal currently comes from omega-3 polyunsaturated fatty acids, which were associated with shorter axial length and lower myopia risk in the Hong Kong Children Eye Study [21]. However, clinicians should frame omega-3 intake as **potentially supportive rather than definitively preventive**, because observational associations do not establish interventional efficacy [21; 9].

Advice about diet should ideally be framed within a broader message about **healthy routines**, not just isolated nutrition targets [12; 5]. A practical counseling model may include encouraging outdoor play, reducing prolonged uninterrupted near work, limiting highly processed snack-heavy routines, providing nutrient-dense meals, and seeking timely eye monitoring when myopia is already present or strongly familial [4; 1]. This better reflects the real-world clustering of diet and myopia-related behaviors and avoids implying that a single nutrient or supplement can solve a clearly multifactorial problem [5; 20; 10; 11].

#### What Clinicians Should Not Overstate

The available evidence does **not** support telling families that diet alone can prevent myopia onset, stop progression, or replace established treatment strategies [10; 11]. At present, the evidence base remains too heterogeneous, observational, and limited in randomized trials to justify strong diet-specific claims for myopia control [10; 9]. The same caution applies to nutritional supplementation. Although some supplements may modestly improve visual fatigue, macular pigment, or related functional measures, they have **not** shown a consistent clinically meaningful reduction in myopia progression overall [9]. Clinicians should therefore avoid presenting over-the-counter nutraceuticals as evidence-based substitutes for established myopia management unless future trials show clearer benefit [9; 11].

#### Integration with Established Myopia Control

The most defensible clinical position is to integrate diet into a **multimodal prevention and management framework**. Current myopia care is strongest when it combines behavioral guidance with validated optical and pharmacologic options tailored to the child's age, risk profile, and progression pattern [11; 4]. This includes increasing outdoor time, regulating near work and screen routines, and, when appropriate, using defocus spectacle lenses, contact-lens-based strategies, orthokeratology, or atropine therapy [4; 11]. Within this framework, diet may be considered an additional modifiable influence that supports broader ocular and systemic resilience rather than a first-line treatment in itself [12; 21].

Families can reasonably be told that healthier dietary patterns may support eye development and reduce exposure to biological conditions associated with myopia, but that established myopia control should still rely primarily on interventions supported by stronger clinical evidence [10; 11]. This kind of measured communication is more useful than either dismissing diet entirely or overpromising its impact [12; 4].

#### Implications for Schools and Public Health

The implications of diet and myopia also extend beyond clinical care, because many relevant behaviors are shaped by institutions rather than by individual visits alone [5]. Schools influence meal patterns, daily schedules, recess and outdoor exposure, screen intensity, and educational pressure, all of which may interact with myopia risk [5; 4]. This means that prevention strategies focused only on clinic-based management may miss broader population-level opportunities [11; 4].

From a public-health perspective, the most realistic implication is not to create nutrition-only myopia programs, but to embed dietary quality within **child eye-health promotion frameworks** that already emphasize outdoor activity, reduced sedentary risk, and early screening [12; 11]. Such frameworks could include school meal quality, protected outdoor recess, parent education, and joint messaging about healthy lifestyle and visual development [1; 5]. Equity should also be considered, since not all families have equal access to healthy food, safe outdoor space, regular eye examinations, or specialized myopia-control interventions [5; 12]. For this reason, advice should be realistic and practical rather than dependent on idealized dietary expectations [1; 12].

#### Implications for Current Practice

Overall, the current implication is not that diet has become a new primary treatment for myopia, but that it has become sufficiently relevant to deserve thoughtful inclusion in comprehensive pediatric eye-health counseling [12; 21]. Clinicians can reasonably encourage healthier dietary patterns, especially those consistent with lower inflammatory burden and better overall ocular nutrition, while remaining transparent that direct myopia-control evidence is still incomplete [20; 1]. At the same time, they should continue to prioritize

interventions with stronger evidence, particularly outdoor-time promotion and evidence-based optical or pharmacologic management when indicated [11; 4].

In this sense, the most defensible clinical position today is one of **measured integration**: diet matters enough to discuss, but not enough to overclaim, and is likely most useful as part of a broader lifestyle and preventive framework [10; 12].

### Limitations of the Current Evidence and Future Research Directions

Any interpretation of the diet–myopia literature must begin with the recognition that the current evidence base remains incomplete, heterogeneous, and methodologically limited [10; 12]. Although recent studies have made the topic more credible and clinically relevant, the literature is still not strong enough to support definitive causal conclusions or highly specific dietary recommendations for myopia prevention and control [10; 9].

#### Observational Evidence and Causality

One of the major weaknesses in the field is the predominance of observational research, much of it cross-sectional rather than longitudinal [10; 19]. Such studies can identify associations between diet and myopia, but they cannot determine whether dietary exposure contributes causally to refractive outcomes, whether myopia-related lifestyles shape diet, or whether both are driven by broader shared factors [20; 10]. This problem is evident in studies of pro-inflammatory dietary patterns, omega-3 intake, saturated fat, and biomarkers such as cis- $\beta$ -carotene [20; 21; 16]. Even when adjustment is used, residual confounding, reverse causation, and unmeasured lifestyle influences remain difficult to exclude [21; 12]. More prospective cohort studies and intervention-based designs are therefore needed to clarify temporal sequence and causal direction [10; 9].

#### Challenges in Dietary and Ocular Measurement

A second major limitation concerns the measurement of diet itself. Most pediatric studies rely on food-frequency questionnaires, dietary recall, or parent-reported intake, all of which are vulnerable to recall bias, reporting error, and imprecision in estimating actual nutrient exposure [21; 12]. This is especially problematic in children, since parents may not fully know what older children consume outside the home or at school [21; 5]. This may partly explain why broad macronutrient meta-analyses often yield inconsistent or null findings while individual studies still detect associations for specific nutrient classes or dietary patterns [10; 21]. Future research would benefit from more standardized dietary assessment, repeated measurement over time, and greater use of biomarkers to complement, rather than replace, self-report data [16; 9]. Even biomarkers, however, are not straightforward, as they may reflect metabolism, supplementation, sunlight exposure, or disease processes rather than diet alone [16; 15].

Another challenge is the heterogeneity in how myopia is defined and measured [10; 19]. Some studies focus on myopia prevalence, others on axial length, spherical equivalent, or high myopia risk, and these outcomes are not interchangeable [21; 16]. In addition, the validity of refractive data depends heavily on methodology, especially whether refraction is cycloplegic in pediatric populations, which is not consistent across datasets [21; 20]. Greater standardization of refractive definitions, outcome reporting, and axial-length measurement would improve interpretability and help clarify whether diet is more closely associated with myopia onset, progression, or structural markers such as axial elongation [11; 4; 21; 22].

#### Residual Confounding and Lifestyle Clustering

Diet is deeply embedded within a cluster of related lifestyle and social factors [5; 12]. Children with poorer diets may also spend less time outdoors, do more near work, and live under different educational or socioeconomic conditions than children with higher-quality diets [5; 20]. Even when studies adjust for some confounders such as outdoor time or parental myopia, it is difficult to capture the full complexity of family routines, school pressure, food environment, and urban context [21; 5]. As a result, some observed diet–myopia associations may reflect shared lifestyle architecture rather than direct nutritional causation alone [12; 10]. Future studies should therefore evaluate diet together with other major behavioral exposures rather than treating these domains separately [5; 12].

#### Limited Trial Evidence and Mechanistic Translation

A particularly important gap is the shortage of high-quality randomized controlled trials testing whether dietary modification or supplementation can meaningfully slow myopia progression [9; 11]. The recent systematic review of randomized trials on nutritional supplementation concluded that although some compounds may improve visual fatigue, accommodative function, or macular pigment, there is not yet consistent evidence of a clinically meaningful reduction in myopia progression [9]. Future trials should therefore focus on outcomes such as axial length change and cycloplegic refractive progression, include adequate follow-up, and distinguish between whole-

diet interventions and isolated supplementation [9; 4]. They may also need to target specific at-risk subgroups rather than assuming uniform effects across all children [21; 11].

Mechanistic plausibility is one of the strengths of this field, but there remains a gap between biological theory and human clinical evidence [17; 21]. Inflammation, choroidal blood flow, scleral hypoxia, and glucose metabolism all provide credible pathways through which diet might influence ocular growth, yet direct proof that changing these pathways through diet alters pediatric refractive outcomes is still limited [17; 13]. Future research should therefore aim for more translational designs that combine dietary assessment with imaging, vascular measures, biomarker analysis, and longitudinal ocular outcomes [22; 17].

#### Population Diversity and Developmental Timing

Another limitation is that much of the most visible recent evidence comes from specific populations, especially East Asian pediatric cohorts and NHANES-based studies in the United States [21; 16]. These studies are valuable, but dietary patterns, food composition, educational systems, outdoor exposure, and genetic backgrounds vary substantially across countries and cultures [5; 12]. Findings from Hong Kong or NHANES therefore cannot be generalized automatically to all child populations [21; 20]. More cross-cultural and multi-country studies are needed, particularly for dietary-pattern research, where the meaning of terms such as “processed food,” “high-fat diet,” or “healthy dietary pattern” may vary by context [5; 19; 12; 1].

Age and developmental stage also matter. Myopia is a developmental condition, and dietary effects may not be uniform across childhood and adolescence [7; 21]. Nutritional influences may differ according to whether the child is pre-myopic, early myopic, rapidly progressing, or approaching high myopia, as well as by school stage or puberty [11; 4]. Future studies should therefore pay closer attention to developmental timing, since any nutritional intervention—if it proves useful—may be more effective as an early preventive strategy than as a late attempt to modify established progression [11; 21; 9; 12].

#### Priority Research Directions

1. Several priorities emerge clearly from these limitations. The field needs:
2. well-designed longitudinal cohorts that repeatedly measure diet, lifestyle, and ocular outcomes over time [10; 5];
3. randomized or quasi-experimental studies testing whether improving dietary quality or specific nutritional exposures can reduce myopia onset risk or axial elongation when added to standard prevention strategies [9; 4];
4. integrated models that evaluate diet together with major behavioral and environmental exposures [5; 12];
5. stronger clarification of whether signals such as omega-3 fatty acids, inflammatory dietary burden, glycemic traits, and biomarkers represent true causal pathways or proxies for broader dietary quality [21; 13]; and
6. better mechanistic-clinical linkage through studies combining diet data with choroidal imaging, perfusion measures, inflammatory markers, and ocular structural or functional outcomes [22; 17]. Research should also include a stronger implementation and equity perspective, examining how food environments, school structures, and socioeconomic conditions shape the feasibility of dietary prevention in real-world settings [5].

#### Discussion

The evidence reviewed in this article suggests that diet is a **plausible but not yet definitively established** modifiable factor in childhood and adolescent myopia. Recent population-based findings strengthen this possibility. In the Hong Kong Children Eye Study, higher dietary omega-3 polyunsaturated fatty acid intake was associated with shorter axial length, less myopic spherical equivalent refraction, and lower odds of myopia, whereas higher saturated fat intake showed the opposite pattern after adjustment for major confounders. However, the broader literature remains inconsistent, and nutrition is still better understood as a **potential rather than confirmed independent determinant** of myopia because diet is closely intertwined with behavioral, environmental, and socioeconomic exposures.

One of the clearest messages from the recent literature is that **dietary patterns may be more informative than isolated nutrients**. A large school-based study in Tianjin found that adherence to a pattern rich in nuts, legumes, whole grains, tubers, and aquatic products was associated with lower myopia risk, whereas adherence to a snack-based pattern dominated by fried foods, fast foods, sugar-sweetened beverages, desserts, and processed meats was associated with higher risk. This is important because children do not consume nutrients in isolation; rather, they eat combinations of foods that reflect family routines, school schedules, food environments, and social conditions. From this perspective, dietary patterns may better capture the real-life context in which refractive development occurs.

The contrast between the Hong Kong findings and the more equivocal macronutrient meta-analytic evidence also deserves attention. A 2024 meta-analysis found no significant overall association between total carbohydrate, protein, or fat intake and myopia risk, spherical equivalent, or axial length in individuals under 18 years of age. By contrast, the Hong Kong cohort suggests that the **quality** of fat intake may matter more than the total amount, since omega-3 fatty acids and saturated fats were associated with opposite ocular outcomes. This indicates that future research should move beyond broad macronutrient categories and examine nutrient subtypes, food sources, and habitual dietary patterns more carefully.

Mechanistically, the literature offers several biologically plausible pathways through which diet could influence ocular growth, including inflammation, altered choroidal blood flow, scleral hypoxia, hyperinsulinemia, oxidative stress, and extracellular matrix remodeling. These mechanisms are credible and help explain why nutrition has become a more serious topic in myopia research. At the same time, most mechanistic support still comes from observational and experimental reasoning rather than from strong pediatric intervention trials.

A key practical implication is that diet should **not** be interpreted as a substitute for established myopia-control measures. Current pediatric protocols emphasize outdoor exposure, near-work regulation, optical interventions, and low-dose atropine, all of which rest on a stronger evidence base than nutrition. The fact that diet is not yet central to current management algorithms does not mean it is irrelevant; rather, it indicates that the evidence is not yet strong enough to justify diet-specific recommendations aimed specifically at slowing myopia progression. At present, the most defensible position is to regard healthy eating as a **potential adjunct**, not a replacement, within broader myopia prevention and control strategies.

This conclusion is reinforced by evidence from randomized controlled trials of nutritional supplementation, which remains limited and heterogeneous. Although some trials reported modest improvements in visual fatigue, contrast sensitivity, accommodative parameters, or macular pigment optical density, there has been **no consistent clinically meaningful reduction in myopia progression** across studies. For that reason, recommending supplements specifically for myopia prevention currently goes beyond what the best available interventional evidence can support.

The topic also has a clear public health and social dimension. Diet in childhood is shaped by parental education, household food affordability, school food environments, urbanization, academic pressure, and time scarcity, all of which also intersect with near work, screen exposure, and outdoor activity. As a result, observed associations between “healthy” or “unhealthy” diets and myopia may partly reflect **wider behavioral clusters** rather than purely nutritional effects. This reinforces the importance of studying diet within an **integrated ecological framework** rather than as an isolated variable.

Several methodological issues also help explain why the literature contains both promising signals and null findings. Many studies are cross-sectional, so temporality cannot be established and reverse causation cannot be ruled out. Dietary exposure is usually measured by food-frequency questionnaires or parent-reported intake, which are vulnerable to recall error and misclassification. Refractive assessment methods are not uniform across studies, and residual confounding is highly likely because diet co-varies with physical activity, academic intensity, body composition, family history, sleep, and urban living conditions.

Future research should therefore prioritize **longitudinal, well-controlled, and ideally multicenter studies** in children and adolescents. Such studies should combine cycloplegic refraction and axial length measurement with standardized dietary assessment, preferably including repeated measures, biomarkers where feasible, and careful adjustment for major lifestyle and socioeconomic factors. It will also be important to distinguish between whole grains and refined carbohydrates, marine versus processed fat sources, and whole-food dietary patterns versus supplementation strategies, because these contrasts appear more informative than crude total macronutrient categories. Ultimately, intervention studies should test whether dietary modification can produce clinically meaningful changes in axial elongation or incident myopia, not merely short-term changes in visual comfort or surrogate outcomes.

In summary, the current evidence supports a balanced conclusion: diet is likely relevant to myopia risk, but the strength of evidence varies considerably by exposure type and study design. The most convincing recent signals point toward a potentially protective role of omega-3-rich and plant-forward dietary patterns, and a potentially adverse role of saturated fat-rich, highly processed snack-based patterns. Nevertheless, because the literature remains dominated by observational studies and supplementation trials have not shown consistent clinically meaningful control of myopia progression, diet should presently be framed as a **promising adjunctive factor within a broader lifestyle approach**, rather than as a stand-alone therapeutic strategy.

## Conclusion

Myopia in children and adolescents is an expanding public health problem that cannot be explained solely by genetics, near work, and reduced outdoor exposure [11; 5]. The evidence reviewed in this narrative article suggests that diet deserves greater attention as a potentially relevant component of the broader environmental and behavioral context shaping refractive development during childhood [12; 20]. Although current evidence does not support the claim that diet alone determines myopia onset or progression, it indicates that nutritional quality may influence biologically meaningful pathways related to inflammation, choroidal blood flow, glucose metabolism, scleral remodeling, and axial elongation [17; 21].

Among nutrient-specific findings, omega-3 polyunsaturated fatty acids appear to have the most promising protective profile, whereas higher saturated fat intake, pro-inflammatory dietary patterns, and possibly high-glycemic or metabolically unfavorable exposures appear more often on the risk-associated side of the literature [21; 19]. By contrast, evidence regarding vitamins, carotenoids, and supplementation remains mixed and insufficient for strong clinical claims [16; 9]. This review also emphasizes that dietary effects are unlikely to act independently, because food patterns are closely interwoven with outdoor time, screen use, educational pressure, family habits, and wider social context [5; 4].

From a clinical perspective, diet should currently be regarded as a potentially modifiable adjunctive factor in pediatric eye health rather than as a proven primary intervention for myopia control [12; 11]. Clinicians may reasonably encourage healthier, nutrient-dense dietary patterns as part of broader child-health and eye-health counseling, particularly alongside established preventive measures such as increased outdoor exposure and evidence-based myopia management [1; 4]. However, current evidence does not justify replacing validated interventions such as atropine, orthokeratology, defocus-based optical correction, or structured behavioral prevention with dietary advice alone [11; 9].

Current evidence remains limited by observational designs, heterogeneous methods, and incomplete mechanistic translation, so future research should prioritize longitudinal pediatric cohorts, improved dietary assessment, standardized ocular outcomes, and interventional studies [10; 22]. In conclusion, diet should not be viewed as a peripheral issue in pediatric myopia research, but neither is it yet a settled one [12; 21]. Rather, it should be understood as part of a multifactorial model of refractive development in which nutrition, daily behavior, and social environment interact during the years when the eye is most developmentally vulnerable [5; 20]. If future high-quality research confirms the associations now emerging, nutritional guidance may become a meaningful component of integrated strategies to reduce the long-term burden of myopia in children and adolescents [4; 11].

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