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Diet Nutrition and Mental Health in Adolescence

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Abstract

Background: Adolescence is a critical period for brain development, coinciding with increased dietary autonomy and exposure to ultra-processed foods. Poor diet quality is associated with cognitive impairment and mental health disorders, yet integrated evidence linking these outcomes remains sparse. Objective: To synthesize evidence from biological mechanisms, observational studies, longitudinal cohorts, and randomized controlled trials (RCTs) examining the relationship between diet quality and cognitive performance, academic achievement, and mental health in children and adolescents.
Methods: Comprehensive narrative review integrating 41 peer-reviewed studies (2009-2025), including 8 systematic reviews/meta-analyses, 8 RCTs and longitudinal cohorts, and 13 cross-sectional observational studies from multiple countries (Brazil, Spain, Canada, Australia, Chile, Iran, China, South Korea, Sweden, Portugal).
Results: Converging evidence demonstrates that poor diet quality (characterized by high consumption of ultra-processed foods and sugar-sweetened beverages, and low consumption of fruits, vegetables, and whole grains) is associated with: [1] impaired cognitive domains (memory, executive function, processing speed); [2] lower academic performance (GPA and standardized test scores); and [3] increased depressive and anxiety symptoms. Three mechanistic pathways emerge: nutrient deficiency impairing structural brain development; ultra-processed foods promoting neuroinflammation, gut dysbiosis, and blood-brain barrier dysfunction; and mental health mediation, whereby depression and anxiety directly impair cognitive capacity. Brief dietary interventions (3-8 weeks) in young adults and adolescents reduce depressive symptoms by moderate-to-large effect sizes (d = 0.36-0.75), sustained at follow-up.
Conclusion: Integrated evidence supports improving diet quality during childhood and adolescence as a scalable strategy to protect cognitive development and reduce mental health burden, with mechanisms operating through nutrient adequacy, reduction of neuroinflammation, and mental health protection.

Introduction

Adolescence is a critical developmental period characterized by profound reorganization of the brain, particularly in the prefrontal cortex, hippocampus, and related regions supporting memory, attention, and executive function [1-3]. This period of heightened neural plasticity coincides with increased dietary autonomy and greater consumption of ultra-processed foods, sugar-sweetened beverages, and fast foods, while intake of fruits, vegetables, legumes, and healthy fats declines [4-6].

Simultaneously, mental health disorders-depression, anxiety, and common mental disorders-have increased in prevalence among youth,alongside declines in academic achievement and quality academic achievement and quality of life [35,37,34,38]. The hypothesis that diet quality influences cognitive development and mental health in youth is supported by multiple biological pathways. The developing brain is highly dependent on specific nutrients; deficiencies in iron, iodine, omega-3 fatty acids, and B vitamins impair myelin development, neurotransmitter synthesis,

and synaptic plasticity, producing lasting cognitive deficits [2,8,9], [19-21]. Beyond micronutrient deficiency, diets rich in ultra-processed foods promote systemic and neuroinflammation, dysbiosis of the gut microbiota, and dysfunction of the intestinal and blood-brain barriers, compromising learning and memory function [4,5,10],[22-24].At the population level, numerous observational studies in children and adolescents document associations between poor diet quality and worse cognitive/academic outcomes and increased mental health symptoms [11-13],[34-38],[41-43],[49,50]. Importantly, randomized controlled trials have begun to provide evidence consistent with causality, demonstrating that brief dietary interventions can reduce depressive symptoms by clinically meaningful amounts [15-18], [25],[42],[48]. Despite advances in nutrition and neuroscience, comprehensive integrative syntheses linking diet quality to both cognitive and mental health outcomes in youth across multiple study designs are sparse. This review aims to fill this gap by integrating mechanistic understanding, observational evidence, longitudinal cohorts, and intervention trials to assess whether and how poor diet quality impairs cognitive development and academic performance in children and adolescents.

Methods

Study Design and Search Strategy: This was a comprehensive narrative and integrative review of the literature for integrative review methodology. A systematic search was performed across PubMed, Scopus, SciELO, PsycINFO, and Google Scholar using keywords: diet quality and cognition, nutrition and adolescent mental health, ultra-processed foods and cognitive performance, academic achievement and diet, depression and dietary patterns, gut-brain axis and mood, and Mediterranean diet and youth mental health. The search was limited to publications from 2009-2025 to capture recent mechanistic understanding and intervention evidence while including foundational works on brain development and nutrition.

Selection Criteria

Studies were included if they:

- Examined associations between diet quality or specific nutrients and cognitive function, academic performance, or mental health outcomes in children or adolescents (ages 6-19 years);
- Employed quantitative designs (cross-sectional, longitudinal, or experimental);
- Provided sufficient statistical reporting for effect size estimation;
- Were published in peer-reviewed journals in English, Portuguese, or Spanish.

Studies were excluded if they:

- Focused exclusively on eating disorders without examining broader diet-cognition-mental health links;
- Involved adult-only samples;
- Lacked outcome measures related to cognition or mental health;
- Were opinion pieces or editorials without empirical data.

Data Extraction and Synthesis: From 73 records examined in full text (including empirical, mechanistic, and contextual sources), 41 empirical studies were selected for detailed analysis and integration in the core synthesis. Data were extracted capturing study design (cross-sectional, longitudinal, RCT), population characteristics (N, age, country), dietary exposure (NOVA classification, dietary patterns, specific nutrients), cognitive or mental health outcomes, adjustment for confounders, and effect sizes.

Synthesis involved:

- Narrative integration organized by mechanism (nutrient deficiency, inflammation/dysbiosis, mental health mediation).
- Organization of observational and intervention evidence by study design.
- Assessment of evidence consistency across populations and geographic regions.
- Discussion of mechanistic plausibility and causal inference.

Additional contextual and mechanistic papers (e.g., on eating disorders, restrictive dieting, obesity, and animal models of ultra-processed food intake) were used to inform the conceptual framework but were not included in the 41 core empirical studies because they did not directly examine diet quality or ultra-processed foods in relation to cognitive, academic, or mental health outcomes in youth.

Researcher positionality: The authors bring interdisciplinary backgrounds in psychology, dentistry, and nutrition training, with longstanding clinical and academic interests in adolescent mental health, eating behavior, and diet-related health outcomes. This lens may have increased sensitivity to studies focusing on ultra-processed foods, psychosocial mechanisms, and common mental disorders in youth. To mitigate interpretive bias, we conducted searches in multiple databases, included diverse study designs (systematic reviews, randomized trials, longitudinal cohorts, and cross-sectional studies), and reported both consistent and inconsistent findings across populations and contexts. We also explicitly considered study quality, methodological limitations, and alternative explanations (such as reverse causation and residual confounding) when interpreting associations between diet, cognition, and mental health.

Results

Biological Mechanisms: How Diet Influences Brain Development and Cognition

Nutrient Deficiencies and Structural Brain Development

Foundational evidence demonstrates that deficiencies of iron, iodine, and omega-3 fatty acids in early childhood produce lasting reductions in hippocampal volume, impaired myelination, and decreased synaptic density, resulting in persistent deficits in language development, attention, and intelligence quotient (IQ) that persist even after nutritional rehabilitation [2],[19-21]. Iron is essential for myelin formation, mitochondrial function, and dopamine and serotonin synthesis; deficiency is associated with poor memory, slower processing speed, and behavioral problems in children [8,9]. Iodine is critical for thyroid hormone synthesis and myelination; deficiency reduces school performance [21].Omega-3 polyunsaturated fatty acids (DHA) are structural components of neuronal membranes and critical for synaptogenesis; low dietary intake is associated with poorer memory and learning [19,47]. Notably, nutrient density during critical developmental windows, particularly prenatal and early childhood, has disproportionate effects on brain structure and programs cognitive capacity across the lifespan [20,21,28]. While older children and adolescents are more resilient to acute

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nutrient shortages, chronically poor diet quality perpetuating micronutrient gaps can impair optimal cognitive refinement during the adolescent period of synaptic remodeling and prefrontal cortex maturation [2].

Ultra-Processed Foods Neuroinflammation and the Gut-Brain Axis

In older children and adolescents, the focus shifts to the effects of overall dietary pattern quality. The "Western" or ultra-processed dietary pattern, characterized by high consumption of sugar-sweetened beverages, packaged snacks, fast foods, and refined carbohydrates, with low consumption of fruits, vegetables, legumes, and whole grains, has a multi-level inflammatory and neurotoxic profile [4-6] [22,29].

Ultra-processed foods promote: [1] systemic low-grade inflammation through high glycemic load, trans fats, and additives activating innate immune signaling and increasing circulating pro-inflammatory cytokines (IL-6, TNF-α, CRP) [4,5,22,26], [2] gut dysbiosis and barrier dysfunction, whereby high sugar and low fiber disrupt commensal bacteria balance, reduce short-chain fatty acid (SCFA) producers, and impair intestinal tight junction integrity, increasing intestinal permeability and permitting bacterial lipopolysaccharide (LPS) to cross into the bloodstream [10,23,30]; and [3] neuroinflammation and blood-brain barrier dysfunction, wherein circulating LPS and pro-inflammatory mediators activate microglial and astroglial cells, promoting a neuroinflammatory state that impairs synaptic plasticity, long-term potentiation (LTP), and dendritic spine density—all essential for learning and memory [10,23,24,30,31].
Recent systematic reviews of RCTs demonstrate that dietary interventions emphasizing vitamin D, omega-3 fatty acids, polyphenols, and microbiota-targeted foods can reduce depressive and anxiety symptoms in youth, with proposed mechanisms involving reduction of inflammation and enhancement of beneficial gut microbiota [18,25,27] [32-34] [40,48].

Blood Glucose Stability and Executive Function

The brain's metabolic dependence on a stable, continuous glucose supply is near-absolute. Rapid blood glucose fluctuations from high-glycemic foods, sugary drinks, or skipped meals transiently impair attention, working memory, and executive function [2,28,35,36]. In school-age children, a low-glycemic breakfast rich in protein and fiber maintains stable glucose and supports sustained attention, whereas skipping breakfast or consuming high-glycemic foods is associated with poorer sustained attention and slower processing speed [2,28].

Sleep Quality and Cognition

Diets high in added sugars, caffeine, and ultra-processed foods are associated with poorer sleep quality, insomnia, and daytime sleepiness in adolescents [14,30,31,33,37]. Since sleep loss impairsmemory consolidation, attention, impulse control, and increases depression risk, poor diet quality may impair cognition partly through sleep disruption [31-33], [39,41].

Observational Evidence: Associations between Diet Quality and Cognitive/Academic Outcomes

Mediterranean and Prudent Dietary Patterns

Cogni-Action Project (Chile): In 1,296 adolescents (10-14 years), adolescents with a Mediterranean-style dietary pattern demonstrated significantly superior performance across cognitive domains (working memory, cognitive flexibility, inhibitory control, fluid reasoning) and higher academic grades compared to Western or other patterns, with effect sizes corresponding to differences of 0.5-1 full letter grade [11].

UP&DOWN Study (Spain): A 2-year longitudinal analysis of 3,692 Spanish adolescents found that greater adherence to Mediterranean-style dietary patterns was associated with better quality of life and higher positive affect, with effects persisting after adjustment for BMI and socioeconomic status [33].

Ultra-Processed Foods and Poor Academic Performance

EHDLA Study (Spain): Among 788 Spanish adolescents (12-17 years), higher ultra-processed food (UPF) consumption was significantly associated with lower GPA across language, mathematics, and English in a dose-response relationship, with adolescents in the highest quartile of UPF consumption having mean GPAs 0.4-0.8 points lower (on a scale of 0-10) than those in the lowest quartile [12]. These associations persisted after adjustment for age, sex, socioeconomic status, physical activity, sleep, and BMI, suggesting the diet-achievement relationship is not merely a proxy for overall health [12].

Diet as a Mediator of Mental Health and Academic Engagement

COMPASS Study (Canada): A prospective analysis of 13,887 Canadian adolescents (12-18 years) followed over 1 year found that higher baseline sugar-sweetened beverage (SSB) consumption predicted significant increases in depressive and anxiety symptoms, while higher fruit and vegetable consumption predicted improvements in well-being [34,45]. The magnitude of effect was clinically meaningful: adolescents in the highest quintile of SSB consumption had approximately 40-50% higher odds of developing new or worsening depressive symptoms over the year compared to the lowest quintile [34].

Multi-Country Observational Evidence: Cross-sectional studies spanning Brazil (n=71,553-94,767), China (n=14,445-8,500), Iran, South Korea (n=51,850), Sweden, and Portugal consistently demonstrate that greater consumption of ultra-processed foods and SSBs is associated with higher prevalence of depressive symptoms, anxiety, common mental disorders, insomnia, and lower quality of life [35-39], [41-43], [49,50].

For example:

- Brazil (PeNSE, 2019): Among 94,767 students, higher UPF consumption was associated with more frequent sadness, irritability, and loneliness [20].
- Since depression and anxiety directly impair cognitive capacity, reducing sustained attention, working memory, cognitive flexibility, and academic motivation, these mental health associations represent a crucial mediating pathway through which diet influences academic performance [34,41,43,45,51].

Randomized Controlled Trials and Longitudinal Cohorts: Evidence for Causality

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Comprehensive Dietary Interventions

Francis et al. (2019): A parallel-group RCT in Australia randomized 76 young adults (17-35 years, majority 18-25) with moderate depressive symptoms to a 3-week dietary improvement intervention (emphasizing fruits, vegetables, whole grains, legumes, lean dairy, lean meats, fish, nuts, olive oil; minimizing ultra-processed foods, sugar, and saturated fats) or control. The intervention group showed significant reductions in depressive symptoms (effect sizes $d \approx 0.65$-0.75, $p < 0.05$), sustained at 3-month follow-up, demonstrating that a brief, structured dietary improvement can produce clinically meaningful reductions in depression in young adults [15]. Tajik et al. (2025): A cluster-randomized school-based intervention in Iran recruited approximately 347 adolescents (~15 years) to 11 schools. The intervention group received 11 sessions over 6 months of lifestyle education (healthy eating, physical activity, stress management). Depressive symptoms (BDI-13) and overall distress (DASS-21) decreased significantly in the intervention group versus control ($p < 0.05$), and healthy eating behaviors increased markedly: more breakfast consumption, higher intake of fruits and vegetables, and lower consumption of fast food and soft drinks [16]. This demonstrates that multi-component lifestyle interventions centered on dietary improvement can reduce mental health symptoms and establish healthier dietary habits at scale in school settings. Campisi et al. (2024): A feasibility pilot trial in Canada enrolled 10 adolescents (12-18 years) with major depressive disorder to a personalized nutrition intervention delivered over 8 weeks, including virtual dietitian counseling, Mediterranean-style meal plans, and food delivery support. Depressive symptoms (CES-DC) showed moderate improvement ($d \approx 0.36$, $p < 0.05$), diet quality (KIDMED) improved significantly, and qualitative interviews indicated improved family meal quality [17].

Specific Micronutrient and Microbiota-Targeted Interventions

Vitamin D: A cluster-randomized trial in rural India (n=451 adolescents) found that high-dose vitamin D3 supplementation (2,250 IU daily for 9 weeks) significantly lowered Beck Depression Inventory scores compared to control [42].

Omega-3 and Polyphenols: Several RCTs have shown that omega-3 supplementation and polyphenol-rich foods (especially berries) can reduce depressive and anxiety symptoms in youth, with proposed mechanisms involving reduction of inflammation [19,25,32,40].

- South Korea (2022, KYRBS): Among 51,850 adolescents, frequent SSB and high-caffeine drink consumption was associated with significantly higher odds of stress, depression, suicidal ideation, and loneliness [39].
- China (Yunnan Province): Among 8,500 adolescents, UPF consumption was positively associated with depressive symptoms, with stronger effects in females [41,42,43].

Microbiota-Targeted Interventions: A meta-analysis of RCTs examining probiotics, prebiotics, fiber, and omega-3 in youth with depression and anxiety found a modest but significant pooled effect suggesting that gut microbiota modulation via diet can improve mood disorders [25,27,34].

Longitudinal Cohorts: Prospective Evidence

Dabravolskaj et al. (2024) - COMPASS, Canada: This prospective cohort of 13,887 adolescents followed over 1 year provides strong evidence that higher baseline consumption of ultra-processed foods and SSBs predicts subsequent increases in depression and anxiety, while healthy diet predicts better mental health trajectories [34,45]. Ferreira et al. (2024) - Early Ultra-Processed Food Consumption, Brazil: A longitudinal study followed Brazilian children assessed for ultra-processed food consumption at ages 3-4 years and tracked to ages 12-13 years. Higher early consumption of ultra-processed foods significantly predicted greater hyperactivity/inattention symptoms in adolescence (assessed via the Strengths and Difficulties Questionnaire), even after adjustment for socioeconomic status and anthropometric factors, suggesting that early dietary patterns have programming effects on neurodevelopment [7,43].

Systematic Reviews and Meta-Analyses

Tucker et al. (2025): A systematic review of 6 RCTs and 13 cohort studies examining diet (Mediterranean-style, vitamin D, omega-3, polyphenols, overall diet quality) and mental health outcomes in adolescents (10-19 years) concluded that while the RCT evidence base is still developing, converging evidence from multiple study designs supports a role for dietary improvement in adolescent mental health, with suggested mechanisms including reduction of neuroinflammation, improved nutrient adequacy, and enhanced gut microbiota health [18]. Hu et al. (2025): A systematic review of 10 RCTs of microbiota-targeted interventions in children and adolescents with depression or anxiety found a modest but significant pooled effect in favor of intervention for reducing depressive and anxiety symptoms, with proposed mechanisms involving reduction of circulating LPS, enhanced GABA production, and reduced systemic and neuroinflammation [25]. Malmir et al. (2023): A meta-analysis of 17 observational studies examining junk food, SSBs, and sweetened snacks in children and adolescents found consistent associations with depression, stress, anxiety, insomnia, and lower happiness/well-being [46]. Firth et al. (2019): A meta-analysis of 16 RCTs of dietary improvement interventions found that interventions to improve diet quality produced significant reductions in depressive symptoms compared to control (pooled standardized mean difference ≈ -0.36, $p < 0.001$), with effects also observed for anxiety [48].

Integrated Conceptual Model

Integrating the evidence reviewed, we propose a three-pathway model linking diet quality to cognitive impairment in youth:

Pathway 1: Nutrient Deficiency and Structural Brain Development

Diets low in micronutrient density fail to support optimal myelination, synaptogenesis, and neurotransmitter synthesis [2,8,9], [19-21] [28]. Particularly during critical developmental windows, such deficiencies produce structural brain alterations and lasting cognitive deficits [2] [19-21] [28].

Even in older children and adolescents, chronic micronutrient insufficiency may compromise the refinement of prefrontal function during adolescent brain reorganization [2].

Pathway 2: Ultra-Processed Foods, Inflammation, Dysbiosis, and Impaired Neural Function

High consumption of ultra-processed foods promotes systemic and neuroinflammation, dysbiosis of the gut microbiota, and dysfunction of the gut barrier [4,5,10,22-24,26,30,31]. These changes impair the integrity of the blood-brain barrier,

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activate microglial and astroglial cells, and reduce synaptic plasticity in regions critical for learning and executive function [10,23,24,30,31]. The result is impaired working memory, attention, processing speed, and cognitive flexibility, manifesting as poorer academic performance [11,12,33,34,38] [41-43].

Pathway 3: Mental Health Mediation

Poor diet quality is associated with increased risk of depression, anxiety, and common mental health disorders, through mechanisms involving micronutrient insufficiency, inflammation, dysbiosis, and poor sleep quality [4-6],[14,19,22,25],[31-33],[35-39],[41-43],[46,50]. These mental health conditions directly impair cognitive capacity, reducing sustained attention, working memory, motivation for learning, and academic engagement, such that the pathway from diet to cognition is partly mediated by mental health status [34,38],[41-43],[45,51]. Notably, these pathways are interconnected and mutually reinforcing: depression induced by poor diet impairs motivation to eat better; poor diet perpetuates inflammation and dysbiosis, worsening mood and cognitive function; cognitive impairment may reduce capacity to plan healthy meals. Thus, intervention at the dietary level may break this negative cycle at multiple points [15-18,25,34,42,43,48].

Discussion

Strength of Evidence and Biological Plausibility

The convergence of mechanistic, observational, longitudinal, and experimental evidence strongly supports the hypothesis that diet quality influences cognitive development, academic performance, and mental health in adolescents [2],[4-6],[10-12],[18-22],[24],[25],[33-35],[38],[41-43],[46-48],[50]. The biological plausibility is high: the brain is metabolically demanding and exquisitely dependent on continuous supply of specific micronutrients (iron, iodine, omega-3, B vitamins) and vulnerable to pro-inflammatory and dysbiosis-promoting effects of ultra-processed foods [2,4,5],[19-22],[23-25],[28,30,31]. Observational studies show dose-response relationships, consistency across many populations and countries, and adjustment for major confounders [11-13],[33-39],[41-43],[49,50]. RCTs provide evidence that brief dietary interventions can produce clinically meaningful reductions in depression within weeks to months, suggesting that the mechanistic pathway is functional and potentially reversible [15-18],[25,27,32,40,42,48]. Of particular note is the temporal precedence established by longitudinal cohorts: baseline consumption of ultra-processed foods predicts subsequent worsening of mental health symptoms [34] and cognitive-behavioral problems [7], supporting a causal direction from diet to outcomes rather than purely reverse causation [34,43,44,45].

Magnitude of Effects and Clinical Significance

The effect sizes observed in dietary intervention RCTs (d = 0.36-0.75 for reduction in depressive symptoms) are moderate to large in magnitude and clinically meaningful [15-17],[18,25,40,42,48].
In EHDLA, the difference in GPA between highest and lowest ultra-processed food consumers was 0.4-0.8 points on a 10-point scale, potentially translating to shifts in letter grades or percentile rankings on standardized tests [12]. In COMPASS, SSB consumption predicted ~40-50% higher odds of developing or worsening depressive symptoms over a year, a relative risk increase that is public-health-relevant [34,45].

Limitations

Directionality in observational studies: While longitudinal cohorts provide stronger evidence for temporal precedence, most cognitive studies are cross-sectional, precluding definitive causal inference. Reverse causation is possible-for example, adolescents with lower academic ability might resort to convenience foods. However, adjustment for major confounders and dose-response associations reduce this as the sole explanation [11-13],[33-39],[41-43],[49,50].

Measurement of dietary exposure: Most studies rely on self-reported questionnaires, subject to recall and social desirability bias. While NOVA classification provides a standardized approach, measurement error is inevitable [22,26,35,46]. Objective biomarkers (e.g., erythrocyte omega-3 levels, plasma micronutrient concentrations) would improve precision [19-21],[28].

Limited RCT data in youth: The RCT evidence base of dietary intervention specifically in children and adolescents, with objective cognitive assessments and follow-up periods beyond 3 months, remains developing. Larger, longer-term RCTs would strengthen causal inferences [18,25,32,40,42,48].

Heterogeneity: There is substantial heterogeneity in intervention types (comprehensive diet improvement vs. nutrient supplementation), duration (3 weeks to 6 months), outcome measures, and populations, complicating direct comparison [15-18],[25,32,40,42,48].

Mechanistic specificity: Few studies simultaneously assess dietary patterns, biomarkers of micronutrient status and inflammation, gut microbiota composition, and cognitive/mental health outcomes. Such studies would better elucidate mechanisms [10],[19-21],[23-25],[28,30,31,34].

Implications for Public Health and Clinical Practice

School-Based Policies: Policies restricting marketing and availability of ultra-processed foods and SSBs in schools, while increasing availability of fruits, vegetables, whole grains, legumes, nuts, and fish, may confer benefits for cognitive development, mental health, and academic achievement [4,11,12,33,35,37,39],[41-43],[49,50]. School-based interventions combining dietary education, physical activity, and stress management merit broader implementation and evaluation [16,18,23,33].

Clinical Assessment for Youth with Mental Health Problems: The RCTs suggest that screening for and improving diet quality should be incorporated into routine clinical assessment and treatment for adolescents with depression, anxiety, or other mental health concerns [15-18],[25,27,32,40,42,48]. Dietary counseling by a registered dietitian, as part of multimodal treatment (alongside psychotherapy and, if indicated, pharmacotherapy), could address an upstream determinant of mental health and potentially lower treatment burden.

Family-Level Interventions: Since food purchasing decisions and meal preparation are largely controlled by caregivers, family-based interventions that educate parents/guardians and modify the home food environment may be more sustainable than individual-level dietary counseling alone [33,37,38,41,45].

Further Research: Larger, longer-term RCTs of comprehensive

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dietary interventions in adolescents with and without existing mental health or cognitive concerns, with objective assessment of cognitive domains and mental health outcomes, are needed [18,25,32,40,42,48]. Studies mechanistically investigating pathways (micronutrient status, inflammatory markers, microbiota composition) would clarify intervention targets [10],[19-21],[23-25], [28,30,31,34].

Conclusion

Integrated evidence from biological mechanisms, observational epidemiology, longitudinal cohorts, and randomized controlled trials converges on the conclusion that poor diet quality—characterized by high consumption of ultra-processed foods and sugar-sweetened beverages, and low consumption of fruits, vegetables, legumes, whole grains, fish, and nuts—is associated with impaired cognitive development, lower academic performance, and increased depressive and anxiety symptoms in children and adolescents [2],[4-6],[10-13],[18-22],[24,25],[33-39],[41-43],[46-50].

Three mechanistic pathways emerge: [1] micronutrient deficiency impairing structural brain development; [2] ultra-processed foods promoting systemic and neuroinflammation, gut dysbiosis, and blood-brain barrier dysfunction; and [3] poor diet quality increasing risk of depression and anxiety, which directly impair cognitive capacity and academic motivation [2,4,5],[8-10],[19-21],[22-25], [28,30,31],[33-35],[38],[41-43],[46-48],[50,51].

RCTs in young adults and adolescents demonstrate that brief to moderate-length dietary interventions, emphasizing. Mediterranean-style or whole-food-based patterns, can produce clinically meaningful reductions in depressive and anxiety symptoms, suggesting a reversible, causal relationship [15-18], [25,27,32,40,42,48]. While the RCT evidence base in youth remains nascent, the consistency of associations across observational studies in multiple countries, the biological plausibility, and positive results of early intervention trials collectively support the hypothesis that improving diet quality during childhood and adolescence is a scalable, potentially cost-effective public health strategy to protect cognitive development, reduce mental health burden, and enhance academic achievement [11-13],[33-39],[41-43],[45,49,50]. Future research should prioritize: [1] larger, longer-duration RCTs of dietary interventions in school and clinical settings; [2] mechanistic studies examining micronutrient status, inflammatory markers, and microbiota changes; and [3] implementation science to translate evidence into sustainable, equity-focused school and clinical policies[18],[19-21],[23-25],[28,30,31,34,40,42,48].

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