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ADHD as a System Disorder: A Narrative Review of Genetic, Nutritional, Psychological and Environmental Interactions

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ABSTRACT

Purpose: Narrative review was conducted to identify, critically appraise, and summarize the current findings on Attention-deficit/hyperactivity disorder (ADHD) and interaction between nutrient status, gene polymorphisms, and environmental exposures in the development and progression of ADHD The review also explores the emerging concept of pseudo ADHD and addresses region-specific challenges, particularly within low- and middle-income contexts such as India.

Materials and Methods: Search was conducted using databases including PubMed, Scopus, and Google Scholar. Articles published between 2000 and 2024 that relevant to ADHD, nutrigenomics, micronutrient deficiency, dopamine regulation, pseudo-ADHD, and public health. A total of 110 unique sources were reviewed, including genetic studies, clinical trials, meta-analyses, and population health reports.

Results: Systematic reviews including individual meta-analyses were included in the umbrella review reporting strong evidence that variants in genes like MTHFR, SLC6A3 (DAT1), FADS2, and DRD4 influence neurodevelopmental trajectories by affecting methylation, fatty acid metabolism, and dopamine signalling.

Conclusion: Strong evidence supports that these genetic susceptibilities are modulated by dietary intake of folate, zinc, iron, and omega-3 fatty acids. Environmental factors—such as sleep disruption, excessive screen time, and ultra-processed food consumption—may lead to behavioural phenotypes that mimic or amplify ADHD symptoms. Indian children are particularly at risk due to the coexistence of undernutrition and modern dietary shifts.

KEYWORDS: ADHD; nutrigenomics; micronutrients; gene–diet interaction; MTHFR; dopamine; pseudo-ADHD; FADS2; folate; omega-3; inflammation; India

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IMPLICATIONS FOR REHABILITATION

- The neurodevelopmental disorder known as Attention Deficit Hyperactivity Disorder (ADHD) can be defined by impulsivity, hyperactivity, and inattention.
- ADHD is frequently identified in children and may last into adulthood, impacting relationships, employment, education, and other facets of life.

WHAT IS ALREADY KNOWN

- ADHD is thought to be caused by a confluence of neurological, environmental, and genetic variables, while its specific aetiology is unknown.
- A family history of ADHD increases the likelihood of developing the illness, indicating the importance of genetic susceptibility.
- Medication, counselling, and educational support are frequently used in conjunction for treatment.

WHAT THIS STUDY ADDS

- Highlights a Gene-Diet Interaction: The paper provides strong evidence that genetic susceptibilities for ADHD (involving genes like MTHFR, SLC6A3, FADS2, and DRD4) are directly influenced by a person's dietary intake of key nutrients like folate, zinc, iron, and omega-3 fatty acids.
- Introduces the "Pseudo-ADHD" Concept: It suggest the idea of "pseudo-ADHD," where symptoms that mimic ADHD are not neurobiological in origin but are instead fueled by modifiable lifestyle factors such as poor sleep, excessive screen time, and unbalanced diets.
- Focuses on the Indian Public Health Context: The review specifically identifies Indian children as a high-risk group due to the "double burden" of coexisting micronutrient deficiencies and a simultaneous increase in the consumption of ultra-processed foods.
- Proposes a "Systems Disorder" Framework: It reframes ADHD not merely as a neurological condition but as a complex "systems disorder". This new framework considers the interchange between genetics, nutrition, and environmental factors, pointing toward precision nutrition as a future therapeutic direction.

INTRODUCTION

Attention deficit hyperactivity disorder (ADHD) is a common neurodevelopmental disorder occurring in 5.9–7.1% (based on two different studies) of children worldwide and impairing academic and social functioning [1,2]. Despite an estimated heritability of attention-deficit/hyperactivity disorder (ADHD) at ~75% from twin and family studies, symptom expression is strongly affected by nutritional factors and the environment [3]. According to a review [4-6], key genetic polymorphisms such as dopamine transporter gene, reuptake (SLC6A3/DAT1), dopamine receptor D4 gene (DRD4), and methylenetetrahydrofolate reductase (MTHFR) alter dopaminergic signalling or DNA methylation and have been consistently implicated in the pathophysiology of ADHD.

India's Emerging ADHD Landscape

The unique setting of India: linear projections between increasing urbanization and dietary transition to rising ADHD diagnoses. Multiple national surveys of Indian children show that deficiencies of iron, zinc, folate and vitamin B12 are common in the population [7]. Simultaneously, the consumptions of ultra-processed foods (UPFs) containing high amounts of sugar and additives is gradually increasing—an established risk factors for neurological impairments [8,9].

The Challenge of Pseudo-ADHD

Many children exhibit ADHD-like symptoms that arise from modifiable behavioural or environmental stressors. This pseudo-ADHD includes attentional and behavioural disturbances driven by poor sleep, excessive screen time, and unbalanced diets, rather than neurochemical abnormalities [10–12]. Misdiagnosing these cases may lead to unnecessary pharmacotherapy and neglect of underlying lifestyle contributors.

MATERIALS AND METHODS

Design

It's a narrative review. Comparing this method to quantitative review methods, the former offers more room for individual insight and literature speculation. Therefore, it could serve as a guide for medical personnel managing and providing high-quality care to individuals with ADHD

Authors' roles in the review process

Following a screening of the titles and abstracts, the first author (RS) identified studies, extracted data, evaluated each study's quality, analysed the data, and derived themes from the review before organising them into a themes matrix. The final list of included papers was evaluated by the second third and fourth authors (AD, AZ and NS), who then discussed any disagreements until an agreement was reached. Additionally, they verified the completeness and quality of the data extraction tables. Fifth author (AK) convened virtually to settle any preliminary disputes. NS and AK went over the theme matrix again and spoke about the differences until they agreed.

Search of key terms

The search terms used in this review included ADHD, biochemical mechanisms, dopamine, Methylation Pathways, Inflammation, and the Gut–Brain Axis

Inclusion criteria

Publications were included in the review if they were (i) original research studies that focused on the lived experiences of attention-deficit hyperactivity disorder in children, adolescents, and adults, (ii) used a qualitative research approach for data collection, (iv) were written in English, and (iii) included data from focus groups or interviews with study participants. Grey

literature, or unpublished scientific data, was excluded. We looked through databases to find research papers released between 2000 and 2024

Data extraction

RS searched through the titles and abstracts of the studies found in the first search. After reading all of the studies deemed possibly eligible, those that met the inclusion and exclusion requirements were kept. The final list of included studies was then evaluated by AD and AZ. For every study that was included, RS, AD and AZ finished the data extraction. NS and AK verified the quality and completeness of the data extraction tables. Disagreements were resolved by re-evaluating and discussing them until the reviewers came to a mutually beneficial conclusion**.**

From each study, the following data were taken: study, nation, goal, design, sample, technique, setting, and important findings**.**

RESULTS

Key findings from the results are discussed under following headings-

Core Biochemical Mechanisms: Dopamine, Methylation, and Nutrient Interactions

- *Dopamine Dysregulation and Genetics*

ADHD has been closely linked to dopaminergic dysfunction, particularly in the front ostriatal circuits responsible for attention and executive function [13]. Polymorphisms in DAT1 and DRD4 modulate synaptic dopamine availability and are associated with impaired behavioural inhibition and increased ADHD risk [4,5,14]. These changes may blunt prefrontal cortex activity and impair reward processing [15].

- *Methylation Pathways and MTHFR*

The MTHFR enzyme controls one-carbon metabolism, which is critical for DNA methylation and neurotransmitter pathways. The C677T polymorphism leads to decreased function of the MTHFR enzyme, which in turn leads to elevated homocysteine and decreased methylation. Recent research is linking MTHFR and methylation functional changes to increased susceptibility to ADHD [6,16,17].

- Micronutrients and Epigenetic Modifiers

Micronutrients (e.g., folate, B_{12}, zinc) function as cofactors in methylation and the production of neurotransmitters. When there is nutrient deficiency, the genes involved in the process may not be fully functioning, increasing oxidative stress or disabling normal neurodevelopment, and therefore amplifying an individual's genetic risk [18–20]. For example, in cases of folate or B12 deficiency, chronically elevated homocysteine levels will occur. In others, the absence of adequate zinc will impair the conversion of phenylalanine to dopamine [21,22]. Together these unique genetic, epigenetic and nutrition routines, support biological plausibility that ADHD has a biological disorder that is considered a neurochemical disorder that is inherited or developed.

Fatty Acids, Inflammation, and the Gut–Brain Axis

- *Omega-3 Deficiency and PUFA Pathways*

Long chain omega-3 fatty acids such as DHA and EPA make membranes fluid and support synaptic transmission [23]. Children with ADHD routinely present with lower blood levels of omega-3 fatty acids and supplementation of omega-3 has shown small, but persistent benefits for behaviour phases [24,25].

- *FADS2 Polymorphisms*

The FADS2 gene involved in the conversion of PUFA has polymorphisms associated with poorer fatty acid profile and increased risk of ADHD [26]. A decrease in conversion from ALA to EPA/DHA may increase neuro inflammation and decreased stability of the membrane in the developing brain.

- Inflammation and Immune Activation

Meta-analyses reveal elevated pro-inflammatory cytokines—such as IL-6 and TNF-α, to be higher in children with ADHD, suggesting that systemic inflammation may contribute to neurocognitive dysfunction [27].

- The Gut–Brain Axis and Microbiota

Gut dysbiosis in early life may disturb brain development through immune activation and altered production of neuroactive compounds. Germ-free animal models show exaggerated stress reactivity and reduced neurogenesis—both linked to ADHD traits [28,29]. Diets high in sugar and low in fibre worsen dysbiosis, whereas probiotics and prebiotics may provide benefit [30]. A summarised table for genetic, biochemical and nutritional reason are mentioned below (Table 1)

Table 1. Genetic, Biochemical and Nutritional reasons involved

Gene/Polymorphism	Biochemical Effect	Key Nutrients Involved	Nutrient Status	Potential Benefit of Adequate Nutrient Intake
MTHFR C677T / A1298C	Altered folate metabolism, reduced methylation capacity, elevated homocysteine	Folate, Vitamin B12, Vitamin B6	Decrease (deficiency common)	Improved methylation efficiency, enhanced neurotransmitter synthesis, reduced oxidative stress
FADS2	Reduced conversion of alpha-linolenic	Omega-3 fatty acids (EPA, DHA)	Decrease (low EPA/DHA status)	Better neuronal membrane fluidity,

	acid to long-chain omega-3 fatty acids			improved synaptic function
SLC6A3 (DAT1)	Altered dopamine transporter activity affecting dopamine reuptake	Zinc, Iron	Decrease (deficiency reported)	Stabilised dopamine signalling, reduction in hyperactivity and impulsivity
DRD4	Variation in dopamine receptor function affecting signal transmission	Zinc, Iron, Tyrosine	Decrease (nutrient insufficiency reported)	Enhanced attention regulation and executive function
COMT Val158Met	Altered catecholamine metabolism influencing dopamine degradation rate	Magnesium	Decrease (low magnesium observed)	Optimised dopamine availability, improved working memory
CLOCK / PER3	Disruption of circadian rhythm regulation	Vitamin D, Melatonin	Decrease (low vitamin D common)	Improved sleep-wake cycle and behavioural regulation
Il-6	Pro-inflammatory cytokine;	Omega-3 fatty acids, Vitamin D, Antioxidants (Vitamin C, E)	Decrease in anti-inflammatory nutrients	Reduced inflammation, improved neuronal signalling
SOD2, GPX1	Altered antioxidant defence capacity	Selenium, Vitamin C, Vitamin E	Decrease (lower antioxidant levels noted)	Reduced oxidative stress, enhanced neuronal protection
TNF-α	Pro-inflammatory cytokine; associated with oxidative stress and neurotransmitter dysregulation	Omega-3 fatty acids, Vitamin D, Antioxidants (Vitamin C, E)	Decrease in anti-inflammatory nutrients	Attenuation of neuro inflammation and oxidative stress

Pseudo-ADHD: Overlooked Lifestyle Drivers

While ADHD is often considered a neurodevelopmental condition with a strong genetic component, many children present with ADHD-like symptoms that originate from non-neurobiological causes. These cases, described here as pseudo-ADHD, share clinical features with true ADHD—such as inattentiveness or hyperactivity—but are triggered by environmental, behavioural or nutritional stressors [31].

- *Sleep Deprivation and Circadian Disruption*

Poor sleep quality and reduced sleep duration can mimic or worsen ADHD symptoms. Children with restricted sleep or circadian misalignment show impaired attention, working memory and emotional regulation [32]. Sleep disturbance also affects dopaminergic functioning and prefrontal cortex functioning, which may replicate the neurobiology of ADHD [33].

- *Excessive Screen Exposure*

Screen Time Advice Increased screen time, especially prior to sleep, is associated with lower melatonin levels, decreased sleep duration and behaviour regulation [34]. A child or young person using screens for more than two hours of recreational time each day, was shown to have increased rates of inattention, impulsivity and irritability [35,36].

In addition, screen use on fast-paced, highly stimulating media may dysregulate executive function and reinforce seeking rewards in the short term which is a core feature of ADHD behaviours [37].

- *High*-Glycaemic Diets and Glycaemic Variability

A diet rich in refined carbohydrates and sugars cause sharp increases and declines in glycaemic levels and may exacerbate control and focus issues [38]. New research has demonstrated high-GI (glycaemic index) meals yield less frontal lobe activation and deficits in attention in children of school age. These pseudo-ADHD presentations highlight the importance of differential diagnosis and indicate that non-pharmacological interventions (such as sleep hygiene education, screen time reduction, dietary plan to achieve low-GI meals) can address symptoms without the use of medication.

The Indian Context: Micronutrient Deficiencies and Ultra-Processed Diets

- Nutritional Shortfalls in Indian Children

Despite economic growth, India faces a persistent burden of micronutrient deficiency, also known as “hidden hunger.” National nutrition surveys reveal widespread shortfalls in iron, zinc, vitamin A, folate, and B12 among children, even in

urban settings [38]. These nutrients are essential for myelination, neurotransmitter synthesis, and DNA methylation during brain development [39].

The reliance on a cereal-based diet with few animal-source foods and even limited dietary diversity, further perpetuates this deficiency. In particular, phytate-rich staples like polished rice and wheat can inhibit zinc and iron absorption [40]. Deficiencies in these nutrients may worsen genetic vulnerabilities, especially in children with MTHFR or FADS2 polymorphisms.

- Increasing Consumption of Ultra-Processed Foods (UPFs)

Alongside undernutrition, India is seeing a surge in the consumption of ultra-processed foods (UPFs), especially among school-aged children. These include packaged snacks, sugary drinks, and fast foods high in refined carbs, trans fats, synthetic dyes and preservatives [41]. Such foods displace nutrient-dense meals and promote neuro inflammation, oxidative stress, and gut dysbiosis—all risk factors for cognitive and behavioural dysfunction [42].

Notably, food dyes like tartrazine (Yellow No. 5) and preservatives such as sodium benzoate have been shown to increase hyperactivity and impulsivity in susceptible children [43]. Meanwhile, UPF-heavy diets are also low in fibre, starving beneficial gut bacteria and weakening the gut–brain axis [44].

- *The Double Burden and Public Health Risk*

India's coexisting burden of micronutrient deficiency and processed food overconsumption represents a dual nutritional threat. This pattern not only increases risk for metabolic disorders but may also exacerbate neurodevelopmental vulnerabilities or create pseudo-ADHD presentations.

Roots of UPF consumption are layered, requiring a surface of solutions: improving maternal and early life nutrition, restricting advertising of UPFs to children, and incorporating precision nutrition education into school health programs.

Precision Nutrition and Future Directions

The emerging science of precision nutrition encompasses genomics, metabolomics, and dietetics to develop dietary strategies for individuals, based on genetic and biochemical profiles [45]. With respect to ADHD, individualisation of nutrition has been proposed as the presence of polymorphisms in genes, such as MTHFR, FADS2, and DAT1, influence the metabolism of nutrients and the neurotransmission of those nutrients; thus, dietary component strategies may produce better therapeutics to diminish symptoms [46,47].

- *Nutrigenetics and Gene–Diet Interactions*

MTHFR variants can inhibit folate metabolism thereby decreasing methylation efficiency and increasing homocysteine. In these patients, activated forms of folate such as L-methylfolate may prove to be more effective than regular folic acid [48].. Likewise, FADS2 polymorphisms that inhibit the insoluble conversion of omega-3 fatty acids may require direct supplementation with DHA/EPA instead of using dietary precursors such as ALA [49]. Gene-nutrient interactions may also be found in dopamine-associated genes. Children's genetic variations in the dopamine transporter gene (DAT1) may lead to differences in attention and executive function [50], and the use of iron or zinc (cofactors of dopamine synthesis) has led to variability in benefit in children with ADHD [51].

- *Integrative Clinical Models*

Incorporating nutrigenetic screening into an ADHD paradigm would allow for the identification of subtypes with methylation deficits, PUFA metabolic inefficiencies, or who are particularly sensitive to oxidative stress. Some pilot studies indicate that procedural and round-spectrum micronutrient interventions with a focus on biochemical individuality may be as effective as stimulants for mild and moderate ADHD [52].

would include biomarker panels as part of clinical work-ups, such as blood homocysteine, profiles of fatty acids, inflammatory markers, markers of physiological states, and so on, to inform supplement selection and dietary patterns [53].

- *Limitations and Ethical Considerations*

Ultimately, there are limitations to precision nutrition: cost, access to genetic testing, and the need for clinical validation, in a variety of large ADHD populations. Considering the ethical implications of genetic risk stratification in children should also be part of the ongoing discussion [54]. Nevertheless, the growing availability of consumer nutrigenomic services and personalised supplement platforms underscores the need for evidence-based guidelines to prevent misinformation and support safe implementation.

Diagnostic Challenges and Comorbidities in ADHD

ADHD diagnosis is grounded in behavioural criteria outlined in the DSM-5. Though ADHD symptoms may overlap with other conditions, potential confounding factors include a number of internalising disorders, learning disabilities and occasionally sociocultural factors that may conceal or produce some of the core ADHD symptoms [55].

- *Psychiatric and Neurodevelopmental Comorbidities*

Over 60% of children with ADHD have at least one comorbid condition, many commonly found with ADHD are anxiety disorders, oppositional defiant disorder (ODD), learning disorders or traits of autism spectrum [56]. Comorbidities may alter the ADHD phenotype—e.g., anxiety may reduce impulsivity but worsen inattentiveness—and influence treatment responsiveness [57]. Emotional dysregulation, once considered peripheral, is now recognised as a core domain in many ADHD presentations, linked to poor outcomes and increased functional impairment [58].

- *Underdiagnoses and Misdiagnosis*

In India, ADHD often goes under-recognized in rural areas due to limited awareness and access to care, while in urban school settings, academic pressure and medicalization may contribute to over diagnosis in children with normal variations in attention and behaviour [59]

Moreover, pseudo-ADHD—arising from screen overload, sleep disruption, or poor nutrition—is frequently mistaken for clinical ADHD in the absence of thorough history-taking and functional assessment [27, 29].

- *Gender Disparities in Diagnosis*

Girls with ADHD tend to present more often with inattentive symptoms and internalizing features such as low self-esteem—patterns that frequently go unrecognized. As a result, they are often underdiagnosed and receive delayed intervention, which can negatively impact academic and emotional functioning [60].

- *Tools and Biomarkers: Where Do We Stand?*

Several neurocognitive tests, rating scales, and emerging biomarker panels have been proposed to supplement clinical assessment. Despite the absence of any validated biomarker for ADHD diagnosis, neuroimaging and genetic risk scores show promise as a biomarker, but still not specific and standardized [61].

Treatment Modalities: Pharmacological and Non-Pharmacological Approaches

ADHD is best managed by using a multimodal approach with a combination of pharmacological, behavioural, and nutritional intervention. Treatment decisions must factor in symptom severity, comorbidity, and family preferences.

- *Pharmacological Treatments*

Stimulants, including methylphenidate and amphetamine-dexamphetamine medications, are considered the first-line medication for ADHD because of the strong efficacy evidence in improving attention and decreasing hyperactivity [62]. Mechanistically, stimulants are mainly norepinephrine and dopamine reuptake inhibitors that increase the synaptic availability of the neurotransmitters in the prefrontal cortex [63].Long-acting formulations of stimulants increase adherence, while also reducing the inevitable rebound symptoms, but can lead some patients to anti-social behaviour, lessened appetite, greater sleep disturbance, headaches, and cardiovascular concerns such as increased heart rate and blood pressure [64]. Once patients have been diagnosed with ADHD and the decision has been made to treat the disorder with pharmacological agents, the treatment would be started in the first instance with stimulants. Non-stimulant medications [65], including atomoxetine (NE-selective reuptake inhibitor) and guanfacine (an α2A-adrenergic agonist) have alternative treatment possibilities for individuals who have tics, anxiety, and/or in individuals who do not respond to stimulants. It should also be important to mention, through meta-analyses, that while stimulants produce better overall responses, some sub-groups may tolerate non-stimulants better. For example, non-stimulants may work better for individuals where emotional regulation was the main concern [66].

- *Non-Pharmacological Interventions*

Behavioural therapies—including parent training, cognitive behavioural therapy (CBT), and classroom behaviour modification—have demonstrated benefits, especially for young children and those with mild-to-moderate symptoms [67]. These approaches improve executive functioning, emotional control, and social skills, and are particularly valuable when medication is not indicated or accessible.

Emerging evidence lends credence to computerised cognitive training, mindfulness and neuro feedback as complementary treatments, though effects are generally smaller and more variable [68].

- *Nutritional and Supplement-Based Therapies*

As discussed in earlier sections, zinc, iron, omega-3 fatty acids, and broad-spectrum micronutrients may benefit select subgroups with biochemical deficiencies. These are best implemented alongside—not in place of—core behavioural or pharmacological treatments [21,52].

Public Health and Policy Implications

ADHD is generally considered a neurodevelopmental disorder requiring clinical diagnosis and pharmacological treatment. However, there is increasing evidence to suggest that ADHD diagnosis and symptom severity is related to social determinants, dietary patterns, health system capacity, and public awareness [69]. This indicates a need for larger public health initiatives beyond the clinical setting.

- *ADHD as a Public Health Concern: An Overlooked Burden*

Even though Attention-Deficit/Hyperactivity Disorder (ADHD) carries large global burdens, it is most often overlooked as a public health concern, particularly in low- and middle-income countries (LMICs) such as India. In LMICs, ADHD symptoms are primarily viewed as behavioural wrongdoing, spiritual disturbance or poor parenting. These perceptions result in delayed diagnosis and thus delayed treatment [70].. In addition, many educational systems in India do not have an inclusive framework or trained personnel, for children with attention or learning difficulties, experiencing stigma in the classroom, poor academic achievement and emotional distress [71].

- *Nutrition Policy Gaps*

India's national nutrition programs (e.g., POSHAN Abhiyaan, Mid-Day Meal Scheme) focus primarily on underweight and stunting, but rarely address neurodevelopmental outcomes linked to micronutrient status [72]. There is no formal screening for iron, zinc, or B12 deficiency in children with attention problems, despite strong biological rationale for such testing [20,21].

Policies should be reoriented to recognise that micronutrient sufficiency is not only about growth—but also cognitive function.

- *ADHD and School Health Programs*

The Rashtriya Bal Swasthya Karyakram (RBSK) in India includes developmental screening, but its operational scope often lacks dedicated psychological or dietary assessment protocols [73]. Training school health personnel to recognize behavioural red flags, evaluate nutrition history, and refer appropriately may also assist in reducing diagnostic timing delays. Adding the guidelines surrounding screen-time, sleeping education, and promoting low-GI meals in school wellness policies may reduce the burden of pseudo-ADHD [74]

- *Access, Equity, and Awareness*

Diagnosis and treatment of ADHD has an urban bias, leaving many rural children with limited access to child psychiatrists,

behavioural therapists, or paediatricians schooled in ADHD [75]. Economic barriers also reduce treatment compliance. These inequities highlight the importance of task-sharing types of models, community mental health engagement, and school support.

Importantly, there need to be social awareness campaigns to decrease stigma from altered but functional children, as well as educating parents, teachers and health workers about the causes of true ADHD and pseudo-ADHD [76].

- *Early-Life Interventions and Maternal Nutrition*

An increasing number of studies suggest that the risk of developing ADHD starts before any human is born, and the risk is modified by maternal nutrition, inflammation, and environmental exposures during pregnancy. To stimulate neurodevelopment and the methylation pathway, nutrients including folate, iron, zinc, iodine, and DHA must be adequate in pregnancy for the best outcome for foetal brain [77].

- *Folate and Methylation*

An adequate amount of peri conceptional folate consumption will help significantly decrease birth defects from neural tube defects and should also curtail the developing attention systems of the foetal brain. Low maternal folate is associated with increased risk of behavioural problems, including hyperactivity and inattention, in early childhood [78,79]. Polymorphisms in MTHFR, which impair folate metabolism, further elevate risk in the context of maternal deficiency. Supplementing with methylated folate forms may be more effective for genetically at-risk mothers [48].

- *Iron and Neurotransmitter Synthesis*

Iron is essential for dopamine synthesis and myelination. Maternal iron deficiency has been linked to reduced cognitive performance, poor inhibitory control, and increased ADHD symptoms in offspring [80]. Animal models show persistent dopaminergic dysregulation when foetal iron supply is inadequate.

- *DHA, Iodine and Brain Maturation*

DHA is required for neuronal membrane development, especially during the third trimester. Inadequate maternal DHA has been associated with poor attention regulation in infancy and increased impulsivity during childhood [81,82]. Iodine, often overlooked, is necessary for thyroid hormone production, which regulates myelination and brain growth. Mild-to-moderate iodine deficiency during pregnancy correlates with lower IQ and higher ADHD symptoms in school-aged children [83].

- *Public Health Opportunity*

Despite these known risks, most antenatal programs focus only on iron and folic acid supplementation. Expanding prenatal guidelines to include zinc, DHA, and iodine, especially in vegetarian or resource-limited populations, could significantly reduce ADHD incidence. Educational campaigns aimed at improving maternal diet diversity and access to fortified foods are essential.

Food Environment, Digital Exposure, and Policy Gaps

ADHD symptom expression is increasingly influenced by modern environmental factors such as dietary commercialisation, media exposure, and urban lifestyle shifts. These factors disproportionately affect vulnerable populations, especially children in low-resource settings.

- *Ultra-Processed Foods and Food Labelling Gaps and dyes*

Ultra-processed foods (UPFs) now constitute a significant portion of urban children's caloric intake in India and globally. These energy-dense, nutrient-poor products—high in additives, refined sugars, and preservatives—have been linked to increased hyperactivity and attention deficits in adolescents [84] Yet most nations, including India, lack comprehensive front-of-pack labelling systems that warn consumers about neurodevelopmental risks. Global reviews have linked excessive ultra-processed food intake to cognitive delay, reduced academic achievement, and increased behavioural problems in children. Conversely, early-life protective factors—such as breastfeeding—are associated with improved neurodevelopmental outcomes [85]. Despite growing evidence, only a handful of countries regulate artificial food colours linked to ADHD-like symptoms. The European Union mandates warning labels on products containing certain synthetic dyes, but India, the U.S., and others have weaker or no restrictions. Policy action to limit the use of azo dyes (e.g., tartrazine) and sodium benzoate in school foods could significantly reduce behavioural dysregulation in susceptible children [86].

- *Digital Media Exposure and Screen Policy*

Children today spend increasing hours with digital screens, often starting before age two. Excessive media use is associated with delayed language, sleep disruption, impulsivity, and executive dysfunction, mimicking ADHD [87,88].

While screen-time guidelines exist from the American Academy of Paediatrics and Indian Academy of Paediatrics, enforcement and awareness remain poor. Public health campaigns targeting media literacy, digital hygiene, and sleep–screen balance are urgently needed.

- *Advertising to Children*

The marketing of sugary cereals, energy drinks, and mobile games directly to children reinforces unhealthy habits and overstimulation. Neurocognitive models show that reward-seeking behaviours promoted by advertising can entrench impulsivity and inattention in children already at risk for ADHD [89]. Restricting junk food and screen-based advertising to children is a low-cost, high-yield intervention supported by evidence from multiple countries.

Future Research Priorities in ADHD and Nutrition

Although the interaction between genetics, nutrition, and ADHD is increasingly recognized, current research remains fragmented. To advance clinical translation and public health application, several key gaps must be addressed.

- *Population-Specific Nutrigenomic Studies*

Most nutrigenomic studies in ADHD are derived from Western populations. There is an urgent need for India-specific data involving polymorphisms in genes like MTHFR, FADS2, SLC6A3 (DAT1), and DRD4. These variations may significantly

influence both nutrient metabolism and treatment response in ADHD [90–92].

- *Longitudinal Nutritional Cohorts*

Long-term data linking prenatal and early-childhood nutrition with ADHD symptoms are needed to separate genetic vulnerability from modifiable triggers. This will also help clarify pseudo-ADHD from true neurodevelopmental forms [93].

- *Precision Nutrition Trials*

Randomised controlled trials must assess the effect of genotype-matched interventions (e.g., L-methylfolate for MTHFR C677T or DHA for FADS2 variants) on symptom control, cognitive development, and emotional regulation [94].

- *Microbiome and Gut–Brain Axis*

Emerging evidence implicates the gut microbiota in neurotransmitter synthesis, HPA axis regulation, and inflammation. Investigating how prebiotics, probiotics, or high-fibre diets may improve ADHD symptoms is an important research priority [95].

- *Early Detection and Biomarkers*

Emerging evidence suggests that serum biomarkers such as homocysteine, vitamin B12, and iron-related parameters may aid in early detection of ADHD and guide personalised nutrition-based interventions. Incorporating such assessments into school health systems could enhance timely identification and support for at-risk children [96].

Gene–Nutrient Interaction Mapping in ADHD: A Functional Overview

The interplay of genetic polymorphisms and micronutrient metabolism informs variability of presentation of symptoms of ADHD, treatment, and prescription response. Understanding these gene–nutrient interactions provides a framework for personalised nutrition and future clinical tools.

- *Functional Gene–Nutrient Matrix*

1. MTHFR – Folate, B12:

The MTHFR gene with C677T and A1298C polymorphisms has been correlated with reduced enzyme activity as well as impairments in folate pathways, and DNA methylation, and has also been related to ADHD and other psychiatric disorders [97].

2. CLOCK – Melatonin, Vitamin D:

Polymorphism of the CLOCK gene have been associated with ADHD risk and sleep–wake disturbance in adolescents, suggesting a possible role for circadian dysregulation in the ADHD pathophysiology [98].

3. DRD4 – Tyrosine, Iron:

The 7-repeat allele of the DRD4 gene has been associated with increased impulsivity in children with ADHD, as well as other extreme forms of behaviours, but in general, it does not impact core measures of attentional performance [99].

4. COMT - Magnesium, B12:

The Val158Met polymorphism in the COMT gene effects the breakdown of dopamine in the prefrontal cortex, which in turn affects executive functions, attention, and behavioural regulation related to ADHD [100].

5. SOD2 / GPX1 – Selenium, Zinc:

Meta-analyses show individuals diagnosed with ADHD are in a state of heightened oxidative stress and the genetic polymorphisms catalysing antioxidant enzymes (SOD2, GPX1, etc.) may exacerbate the imbalance in redox state and symptomatology severity [101].

6. CLOCK / PER3 – Melatonin, Vitamin D:

Circadian rhythm genes (CLOCK, PER3) variants were correlated with ADHD and coexistent sleep impairment suggesting that ADHD symptoms and sleep problems are routed in the same neurobiological pathways [102].

7. IL-6 / TNF-α – Omega-3s, Antioxidants:

Elevated levels of pro-inflammatory cytokines such as IL-6 and TNF-α were reported in ADHD subtypes providing further evidence that neuro inflammation and glial dysfunction is part of the pathways to ADHD [103].

Clinical Implications

This matrix informs the considerations for biochemical screenings and genotype-informed supplementation in children with ADHD. For instance, children with MTHFR C677T homozygosity may be treated with L-methylfolate instead of standard folic acid or children with low zinc and DAT1 genotype may also be most improved with zinc supplementation. Poor PUFA converters (FADS2 G allele) may require preformed DHA/EPA, not plant-based ALA. Such evidence supports the emerging model of precision nutrition in managing ADHD.

DISCUSSION

Expanding the ADHD Framework: Integrative and Trans diagnostic Perspective.The traditional framing of ADHD as a purely behavioural disorder is rapidly evolving into a multidimensional neurodevelopmental model. This section highlights broader theoretical and research-based shifts that further justify a systems-based, integrative approach.

ADHD as a Systems Disorder

There is mounting evidence that ADHD is not simply a dysfunction of prefrontal dopaminergic signalling, but engages systems involving neuro immune, neuroendocrine, and metabolic signalling networks. Higher levels of inflammatory cytokines, higher oxidative stress, and disruption to circadian cycles, interact with genetic predispositions and nutritional influences to construct a systems-level state of dysregulation [104,105].

Glycaemic Instability and Reward Dysregulation
Beyond classical neurotransmitter explanations for ADHD, emerging research includes blood sugar fluctuations as mediators of impulsivity and attentional fatigue. High-glycaemic dietary influences may introduce difficulty in front ostriatal regulation, contributing to reward-seeking behaviours and mood lability [106].

Nutritional Psychiatry in Youth
Extensive articles of review literature showing significant associations between the quality of diet and mental health in children and adolescents exist. Western dietary patterns—characterised by processed food, low micronutrient density, and food additives—are linked to increased risk of ADHD, depression, and anxiety [107,108].

Developmental Origins of ADHD
Prospective studies provide corpus of evidence supporting the hypothesis that early-life nutritional inadequacies (e.g. DHA, iron, iodine, folate) are detrimental to brain development influencing the risk of attention and emotional regulation problems. [109].

Psychological aspects
As far as "Anxiety or depression" and "Oppositional-Defiant, Conduct Disorder" are concerned, findings indicate a significant difference between the two behavioural groups of children with ADHD. The boys show more consistency in "Oppositional-Defiant & Conduct Disorder" and "Anxiety or depression," but the girls only show one of the two behavioural disorders. The greatest way to improve their children's expected outcomes is to provide them with school counselling and parental behaviour management therapy. These actions are necessary to address troublesome children in schools and households and help them get past their behavioural problems. [110]

Toward a Trans diagnostic Research Model
As we move toward a precision psychiatry framework, we can view ADHD as not an isolated disorder but rather intertwined with anxiety and autism and learning disability and mood disorders, all subject to genetic as well nutritional as environmental influences. This trans diagnostic approach promotes shared preventive interventions and comprehensive screening across various domains [111].

Attention-deficit/hyperactivity disorder (ADHD) is a multifaceted neurodevelopmental disorder, grounded in biological, genetic, environmental, and biochemical aetiology. While the biological exploration of ADHD is focused on dopamine dysregulation and genetic polymorphisms, new evidence highlights the potential for modifiable exposures—particularly nutrition, inflammation, and lifestyle—to influence the expression disability and symptom severity.

Modern dietary patterns, rich in ultra-processed foods and poor in essential micronutrients, create a biochemical environment that may amplify underlying genetic vulnerabilities. At the same time, behavioural contributors such as sleep disruption, excessive screen exposure, and high-glycaemic diets may generate pseudo-ADHD presentations that mimic core symptoms. India is facing the "double burden" of malnutrition and a shift in lifestyle, which warrants the immediate consideration of its effects from clinical and public health perspectives. The systematic application of precision nutrition in conjunction with developmental screening, monitoring systems, and family-based interventions along with existing health and education sectors may provide a suitable approach to ameliorate situations. This will require a shift of paradigm; rather than have ADHD as a reactive phenomenon concerning symptom management, we begin to adopt a proactive systems-based approach to prevention. ADHD can no longer be perceived merely as a fixed neurological disorder; instead, ADHD should be viewed as an ongoing condition that interacts with factors across the lifespan that are modifiable.

Conflict of Interest
Declared none

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Data availability statement
The data that supports the study's conclusions can be found in the journal [and/or] its additional sources, as the authors confirm.

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