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THE ROLE OF DIETARY AND SUPPLEMENTATION INTERVENTIONS IN ALLEVIATING ADHD SYMPTOMS

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ABSTRACT

Introduction: Attention-Deficit/Hyperactivity Disorder (ADHD) is a common neurodevelopmental disorder affecting both children and adults. Standard pharmacological treatment, while effective, carries the risk of adverse effects, prompting patients to seek complementary therapies. Modifiable factors, such as diet and supplementation, are increasingly recognized as significant elements regulating central nervous system functioning.
Objective: The aim of this review is to synthesize and critically analyze current scientific evidence regarding the impact of dietary interventions (elimination diets, dietary patterns) and supplementation (fatty acids, micronutrients, vitamins) on the severity of ADHD symptoms.
Methodology: A review of available scientific literature was conducted using PubMed, Scopus, and Google Scholar databases. The analysis included meta-analyses, systematic reviews, and randomized controlled trials (RCTs) published over the last 20 years, focusing on publications with high methodological rigor.
Results: The review indicates that the oligoantigenic diet demonstrates high efficacy in a specific subgroup of patients with food hypersensitivity, while the elimination of artificial food colors yields a small but significant effect in the general population. Unhealthy dietary patterns (Western diet) correlate with a higher risk of ADHD diagnosis. Regarding supplementation, the strongest evidence supports omega-3 fatty acids (with a predominance of EPA). Supplementation with iron, zinc, magnesium, and vitamin D is clinically effective primarily in cases of correcting confirmed deficiencies.
Conclusions: Endogenous processes regulating ADHD symptoms are modulated by a complex interaction of nutritional factors. Although diet and supplementation do not replace pharmacotherapy in severe cases, they constitute a valuable adjunctive (additive) strategy. A holistic approach, incorporating personalized nutrition and safety monitoring, should be considered an element of standard care for patients with ADHD.

KEYWORDS

ADHD, Diet, Supplementation, Omega-3, Zinc, Iron, Elimination Diet, Gut Microbiota, Mental Health

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1. Introduction

Attention-Deficit/Hyperactivity Disorder (ADHD) is one of the most common neurodevelopmental disorders, with a global prevalence in the child and adolescent population estimated at 5–7% [1]. The disorder persists into adulthood for a significant proportion of patients, affecting approximately 2.5–4% of the general adult population [1, 2]. According to current diagnostic classifications, ADHD is characterized by a persistent and developmentally inappropriate pattern of behavior comprising symptoms from three main domains: inattention (difficulty sustaining focus, losing items, distractibility), hyperactivity (excessive motor activity, inability to stay seated), and impulsivity (difficulty delaying gratification, making rash decisions) [2]. These symptoms lead to clinically significant functional impairments in academic, occupational, social, and emotional spheres, generating a substantial burden for patients, their families, and healthcare systems [2]. The standard therapeutic approach in ADHD is multimodal, primarily including pharmacotherapy and psychosocial interventions (e.g., cognitive-behavioral therapy, parent management training) [3]. First-line pharmacological therapy, based mainly on stimulants (methylphenidate and amphetamine derivatives) as well as non-stimulants (e.g., atomoxetine), demonstrates high efficacy in reducing key disorder symptoms. However, as indicated by a comprehensive network meta-analysis by Cortese et al. [3], despite proven efficacy, pharmacotherapy is associated with significant limitations. It is estimated that up to 30% of patients do not achieve an optimal response to treatment or experience clinically significant adverse effects, such as sleep disturbances (insomnia), decreased appetite and weight loss, headaches, or cardiovascular risks [3]. Furthermore, a growing number of patients and their caregivers express a preference for non-pharmacological methods or seek complementary

and alternative medicine (CAM) therapies that could safely support standard treatment [4]. These limitations constitute the primary motivation for intensive research into the role of lifestyle factors, including nutritional interventions, in managing ADHD symptoms. The etiology of ADHD is complex and multifactorial. Underlying it are strong genetic predispositions (heritability estimated at 70–80%), which interact with various environmental factors [5]. From a neurobiological perspective, ADHD is primarily associated with dysfunction in the neuronal networks of the prefrontal cortex and the pathways connecting it to subcortical structures. A key role is played by the dysregulation of neurotransmitter systems—mainly dopaminergic and noradrenergic [5, 6]. The link between diet and brain function is well documented, and several biological mechanisms directly connect nutritional status with the pathophysiology of ADHD. The synthesis of neurotransmitters such as dopamine and norepinephrine is a metabolic process requiring not only precursor amino acids (e.g., tyrosine) but also numerous enzymatic cofactors, including zinc, iron, magnesium, and B vitamins [7]. Scientific data suggest that deficiencies in these micronutrients, which may occur more frequently in children with ADHD, potentially disrupt optimal neurotransmission [7]. Equally important appears to be the role of polyunsaturated fatty acids (PUFA), especially omega-3 fatty acids: docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA). The brain is an organ exceptionally rich in lipids; DHA is a key structural component of neuronal membranes, influencing their fluidity and receptor function, while EPA modulates inflammatory processes. Numerous studies, including meta-analyses, indicate statistically significantly lower blood concentrations of omega-3 fatty acids in patients with ADHD compared to the healthy population, suggesting potential benefits from their supplementation [8]. Other hypotheses focus on the influence of diet on the gut-brain axis. The composition of the gut microbiota, heavily dependent on dietary habits, affects central nervous system functioning through immunological, neuronal, and metabolic pathways (e.g., production of short-chain fatty acids, SCFA) [9]. Gut dysbiosis and associated systemic inflammation are considered factors that may contribute to neuroinflammation underlying certain neurodevelopmental disorders [9]. Simultaneously, since the 1970s, the hypothesis of hypersensitivity to specific dietary components has been investigated. This particularly concerns artificial food colors (AFCs) and preservatives (e.g., sodium benzoate), which in genetically susceptible individuals may trigger or exacerbate behavioral symptoms [10]. Finally, the modern Western dietary pattern, characterized by high consumption of simple sugars and processed fats and low nutrient density, is linked to promoting inflammation, oxidative stress, and generally poorer cognitive functioning [11]. In the context of these theoretical premises, the growing interest in nutritional approaches to ADHD is fully justified. Diet is one of the most fundamental and modifiable lifestyle factors. For patients and their families, dietary and supplementation interventions offer the potential for safe and accessible support in symptom management, often perceived as a more "natural" approach [4]. However, a rigorous, evidence-based assessment of available research is essential to separate interventions with proven efficacy from those that remain hypothetical.

2. Dietary Interventions in ADHD

Interest in the influence of nutrition on the etiopathogenesis and clinical course of ADHD has not waned for decades, constituting one of the most dynamically developing areas of nutritional psychiatry. Modern science is decisively moving away from anecdotal reports in favor of rigorous clinical trials, attempting to understand how food modulates neuroplasticity and neurochemistry of the central nervous system. Literature analysis allows for the distinction of two main, complementary research streams: elimination diets, focused on removing potentially toxic or allergenic factors, and diets based on health-promoting dietary patterns, concentrating on optimizing brain metabolism by supplying necessary cofactors [10].

2.1. Restrictive and Elimination Diets

These approaches verify the hypothesis that specific dietary components—both synthetic additives and natural allergens—may induce neurobiological reactions (immunological or pharmacological) in susceptible individuals, manifesting as behavioral disturbances that mimic or exacerbate core ADHD symptoms.

2.1.1. Oligoantigenic Diets (Few-Foods Diets) – The Diagnostic "Gold Standard"

The oligoantigenic diet (also called the "few-foods diet") constitutes the most radical, yet most effective form of nutritional intervention in a selected group of patients. It is based on the assumption that in some children, behavioral symptoms are a manifestation of systemic food hypersensitivity, which does not necessarily proceed via the classic IgE-dependent allergy mechanism (therefore standard skin or blood tests often yield false-negative results and are not useful for qualification) [12]. This procedure is strictly medical and diagnostic, not prophylactic. It consists of two rigorous stages. The first is a strict elimination phase

(usually lasting 2–5 weeks), during which the patient consumes only a narrow group of foods with the lowest allergenic potential. In classic protocols, these are typically: rice, turkey or lamb meat, selected cruciferous vegetables (e.g., broccoli), pears, and water. All other products, including dairy, gluten-containing grains, citrus fruits, and food additives, are completely excluded. Key evidence for the efficacy of this approach is the breakthrough "INCA" study (Impact of Nutrition on Children with ADHD) published in *The Lancet* by Pelsser et al. (2011). This randomized controlled trial demonstrated that 64% of children subjected to a strict oligoantigenic diet experienced a spectacular reduction in ADHD symptoms (by at least 40% on the ARS scale). Significantly, the authors also noted that in children responding to the diet (so-called responders), concurrent somatic symptoms often resolved, such as abdominal pain, headaches, eczema, or sleep disturbances, suggesting the systemic nature of the hypersensitivity reaction [12]. Despite impressive results, great caution should be exercised in the interpretation and widespread implementation of this method. As indicated by a systematic review by Sonuga-Barke et al. (2013), the therapeutic effect of the oligoantigenic diet is much stronger when assessments are made by parents (unblinded study, susceptible to placebo effects and expectations) and weakens significantly—though remains statistically significant—when assessors are "blinded" observers (e.g., teachers or researchers analyzing coded video recordings) [16]. Furthermore, this diet is extremely burdensome for the patient and their family, leads to social isolation of the child (e.g., inability to eat at school), and carries the risk of serious micronutrient deficiencies. For this reason, experts such as Nigg and Holton (2014) recommend that the oligoantigenic diet should not be used routinely but treated as a specialized "second-line" diagnostic procedure, conducted exclusively under strict medical and dietetic supervision, in severe cases resistant to standard pharmacological treatment [10].

2.1.2. Elimination of Artificial Colors and Preservatives – Toxicology or Allergy?

The hypothesis linking artificial food colors (AFCs) with hyperactivity, popularized in the 1970s by Dr. Benjamin Feingold, sparked controversy for years, but modern research with high methodological rigor confirms its partial validity. A breakthrough in this field was the Southampton study (McCann et al., 2007), a randomized, double-blind, placebo-controlled trial on a large, representative group of children from the general population. Researchers tested two mixtures of additives (Mix A and Mix B), containing commonly used colors (including Quinoline Yellow, Allura Red, Tartrazine, Sunset Yellow) and a preservative (sodium benzoate). The results showed that consumption of these mixtures caused a measurable increase in hyperactivity and attention deficits in both 3-year-old and 8/9-year-old children [13]. It is important to emphasize a fundamental conclusion from this study: the influence of these substances on children's behavior was demonstrated *independently* of having a formal ADHD diagnosis. This suggests a general toxicological effect of these compounds on the developing nervous system, rather than a specific allergic reaction limited to a narrow group of patients. These results were systematized in a meta-analysis by Nigg et al. (2012), which confirmed that AFC elimination yields a small but statistically significant clinical improvement. Although for a single patient this effect may be subjectively imperceptible, on a population scale, it has significant public health importance, comparable to the impact of lead on children's IQ [14]. The mechanism of this phenomenon is the subject of intensive research. It is estimated that genetic sensitivity to dyes concerns about 8% of children with ADHD and may be linked to polymorphisms in genes responsible for histamine degradation (e.g., HNMT - histamine N-methyltransferase). In susceptible individuals, consumption of synthetic dyes may inhibit histamine-degrading enzymes, leading to its accumulation in the body. Histamine, acting as a neurotransmitter in the CNS, can lead to excessive psychomotor agitation and concentration disturbances in excess [17]. In light of these data, simple elimination of highly processed products containing these additives is a safe, inexpensive, and recommended preliminary intervention for all children with ADHD symptoms.

2.1.3. Sugar and Behavior – Analysis of Myths, Facts, and Metabolic Mechanisms

The widespread belief among parents and teachers that sugar (sucrose) causes a so-called "sugar rush" and directly, immediately triggers ADHD symptoms is one of the most persistent medical myths. However, this belief is not supported by high-quality experimental studies. A classic meta-analysis by Wolraich et al. (1995), published in *JAMA*, summarizing 16 double-blind RCTs, showed no statistically significant effect of sugar consumption on the behavior or cognitive functions of children, regardless of whether they had an ADHD diagnosis or not [18]. The correlation observed by parents is most often explained by the phenomenon of "illusory correlation" or the situational context of consuming sweets (birthdays, holidays, family gatherings), where children are excited for other reasons (emotions, lack of structure, noise). Nevertheless, the topic of sugar returns in newer scientific analyses in the context of long-term mechanisms and insulin metabolism, rather than immediate behavioral reactions. Johnson et al. (2011) point out that although sugar *per se* is not a direct cause of ADHD, its chronic, excessive consumption may worsen patient functioning through two key mechanisms. First, a diet high in simple sugars (high

glycemic load) leads to rapid fluctuations in blood glucose: rapid hyperglycemia followed by reactive hypoglycemia. The brain, especially the prefrontal cortex responsible for executive functions, is extremely sensitive to drops in glucose supply, which can result in irritability, decreased concentration, and weakened emotional control [19]. Second, chronic sugar abuse stimulates the dopaminergic system (reward system) in a manner resembling the mechanism of substance addiction. In children with dopamine deficits, which is a neurobiological feature of ADHD, this can lead to compulsive eating behaviors (binge eating) and deepening emotional instability via receptor neuroadaptation [19].

2.1.4. Gluten-Free and Casein-Free Diet (GFCF) – Hopes vs. Evidence

Diets excluding gluten (cereal protein) and casein (milk protein) have gained significant popularity, often being a direct extrapolation of recommendations used in Autism Spectrum Disorders (ASD). Proponents of this theory suggest the existence of a "leaky gut syndrome" in children with neurodevelopmental disorders, through which undigested peptides with opioid activity (gliadorphin from gluten and casomorphin from milk) enter the bloodstream. These compounds are purported to cross the blood-brain barrier and toxically affect CNS functioning. However, a comprehensive literature review by Millichap and Yee (2012) indicates a lack of sufficient clinical evidence justifying the routine use of the GFCF diet in the ADHD population [20]. Positive behavioral effects on a gluten-free diet are observed almost exclusively in patients with coexisting, diagnosed celiac disease or confirmed non-celiac gluten sensitivity (NCGS). It is worth noting that screening studies have not found a significantly increased prevalence of celiac disease in the ADHD population compared to the general population. Considering the high cost of a gluten-free diet, difficulties in balancing it (risk of fiber and B-vitamin deficiencies), and the risk of the child's social stigmatization, experts strongly advise against its empirical introduction without prior, reliable gastrointestinal and allergological diagnostics [20].

2.2. Dietary Patterns and Diet Quality

Modern nutritional psychiatry shifts the emphasis from eliminating single ingredients to promoting healthy dietary patterns. This approach assumes that for proper development and functioning, the brain needs a constant, balanced supply of a broad spectrum of nutrients, and their chronic deficiencies or excesses can permanently disrupt neuroplasticity and synaptogenesis.

2.2.1. The "Western" Diet as an Environmental Risk Factor

Numerous observational and epidemiological studies involving thousands of participants indicate a significant negative correlation between the "Western" diet and the severity of ADHD symptoms. This diet, characterized by high consumption of ultra-processed foods, saturated and trans fats, refined sugars, and low content of fiber, vitamins, and phytochemicals, is considered a strong factor promoting metabolic dysregulation. A study by Howard et al. (2011) on a large cohort of Australian adolescents showed that this dietary pattern was strongly associated with an ADHD diagnosis, even after adjusting for many confounding variables, such as parental socioeconomic status or education [11]. These conclusions were confirmed in an extensive systematic review and meta-analysis published in the *Journal of Affective Disorders* by Del-Ponte et al. (2019). The authors analyzed 14 studies on dietary patterns and demonstrated a consistent link between an "unhealthy" dietary profile (high in refined sugars, saturated fats) and an increased risk of ADHD occurrence, simultaneously indicating the protective nature of a "healthy" diet [21]. The mechanism of this relationship is complex and likely bidirectional. On the one hand, a micronutrient-poor diet does not provide necessary cofactors for the synthesis of key neurotransmitters, and pro-inflammatory components may damage delicate structures of the prefrontal cortex. On the other hand, impulsivity inherent in the clinical picture of ADHD favors making poorer, impulsive dietary choices, closing the metabolic vicious cycle [10, 11].

2.2.2. Neuroprotective Potential of the Mediterranean Diet

As a counterweight to the destructive Western model, scientific literature increasingly points to the Mediterranean Diet (MedDiet) as an optimal nutritional model supporting the mental health of children and adults. A cross-sectional study by Ríos-Hernández et al. (2017) conducted in Spain showed that children with low adherence to the Mediterranean diet (KIDMED index) had a significantly higher risk of ADHD diagnosis, and this relationship was independent of other environmental factors [15]. The protective effect of this diet results from the synergy of its elements. The richness of vegetables, fruits, and olive oil provides potent antioxidants and polyphenols that reduce oxidative stress in the brain. Marine fish, nuts, and seeds are sources of essential unsaturated fatty acids (especially omega-3), crucial for neuronal membrane fluidity and signal transmission speed. Furthermore, the high content of dietary fiber from whole grains and legumes modulates the gut microbiota. A healthy microbiome stimulates the production of short-chain fatty acids (SCFA), which cross the blood-brain barrier and exhibit anti-inflammatory and neurotrophic effects (stimulating BDNF factor) [9]. The safety and general health benefits of the Mediterranean diet make it a recommended baseline intervention for all patients with ADHD.

3. Supplementation and Nutraceuticals in ADHD

Despite the high efficacy of pharmacotherapy, in some patients, the response to treatment is insufficient or associated with the occurrence of bothersome adverse effects. Consequently, dietary supplementation has become the most commonly used complementary intervention in ADHD. However, literature analysis indicates that rational supplementation should not be based on the random administration of multivitamins, but on targeted, precise correction of specific metabolic deficits that may disrupt neuroplasticity, myelination, and neurotransmitter synthesis.

3.1. Polyunsaturated Fatty Acids (PUFA) – Omega-3 and Omega-6

The best-documented and most widely studied nutraceutical intervention in ADHD is supplementation with long-chain polyunsaturated fatty acids (LC-PUFA), specifically omega-3 fatty acids: eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA).

3.1.1. Mechanism of Action and Clinical Evidence

The theoretical basis for using omega-3 fatty acids stems from their fundamental role in the structure and functioning of the central nervous system. DHA constitutes a key building block of neuronal membrane phospholipids (especially in synapses), determining their fluidity, permeability, and the functionality of receptors and ion channels. EPA plays a primary role as a regulator of inflammatory processes, inhibiting the production of pro-inflammatory cytokines that can negatively affect neurogenesis and synaptic plasticity [22, 23]. A landmark meta-analysis by Bloch and Qawasmi (2011), published in the prestigious *Journal of the American Academy of Child & Adolescent Psychiatry*, covering 10 randomized controlled trials, showed that omega-3 supplementation yields a small but statistically significant and replicable improvement in ADHD symptoms. Although this effect is smaller than that of stimulant medications, it is greater than that of other natural therapies, making omega-3 the most important element of nutritional support [22].

3.1.2. Importance of Dose and EPA:DHA Ratio

A key conclusion from newer analyses is the fact that not every omega-3 preparation is effective. A meta-analysis by Chang et al. (2018), published in *Neuropsychopharmacology*, shed new light on the composition of preparations. It was shown that clinical improvement is observed exclusively in studies using high doses of EPA. The authors identified that preparations with an EPA:DHA ratio higher than 1.5:1 (with a clear predominance of EPA) were effective in reducing symptoms, while those with a predominance of DHA did not bring significant behavioral benefits [23]. This suggests that the anti-inflammatory and modulating action of EPA may be more important in short-term symptom reduction than the structural role of DHA. Derbyshire (2017) in her review suggests a minimum effective daily dose of 500 to 1000 mg of combined EPA and DHA, while maintaining appropriate proportions [24].

3.2. Key Micronutrients: Iron, Zinc, Magnesium

Micronutrients function as essential enzymatic cofactors in the synthesis pathways of monoamine neurotransmitters (dopamine, norepinephrine, serotonin). Their deficiencies, often observed in children with ADHD, may constitute a reversible cause of cognitive dysfunction.

3.2.1. Iron and Ferritin

Iron is a key cofactor for tyrosine hydroxylase—the rate-limiting enzyme in dopamine synthesis from tyrosine. Cortese et al. (2012) showed in a systematic review that children with ADHD have significantly lower levels of serum ferritin (iron storage protein) compared to controls, even in the absence of overt anemia [25]. A comprehensive meta-analysis by Villagomez and Ramtekkar (2014) confirmed a strong correlation between low ferritin levels and ADHD symptom severity. Intervention studies indicate that iron supplementation (e.g., 80 mg/day) in children with low ferritin levels (<30 ng/mL) leads to significant improvement in ADHD symptom scales (Conners' Rating Scale) compared to placebo. However, it must be emphasized that iron supplementation in children with normal stores of this element provides no benefit and may be toxic (risk of hemochromatosis); therefore, this intervention strictly requires prior determination of ferritin levels [26, 39].

3.2.2. Zinc

Zinc is a modulator of NMDA receptors and regulates the activity of the dopamine transporter (DAT), influencing dopamine availability in the synaptic cleft. The meta-analysis by Villagomez and Ramtekkar (2014) showed that serum zinc levels in children with ADHD are significantly lower compared to healthy peers, suggesting the prevalence of this deficiency in this patient group [26]. Clinical trials, such as the double-blind study conducted by Arnold et al. (2011), suggest that zinc supplementation (e.g., 15–30 mg/day) may reduce symptoms of hyperactivity and impulsivity. Clinically significant is the finding that zinc addition may potentiate the effect of methylphenidate, allowing for the use of lower drug doses while maintaining therapeutic efficacy [38].

3.2.3. Magnesium

Magnesium acts as a physiological calcium antagonist and NMDA receptor channel blocker, translating into a calming and neuroprotective effect. Magnesium deficiency manifests as neuromuscular hyperexcitability, anxiety, tics, and sleep disturbances. A review by Effatpanah et al. (2019) indicates that magnesium supplementation (often combined with vitamin B6 for better cellular absorption) leads to a reduction in hyperactivity in children with confirmed deficiency of this element (hypomagnesemia), although evidence for populations with normal magnesium levels is weaker [27].

3.3. Neuroprotective Vitamins (Vitamin D and B Group)

3.3.1. Vitamin D3

Vitamin D is currently recognized as a neurosteroid influencing the expression of neurotrophic factors (NGF, GDNF) and protecting dopaminergic neurons against oxidative stress. A meta-analysis by Khoshbakht et al. (2018) showed that children with ADHD have significantly lower serum 25(OH)D levels compared to the healthy population [28]. Preliminary intervention studies (RCTs) suggest that normalizing vitamin D levels (supplementation of around 2000 IU/day) may support pharmacological treatment, improving executive functions and reducing anxiety symptoms [28].

3.3.2. B Vitamins

Vitamins B6 (pyridoxine), B12 (cobalamin), and folic acid are crucial in the one-carbon cycle (methylation), essential for serotonin and dopamine production. A study by Landaas et al. (2016) on a large group of adults with ADHD showed a correlation between low levels of B vitamins and symptom severity. However, evidence for the efficacy of high-dose B-complex supplementation (beyond correcting evident deficiencies) remains inconclusive and requires further large-scale clinical trials [29].

3.4. Gut-Brain Axis: Probiotics

The microbiota-gut-brain axis is a new, extremely promising direction of research in psychiatry. Gut dysbiosis may affect the CNS through the production of bacterial metabolites (neurotransmitters), pro-inflammatory cytokines, and vagus nerve modulation. A breakthrough prospective study by Pärtty et al. (2015) showed that early administration of the probiotic *Lactobacillus rhamnosus GG* in the first 6 months of life reduced the risk of developing ADHD and autism spectrum disorders in later childhood [30]. A systematic review by Bundgaard-Nielsen et al. (2020) suggests that although evidence for treating already diagnosed ADHD with probiotics is still preliminary, these interventions are safe and may improve patients' general health and alleviate coexisting symptoms (e.g., gastrointestinal complaints) [31].

3.5. Other Compounds: Plant Extracts and Melatonin

3.5.1. Pycnogenol

Pycnogenol, a standardized extract from French maritime pine bark (*Pinus pinaster*), rich in bioflavonoids, is studied for its strong antioxidant properties and ability to increase nitric oxide (NO) levels, which may modulate cerebral blood flow (perfusion). A randomized study by Trebatická et al. (2006) showed that Pycnogenol supplementation (1 mg/kg body weight) for one month led to a significant reduction in hyperactivity and improvement in concentration compared to placebo, with a simultaneous decrease in urinary oxidative stress markers (8-OHdG) [32].

3.5.2. Melatonin

Although melatonin does not directly treat core daytime ADHD symptoms, it plays a key role in managing sleep disorders (especially delayed sleep phase), which affect up to 70% of patients with this diagnosis. Hollway and Aman (2011) confirmed in their meta-analysis that melatonin effectively and safely shortens sleep latency (time to fall asleep) in children with ADHD. Improvement in sleep quality and duration secondarily translates into better cognitive functioning, reduced irritability, and better concentration at school the next day [33].

4. Safety, Interactions, and Clinical Aspects

Introducing dietary and supplementation interventions into an ADHD treatment plan requires not only knowledge of their potential benefits but primarily an awareness of risks, drug interactions, and safety principles.

4.1. Supplement-Drug Interactions

One of the most clinically significant aspects, often overlooked by patients, is the interaction of vitamin C (ascorbic acid) and other urinary acidifying agents with stimulant medications (amphetamine derivatives). As indicated by Hodgkins et al. (2012) in a review of amphetamine pharmacokinetics published in *Mayo Clinic Proceedings*, renal elimination of these drugs is strongly dependent on urinary pH. Amphetamine is a weak base; in an acidic environment, it exists in an ionized form, which drastically limits its reabsorption in renal tubules. Acidification of urine (e.g., by high doses of vitamin C or citrus juices consumed around the time of drug administration) significantly accelerates drug excretion, shortening its half-life and weakening the therapeutic effect [34]. For this reason, avoiding high doses of vitamin C one hour before and after taking stimulant medication is recommended.

4.2. Safety and Toxicity of Supplementation

Although supplements are often perceived by parents as "completely safe and natural," they are not free from adverse effects and toxicity risks. **Omega-3 Fatty Acids:** Generally considered safe, but in high doses may cause gastrointestinal discomfort (belching, fishy aftertaste, loose stools). A significant problem is preparation quality—fish oils are susceptible to oxidation (rancidity), rendering them worthless or harmful. Sarris et al. (2011) emphasize the necessity of using certified products free from heavy metals (mercury, lead) and PCBs, which can accumulate in fish fat [35]. **Zinc and Copper:** Long-term supplementation with high doses of zinc (>40–50 mg/day) may competitively inhibit copper absorption in the intestines, leading to iatrogenic copper deficiency, microcytic anemia, and neutropenia. Therefore, during long-term zinc therapy, monitoring copper levels or concurrent, balanced supplementation (in a ratio of e.g., 15:1) is recommended [36]. **Fat-Soluble Vitamins:** Unlike B vitamins, which are excreted in urine, vitamins A, D, E, and K accumulate in adipose tissue and the liver. Vitamin D supplementation should always be monitored by blood 25(OH)D levels to avoid hypercalcemia and renal toxicity [28].

4.3. Clinical Implications and Safety Summary

A systematic review by Cochrane (Gillies et al., 2012) concludes that although fatty acids and certain minerals show promising effects, evidence for their efficacy is not strong enough to replace standard pharmacotherapy [37]. Lange et al. (2023), in their latest review, propose an "additive therapy" model. In this model, diet and supplementation constitute the foundation (providing "fuel" for the brain) supporting pharmacological treatment, allowing in some cases for a reduction in stimulant doses and minimization of their side effects [40]. Crucial for patient safety is that every supplementation intervention be consulted with the attending physician. The "natural" origin of substances does not exempt one from maintaining dosage rigor, monitoring biochemical parameters, and considering potential interactions with prescribed medications.

5. Discussion

5.1. Synthesis of Evidence and Hierarchy of Efficacy

A review of available literature allows for the formulation of a hierarchy of nutritional interventions based on the strength of scientific evidence (Evidence-Based Medicine). The strongest support in meta-analytic studies is held by omega-3 supplementation (specifically EPA) [22, 23] and the oligoantigenic diet, though the latter is limited to a specific subgroup of patients with food hypersensitivity [12, 16]. In the case of micronutrients (iron, zinc, vitamin D), evidence unequivocally points to their therapeutic efficacy *exclusively* in situations of laboratory-confirmed deficiency [26, 40]. Interventions involving sugar or dye elimination, although popular, demonstrate a small clinical effect in the general ADHD population, having mainly prophylactic significance [14, 18].

5.2. Heterogeneity of Response and Personalization

A key conclusion drawn from the analyzed works is the observation of enormous heterogeneity in patient response. ADHD is not a homogeneous disorder but a spectrum with diverse genetic and environmental etiologies. As suggested by Lange et al. (2023), a "universal ADHD diet" does not exist [40]. Intervention efficacy depends on the patient's individual metabolic profile (e.g., polymorphisms in fatty acid metabolism genes FADS1/2 or histamine genes HNMT) [17]. This shifts the treatment paradigm towards precision psychiatry, where supplement selection is preceded by biomarker diagnostics.

5.3. Biological Mechanisms

The common denominator for the discussed interventions appears to be the reduction of systemic inflammation and oxidative stress, as well as the optimization of neurotransmitter synthesis. Both the Mediterranean diet and omega-3 fatty acids or zinc target neuroimmunological and neurotrophic pathways (BDNF) that are disrupted in ADHD [21, 23]. This confirms the hypothesis that ADHD may have an inflammatory or metabolic component in a portion of patients.

5.4. Limitations and Practical Implications

Despite promising results, many studies are burdened with methodological errors: small sample sizes, short observation periods, or difficulties in blinding dietary trials. For clinicians, the implication is to treat diet and supplementation as adjunctive therapy. It does not replace pharmacotherapy in moderate to severe cases, but may allow for drug dose reduction, decreased side effects, and improvement in the patient's overall functioning [37, 40].

6. Conclusions

1. **Role of Diet:** Healthy dietary patterns (e.g., the Mediterranean diet) and the elimination of highly processed foods rich in artificial colors constitute the foundation of supportive management in ADHD, beneficially affecting general health and potentially alleviating behavioral symptoms.
2. **Targeted Supplementation:** Supplementation with omega-3 fatty acids (with high EPA content) demonstrates consistent, though moderate efficacy in reducing ADHD symptoms. Supplementation with micronutrients (iron, zinc, magnesium, vitamin D) is clinically justified and effective only in cases of correcting confirmed deficiencies.
3. **Safety:** Nutritional interventions are generally safe but require awareness of potential interactions (e.g., vitamin C vs. stimulant drugs) and monitoring for toxicity during long-term use.
4. **Integrated Approach:** Patients derive the greatest benefit from a multimodal model combining standard medical and psychological care with lifestyle and nutritional optimization.

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