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Ketogenic Therapies and ADHD in Children: A Narrative Review of Possible Mechanisms, Indirect Evidence, and Research Gaps

Abubkr Ahmed, Mohamed Taha, Rogaya Idris, Rahma M. Abd El-Aziz

Independent Research, Navan, Ireland
Email: drmohamed.mustafamohamed@gmail.com

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Abstract

Attention-deficit/hyperactivity disorder (ADHD) is a common neurodevelopmental disorder affecting children and adults. Recently, there has been growing interest in the potential role of diet in the management of ADHD symptoms. This narrative-based literature review describes the biological plausibility of the ketogenic diet (KD), examines studies looking at the role of the KD in the management of ADHD symptoms, and identifies key areas for future research. No randomized controlled trials (RCTs) were found that investigated children with a primary ADHD diagnosis who underwent verified nutritional ketosis. Current literature in this area derives from three primary sources—dietary pattern studies, observations in existing pediatric epilepsy cohorts, and animal studies—yet such research may not necessarily confirm whether subjects entered ketosis and often relies on activity-based outcome measures. To properly establish the potential function of the KD in the care of children with ADHD, scientific validation through carefully designed controlled trials conducted within strict ethical frameworks is essential.

Keywords

Ketogenic Diet, ADHD, Hyperactivity Disorder

1. Attention-Deficit/Hyperactivity Disorder (ADHD)

1.1. Definition

Attention-deficit/hyperactivity disorder (ADHD) is among the most prevalent neurodevelopmental disorders of childhood. Global prevalence estimates range from 3% - 6% [1], with approximately 5% of children and adolescents and 2.5% of adults affected worldwide [2]. Although reported prevalence varies across re-

gions, these differences are widely attributed to heterogeneity in diagnostic criteria, assessment methodologies, and study design rather than to true geographic variation in disease burden. Clinically, ADHD is characterized by persistent patterns of inattention, hyperactivity, and impulsivity. Marked interindividual variability in symptom expression and treatment response reflects the disorder's complex and multifactorial etiology, which encompasses genetic susceptibility, environmental exposures, and neurobiological mechanisms [1].

Longitudinal evidence indicates that ADHD frequently persists beyond childhood, with approximately 60% - 70% of affected children continuing to exhibit symptoms during adolescence and nearly 50% experiencing clinically significant symptoms into adulthood [3]. Symptom persistence is associated with substantial adverse effects on academic attainment, occupational functioning, and interpersonal relationships. Despite increasing recognition, adult ADHD remains underdiagnosed in many settings, partly due to limited access to specialized assessment services and variability in clinician training and care pathways [1].

At the neurobiological level, ADHD has been linked to dysregulation of key neurotransmitter systems, particularly dopaminergic (DA), serotonergic (5-HT), and norepinephrinergic (NE) pathways, which play central roles in attention, impulse control, and executive functioning [4]-[6].

1.2. Pharmacological Management and Limitations

Owing to their robust short-term efficacy, stimulant medications remain the first-line pharmacological treatment for ADHD. Amphetamine-based formulations demonstrate high effectiveness in adults, whereas comparative evidence suggests that methylphenidate (MPH) is generally the preferred first-line agent for children and adolescents. Treatment selection is typically guided by clinical practice guidelines that integrate patient age, comorbid conditions, and risk of adverse effects [7] [8]. Non-stimulant agents, including atomoxetine and extended-release viloxazine, offer alternative therapeutic options when stimulants are ineffective, poorly tolerated, or contraindicated, although their use is often limited by delayed onset of action and distinct adverse-effect profiles [9]. Advances in genetic and neurobiological research have further informed pharmacogenetic frameworks by linking ADHD pathophysiology to dopaminergic, noradrenergic, and serotonergic signaling pathways.

Stimulant medications and certain non-stimulant agents are associated with modest increases in heart rate and blood pressure. While serious cardiovascular events are uncommon in appropriately screened individuals, comprehensive baseline evaluation and ongoing monitoring remain standard components of clinical care [8] [10]. Rare but clinically meaningful psychiatric adverse effects, including psychotic or manic symptoms, have also been reported, particularly among individuals with underlying vulnerability or at higher medication doses [11].

Given the substantial functional burden of ADHD and its far-reaching healthcare, educational, and societal costs, there is sustained interest in adjunctive and non-

pharmacological treatment strategies. Economic analyses conducted in European contexts demonstrate that ADHD is associated with considerable annual expenditures, with pharmacotherapy accounting for only one component of both direct and total costs [11] [12].

In addition to pharmacological approaches, ADHD management commonly incorporates non-pharmacological interventions such as behavioral parent training, school-based accommodations, and selected psychological therapies [13] [14]. Evidence suggests that behavioral interventions combined with stimulant medication yield greater therapeutic benefit than pharmacological or behavioral treatment alone [13]. Dietary strategies have also received growing attention, with interventions primarily focusing on restrictive or elimination diets, microbiome-targeted approaches involving pre-, pro-, and synbiotic supplementation, and the use of dietary supplements containing vitamins, minerals, and polyunsaturated fatty acids (PUFAs).

1.3. Evidence-Based Dietary Interventions in ADHD

A clear understanding of the current evidence supporting established dietary interventions in ADHD is essential for contextualizing emerging metabolic approaches.

1.4. The DASH Diet

Less severe ADHD symptomatology has been associated with adherence to the Dietary Approaches to Stop Hypertension (DASH) diet, which emphasizes high intake of fruits, vegetables, and low-fat dairy products while limiting simple sugars. In a 12-week randomized controlled trial (RCT), multiple behavioral outcome measures, including the Swanson, Nolan, and Pelham-IV Questionnaire (SNAP-IV) and the Abbreviated Conners Scale (ACS), demonstrated statistically significant improvements among participants following the DASH dietary pattern [15]. Despite these encouraging findings, the reported clinical effects remain preliminary. Replication in larger cohorts, incorporation of blinded outcome assessments, and longer follow-up durations are necessary before definitive clinical recommendations can be established [16]-[18].

1.5. Elimination and "Few-Foods" Diets

Elimination diets represent one of the most extensively investigated nutritional interventions in ADHD management [19] [20]. These protocols typically employ a structured, multiphase design. First, a defined set of potential dietary triggers is removed under professional dietary supervision during a brief and highly controlled elimination phase [21]. This is followed by a systematic reintroduction phase aimed at identifying specific symptom-provoking foods, ideally using predefined outcome criteria and, when feasible, blinded evaluation procedures [21] [22].

Within this framework, the INCA study identified a subgroup of "clinical re-

sponders," defined by improvements exceeding 40% on the ADHD Rating Scale (ARS). In addition, adherence to the Few-Foods Diet (FFD) has been associated with reductions in comorbid somatic complaints, including gastrointestinal symptoms and sleep disturbances [23]. Meta-analytic evidence further supports these findings, reporting a large effect size of 0.80 for parental assessments of ADHD symptoms following restrictive dietary interventions [20].

However, the demanding nature of both elimination and reintroduction phases, particularly in protocols such as the oligoantigenic diet, requires substantial parental involvement and close professional supervision [24]. Symptom recurrence during food reintroduction is common among responder subgroups, underscoring considerable interindividual heterogeneity and highlighting the challenges associated with sustaining highly restrictive dietary regimens over extended periods.

Although elimination-based dietary approaches have provided valuable insights into nutrition-related symptom modulation in ADHD, their methodological heterogeneity and practical limitations emphasize the need to explore alternative metabolic strategies. Ketogenic therapies, initially developed for pediatric epilepsy, provide a biologically grounded framework for investigating whether sustained alterations in cerebral energy metabolism and associated signaling pathways may influence ADHD-related phenotypes [25]. Accordingly, this review focuses on mechanistic plausibility, indirect evidence, and existing research gaps, without interpreting the current literature as evidence of clinical efficacy for ketogenic therapy in primary ADHD.

2. Methods

2.1. Data Acquisition

This review was conducted as a structured narrative review employing transparent literature identification and screening procedures. Systematic searches were performed in PubMed and ScienceDirect using predefined search terms. Titles and abstracts were initially screened for relevance, followed by targeted full-text evaluation of records meeting eligibility criteria. An extended Boolean search strategy was developed by combining terms related to the target condition ("ADHD," "Attention-Deficit/Hyperactivity Disorder," "Hyperactivity") with intervention-related terms ("Ketogenic Diet," "High-Fat Diet," "MCT").

Publications released between January 2000 and January 2026 were considered, encompassing both foundational studies and more recent clinical investigations. Inclusion criteria were predefined to capture: [1] studies evaluating structured ketogenic therapy, defined by explicit macronutrient prescription and/or confirmed biochemical ketosis, particularly β-hydroxybutyrate (BHB); and [2] both pediatric clinical studies and preclinical investigations employing validated animal models, such as the Spontaneously Hypertensive Rat (SHR). Non-research publications, including editorials and correspondence lacking sufficient methodological detail, were excluded to maintain scientific rigor.

2.2. Study Selection and Data Extraction

The literature search was updated in January 2026. A comprehensive search across PubMed and ScienceDirect initially yielded 385 records. After removing duplicates and conducting a preliminary screening of titles and abstracts, the most relevant full-text articles were evaluated for eligibility. Ultimately, 105 key sources—including clinical trials, observational studies, and mechanistic reviews—were selected for inclusion based on their direct relevance to the neurobiological and metabolic aspects of ADHD. This selection ensures a robust evidence base for the current synthesis while acknowledging the inherent qualitative nature of this narrative review.

Remaining studies were screened to assess relevance to the neurobiological and behavioral dimensions of ADHD. Full-text review was subsequently undertaken to confirm that eligible studies addressed mechanistic pathways of interest, including the microbiota-gut-brain axis and modulation of neurotransmitter systems (DA, NE, 5-HT). Due to substantial heterogeneity in outcome measures and dietary protocols, data were synthesized qualitatively rather than subjected to quantitative aggregation.

2.3. Evidence Hierarchy in This Review

To preserve interpretive accuracy and avoid overstating the strength of existing findings, the evidence reviewed was categorized according to the following hierarchy:

Direct clinical evidence: Studies involving children with primary ADHD undergoing confirmed nutritional ketosis; no published RCTs were identified.

Indirect clinical evidence: Research examining dietary modifications, including low-glycemic index, Mediterranean, and elimination diets, in ADHD populations (available; variable methodological quality).

Cross-population evidence: Behavioral outcomes associated with ketogenic therapy in pediatric epilepsy cohorts (indirect; potentially confounded by seizure control and related factors).

Mechanistic animal evidence: Studies employing ADHD-relevant animal models, such as the SHR, intended to generate mechanistic hypotheses rather than establish clinical efficacy.

General preclinical data: Activity-based studies conducted in neurotypical (non-ADHD) rodent models, representing the lowest level of translational relevance.

2.4. Operational Definitions

In this review, the operational criterion for "verified ketogenic therapy" requires structured dietary protocols intended to induce sustained nutritional ketosis, specifically confirmed by circulating ketone concentrations (e.g., blood BHB levels ≥ 0.5 mmol/L) [26]. Accordingly, studies described as "ketogenic" that lack this biochemical validation are classified as "ketogenic-format" interventions. This distinction ensures that the metabolic effects of true physiological ketosis are not

conflated with those of a general low-carbohydrate diet.

3. General Dietary Patterns and ADHD

Diet as an Alterable Environmental Factor

Studies examining overall dietary patterns in relation to ADHD risk and symptom severity have yielded mixed results, largely reflecting heterogeneity in study design, control of confounding variables, and outcome assessment methods [11].

Rather than focusing on isolated foods or individual nutrients, dietary pattern analysis offers a more integrative framework for understanding how habitual dietary exposures collectively influence neurodevelopmental outcomes. Broader societal shifts have contributed to a global nutrition transition, characterized by reduced time for home meal preparation and shared family meals, with corresponding changes in eating behaviors and food choices [27]. These shifts have been accompanied by increased consumption of processed and fast foods, edible oils, and sugar-sweetened beverages, trends that have been associated with worsening health outcomes across populations [11].

Evidence categorizing dietary patterns as "healthy" or "unhealthy" further supports these associations. Meta-analytic data indicate that unhealthy dietary patterns are associated with higher odds of ADHD, independent of study methodology or geographic region [28]. In parallel, children with ADHD consistently demonstrate significantly lower adherence to healthy dietary patterns compared with neurotypical peers [28]. For example, a Spanish case-control study reported a strong association between ADHD diagnosis and poor adherence to the Mediterranean diet (OR: 7.07; 95% CI: 2.65 - 18.84) [29]. Similarly, a large population-based survey of 14,912 children in China found that "Processed" and "Snack" dietary patterns were associated with higher odds of ADHD symptoms (OR: 1.56 and 1.76, respectively), whereas a "Vegetarian" dietary pattern was associated with lower odds of symptom presence (OR: 0.67) [30].

Artificial food colorings (AFCs), whose consumption has increased substantially over the past five decades, represent a notable component of unhealthy dietary patterns. In a double-blind, placebo-controlled challenge study, AFC exposure was shown to alter brainwave activity and exacerbate ADHD symptoms [31]. However, a broader meta-analysis estimated a modest overall effect size of 0.283 for AFCs on hyperactivity, which was attenuated to 0.210 after exclusion of lower-quality trials [11] [32].

A comprehensive systematic review and meta-analysis further underscore the relationship between dietary patterns and ADHD vulnerability. Adherence to a "Healthy" dietary pattern rich in fruits, vegetables, seafood, and micronutrients such as zinc and magnesium was associated with lower odds of ADHD (OR: 0.63; 95% CI: 0.41 - 0.96) [33]. In contrast, "Western" and "Junk Food" dietary patterns were associated with substantially increased risk. Specifically, junk food consumption was linked to higher odds of ADHD (OR: 1.51; p: 0.024), while the Western dietary pattern, characterized by high intake of processed meats and hydrogenated

fats, was associated with higher odds of ADHD (OR: 1.92; p: 0.016) [33].

Despite these findings, much of the existing literature has largely overlooked the independent and interactive effects of macronutrient composition, nutrient-nutrient interactions, and broader socioeconomic determinants of dietary exposure, instead emphasizing specific foods or named dietary patterns in the context of ADHD management. Moreover, many studies rely on relatively small cohorts, limiting generalizability. The potential protective effects of broader food environments and population-level dietary exposures remain insufficiently characterized. Given the growing global prevalence and impact of ADHD, addressing these gaps through population-level and cross-cultural research may provide valuable insights for both prevention strategies and therapeutic development.

4. High-Fat Dietary Interventions and the Ketogenic Context

4.1. The Bridge between Low-Carbohydrate/High-Fat Diets and ADHD Symptoms

Human Observational Evidence

Despite growing evidence linking overall dietary patterns to neurodevelopmental health, clinical studies directly evaluating the therapeutic potential of a structured ketogenic diet (KD) in children with ADHD are notably lacking. Most dietary research in ADHD has emphasized overall diet quality or targeted exclusions, such as refined sugars or food additives, rather than interventions specifically designed to induce and sustain nutritional ketosis [34] [35]. Consequently, whether a ketosis-driven metabolic state meaningfully influences core ADHD symptom domains remains an unresolved clinical question.

In this review, the term ketogenic therapies refers to structured dietary protocols intended to induce sustained nutritional ketosis, ideally confirmed through measurement of circulating ketone concentrations, such as BHB. These protocols include the classic KD, typically prescribed using fat-to-protein-plus-carbohydrate ratios of 3:1 or 4:1, medium-chain triglyceride (MCT)-based variants, and modified ketogenic approaches commonly applied in pediatric neurology [36]. In contrast, community-adopted "keto" diets used primarily for weight management often rely on less stringent macronutrient targets and do not reliably achieve sustained ketosis. Accordingly, studies described as "ketogenic" in the absence of biochemical confirmation are classified here as ketogenic-format interventions rather than verified ketogenic therapy [37].

The KD is characterized as a high-fat, adequate-protein, and low-carbohydrate dietary regimen, typically restricting carbohydrate intake to approximately 5% - 10% of total daily energy intake, with protein and fat contributing roughly 10% - 30% and 60% - 80% of total caloric intake, respectively [38]. The classic KD, which has been used clinically in epilepsy since 1921, is distinguished by an even higher fat content and further restriction of protein and carbohydrates [39] [40]. The primary objective of ketogenic therapy is to induce a metabolic state in which glucose derived from dietary carbohydrates is largely replaced by fatty acids and ke-

tone bodies as the principal energy substrates [41]. Reduced carbohydrate intake leads to lower circulating glucose levels, resulting in decreased insulin secretion and increased glucagon release, thereby promoting lipolysis, defined as the breakdown of triglycerides into free fatty acids. Through hepatic β-oxidation, these free fatty acids are converted to acetyl-CoA. When oxaloacetate availability is limited, excess acetyl-CoA is diverted toward ketogenesis, producing ketone bodies, including acetone, acetoacetate, and BHB, the predominant circulating ketone. Under these conditions, ketone bodies replace glucose as a major alternative energy source for individuals adhering to a KD [42]-[46].

In neurological disorders, particularly epilepsy, ketogenic therapy has been associated with changes in mitochondrial function and neuronal excitability in both clinical and experimental models; however, its relevance to ADHD pathophysiology remains uncertain [47].

Additional support for a molecular link between metabolic status and ADHD symptomatology comes from studies examining micronutrient deficiencies. Alterations in iron and zinc status have been associated with greater symptom severity in some cohorts, underscoring the clinical relevance of micronutrient assessment, particularly in children consuming restrictive diets [48] [49]. At the same time, poorly planned ketogenic interventions may increase the risk of micronutrient insufficiency, highlighting the necessity of dietetic supervision and proactive nutritional monitoring in any proposed metabolic therapy [50].

Despite advances in understanding the biological underpinnings of neurodevelopmental disorders, these insights have not yet translated into novel, widely effective therapeutic strategies. In established clinical contexts such as drug-resistant epilepsy, ketogenic therapies can be implemented safely under specialist dietary supervision with systematic monitoring. By contrast, the long-term safety profile and overall risk-benefit balance of ketogenic therapy in children with primary ADHD remain unknown and cannot be inferred directly from epilepsy populations.

4.2. Metabolic Links: Glucose Oscillations and ADHD

The potential relevance of macronutrient redistribution in ADHD management is illustrated by recent dietary intervention studies. In a 5-week structured program involving 47 newly diagnosed children aged 6 - 9 years with ADHD, significant improvements were observed across all subscales of the Conners' Parent Rating Scale (CPR-RS), including hyperactivity, impulsivity, and learning difficulties [51]. Importantly, this intervention did not employ a strict ketogenic protocol but instead involved substantial dietary modification.

Notably, the study did not demonstrate induction of nutritional ketosis and therefore does not constitute a ketogenic trial. The intervention emphasized increased fat intake, rising from 73 g to 84.9 g per day, alongside a marked reduction in refined carbohydrate consumption. This dietary shift aligns with prevailing neurological hypotheses suggesting that high-fat dietary patterns may exert therapeutic effects across a range of neurological conditions [52].

The findings revealed a strong positive association between baseline carbohydrate intake and the severity of hyperactivity and learning difficulties. This relationship has been hypothesized to involve dysregulated "glucose oscillation" events. Although evidence specific to ADHD remains mixed, high-glycemic-load dietary patterns have been proposed to contribute to behavioral dysregulation in susceptible children [53].

The intervention also addressed the frequent comorbidity of obesity in ADHD, which affected more than 50% of participants in this cohort. Given this high prevalence, dietary modification was associated with changes in systemic inflammatory markers, including interleukin-6 (IL-6). These observations are consistent with broader hypotheses linking metabolic status and inflammatory signaling to neurobehavioral regulation. Nevertheless, it is important to emphasize that this study does not establish a causal relationship between changes in inflammatory markers and improvements in ADHD symptoms [54] [55].

Rather than aiming to induce physiological nutritional ketosis, the investigators hypothesized that reduced carbohydrate intake combined with increased fat consumption contributed to symptom mitigation. This "fat-dominant" dietary pattern provides a plausible molecular framework for continued exploration of structured ketogenic therapies [56]. Collectively, these preliminary findings suggest that such metabolic interventions warrant careful clinical evaluation in pediatric ADHD populations, particularly where moderate carbohydrate restriction is associated with measurable behavioral change.

4.3. The KD: From Epilepsy to ADHD Management

4.3.1. Cross-Population Evidence: Pediatric Epilepsy

ADHD and epilepsy are both common pediatric neurological disorders. Epilepsy affects approximately 1% of children and adolescents and is defined by the occurrence of two or more unprovoked, nonfebrile seizures, whereas the prevalence of ADHD in children ranges from 7.5% - 16% [57]. It is because of differences in diagnostic criteria and the populations studied that the prevalence estimates differ, even from the 3% - 6% [1] quoted earlier in this review. The relationship between epilepsy and ADHD is complex and remains incompletely understood. Epidemiological studies indicate that children with epilepsy have a 2.5 - 5.5-fold higher risk of ADHD compared with healthy controls [58].

Within pediatric epilepsy cohorts, several studies have reported improvements in attention, behavior, or social functioning during ketogenic therapy. However, interpretation of these findings is complicated by multiple confounding factors, including seizure reduction, improved sleep quality, adjustments in antiseizure medications, changes in family mealtime structure, and expectancy effects related to behavioral observation [59]-[61].

To ensure internal validity in future trials, several key variables must be controlled. First, changes in antiseizure medications (ASMs) may independently improve behavior; future research should incorporate a "stable medication period"

of at least 4 - 8 weeks prior to dietary initiation. Second, the KD often improves sleep architecture [62], which can indirectly mitigate ADHD symptoms; therefore, objective sleep measures (e.g., actigraphy or sleep logs) should be utilized to isolate these effects. Finally, the structured nature of KD therapy often introduces changes in family mealtime routines and observer expectancy; mitigating this requires the use of blinded raters—such as teachers or clinicians who are unaware of the dietary status—to provide objective behavioral assessments.

Given that sleep disturbances are common in children with ADHD and can exacerbate inattentive and hyperactive symptoms [63], such findings should be interpreted as cross-indication signals rather than evidence of therapeutic efficacy in primary ADHD populations [64].

In this context, Pulsifer *et al.* (2001) reported that children receiving a KD for one year demonstrated statistically significant improvements in social behavior and attention, providing an informative, albeit indirect, metabolic signal relevant to ADHD [65].

4.3.2. Animal Model Evidence

Comparative medicine approaches have further contributed to the exploration of metabolic pathways linking KDs and behavioral regulation. In a 6-month prospective, randomized, double-blind, crossover trial, the effects of a medium-chain triglyceride-based KD were evaluated in 21 dogs with idiopathic epilepsy and concurrent ADHD-like behaviors [66]. Although serum β-hydroxybutyrate levels were assessed, the specific mechanisms underlying alterations in brain energy metabolism were not fully elucidated. Nonetheless, these findings support the concept that ketogenic-format diets may influence behavioral phenotypes through metabolic stabilization, while acknowledging the inherent limitations of interspecies translation [52]. Importantly, this evidence derives from non-human subjects with comorbid epilepsy and should therefore be regarded as hypothesis-generating for human ADHD rather than as direct clinical proof.

5. Mechanistic Insights and the Microbiota-Gut-Brain Axis

5.1. The Role of Gut Microbiota in ADHD Pathogenesis

Animal Model Evidence: Mechanistic Insights

To understand how metabolic shifts induced by dietary interventions may translate into behavioral outcomes, increasing attention has been directed toward the microbiota-gut-brain axis. The influence of gut microbiota on human physiology, immune regulation, and neurological function is now well recognized, particularly in relation to complex neurodevelopmental and neurodegenerative conditions, including ADHD, Parkinson's disease, and Alzheimer's disease [67] [68]. Clinical studies in children and adolescents with ADHD have consistently reported alterations in gut microbiome composition and diversity compared with neurotypical controls. Specifically, adolescents with ADHD exhibit a marked reduction in *Faecalibacterium* abundance [69], and lower gut microbial alpha di-

versity has been negatively associated with ADHD symptom severity [70]. Collectively, these findings support the hypothesis that the microbiota-gut-brain axis contributes to central nervous system homeostasis and behavioral development, potentially through its influence on neurotransmission and neuroplasticity [71] [72].

5.2. Neurotransmission and Excitation-Inhibition Balance

The SHR, a validated preclinical model of ADHD, has been widely employed to explore mechanistic pathways linking metabolic interventions and behavioral phenotypes [73]. In a comparative SHR study evaluating an oral KD against MPH, investigators examined the potential of dietary intervention as a metabolic modulator of ADHD-like behaviors. The findings suggested that ketogenic dietary exposure was associated with reductions in ADHD-like behaviors, potentially mediated through alterations in the microbiota-gut-brain axis [74].

Further analysis within the SHR model demonstrated that ketogenic intervention significantly increased the relative abundance of several beneficial bacterial taxa, including *Ruminococcus_gauvreauii_group, Bacteroides*, and *Bifidobacterium*. Of particular relevance, Bifidobacterium is known to contribute to gamma-aminobutyric acid (GABA) production, the primary inhibitory neurotransmitter within the cerebral cortex. Reduced cortical GABA levels have been linked to impulsivity and are frequently observed in children with ADHD [75]. Beyond its role in inhibitory neurotransmission, *Bifidobacterium* has been proposed as a potential microbial biomarker due to its involvement in dopaminergic reward signaling pathways [76] [77]. In addition, short-chain fatty acids (SCFAs), which exhibit neuroactive and anti-inflammatory properties, are efficiently produced by taxa such as *Ruminococcus* and *Bacteroides* [78]. These metabolites are thought to support microglial homeostasis and modulate the expression of brain-derived neurotrophic factor (BDNF), deficits of which have been associated with impairments in working memory [79]. Together, these preclinical observations suggest that ketogenic dietary intervention may attenuate ADHD-like behaviors through microbiota-mediated neurobiological mechanisms [74] [80].

5.3. Neuroenergetics and Metabolic Stabilization

Ketogenic dietary interventions have also been hypothesized to influence amino acid metabolism pathways relevant to neuropsychiatric function. Several studies in pediatric and adolescent ADHD populations have reported alterations in amino acid availability and metabolism, particularly involving precursors of monoamine neurotransmitters [81]. However, findings across these studies remain inconsistent and do not support the presence of a uniform or persistent biochemical deficiency characteristic of ADHD. While short-term supplementation with specific amino acids, such as DL-phenylalanine or S-adenosyl-L-methionine, has been associated with transient improvements in mood and hyperactivity, ketogenic-format interventions may offer a more integrated metabolic modulation. Concurrently, KDs inherently suppress glucose-dependent metabolic pathways, a feature

of potential relevance given that high intake of refined carbohydrates has been associated with behavioral dysregulation in ADHD [74] [82]. Evidence suggests that replacing energy-dense, low-nutrient foods with structured dietary patterns may correlate with symptom improvement; however, carbohydrate restriction alone is often insufficient to produce sustained clinical benefit [83]. Taken together, the combined capacity of KDs to modulate gut-mediated amino acid metabolism while imposing stringent limitation on refined carbohydrate intake positions ketogenic therapy as a biologically plausible, yet still insufficiently studied, intervention in ADHD management [74].

5.4. Evidence from the Animal Models

Previous investigations have reported that rodents maintained on a KD exhibit reduced locomotor activity compared with animals fed a standard diet; however, the duration of dietary exposure required to elicit this effect has not been clearly established. Experimental studies have therefore sought to examine the temporal dynamics of KD-associated changes in activity levels and to explore potential relationships between altered locomotion and anxiety-related behaviors. Locomotor activity has typically been assessed using the open-field test [84] [85], while anxiety-like behavior has been evaluated using the elevated plus maze [86] [87].

Murphy and Burnham (2006) reported that exploratory behavior and overall activity levels in Long-Evans rats were significantly reduced after 7 days of exposure to a KD [35]. However, because confirmation of sustained metabolic ketosis was limited, these findings warrant cautious interpretation. In a separate study, Wistar rats were assigned to a conventional diet, a balanced KD, or a standard control diet for 18 - 19 days, with results demonstrating that rats receiving the KD were consistently less active than those maintained on the usual diet [88]. Although these and other preclinical studies suggest that KD exposure influences general behavioral activity in rodents, interpretation remains constrained by the limited availability of experimental animal models that accurately capture core features of ADHD [89].

Collectively, existing data support the hypothesis that ketogenic-based diets may reduce motor activity in animal models. However, reductions in locomotion should not be interpreted as evidence of improvement in core ADHD symptoms, such as attentional regulation, executive functioning, or impulse control. Moreover, many of these studies focused primarily on measures related to impulsivity or activity and did not assess broader ADHD-relevant behavioral domains, limiting the extent to which findings can be generalized to ADHD treatment. While KDs have demonstrated clear efficacy in animal models of partial seizure disorders [35] [66] [88], their association with reduced motor activity does not directly translate into clinical efficacy for ADHD symptomatology in humans. Comprehensive evaluation of the potential role of KD interventions in ADHD will therefore require rigorously designed human clinical trials alongside the development and application of ADHD-specific animal models [90].

More recent animal studies by Carreón-Trujillo *et al*. (2024) and Liu *et al*. (2023) have further explored the behavioral effects of ketogenic interventions [74] [91]. A major limitation shared by both investigations is the absence of confirmation of physiological ketosis, such as measurement of serum β-hydroxybutyrate concentrations or equivalent biomarkers. Additionally, Liu *et al*. (2023) did not clearly define the macronutrient composition of the KD employed, thereby limiting comparability across studies and reducing interpretive rigor [74].

5.5. Translational Constraints of Animal Models in ADHD Ketogenic Research

Despite reports of behavioral changes in rodent models, several translational challenges remain. First, reduced hyperactivity observed in open-field paradigms does not necessarily reflect improvements in executive control or sustained attention, which are central diagnostic features of ADHD in humans [92]. Second, although the SHR model provides valuable insights, it does not fully recapitulate the heterogeneity and complexity of the human ADHD phenotype. Furthermore, inconsistent reporting of dietary composition and the frequent lack of confirmation of physiological ketosis, particularly through serum BHB measurements, limit reproducibility and cross-study comparability [93]. These methodological limitations underscore the need for cautious interpretation of preclinical findings when considering translation to human research. Future animal studies should prioritize ADHD-relevant cognitive endpoints, including sustained attention, response inhibition, and cognitive flexibility, rather than relying predominantly on locomotor activity measures. A comprehensive overview of the preclinical evidence regarding ketogenic interventions and their effects on ADHD-related phenotypes is presented in the summary **Table 1** below.

Table 1. Preclinical evidence on ketogenic-format dietary interventions relevant to ADHD-related phenotypes.

Limitations & Obstacles	Behavioral & Neurochemical Findings	ADHD Domain Assessed	Ketosis Confirmed (BHB)?	Animal Model	Reference
[35] [88]	Use of non-ADHD models; absence of serum BHB measurement.	Significant reductions in exploratory behavior and overall activity.	Activity/ Exploration	No	Long-Evans & Wistar
[91]	Subcutaneous administration likely insufficient; no BHB validation.	No significant effects on locomotor activity or spatial memory.	Locomotor activity	No	Wistar (6-OHDA)
[74]	KD composition not specified; lack of biochemical confirmation of ketosis (BHB).	Improved open-field performance with reported neurotransmitter upregulation.	Activity & Gut–Brain Axis	No	SHR
[90]	Limited data directly linking behavioral effects to ketosis status.	Ketogenic-format diets may reduce hyperactivity in preclinical settings.	Hyperactivity/Metabolic	Varies	Systematic Review

In conclusion, although the majority of preclinical evidence suggests a "calming" effect of ketogenic-format diets on hyperactivity, the existing data remain primarily hypothesis-generating. Future investigations should prioritize the use of validated ADHD-specific animal models and incorporate rigorous biochemical characterization, including measurements of ketone bodies, glucose, and lipid profiles, to strengthen translational relevance and better link rodent behavioral outcomes to potential human clinical applications.

6. Methodological and Ethical Barriers to Clinical Research in Children with ADHD

Ketogenic therapy is most frequently applied in pediatric clinical practice for drug-resistant epilepsy, a context in which the anticipated benefits often outweigh dietary burden and monitored medical risks. In contrast, ADHD is characterized by the availability of multiple established, evidence-based pharmacological and behavioral interventions, thereby raising the ethical threshold for evaluating highly restrictive dietary approaches in children during critical periods of growth and development [94] [95].

6.1. Hierarchy of Clinical Necessity

Within pediatric neurology, the KD is widely regarded as a "metabolic rescue" therapy for refractory epilepsy [96]. In this setting, the risk-benefit balance is comparatively clear, as the urgent need to prevent seizures, neurocognitive decline, and status epilepticus supersedes the potential adverse effects associated with sustained ketosis. By contrast, ADHD is typically conceptualized as a condition primarily managed through behavioral and pharmacological strategies rather than as an immediately life-threatening disorder [97]. Consequently, when effective alternative treatments are available, Institutional Review Boards (IRBs) generally exercise heightened caution in approving trials involving highly restrictive dietary protocols for growing children.

6.2. Executive Dysfunction and the Compliance Trap

From a methodological standpoint, ADHD presents unique challenges related to treatment adherence. Core features of the disorder, including impulsivity and executive dysfunction, are inherently misaligned with the strict precision required to maintain a therapeutic ketogenic regimen. The fluctuating nature of ADHD symptoms further complicates long-term adherence to rigid fat-to-carbohydrate ratios, such as 3:1 or 4:1, thereby posing threats to internal validity in longitudinal trials [98]. This contrasts with epilepsy research, where parental adherence is often reinforced by the immediate and tangible risk of seizure recurrence.

6.3. Limited Mechanistic Anchors and Biomarkers

Although ADHD is etiologically heterogeneous, the KD demonstrates established efficacy in epilepsy through well-characterized mechanisms involving seizure

threshold modulation and GABAergic stabilization [99]. In ADHD, proposed mechanisms, such as stabilization of dopaminergic pathways or enhancement of mitochondrial function within the prefrontal cortex, remain largely theoretical. A major methodological limitation is the absence of a definitive, quantitative biomarker that directly links dietary ketosis to attentional or behavioral outcomes, analogous to the suppression of epileptiform activity observed on electroencephalography in epilepsy.

6.4. Financial Constraints and Commercial Inertia

Dietary interventions pose inherent challenges for large-scale clinical investigation, as they are difficult to patent and require substantial delivery infrastructure, including specialized dietetic expertise, ongoing monitoring, and sustained family engagement. These factors may limit access to commercial funding relative to pharmaceutical trials [100]. As a result, research support often relies on public or charitable funding mechanisms, which can constrain both trial duration and sample size.

6.5. Developmental and Growth Concerns

Potential pediatric risks associated with prolonged ketogenic therapy include alterations in growth velocity, bone health, lipid profiles, and micronutrient sufficiency. In clinical practice, the implementation of highly restrictive diets necessitates robust governance structures, encompassing specialized dietary supervision, scheduled nutritional monitoring, and predefined contingency plans when clinical benefit is not observed within a specified timeframe, particularly in children with selective eating behaviors [101]. In the context of ADHD, where extended treatment durations may be contemplated, ethically sound trial design requires comprehensive safety monitoring, including growth parameters, bone health indicators, lipid profiles, and micronutrient status, alongside clearly defined stopping criteria.

6.6. Safety Monitoring and Clinical Contraindications

To ensure participant safety in pediatric ADHD trials, researchers must account for the well-documented adverse effect profile of therapeutic ketogenic diets [102]. Beyond growth velocity and lipid profiles, common clinically monitored adverse effects include:

- Gastrointestinal Distress: Constipation, nausea, and abdominal pain are frequent, particularly during the induction phase.
- Metabolic Derangements: Transient hypoglycemia, hyperuricemia, and metabolic acidosis.
- Long-term Risks: Nephrolithiasis (kidney stones) and possible decreases in bone mineral density.

Minimum Safety Labs and Monitoring Cadence: For any proposed ADHD ketogenic trial, a standardized monitoring cadence is essential to mitigate these risks

[103]. We recommend the following minimum protocol:

- Baseline: Comprehensive metabolic panel (CMP), lipid profile, CBC, vitamin D, and renal ultrasound (if clinically indicated).
- Monthly (first 3 months): Monitoring of growth parameters (height, weight, BMI), blood glucose, and capillary BHB levels.
- Quarterly (every 3 months): Full laboratory reassessment including lipid panels, renal function tests (BUN/creatinine), urine calcium-to-creatinine ratio (to screen for lithiasis risk), and micronutrient status (iron, zinc, selenium).
- Clinical Contraindications: Absolute contraindications include primary carnitine deficiency, fatty acid oxidation disorders, and pyruvate carboxylase deficiency, all of which must be ruled out via baseline metabolic screening.

7. Limitations of This Review

Careful consideration of the limitations of this narrative review is essential for appropriate interpretation of the findings. First, this review was not conducted as a systematic review and therefore may be subject to selection bias, as the literature was identified and synthesized qualitatively rather than through a PRISMA-compliant quantitative meta-analytic framework. In addition, a formal risk-of-bias assessment was not performed, and outcome data were not extracted in a manner suitable for quantitative synthesis.

Second, substantial heterogeneity exists across the included studies with respect to dietary protocols, macronutrient compositions, and behavioral outcome measures, limiting the ability to perform direct comparisons or draw unified conclusions. Third, much of the current evidence is derived from extrapolation of findings in pediatric epilepsy cohorts and preclinical animal models, which may not adequately capture the neurobiological and behavioral complexity of primary ADHD in children. Finally, there remains a notable lack of long-term pediatric interventional data evaluating both the safety and efficacy of structured ketogenic therapies in children with primary ADHD in the absence of comorbid epilepsy.

8. Conclusion

This narrative review identifies consistent associations between overall dietary quality and ADHD risk and symptom burden, with Western or highly processed dietary patterns generally associated with worse outcomes, and healthier dietary patterns associated with fewer symptoms. However, there is currently no direct clinical trial evidence demonstrating that verified ketogenic therapy improves primary ADHD in pediatric populations. Existing ketogenic-related evidence arises from indirect dietary modification studies, epilepsy cohorts that introduce important confounding variables, and preclinical animal models that are frequently constrained by incomplete dietary reporting and lack of biochemical verification of ketosis. Accordingly, ketogenic therapies should be regarded as experimental in the context of primary ADHD and explored only within rigorously designed and ethically justified clinical research frameworks.

9. Recommendation

Future investigations should prioritize feasibility- and safety-focused RCTs in carefully selected pediatric ADHD cohorts. Study designs should compare a verified ketogenic therapy protocol, with clearly specified dietary format and delivery under professional dietetic supervision, against an active dietary comparator such as Mediterranean or low-glycemic index counseling in addition to usual clinical care. Primary endpoints should include multi-informant ADHD symptom ratings (e.g., Conners-4 or ARS-5 completed by parents and teachers) [104], complemented where feasible by objective measures of attention and inhibitory control. Specifically, the "Sustained Attention" domain should be assessed using objective computerized tasks such as the Continuous Performance Test (CPT), while the "Behavioral Inhibition" domain should be mapped to paradigms such as the Go/No-Go or Stroop Task [105]. Combining these objective metrics with multi-informant scales will provide a robust framework to evaluate the metabolic impact on core ADHD domains. Given the high prevalence of sleep disturbances in children with ADHD, structured sleep assessment should be incorporated into dietary intervention trials. Biological engagement of the intervention must be confirmed through scheduled ketone measurements, using capillary or serum BHB, with sustained adherence supported by structured meal planning, coordination with school environments, and predefined adherence thresholds. Comprehensive safety monitoring should include assessment of growth velocity, BMI trajectories, lipid profiles, and micronutrient status, including iron indices, vitamin D, folate and vitamin B12, and zinc and selenium.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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