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REVIEW

Vitamin Interventions in ASD and ADHD: Systematic Review and Meta-Analysis

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Background: Vitamin interventions have emerged as a cost-effective and accessible approach to managing Autism Spectrum Disorder (ASD) and Attention-Deficit/Hyperactivity Disorder (ADHD), primarily for alleviating gastrointestinal symptoms such as constipation. Recent studies suggest vitamins may also improve core symptoms, yet most existing research focuses on comparisons between patients and healthy controls, lacking clinically relevant, evidence-based insights.

Methods: A meta-analysis was conducted using studies retrieved from PubMed, Web of Science, and the Cochrane Library, focusing on vitamin interventions in ASD and ADHD populations.

Results: The findings indicate that vitamin supplementation significantly improves symptoms in both ASD and ADHD. However, the effects vary by vitamin type and disorder. Vitamin B supplementation was particularly effective in reducing ASD-related symptoms, while vitamin D supplementation showed greater efficacy in improving ADHD symptoms.

Conclusion: Different vitamins exert disorder-specific therapeutic effects, suggesting their potential role in guiding tailored clinical interventions for ASD and ADHD.

Keywords: vitamin therapy, ASD, ADHD, meta-analysis

Introduction

Autism spectrum disorder (ASD) is characterized primarily by impairments in social communication and interaction, together with restricted/repetitive behaviors and sensory anomalies while Attention-deficit/hyperactivity disorder (ADHD) presents primarily with inattention, hyperactivity, and impulsivity. ADHD is primarily manifested by symptoms such as inattention, hyperactivity, and impulsivity, whereas ASD is defined by deficits in social communication, restricted interests, and repetitive behaviors.[1] Despite their diagnostic differences, a growing body of research has revealed overlapping features between ADHD and ASD in terms of clinical presentation, neurobiological mechanisms, and genetic underpinnings.[2–6] Both conditions frequently involve impairments in executive functioning, emotional regulation, and sensory processing, providing a theoretical foundation for potential shared intervention strategies.

In recent years, vitamins have gained increasing attention in the field of neurodevelopmental disorders due to their critical roles in metabolism, immune regulation, and neurodevelopment.[7–9]Studies have indicated that deficiencies or metabolic abnormalities in vitamins such as vitamin D, B6, B12, and folate are common in individuals with ADHD and ASD, and are closely associated with impairments in behavior, cognitive function, and brain development.[2,10–12] Recent evidence indicates that children with ASD often show alterations in several micronutrients—such as vitamin D, B-vitamins, and folate—which can affect neurotrophic factors, synaptic protein expression, oxidative stress, and inflammatory signaling, thereby influencing neurodevelopment and synaptic plasticity.[13] These mechanisms provide a plausible rationale for investigating vitamins as adjunctive interventions for behavioral outcomes in ASD. Meanwhile,

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nutritional evidence links B-vitamins—especially the active form of vitamin B6 (pyridoxal-5-phosphate, PLP)—and vitamin B12/folate to cognitive and emotional functions; meanwhile, omega-3 polyunsaturated fatty acid imbalances (eg, elevated omega-6/omega-3 ratio) are repeatedly observed in ADHD, and multiple RCTs and meta-analyses report that omega-3 supplementation can improve attention-related symptoms.[14,15]

Nutritional interventions involving vitamin supplementation have shown promising effects in alleviating core symptoms—such as improving attention and impulse control in children with ADHD, and enhancing communication skills, language development, and reducing stereotyped behaviors in individuals with ASD.[13–16]Notably, recent evidence suggests that the benefits of vitamin interventions may extend beyond symptom relief, potentially involving modulation of the gut-brain axis. This includes regulation of systemic inflammation, gut microbiota composition, and neurotransmitter synthesis.[17,18] Given the high prevalence of chronic inflammation and neuroinflammation in both ASD and ADHD populations, vitamin-based interventions may offer a multi-targeted approach with potential benefits for emotional stability, cognitive enhancement, and restoration of social functioning.[8,11,13]

Although studies on vitamin interventions in neurodevelopmental disorders are accumulating, there is still a lack of systematic conclusions regarding intervention protocols, dosage, duration, or mechanisms of action in both ASD and ADHD. Besides, although ASD and ADHD differ in etiology and clinical phenotype, they are both highly prevalent in child and adolescent psychiatric practice and frequently co-occur, which poses overlapping diagnostic and treatment challenges.[19] Considering the partially shared pathophysiological foundations and intervention targets of the two disorders, developing a common nutritional intervention model may not only increase clinical efficiency but also help alleviate healthcare burdens, especially in resource-limited settings. Therefore, it is imperative to synthesize the existing clinical evidence through a meta-analytic approach.

This study aims to systematically review and meta-analyze the current clinical trials involving vitamin interventions in ASD and ADHD. By assessing their effects on symptom improvement, emotional regulation, or cognitive function, we seek to clarify the shared and distinct therapeutic outcomes, provide evidence-based guidance for transdiagnostic interventions, and offer theoretical and practical implications for nutritional therapy in neurodevelopmental disorders.

Methods

Protocol and Registration

This review adhered to the guidelines of the Preferred Reporting Item for Systematic Reviews and Meta-Analysis (PRISMA).[20,21] The protocol was registered in the database of the International Prospective Register of Systematic Reviews (PROSPERO, CRD420251019077).

Search Strategy

This study aims to evaluate the clinical efficacy of vitamin supplementation in improving symptoms of autism spectrum disorder (ASD) and attention-deficit/hyperactivity disorder (ADHD). To this end, we systematically searched three databases (ie, PubMed, Web of Science, and Cochrane Library) for relevant studies published up to February 1, 2025. The search terms included: (“Autism spectrum disorder” OR “ASD” OR “Autism” OR “Attention Deficit Hyperactivity” OR “ADHD”) AND (“Vitamin” OR “Vitamin supplement” OR “Nutritional Supplementation” OR “Dietary Supplement” OR “Micronutrient” OR “Nutritional Intervention”) AND (“Clinical trials” OR “randomized control”).

Two independent reviewers screened the titles and abstracts of the retrieved articles based on predefined inclusion and exclusion criteria. In cases where the abstract lacked sufficient information, the full text was reviewed. Full texts of potentially eligible studies were then assessed for inclusion. Any disagreements between reviewers were resolved through discussion and consensus. Additionally, we manually searched the reference lists of included studies and relevant reviews to identify potentially missing studies. For studies with incomplete data, the corresponding authors were contacted via Email to request the necessary information.

Selection Criteria

The inclusion and exclusion criteria for the literature were determined based on the PICOS framework outlined in the PRISMA guidelines. The specific criteria were as follows: (1) Participants: Studies were included if the participants were diagnosed with autism spectrum disorder (ASD), attention-deficit/hyperactivity disorder (ADHD), or comorbidity of both. Studies involving healthy controls or non-human subjects were excluded. (2) Intervention: Eligible studies employed vitamin supplementation as the primary intervention. To minimize confounding effects, other concurrent treatments (eg, pharmacological therapies) were required to remain unchanged or be absent during the intervention period. (3) Comparisons: Studies were included if they provided both pre- and post-intervention measurements. Studies lacking baseline assessments and/or appropriate control conditions were excluded. (4) Outcomes: Clinical outcomes were defined as quantifiable measures of symptom improvement or functional enhancement, assessed using validated rating scales. ASD-specific outcomes included social communication skills (eg, ADOS, SRS), restricted and repetitive behaviors (eg, RBS-R), and overall symptom severity (eg, CGI, CARS). ADHD-specific outcomes included attention deficit and hyperactivity/impulsivity symptoms (eg, ADHD-RS, Conners Rating Scale) and executive function measures. Comorbidity or non-specific outcomes included emotional regulation (eg, CBCL) and adaptive functioning (eg, Vineland Adaptive Behavior Scales). Outcome extraction followed a predefined hierarchy: (1) core symptom rating scales; (2) functional measures; (3) emotional and behavioral measures. Only studies reporting symptom-related outcomes were included; those focusing solely on unrelated outcomes were excluded. (5) Study Design: Eligible studies employed a standard control group, sham control group, crossover design, parallel group, or other forms of clinical trial design. For crossover studies, only data from the first phase were used to avoid potential carryover effects. (6) Data Reporting: Studies were required to report sufficient statistical information (eg, means and standard deviations) to allow for the calculation of effect sizes using Hedges' g. Studies with incomplete, missing, or uncomputable data were excluded. (7) Publication Type: Only peer-reviewed articles published in English were considered (See Supplemental Table 1).

Assessment of Risk of Bias

The quality of the included studies was assessed using the Cochrane Risk of Bias Tool (ROB2).[22,23] Five domains of bias were evaluated: bias due to randomization, bias due to deviations from intended intervention, bias due to missing data, bias due to outcome measurement, bias due to selection of reported results. Concretely, bias due to randomization assesses whether the randomization sequence was adequately generated and concealed to prevent baseline imbalances. Bias due to deviations from intended intervention evaluates whether participants adhered to the intended intervention and whether any deviations introduced bias. Bias due to missing data examines the extent and reasons for missing outcome data and their potential impact on the results. Bias due to outcome measurement assesses whether outcome measurements were objective, consistent, and blinded where appropriate. Finally, bias due to selection of reported results evaluates whether the reported outcomes were selectively chosen from among multiple prespecified analysis.

Data Extraction

Following training and calibration exercises to ensure inter-rater reliability, two reviewers independently and in duplicate extracted data from each eligible study. Extracted data included sample sizes, change scores, t-values, p-values, as well as means and standard deviations of pre- and post-intervention outcomes for effect size calculation. Additional data such as the number of participants, mean age, and specific methodological details were also collected. Notably, a wide range of assessment scales were employed across the included studies. To ensure relevance and comparability, we selected those scales that specifically targeted core symptoms of ASD or ADHD. Furthermore, among the commonly used rating sources—namely, parent-reported, teacher-reported, and clinician-administered scales—we prioritized clinician-administered evaluations, and teacher-reported later, last is the parent-reported due to their higher objectivity and diagnostic reliability.

Effect Size Calculation

The Standardized Mean Difference (SMD) was used as the primary effect size metric during the meta analysis, as the included studies assessed similar psychological or behavioral constructs using different measurement instruments.[24–26] For each study, we extracted the pre-, post-intervention means, standard deviations, and sample sizes for both the intervention and control groups to calculate the SMD. When standard deviations were not directly reported, we estimated them using available statistics such as standard errors, confidence intervals, t-values, or p-values, following the guidelines provided in the Cochrane Handbook.[22] To reduce potential bias in studies with small sample sizes, we applied a correction to obtain Hedges' g,[27] an adjusted version of SMD that accounts for small-sample bias. In studies that reported only pre-post change scores, we prioritized extracting post-intervention data to avoid confounding effects due to baseline differences.

Data Synthesis and Subgroup Analysis

The choice between a random-effects model and a fixed-effects model depends on the degree of heterogeneity among the included studies. When heterogeneity is low, a fixed-effects model is appropriate, as it assumes that all studies estimate the same underlying effect size. Conversely, when substantial heterogeneity exists, a random-effects model is preferred, as it accounts for variability both within and between studies by incorporating between-study variance into the analysis. SMD was used as the primary metric for standardized mean differences. All effect sizes were reported with corresponding 95% confidence intervals (CIs). Subgroup analyses were conducted based on key study characteristics, such as type of disorder (ie,ASD, ADHD) or vitamin type, to explore possible moderators of treatment effects.

Assessment of Heterogeneity

In systematic reviews and meta-analyses, it is often unclear whether observed results are consistent across studies. Therefore, testing for heterogeneity is essential to assess the degree of variability among study outcomes.[28] In line with previous meta-analyses,[29] heterogeneity was evaluated using the I^2 statistic and tau-squared (τ^2) estimate. The I^2 statistic quantified the proportion of total variability due to heterogeneity rather than chance,[30] with values of 25%, 50%, and 75% interpreted as representing low, moderate, and high heterogeneity, respectively. Tau-squared (τ^2) represents the between-study variance in a random-effects model, reflecting the extent of true effect size differences across studies.

Publication Bias and Sensitivity Analysis

Publication bias can distort the conclusions of a meta-analysis by overrepresenting studies with positive or significant results. To evaluate potential bias, several complementary methods were employed. Funnel plots were first visually inspected, plotting effect sizes against their standard errors based on SMD.[31] In an unbiased distribution, smaller studies (with larger standard errors) scatter widely at the bottom, while larger studies cluster near the top, forming a symmetrical inverted funnel shape.

However, because funnel plots are subjective and do not provide statistical inference, further analyses were conducted. Egger's regression test[32] was used to statistically assess funnel plot asymmetry. A p-value less than 0.05 indicated significant evidence of publication bias.

Results

After conducting a systematic search of the PubMed, Cochrane Library, and Web of Science databases, a total of 982 articles were initially identified. Following a rigorous screening process based on predefined inclusion and exclusion criteria, 22 studies were deemed eligible for inclusion (See Figure 1 and Supplemental Table 2).

The Vitamin Therapy Results on Both Neurodevelopmental Disorders

The total of 22 studies reported the effects of vitamin interventions on ASD or ADHD, including 12 studies on ASD involving 652 patients and 10 studies on ADHD involving 852 patients. The intervention groups included 787 participants, while the control groups included 717 participants. Heterogeneity analysis revealed a high level of

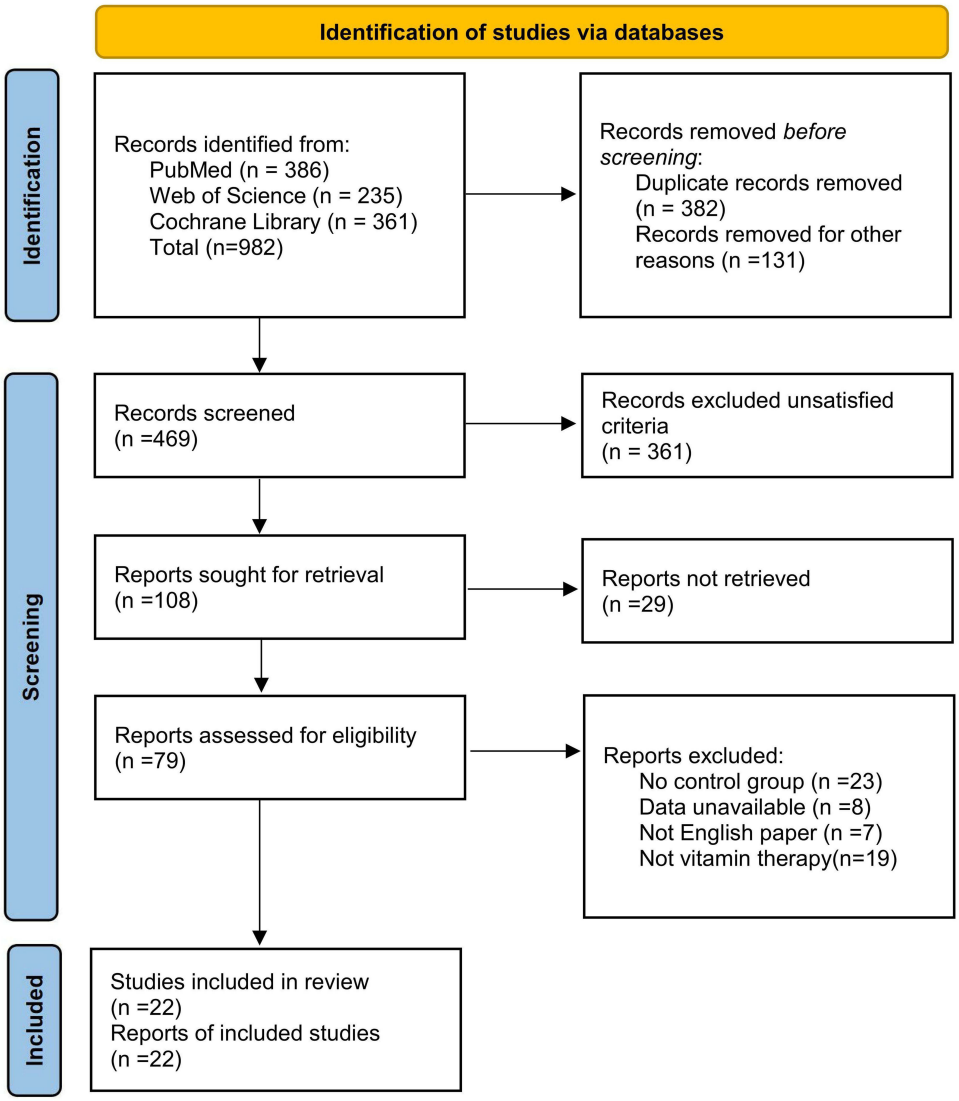

Figure 1 PRISMA flow diagram illustrating the study selection process. The diagram depicts the number of records identified through database searching and other sources, the number of records after duplicates were removed, the number of records screened and excluded, the number of full-text articles assessed for eligibility, and the number of studies included in the final qualitative and quantitative synthesis.

heterogeneity among the included studies (I^2=92.0%, τ^2 =0.8336, p< 0.001). Therefore, a random-effects model was applied for data synthesis. The pooled effect size was 0.41 (95% CI:0.01 to 0.81), which was statistically significant (p < 0.05), indicating that vitamin interventions had a significant therapeutic effect on patients with ASD and ADHD (See Supplemental Figure 1).

The Vitamin Therapy Results on ADHD and ASD

12 studies on ASD involving involving a total of 652 participants, with 339 in the intervention group and 313 in the control group (See Figure 2A). A high degree of heterogeneity was observed across studies (I^2= 95.9%, τ^2 =1.5771, p< 0.001), prompting the use of a random-effects model for meta-analysis. The pooled effect size was 0.38 (95% CI:–0.41 to 1.18), which was not statistically significant (p> 0.05). These findings indicate that vitamin supplementation did not yield a significant therapeutic benefit for individuals with ASD.

A total of 10 studies reported the effects of vitamin interventions on ADHD, including 852 patients, with 448 in the intervention group and 404 in the control group (See Figure 2B).Heterogeneity analysis indicated substantial heterogeneity among studies (I^2=74.6%, τ^2=0.2739, p< 0.001). Therefore, a random-effects model was applied for evidence

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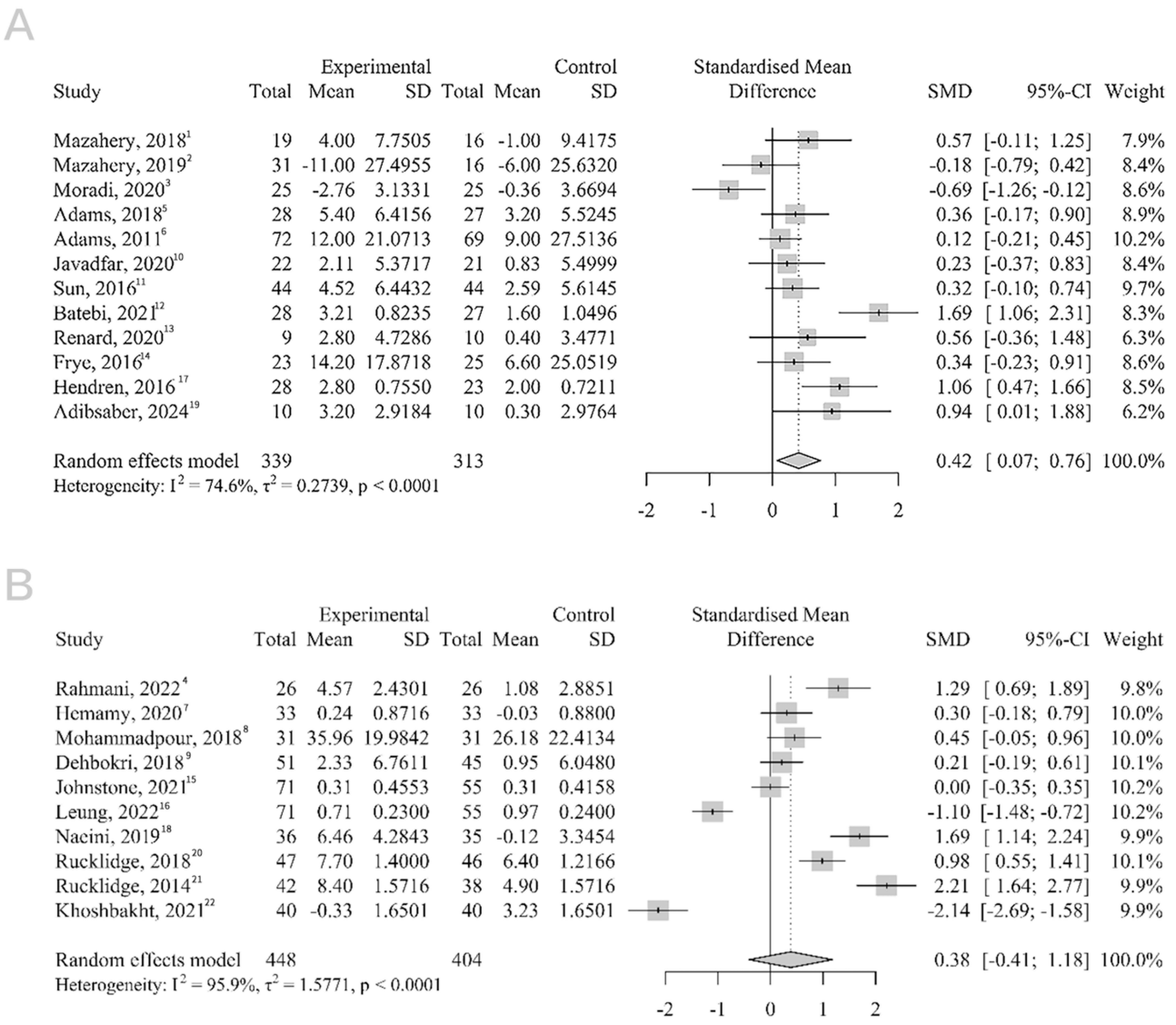

Figure 2 The forest results of vitamin therapy results on ADHD and ASD. The forest plots (**A**) showed the vitamin therapy results on ASD while the forest plot (**B**) indicated the vitamin therapy results on ADHD. All referred articles are mentioned in Supplemental Table 2.

synthesis. The pooled effect size was 0.42 (95% CI:0.07 to 0.76), which was statistically significant ($p< 0.05$), suggesting that vitamin interventions had a significant therapeutic effect on patients with ADHD.

Different Type of Vitamin Therapy Results

Among the 22 included studies, 5 of them investigated vitamin B therapy, 10 of them focused on vitamin D supplements, and other 7 articles examined multivitamin interventions. Subgroup analyses based on the type of vitamin intervention revealed that both vitamin D and vitamin B showed significant therapeutic effects in individuals with ASD and ADHD, with pooled SMDs of 0.47 (95% CI:0.04 to 0.90) and 0.41 (95% CI:0.01 to 0.81), respectively ($p< 0.05$ for both). In contrast, the pooled effect size for multivitamin interventions was 0.06 (95% CI:–0.97 to 1.09) and did not reach statistical significance ($p> 0.05$), suggesting limited or no observable benefit of multivitamin supplementation in this population (See Supplemental Figure 2).

Different Type of Vitamin Therapy Results on ASD and ADHD

Among the 12 studies investigating vitamin interventions for ASD, 5 studies involved vitamin B supplementation, 5 of them focused on vitamin D, and the last 2 articles examined multivitamin use. Subgroup analyses based on intervention

type indicated that vitamin B supplementation had a significant therapeutic effect on individuals with ASD, with a pooled SMD of 0.42 (95% CI:0.07 to 0.76, $p< 0.05$). Besides, the effects of both multivitamin and vitamin D interventions were not statistically significant, with pooled SMDs of 0.19 (95% CI:–0.09 to 0.47) and 0.12 (95% CI:–0.42 to 0.66), respectively ($p> 0.05$), suggesting limited efficacy of these interventions in this subgroup (See Figure 3).

Among the 10 studies assessing vitamin interventions for ADHD, 5 of them examined multivitamin supplementation and 5 of them focused on vitamin D. No studies included vitamin B interventions (See Figure 4). Subgroup analyses based on intervention type revealed that vitamin D supplementation had a significant therapeutic effect on individuals with ADHD, with a pooled SMD of 0.77 (95% CI: 0.20 to 1.35, $p< 0.05$). In contrast, multivitamin supplementation did not show a statistically significant effect, with a pooled SMD of –0.01 (95% CI:–1.50 to 1.47).

Sensitivity Analysis and Publication Bias

Sensitivity analysis showed that the overall effect size remained relatively stable (around 0.41) when each of the included studies was removed one at a time. However, the statistical significance of the meta-analysis results changed when 16 of the studies were individually excluded, indicating that the overall findings for both ADHD and ASD patients were robust generally (See supplemental Figure 3).

The funnel plot revealed that 10 studies lay outside the funnel, while 12 were within it. The distribution of studies was roughly symmetrical on both sides of the central line. Most studies (n = 20) had moderate standard errors, with only 2 studies showing small standard errors at the bottom of the funnel. No studies were located at the top, indicating a lack of very large sample sizes. Further assessment using Egger's test yielded t = 1.39, P = 0.1785, suggesting no evidence of publication bias. (See supplemental Figure 4).

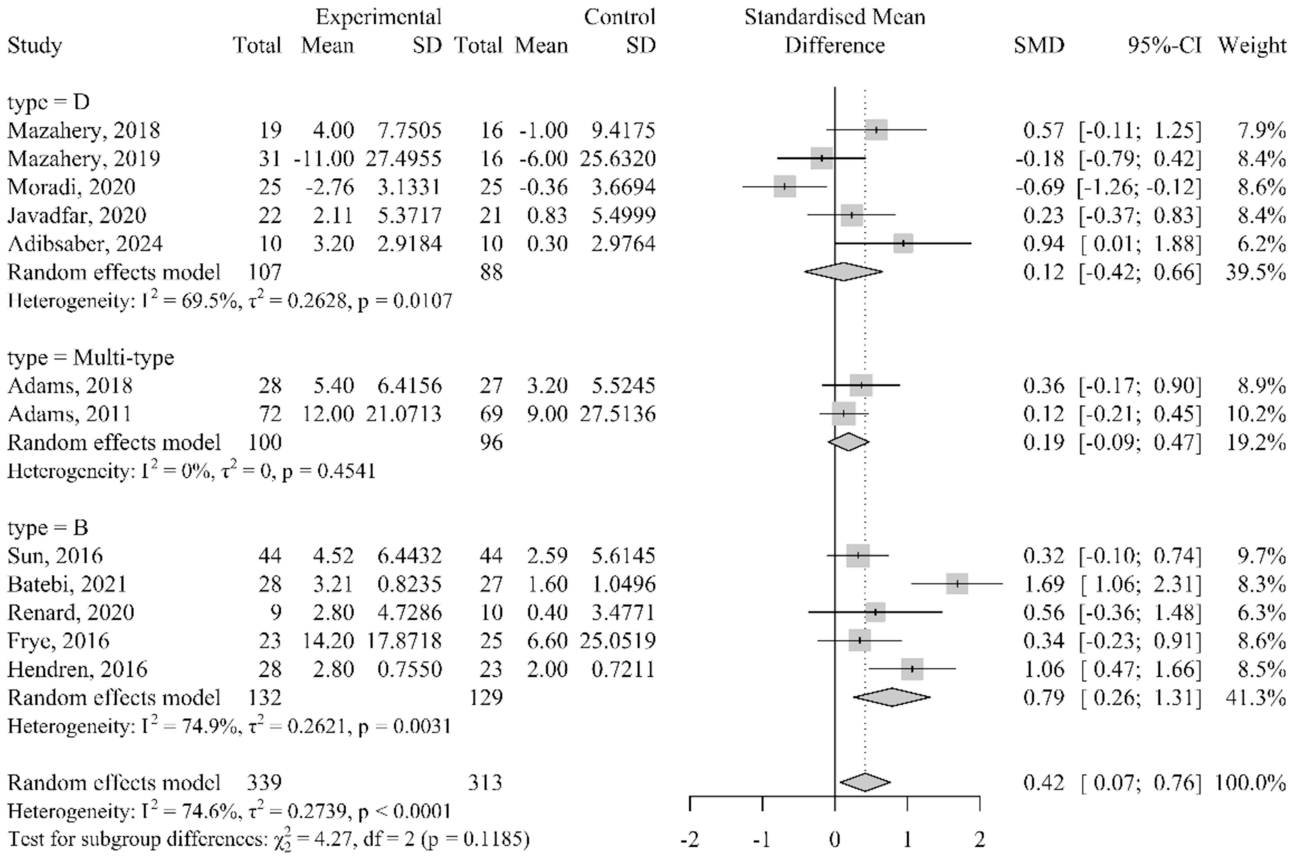

Figure 3 The forest results of different type of vitamin therapy on ASD. The forest plot indicated the meta analysis results of different type vitamin therapy of ASD. The type equals to D indicated vitamin D therapy results on ASD, the type equals to multi-type suggested the muti vitamin supplementation results on ASD, and the type equals to B illuminated the vitamin B therapy results on ASD. All referred articles are mentioned in Supplemental Table 2.

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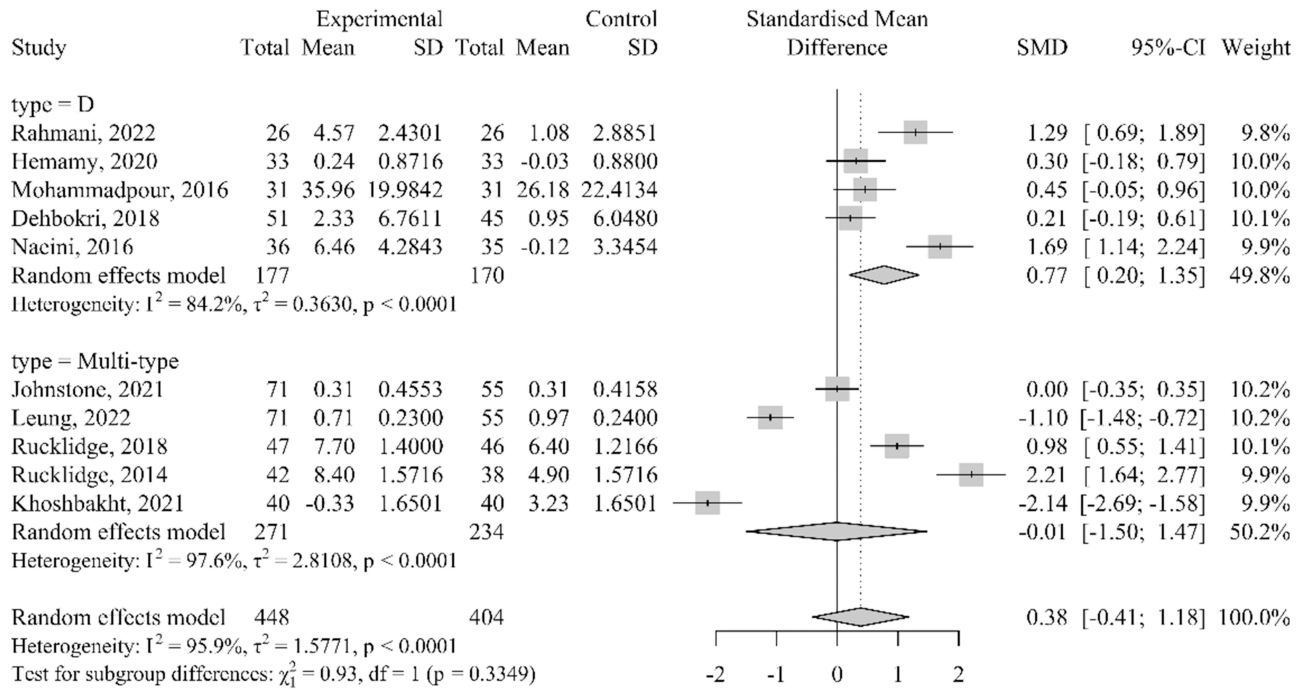

Figure 4 The forest results of different type of vitamin therapy on ADHD. The forest plot indicated the meta analysis results of different type vitamin therapy of ADHD. The type equals to D indicated vitamin D therapy results on ADHD, and the type equals to multi type suggested the muti vitamin supplementation results on ADHD. All referred articles are mentioned in Supplemental Table 2.

Assessment of Risk Bias

The risk of bias was assessed using the ROB 2.0 tool, and visualized with the "robvis" package in R. As shown in Supplemental Figures 5 and 6, only one study was rated as having "some concerns" in the domain of randomization, while the remaining 21 studies were judged to have low risk. In the domain of deviations from intended interventions, 5 studies were rated as having some concerns, and 17 were considered low risk. For missing outcome data, 1 study had some concerns, while 21 were at low risk. Regarding the measurement of the outcome, 4 studies were assessed as having some concerns, and 18 as low risk. In terms of selective reporting, all 22 studies were rated as low risk.

Overall, 8 studies were judged to have some concerns, and 14 were rated as low risk across all domains. No studies were assessed as high risk. The moderate risk ratings were mainly due to insufficient reporting, such as unclear randomization procedures, allocation concealment, or lack of detailed descriptions regarding blinding methods. These limitations made it difficult to fully evaluate the methodological rigor of some trials. Nevertheless, the absence of studies with a high risk of bias, coupled with the predominance of low-risk assessments, indicates that the overall quality of the included randomized controlled trials was acceptable Therefore, it is unlikely that the potential biases significantly influenced the meta-analysis results.

Discussion

In this meta-analysis, we explored the effectiveness of vitamin therapy in alleviating symptoms of ASD and ADHD, with the goal of providing practical clinical guidance. Our results show that vitamin supplementation can meaningfully improve symptoms in both conditions, although different types of vitamins appear to have different effects depending on the disorder.

For individuals with ASD, vitamin B supplementation was especially beneficial, leading to improvements in emotional regulation and stereotyped behaviors. On the other hand, patients with ADHD showed more positive responses to vitamin D, particularly in reducing behavioral issues and improving focus. Interestingly, both vitamin B and D demonstrated potential cross-disorder benefits, though to varying degrees.

These findings align with previous research showing that vitamin supplements can help reduce impulsivity and irritability in both ASD and ADHD.[12,33,34] Some studies have also reported that vitamin therapy may lessen emotional symptoms in ADHD and reduce repetitive behaviors in ASD.[35,36] Our results support these earlier findings and further emphasize that specific vitamins may be more effective for certain conditions—namely, vitamin B for ASD and vitamin D for ADHD.

Our findings that vitamin B supplementation is particularly beneficial for individuals with ASD and vitamin D shows more pronounced effects in ADHD are supported by underlying neurophysiological mechanisms. Vitamin B, especially in its active form pyridoxal-5-phosphate, plays a vital role in synthesizing key neurotransmitters such as GABA, serotonin, and dopamine, which are involved in emotional regulation and behavior—domains often impaired in ASD.[15] Additionally, B vitamins participate in methylation processes affecting gene expression and neurodevelopment, potentially contributing to symptom improvement.[19]

Vitamin D functions as a neurosteroid, influencing brain regions such as the cerebellum, striatum, and hippocampus. It regulates synaptic proteins, neurotrophic factors, and signaling pathways like mTOR and the oxytocin system, which are implicated in attention and behavioral regulation, thereby offering a plausible explanation for its observed therapeutic effects in ADHD.

One reason past studies may not have explored this in depth is because most research focused on comparing patients to healthy controls, rather than examining how vitamin deficiencies relate to symptom improvements.[11,37,38] Additionally, many studies focused on just one vitamin, without considering multivitamin combinations or comparing different types of vitamins across disorders. Vitamin therapy is often perceived primarily as a nutritional supplement rather than a formal therapeutic intervention, which may contribute to relatively limited in-depth research in this area. Additionally, the low commercial value of vitamin formulations and restricted access to high-quality products may further reduce academic attention and investment. These factors together influence the clinical adoption and research intensity of vitamin therapy, warranting further investigation and validation in future studies.

However, it's exactly this simplicity and accessibility that make vitamin therapy so valuable—especially in today's overstretched healthcare systems. Our study demonstrates, through rigorous randomized placebo-controlled meta-analysis, that specific vitamins can have targeted benefits for different conditions. This makes vitamin therapy a cost-effective, easy-to-implement option that clinicians should seriously consider as part of routine care for ASD and ADHD.

Besides, one of the limitations of the present study arises from the scarcity of high-quality placebo-controlled trials that examine the effects of vitamins beyond B and D in ASD and ADHD. Another limitation is that the present study does not aim to provide a detailed review of all intricate biological mechanisms behind the observed improvements. Given that the pleiotropic effects of vitamin supplementation on various processes, the biological mechanism of ASD and ADHD should also be considered in the future.

That said, our study does have limitations. There's still a shortage of high-quality placebo-controlled trials looking at the effects of various vitamins in ASD and ADHD, particularly for vitamins beyond B and D. This limited the range of vitamins we could evaluate. We also lack a solid understanding of the underlying biological mechanisms behind these improvements. Finally, since different studies focus on different symptoms, it's hard to pinpoint which specific symptoms are most responsive to which vitamins—especially shared symptoms across both disorders. Future research should dive deeper into these questions, exploring both the mechanisms of action and the symptom-specific impacts of different vitamin therapies.

Moreover, there was significant heterogeneity among the included studies ($I^2 = 92\%$). However, due to inconsistent and insufficient reporting on participant age, disorder type, vitamin dosage and intervention duration, and outcome measures, only limited subgroup analyses were possible, and meta-regression analyses to explore sources of heterogeneity were not conducted. Second, we did not provide comprehensive tables of primary data (eg, means, standard deviations, and sample sizes), which affected the transparency and reproducibility of the analysis. Future studies should adopt stricter reporting standards, collect more detailed moderator information, and apply multivariate statistical methods such as meta-regression to more thoroughly investigate the heterogeneity of intervention effects and their underlying mechanisms, thereby enhancing the robustness of conclusions and their clinical relevance.

Conclusion

This meta-analysis supports the use of vitamin supplementation as a promising adjunctive treatment for ASD and ADHD. Vitamin B showed greater benefits in improving symptoms of ASD, while vitamin D was more effective in managing ADHD-related behaviors. These findings suggest that specific vitamins may target disorder-specific symptoms. Despite limitations such as the lack of trials on other vitamins and limited understanding of underlying mechanisms, vitamin therapy remains a low-cost, accessible option. Future research should further investigate the symptom-specific effects and biological pathways involved, to better inform personalized interventions for neurodevelopmental disorders.

Data Sharing Statement

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

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Disclosure

The authors report no conflicts of interest in this work.

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