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THE IMPACT OF VITAMIN B12 DEFICIENCY ON LEARNING PERFORMANCE AND NERVOUS SYSTEM FUNCTION IN SCHOOL-AGED CHILDREN

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ABSTRACT

Vitamin B12 (cobalamin) plays a critical role in the development of the central nervous system, myelin synthesis, and DNA biosynthesis. Recent epidemiological and clinical studies indicate that vitamin B12 deficiency in children is associated not only with hematological disorders but also with impairments in cognitive functions and academic performance. Evidence suggests that children with B12 deficiency exhibit significant reductions in attention, memory, and information processing speed, along with poorer academic achievement. For instance, children with low B12 levels have been reported to have a 2.36-fold higher risk of grade repetition. Neurologically, this deficiency may lead to peripheral neuropathy, ataxia, developmental delay, and impaired concentration. This article analyzes the impact of vitamin B12 deficiency on cognitive development, learning performance, and nervous system function in children based on contemporary scientific literature.

KEYWORDS

Vitamin B12, cobalamin, children, cognitive development, academic performance, nervous system, neurological disorders, myelination, attention, memory

Introduction

Vitamin B12, or cobalamin, is a water-soluble vitamin that serves as an essential cofactor in DNA synthesis, one-carbon metabolism, erythropoiesis, and, most importantly, the normal functioning of the central nervous system. Cobalamin is involved in the activity of methionine synthase and methylmalonyl-CoA mutase; therefore, its deficiency leads to disruptions in methylation processes, accumulation of homocysteine and methylmalonic acid, impaired myelin synthesis, and reduced neuronal signal transmission. Consequently, vitamin B12 deficiency should be regarded not merely as a hematological disorder but as a complex metabolic condition with significant neurological and neurocognitive consequences. According to the NIH Office of Dietary Supplements, neurological manifestations of B12 deficiency may occur even in the absence of anemia, underscoring the importance of early detection.

Childhood, particularly the school-age period, represents a critical phase of ontogenesis during which the cerebral cortex, executive functions, working memory, attention regulation, processing speed, and academic adaptation undergo active development. During this period, chronic micronutrient deficiencies may directly or indirectly impair key components of learning performance, including

sustained attention, task planning, memory retention, language processing, and consistent academic achievement. Vitamin B12 occupies a unique position in this context due to its close involvement in neurometabolism and myelination. A comprehensive review analyzing the relationship between cognition and B12 in children emphasized its essential role in brain development, nerve fiber myelination, and cognitive function.

The relevance of this issue is further reinforced by its widespread prevalence. The population prevalence of vitamin B12 deficiency varies considerably depending on the biomarker used, cutoff thresholds, and age group. According to NIH data, while clinically overt B12 deficiency is relatively rare, subclinical or marginal deficiency is much more common, reaching up to 40% in certain Western populations. In low- and middle-income countries, the risk is generally higher due to limited consumption of animal-derived foods. A multicenter cross-sectional study conducted among primary school children in Eastern Ethiopia reported that approximately 34% of participants had B12 deficiency. The authors further noted that the prevalence among children in developing countries often ranges between 21% and 45%.

These variations in epidemiology are not incidental. First, assessing B12 status in children requires not only total serum cobalamin but also functional biomarkers such as holotranscobalamin, methylmalonic acid, and total homocysteine. Second, dietary patterns, socioeconomic status, intestinal malabsorption, vegetarian diets, parasitic and inflammatory diseases, and certain medications may all influence B12 levels. BOND consensus reports and review articles highlight that differences in biomarker selection and non-standardized cutoff values complicate direct comparison across studies, necessitating methodological caution in interpreting findings.

From a practical standpoint, the significance of B12 deficiency in school-aged children is most clearly reflected in its association with academic performance. A cohort study conducted among schoolchildren in Colombia demonstrated that children with plasma B12 levels below 148 pmol/L had a 2.36-fold higher risk of grade repetition and a 1.89-fold higher risk of school absenteeism. These findings suggest that B12 deficiency may be directly linked to measurable educational outcomes. Importantly, while cognitive test scores do not always fully correspond to real-world academic success, indicators such as grade repetition and absenteeism represent more robust measures of educational adaptation.

Recent studies have further clarified the relationship between vitamin B12 and executive functions. Data from the GUSTO cohort (2025) indicate that higher plasma B12 levels in children aged 7–11 years are associated with better cognitive flexibility and working memory. The authors also reported potential associations between B12 levels and certain parameters of functional brain connectivity. These findings allow vitamin B12 to be conceptualized not merely as a general “cognitive vitamin,” but as a biologically relevant factor linked to specific components of executive functioning. From an educational perspective, working memory, cognitive flexibility, and inhibitory control are central to task execution, reading comprehension, and mathematical problem-solving.

Objective of the Study

To conduct a systematic analysis of existing scientific evidence on the impact of vitamin B12 deficiency on learning performance and central nervous system functions in school-aged children, and to evaluate its clinical and hygienic significance.

Materials and Methods

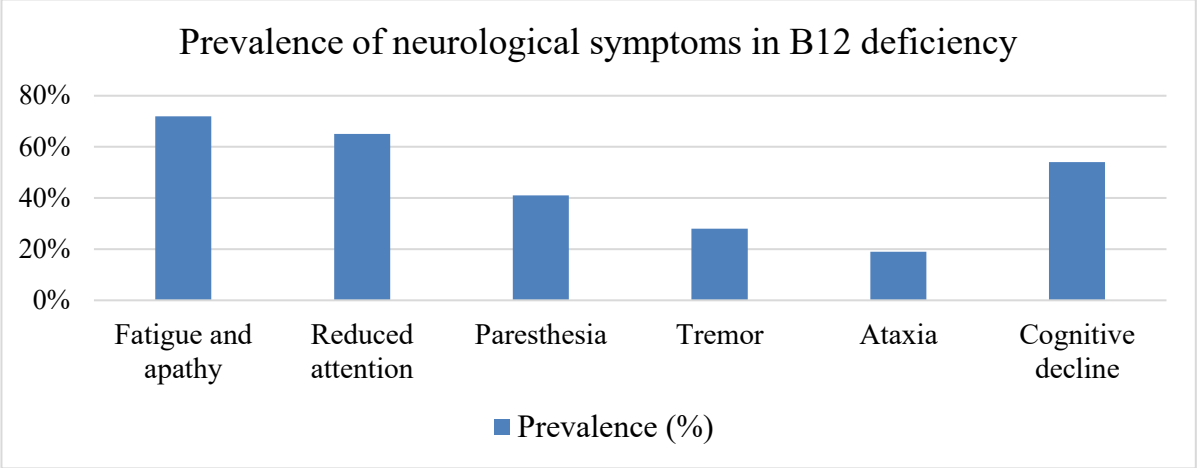
The literature review was conducted using scientific publications indexed in PubMed, Scopus, Web of Science, and ScienceDirect databases. The analysis included epidemiological studies, cohort observations, randomized controlled trials, and systematic reviews published between 2005 and 2025.
The inclusion criteria comprised: school-aged children (6–18 years); assessment of the relationship between vitamin B12 levels and cognitive functions; and evaluation of academic performance indicators, including memory, attention, and IQ scores.
Both qualitative and quantitative findings were assessed using a comparative analytical approach.

Results

The analysis of the literature indicates that vitamin B12 deficiency in school-aged children affects nervous system function and academic performance across several domains: cognitive functions, academic outcomes, and neurological symptomatology.
Association between B12 levels and cognitive functions. Numerous epidemiological and cohort studies demonstrate a positive correlation between vitamin B12 levels and cognitive performance.
In the GUSTO cohort study conducted in Singapore (children aged 7–11 years), higher plasma B12 levels were significantly associated with better working memory and cognitive flexibility ($p < 0.05$).
Similarly, studies conducted in India and Nepal have linked low B12 levels with reduced attention span, slower information processing speed, and impaired short-term memory.
These findings are explained by neurometabolic mechanisms, including impaired myelination and elevated homocysteine levels.
Vitamin B12 deficiency and academic performance. A cohort study among schoolchildren in Colombia demonstrated the following associations:
Association between B12 levels and academic outcomes

Indicator	Normal B12	Low B12 (<148 pmol/L)	Relative Risk (RR)
Grade repetition	8.5%	19.7%	2.36
School dropout	5.2%	9.8%	1.89
Low academic performance	14.3%	28.1%	1.96

These findings indicate that B12 deficiency significantly affects real-world educational indicators such as retention and dropout rates.
B12 levels and neurological symptoms. Based on pediatric clinical observations, the following neurological manifestations have been reported in children with B12 deficiency.



These symptoms interfere with active participation in the learning process and are often misinterpreted as purely psychological conditions.

Biochemical markers and cognitive outcomes. Elevated homocysteine levels represent a key pathogenetic mechanism in B12 deficiency. Studies indicate that increased homocysteine is associated with lower IQ scores, while elevated methylmalonic acid contributes to neuronal dysfunction.

Biochemical markers and cognitive effects

Parameter	**Change**	**Cognitive impact**
Vitamin B12	↓	Decreased memory and attention
Homocysteine	↑	Neurotoxic effects
Methylmalonic acid	↑	Impaired myelination

Findings from interventional studies. Results from randomized controlled trials are inconsistent. Some studies report improvements in memory and attention following B12 supplementation, whereas others show no statistically significant effects.

For example, a randomized trial conducted in Nepal found no significant improvement in cognitive outcomes among children receiving B12 and folate supplementation ($p > 0.05$).

This inconsistency may be explained by several factors:

– the presence of multiple micronutrient deficiencies

– short duration of follow-up

– baseline differences in cognitive performance among study groups

Discussion

The analyzed literature indicates that vitamin B12 deficiency may influence learning performance and nervous system function in school-aged children; however, the magnitude, direction, and clinical expression of this effect are not consistent across studies. A key observation is that the available evidence operates on two distinct levels: on one hand, the biological mechanisms are highly robust; on the other, population-based and clinical outcomes remain heterogeneous. Vitamin B12 plays a central role in neurometabolism, DNA synthesis, methylation reactions, myelin formation, and homocysteine metabolism. Therefore, its deficiency is expected—both theoretically and physiologically—to be associated with cognitive decline, reduced attention, and neurological manifestations. A major review

published in 2016 similarly concluded that while B12 is essential for brain development and cognitive function, cognitive outcomes in children vary substantially across studies.

The practical significance of these findings is most clearly demonstrated in studies examining academic outcomes. A prospective study conducted in Bogotá reported that B12 deficiency was associated with a 2.36-fold increase in the risk of grade repetition and a 1.89-fold increase in school absenteeism. Importantly, these associations persisted even after adjustment for folate, iron, zinc, and vitamin A biomarkers. This suggests that B12 deficiency may not merely reflect generalized poor nutrition but could act as an independent contributor to academic performance. From this perspective, academic achievement should not be viewed solely as a pedagogical or social construct; in certain cases, it may also be influenced by underlying biochemical factors.

However, oversimplifying this relationship would be scientifically inappropriate. The association between B12 deficiency and poor academic performance cannot be immediately interpreted as causal. B12 status is frequently correlated with confounding variables such as socioeconomic disadvantage, low intake of animal-source foods, limited parental education, chronic infections, malabsorption syndromes, and coexisting micronutrient deficiencies. A study among Colombian schoolchildren demonstrated a strong association between B12 levels, socioeconomic status, and dietary patterns. Consequently, part of the observed decline in cognitive or academic outcomes may be attributable not directly to B12 deficiency but to broader social and nutritional determinants that coexist with it. In this context, attributing complex developmental outcomes to a single micronutrient risks oversimplification.

From a neurological standpoint, the evidence further strengthens the clinical relevance of B12 deficiency. Pediatric clinical observations have documented symptoms such as dizziness, syncope, seizures, tremor, ataxia, paresthesia, visual disturbances, fatigue, and impaired concentration in children with B12 deficiency. These findings suggest that non-specific complaints commonly observed in school-aged children—such as fatigue, inattentiveness, and poor memory—may, in some cases, reflect underlying neurometabolic disturbances rather than purely behavioral or psychological issues. In clinical practice, this represents a critical point of potential diagnostic error: manifestations often interpreted as behavioral or academic problems may, in fact, be expressions of a biochemical deficiency requiring laboratory evaluation.

More recent evidence has begun to characterize the relationship between B12 and cognition at a more refined level. Data from the GUSTO cohort indicate that higher plasma B12 levels in children aged 7–11 years are associated with specific executive functions, particularly working memory and cognitive flexibility. This distinction is important, as academic performance is driven less by global IQ and more by executive processes such as task planning, rule switching, sustained attention, and temporary information processing. Thus, the impact of B12 deficiency may not manifest as generalized intellectual decline but rather through subtle impairments in cognitive domains that are critical for effective learning.

At the same time, interventional studies do not consistently confirm the associations observed in observational research. A study from India, combining observational and randomized designs with follow-up into mid-childhood, found that early-life supplementation with B12 and/or folate did not produce strong or consistent improvements in later cognitive outcomes. A key implication of this work is that biological plausibility and observational associations do not always translate into measurable clinical benefits. Several explanations may account for this discrepancy: the timing of supplementation

may be suboptimal (i.e., intervention initiated too late), cognitive development may depend on complex interactions within a broader socio-biological environment, and baseline deficiency levels in the intervention group may not have been sufficiently severe to demonstrate a measurable effect. Therefore, the assumption that B12 supplementation alone can universally improve academic performance is not currently supported by robust evidence.

Additionally, methodological variability in assessing B12 status represents a major source of inconsistency across studies. Many investigations rely solely on total serum B12 measurements, whereas functional deficiency is more accurately reflected by biomarkers such as methylmalonic acid, total homocysteine, and holotranscobalamin. The BOND review specifically highlights that differences in biomarker selection and cutoff thresholds significantly limit comparability between studies. As a result, a child classified as "deficient" in one study may be considered "borderline" or even "normal" in another. Under such conditions, statistical heterogeneity is not only expected but inevitable.

Conclusion

Vitamin B12 deficiency in school-aged children represents a condition of significance not only from a hematological perspective but also in terms of neurological and cognitive outcomes. The reviewed literature indicates that this deficiency may be associated with impairments in attention, working memory, academic performance, and certain aspects of nervous system function; however, this relationship is not consistently confirmed across all studies.

1. The biological basis of vitamin B12 deficiency is well established. Cobalamin plays a central role in DNA synthesis, methylation reactions, homocysteine metabolism, and the myelination of nerve fibers. Consequently, its deficiency may lead to neurometabolic disturbances, slowed neural impulse transmission, and reduced cognitive performance. The literature reports that affected children may exhibit decreased attention, increased fatigue, impaired memory, paresthesia, dizziness, and, in some cases, ataxia. These findings support the interpretation of B12 deficiency as a neurobiological factor that may directly, rather than indirectly, influence learning performance.

2. Available epidemiological and clinical data suggest that children with low B12 levels are more likely to demonstrate poorer academic outcomes. Cohort studies have linked B12 deficiency with increased rates of grade repetition, reduced school attendance, and impaired executive functioning. In particular, working memory, cognitive flexibility, and sustained attention—key determinants of everyday academic success—appear to be sensitive to B12 status. At the same time, it is essential to account for confounding factors such as socioeconomic conditions, dietary quality, coexisting micronutrient deficiencies, and overall health status. Thus, while B12 deficiency is unlikely to be the sole cause of poor academic performance, it may represent an important modifiable risk factor.

3. More methodologically robust research is required to clarify the impact of B12 deficiency on learning performance and nervous system function in children. A major limitation of existing studies lies in the heterogeneity of biomarkers, variability in cutoff values, and the frequent reliance on total serum B12 alone. In contrast, functional indicators such as methylmalonic acid, homocysteine, and holotranscobalamin provide a more accurate assessment of deficiency. Furthermore, randomized controlled trials have produced inconsistent findings regarding the cognitive benefits of B12 supplementation. Future research should therefore incorporate standardized laboratory criteria, long-term follow-up, and objective educational outcomes.

In summary, vitamin B12 deficiency is a clinically and hygienically significant condition that may affect both nervous system function and academic performance in school-aged children. Early detection, identification of at-risk groups, dietary optimization, and appropriate correction strategies constitute essential preventive measures aimed at preserving cognitive potential and improving educational outcomes.

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