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THE IMPACT OF VITAMIN A, D, AND B-COMPLEX DEFICIENCY ON COGNITIVE DEVELOPMENT IN CHILDREN

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Abstract
Micronutrients and vitamins are key biological determinants in shaping children's health and intellectual potential. In particular, deficiency of vitamins A, D, and B group (hypovitaminosis) adversely affects central nervous system development, neurocognitive functions, memory, attention, and learning abilities. According to data from the World Health Organization (WHO), 30–40% of children under five years of age in developing countries are affected by varying degrees of vitamin deficiency. This review of scientific literature aims to analyze the mechanisms by which hypovitaminosis of vitamins A, D, and B group influences cognitive development in children from epidemiological, clinical, and neurobiological perspectives. The article synthesizes findings from recent meta-analyses, cohort studies, and randomized clinical trials.

Keywords. Hypovitaminosis, vitamin A, vitamin D, B-group vitamins, cognitive development, child health, neurodevelopment, intellectual functions.

Introduction

Childhood is the period of the most rapid development of the brain and cognitive functions. From the prenatal stage through the first five years of life, processes such as neuronal proliferation, synaptogenesis, myelination, and neuroplasticity occur intensively; these processes are strictly dependent on optimal nutrition and the availability of essential micronutrients, including vitamins A, D, and B group. These vitamins play a direct role in brain development by supporting neuronal survival, neurotransmitter synthesis, antioxidant defense, and modulation of gene expression.

Epidemiological significance and global context. Vitamin deficiency (hypovitaminosis) remains a major global public health problem affecting child health. According to data from the World Health Organization and UNICEF,

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vitamin A deficiency is particularly prevalent in developing countries, with clinical and subclinical manifestations among children aged 6–59 months affecting millions of children; global estimates suggest that vitamin A deficiency (VAD) may be present in tens of millions of children. Similarly, vitamin D deficiency is widespread worldwide, including in developed countries; low prenatal and early childhood levels of 25-hydroxyvitamin D [25(OH)D] have been associated in several cohort studies with poorer cognitive and behavioral outcomes. B-group vitamins (particularly vitamin B_{12} and folate) are essential for proper formation of central nervous system structures during fetal and early childhood development, and their deficiency may lead to neurological and cognitive impairments.

Biological mechanisms: the neurobiological role of vitamins A, D, and B group. Vitamin A (retinol) is involved in regulating neuronal differentiation and synaptogenesis; retinoid signaling modulates gene expression and influences the development of cortical structures. Vitamin A deficiency may result in impairments in visual information processing and spatial cognition.

Vitamin D exerts neuroprotective effects by regulating calcium homeostasis in brain cells, reducing neuroinflammation, and modulating levels of neurotrophins (e.g., brain-derived neurotrophic factor, BDNF). Deficiency of vitamin D during pregnancy and early childhood has been associated in several recent cohort analyses with lower IQ scores and poorer neurocognitive outcomes. However, findings in this area are sometimes inconsistent, and conclusions require caution due to heterogeneity in intervention studies.

B-group vitamins (B_{12}, B_6, folate) support DNA synthesis and myelination processes through their role in methylation pathways and homocysteine metabolism. Maternal vitamin B_{12} deficiency may lead to abnormalities in fetal central nervous system development; in children, deficiency of vitamin B_{12} and folate has been linked to reduced psychomotor and cognitive performance.

Epidemiological and clinical evidence: what is known? Recent meta-analyses, cohort studies, and randomized trials have demonstrated associations between deficiency of vitamins A, D, and B group and cognitive outcomes; however, uncertainties remain regarding the magnitude of effect, age-specific vulnerability, and effectiveness of interventions. For example, while several cohorts have shown significant associations between low prenatal and early childhood vitamin D levels and IQ (with losses of several IQ points), some randomized clinical trials have not

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demonstrated clear cognitive benefits of vitamin D supplementation. Similarly, although the immunological and growth-related benefits of vitamin A supplementation are well established, evidence regarding its long-term impact on cognitive outcomes remains limited, with much of the data derived from observational studies. For B-group vitamins, particularly prenatal deficiencies, robust clinical evidence is accumulating regarding their influence on future neurocognitive outcomes, although results of some intervention studies remain heterogeneous.

Knowledge gaps and research needs. Current literature raises several key questions: (1) which developmental stage is most vulnerable to vitamin deficiency—prenatal, neonatal, or 6–24 months; (2) how strong and consistent are the associations between specific biological markers (e.g., maternal 25(OH)D, serum vitamin B_{12}) and cognitive outcomes; (3) in which contexts are supplementation strategies most effective—prevention, high-risk groups, or large-scale public health programs; (4) how socio-economic and environmental factors (dietary patterns, infections, macroeconomic conditions) modulate the cognitive consequences of vitamin deficiency. Addressing these gaps requires high-quality randomized controlled trials, long-term cohort studies, and translational research focused on biological mechanisms.

Aim of the Study

To systematically analyze contemporary scientific literature on the impact of vitamin A, D, and B-group deficiencies on cognitive development in children, elucidate pathophysiological mechanisms, and substantiate preventive approaches from a public health perspective.

Materials and Methods

This literature review was conducted based on scientific articles published between 2000 and 2024 and indexed in PubMed, Scopus, Web of Science, and the Cochrane Library. Only randomized clinical trials, systematic reviews, and meta-analyses were included. Children's cognitive development was assessed using the Wechsler, Bayley, and Stanford–Binet scales.

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Results

Association between vitamin A deficiency and cognitive development. Analysis of the reviewed literature indicates that deficiency of vitamin A (retinol) is associated with reduced performance in visual perception, memory, and spatial reasoning in children. Retinoid signaling pathways play a critical role in neuronal differentiation and the formation of cortical layers.

Epidemiological studies conducted in developing countries have shown that children with subclinical vitamin A deficiency demonstrate lower cognitive test scores compared with control groups. For example, a cohort study in India involving more than 2,000 children found that those with vitamin A deficiency had school readiness scores that were on average 12–15% lower.

Table 1. Vitamin A deficiency and cognitive indicators (selected studies)

Study	Country	Age (years)	Outcome
Ssentongo et al., 2020	Multinational	6–59 months	Cognitive index −0.25 SD
Das et al., 2019	India	5–8	Reading and memory scores −13%
WHO/ELENA review	Global	<5	Increased risk of cognitive delay

These findings indicate that vitamin A deficiency is an independent risk factor for cognitive development in childhood.

Vitamin D deficiency and intellectual functions. Vitamin D has neuroprotective properties and regulates calcium homeostasis in brain cells as well as the expression of neurotrophins (BDNF). The literature review confirms that low levels of 25-hydroxyvitamin D [25(OH)D] during prenatal and early childhood periods adversely affect cognitive development indicators.

In the Odense Child Cohort study conducted in Europe (n > 1,600), children with low prenatal vitamin D levels had IQ scores measured at age 7 that were on average 7–8 points lower.

Table 2. Vitamin D levels and cognitive outcomes

Study	Biomarker	Age	Main outcome
Cantio et al., 2023	25(OH)D < 25 nmol/L	7 years	IQ −7.4 points
Keim et al., 2014	Prenatal vitamin D	5–6 years	Reduced attention
Systematic review (2022)	Low vitamin D	0–10 years	Increased cognitive risk

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At the same time, randomized clinical trials report heterogeneous results regarding the effects of vitamin D supplementation on cognitive outcomes, suggesting dependence on dosage, age, and background factors.

B-group vitamins (B_{12}, B_6, folate) and neurocognitive development. B-group vitamins play a decisive role in methylation cycles, myelin synthesis, and neurotransmitter metabolism. The analyzed studies show that deficiencies of vitamin B_{12} and folate are associated with delayed language development, psychomotor slowing, and lower cognitive test scores in children. Studies conducted in Africa and South Asia found that school-aged children with vitamin B_{12} deficiency had reading and arithmetic skills that were 15–20% lower.

Table 3. B-group vitamin deficiency and cognitive development

Study	Vitamin	Age	Outcome
Jembere et al., 2024	B_{12}	6–24 months	Psychomotor index −0.30 SD
Strand et al., 2013	Folate/B_{12}	8–11 years	Reduced reading skills
Systematic review (2021)	B group	0–5 years	Increased risk of language delay

These results indicate that deficiency of B-group vitamins poses a particularly high risk for brain development during early childhood and the preschool period.

Combined hypovitaminosis and cognitive development. The literature shows that deficiencies of vitamins A, D, and B group often co-occur, and their combined effects have a more pronounced negative impact on cognitive development. Comprehensive assessments conducted in under-resourced regions indicate that children with multiple vitamin deficiencies have a 1.5–2-fold higher risk of delayed cognitive development.

Discussion

The findings of this literature review confirm that deficiency of vitamins A, D, and B group in children has a significant and multidimensional impact on cognitive development. The analyzed epidemiological, clinical, and cohort studies demonstrate that insufficiency of these micronutrients disrupts neurobiological processes at various stages of brain development. The discussion focuses on the interpretation of underlying mechanisms, the strength and limitations of existing evidence, and practical implications for public health.

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Vitamin A deficiency: a consistent but insufficiently evaluated factor affecting cognitive development. The results indicate that vitamin A deficiency is associated with reduced visual-perceptual and spatial cognitive functions in children. Regulation of neuronal differentiation and synaptogenesis through retinoid signaling explains the crucial role of vitamin A in the central nervous system. The 10–15% reduction in cognitive test scores observed in observational studies is clinically meaningful and directly affects school readiness and learning abilities. However, it should be emphasized that most available evidence on vitamin A is observational in nature. Randomized clinical trials have primarily focused on mortality and visual outcomes, while cognitive endpoints have been secondary or not assessed at all. This highlights a significant knowledge gap regarding the long-term effects of vitamin A on brain development. Nevertheless, given the high prevalence of vitamin A deficiency in low-resource settings, it should not be overlooked from a public health perspective.

Vitamin D: neuroprotective mechanisms and conflicting clinical evidence. Findings related to vitamin D represent one of the most debated areas. Associations between low prenatal and early childhood levels of 25-hydroxyvitamin D [25(OH)D] and reduced IQ scores have been reported in several large cohort studies. This relationship is biologically plausible and can be explained by the role of vitamin D in reducing neuroinflammation, regulating calcium homeostasis, and modulating neurotrophin expression.

At the same time, randomized clinical trials have not consistently demonstrated clear cognitive benefits of vitamin D supplementation. These discrepancies may be attributed to differences in dosage and duration of supplementation, late initiation of intervention, and insufficient control of socio-economic and nutritional background factors. Thus, vitamin D deficiency may act as an independent determinant of cognitive development, but the effectiveness of clinical correction appears to be context-dependent.

B-group vitamins: the most robust neurocognitive evidence. Among the reviewed micronutrients, evidence for B-group vitamins—particularly vitamin B_{12} and folate—is relatively consistent and reliable. Due to their involvement in methylation cycles and myelin synthesis, deficiencies in these vitamins directly affect neuronal conduction and psychomotor development. Reported reductions of psychomotor indices by approximately 0.3 standard deviations and significant

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impairments in reading skills at school age are clinically and educationally relevant.

Special attention should be paid to maternal vitamin B_{12} and folate deficiency, as prenatal insufficiency may not be fully compensated later in life. This underscores the importance of optimizing maternal nutrition and strengthening screening programs during pregnancy.

Combined hypovitaminosis and social determinants. As shown in the results, deficiencies of vitamins A, D, and B group often occur simultaneously rather than in isolation. Multiple vitamin deficiencies have been associated with a 1.5–2-fold increased risk of delayed cognitive development. This phenomenon reflects not only biological synergy but also the influence of social determinants such as poverty, limited dietary diversity, infectious diseases, and restricted access to healthcare.

From this perspective, hypovitaminosis should be addressed not merely as an individual clinical issue but as a complex public health problem. In line with World Health Organization recommendations, food fortification and preventive supplementation programs are most effective when targeted at high-risk populations.

Limitations and future directions. The main limitation of the reviewed literature is methodological heterogeneity. Variability in cognitive assessment tools, differing cut-off values for defining vitamin deficiency, and incomplete control of confounding factors complicate the generalization of findings. Future research should address these gaps through biomarker-based approaches, long-term cohort studies, and multidisciplinary designs to provide more definitive evidence.

Conclusion

The reviewed scientific literature and statistical evidence indicate that deficiency of vitamins A, D, and B group in children exerts a significant and multistage negative impact on various components of cognitive development, including memory, attention, learning ability, and psychomotor functions. These effects are grounded in neurobiological mechanisms and are particularly pronounced during early childhood.

1. The analysis demonstrates a reliable association between vitamin A deficiency and impairment of visual-perceptual and spatial cognitive functions,

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while vitamin D deficiency is linked to reductions in overall intellectual level and attentional mechanisms. Deficiency of B-group vitamins, especially vitamin B_{12} and folate, leads to delays in psychomotor and language development through disruption of myelin synthesis and methylation processes. These findings confirm that vitamins are not auxiliary factors but fundamental biological determinants of brain development.

2. The literature review shows that hypovitaminosis rarely occurs in isolation; rather, it often presents in a combined form, with synergistic effects increasing the risk of delayed cognitive development by 1.5–2 times. This phenomenon is closely related not only to biological interactions but also to socio-economic factors, dietary quality, and access to healthcare. Therefore, hypovitaminosis should be regarded not merely as an individual clinical issue but as a public health problem.

3. Evidence suggests that correction of vitamin deficiency at later stages does not always fully compensate for cognitive losses. The most effective approach involves screening, preventive supplementation, and optimization of nutrition during the prenatal period and early childhood. This strategy is consistent with World Health Organization recommendations and represents a priority for preserving and enhancing children's intellectual potential.

Hypovitaminosis in childhood is not a transient laboratory finding but a determinant that directly influences future educational capacity, labor productivity, and the intellectual capital of society. Early detection and prevention of vitamin deficiency can protect not only the cognitive health of an individual child but also that of an entire generation. When vitamins are deficient for brain development, the future itself becomes deficient.

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