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Vitamin B12 Deficiency in School-Aged Children: Prevalence, Causes, Clinical Consequences, and Treatment Recommendations

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Abstract

Vitamin B12 deficiency in school-aged children is a common but underdiagnosed condition. Depending on the region and dietary habits, the prevalence of subclinical deficiency in at-risk groups reaches 20-40%, which globally corresponds to tens of millions of children. The main causes of deficiency are nutritional factors (insufficient intake of animal products), malabsorption due to gastrointestinal diseases (coeliac disease, Crohn's disease, autoimmune gastritis), competitive consumption (small intestinal bacterial overgrowth, parasitic infestations), drug-induced causes (proton pump inhibitors, metformin), as well as rare hereditary disorders of cobalamin transport and metabolism. The key clinical feature of B12 deficiency in schoolchildren is its ability to present as isolated cognitive impairment (reduced attention, working memory, and information processing speed) in the absence of anaemia and with normal total serum B12 levels. The most sensitive diagnostic method is the measurement of active B12 (holotranscobalamin) in combination with homocysteine. Treatment includes parenteral (500-1000 µg of cyanocobalamin or methylcobalamin) or oral (500-1000 µg/day) administration of the vitamin depending on the severity of deficiency and the presence of malabsorption. Neurological manifestations are reversible with timely therapy. The main barriers to timely diagnosis are low awareness among physicians and the general public regarding the cognitive manifestations of B12 deficiency, the absence of screening protocols for at-risk groups, and insufficient laboratory provision. Vitamin B12 is one of those micronutrients whose deficiency can be completely corrected by affordable means; timely detection and adequate therapy can restore cognitive function and prevent long-term consequences for academic performance and psychoemotional development in children.

Keywords: Vitamin B12, cobalamin, Deficiency, School-aged children, Cognitive impairment, Holotranscobalamin

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Introduction

Vitamin B12 (cobalamin) is a water-soluble micronutrient unique in its chemical structure, which contains a cobalt ion (Green *et al*., 2017; Paez-Hurtado *et al*., 2023). Unlike most water-soluble vitamins, B12 can accumulate in the body, with the liver serving as the main depot, storing 2 to 5 mg of the vitamin in a healthy adult (Smith *et al*., 2018; Aşkın *et al*., 2021). Due to efficient enterohepatic recirculation and slow excretion, when B12 intake completely ceases, the body stores are depleted over several years in adults; however, in children, given their younger age and consequently smaller total pool, deficiency may develop more rapidly (Hasbaoui *et al*., 2021; Patel & McGuirk, 2025). The

physiological role of B12 in humans is mediated through its participation as a cofactor for two key enzymes: methionine synthase and methylmalonyl-CoA mutase (Mascarenhas *et al*., 2022; McCorvie *et al*., 2023). In the first case, B12 is required for the conversion of homocysteine to methionine, a reaction that underlies methionine synthesis and, through it, the synthesis of many other biologically active compounds, while also playing a central role in the folate cycle and DNA synthesis. In the second case, B12 is involved in fatty acid metabolism and maintaining the integrity of the myelin sheath of nerve fibres (Kräutler, 2012; Bito *et al*., 2020; Sokolovskaya *et al*., 2021). Thus, B12 deficiency can lead to disturbances in three key systems: haematopoietic (with development of megaloblastic anaemia), nervous (with demyelination, peripheral neuropathy and damage to the posterior and lateral columns of the spinal cord – subacute combined degeneration of the spinal cord) and gastrointestinal (atrophic glossitis, malabsorption) (Bito & Watanabe, 2016; Osmola *et al*., 2024; Tagore *et al*., 2025). In addition, growing research interest has been directed toward natural bioactive compounds and plant-derived molecules with potential systemic biological effects, including modulation of metabolic and neurological processes (Hamad *et al*., 2018; Ahmed *et al*., 2020).

Critically important for understanding the modern diagnosis of B12 deficiency is the distinction between total serum B12 and its biologically active fraction, known as holotranscobalamin or active B12 (Tufail *et al*., 2024). Two main forms of B12 circulate in the blood, bound to different carrier proteins (Brokner *et al*., 2017; Verma *et al*., 2023; Al-Mousawi *et al*., 2024). The main differences between total and active B12 are summarised in **Table 1**.

Table 1. Comparative characteristics of total and active vitamin B12 in the diagnosis of deficiency

Parameter	Total serum B12	Active B12 (holotranscobalamin, HoloTC)
Carrier protein	Haptocorrin (HC)	Transcobalamin (TC)
Proportion of total blood B12	70-80%	20-30%
Cellular availability	No (complex not recognized by cellular receptors)	Yes (specific receptors on all cells)
Metabolic activity	Inert form (liver depot)	Only the biologically active form
What it reflects	Total body stores (including "storage" pool)	True tissue-level deficiency
When it decreases	With prolonged depletion of stores (months to years)	In the early stages of deficiency (weeks)
Deficiency threshold (in children)	<200-250 pg/mL (laboratory-dependent)	<25-35 pmol/L
Optimal level	> 400-500 pg/mL	> 50 pmol/L

As follows from **Table 1**, only the B12-transcobalamin complex (holotranscobalamin) is able to bind to cellular receptors and enter tissues, where its metabolic activity is realised. Total serum B12 mainly reflects the pool bound to haptocorrin, which is metabolically inert and can remain within the normal range even when tissue deficiency has already developed (Hannibal *et al*., 2016; Zhang *et al*., 2022). In contrast, the level of active B12 decreases early and serves as the most sensitive marker of true deficiency at a stage when total B12 has not yet fallen below the reference values (Lorde *et al*., 2024; Harrington *et al*., 2025). Reference intervals for active B12 in children are not sufficiently unified; however, most laboratories use a deficiency threshold below 25-35 pmol/L, while values above 50 pmol/L are generally considered normal (Wiedeman *et al*., 2023; Kalayci & Yigit, 2024; Ata & Tas, 2025).

An additional and extremely important metabolic marker is homocysteine (Zaric *et al*., 2019; Hermann & Sitdikova, 2021; Smith & Refsum, 2021). Since B12 acts as a cofactor for methionine synthase, which converts homocysteine to methionine, B12 deficiency leads to the accumulation of homocysteine in the blood. Elevated homocysteine is regarded as a functional marker of B12 deficiency that may appear earlier than the decrease in the vitamin itself, and it possesses its own toxicity: hyperhomocysteinaemia damages the vascular endothelium, promotes thrombosis, and exerts a direct neurotoxic effect (Kim *et al*., 2018; Guieu *et al*., 2022; Guéant *et al*., 2023). In children, threshold values for homocysteine are less standardised than in adults, but levels above 10-12 μmol/L are often considered to warrant attention, especially in the presence of neurological symptoms or anaemia (Marroncini *et al*., 2024; Huang *et al*., 2025).

The prevalence of B12 deficiency among school-aged children varies widely depending on the geographic region, dietary habits, and socioeconomic conditions (Aureli *et al*., 2023). A study conducted in Istanbul on a sample of 234 children aged 6-18 years found a mean B12 level of 261.5±162.9 pg/mL, with deficiency detected in 40.6% of participants, significantly more often in boys and in adolescents aged 12-18 years. The prevalence of anaemia in the same sample was 9.4%, with a marked predominance among girls (13.4% vs. 5.2% in boys) (Culha Hosceylan *et al*., 2025). Within the large-scale BRINDA project, which covered 54,534 children from 17 studies in 16 countries, the prevalence of B12 deficiency among children aged 5-19 years ranged from 0% in the United Kingdom and the United States to 3.1% in Colombia and 0.3% in Mexico (Young *et al*., 2020; Werner *et al*., 2025). However, these data mainly reflect countries with a high intake of animal products. In sub-Saharan Africa, the situation is much worse: among children aged 3-5 years, B12 deficiency reached 27.3% in Côte d'Ivoire, 29.4% in Liberia and 16.3% in Cameroon. The overall prevalence of anaemia among children aged 5-19 years, according to BRINDA, was a median of 16%, with a marked increase in the 15-19 years age group to 22%, especially among girls. It is important to emphasise that the direct contribution of B12 deficiency to the anaemia burden varies across regions; in most countries with a high intake of animal protein, the association between low B12 and anaemia is weak, whereas in populations with a high prevalence of vegetarianism or veganism, this

association becomes significant (Rizzo *et al*., 2016; Fernandes *et al*., 2024).

Studies conducted in various regions of Russia indicate that the problem of deficiencies of B vitamins, including B12, is also relevant for the Russian paediatric population. A study analysing blood parameters of 146 children attending preschool educational institutions showed that half of the examined children had year-round vitamin B12 deficiency (Yambulatov, 2018). The authors also noted that 75-85% of the children had a deficiency of two or more vitamins, and 40% had polyhypovitaminosis. In the Voronezh region, among children with anaemia hospitalised in inpatient settings, the proportion of B12-deficient and folate-deficient conditions was 0.26%, with the vast majority (86.7%) occurring in the older age group, which the authors attribute to nutritional factors and vegetarian diets among adolescents (Bogatikova *et al*., 2025). Children with restrictive dietary patterns deserve special attention. In a study of school-aged children living in Donbas who followed vegetarian and dairy-free diets, B12 deficiency was detected in 15% of children on a dairy-free diet and in 30% of vegetarian children (Svistunova *et al*., 2023).

Thus, despite the absence of a unified federal epidemiological registry, regional studies and data from laboratory services convincingly demonstrate that vitamin B12 deficiency occurs among Russian children, increases in older age groups, and is closely related to dietary patterns. This review aims to systematize current data on the prevalence, etiology, clinical consequences, and approaches to the diagnosis and treatment of vitamin B12 deficiency in school-aged children, as well as to assess the socio-economic significance of this problem for paediatric practice and public health.

Causes of Vitamin B12 Deficiency in School-Aged Children

Understanding the aetiology of vitamin B12 deficiency in school-aged children is of fundamental importance for developing effective prevention and treatment strategies. Unlike in infants, where maternal status plays a leading role (Behere *et al*., 2021; Keskin *et al*., 2022; Finkelstein *et al*., 2024), in school-aged children the dominant factors are the vitamin stores accumulated by this age and the pattern of nutrition (Bawaskar & Bawaksar, 2020). However, it is important to note that children who experienced B12 deficiency in utero or during breastfeeding may have initially lower reserves and, consequently, increased vulnerability to deficiency at an older age in the setting of unfavourable dietary habits (Smith, 2018; Strand *et al*., 2018; Finkelstein *et al*., 2021). Thus, the influence of maternal deficiency in school-aged children is no longer a direct cause but may act as a modifying risk factor. A rational diagnostic and therapeutic approach is based on an aetiological classification that identifies five main mechanisms of B12 deficiency development in school-aged children.

Nutritional (Alimentary) Deficiency

The most common cause of B12 deficiency in school-aged children worldwide is insufficient dietary intake of the vitamin. B12 is found exclusively in animal-derived foods: meat, fish, eggs and dairy products (Obeid *et al*., 2019; Temova Rakuša *et al*., 2022; Taeger & Thiele, 2024). Prolonged exclusion of these foods from the diet leads to alimentary deficiency. Vegetarian and vegan diets represent the most obvious risk group: the stricter the dietary restrictions, the higher the probability of deficiency (Łuszczki *et al*., 2023; Wang *et al*., 2023; Malhotra & Lakade, 2025). However, even with a traditional diet, if the child's diet is poor in animal proteins and contains little meat and dairy products, the risk of deficiency is also increased. In school-aged children, particularly during adolescence, an additional risk factor is the increased requirement for the vitamin due to active growth, hormonal changes and increased muscle mass (Jensen, 2023; Lotti *et al*., 2026). Furthermore, at this age, eating pattern disturbances are common: skipping meals (especially breakfast), choosing foods of low nutritional value (fast food, processed products), and dieting for body weight control, particularly among adolescent girls. All these factors create preconditions for the development of occult or overt B12 deficiency even with a formally non-vegetarian diet.

Malabsorption (Impaired Absorption)

B12 absorption is a complex multi-step process. Normally, the vitamin ingested with food is released from protein binding in the stomach, where it binds to R-proteins (haptocorrin). Subsequently, in the small intestine under the action of pancreatic proteases, it is transferred to intrinsic factor – a glycoprotein secreted by gastric parietal cells (Green & Datta Mitra, 2017; Guéant *et al*., 2022). Only the B12-intrinsic factor complex is recognised by specific receptors in the terminal ileum and absorbed into the bloodstream. Disruption of any of these steps can lead to deficiency even with adequate dietary vitamin intake (Choudhury *et al*., 2023; Jajoo *et al*., 2024). In school-aged children, malabsorption of B12 can be caused by chronic gastritis, especially with mucosal atrophy (including autoimmune gastritis), which, although considered rare in children, may occur particularly in adolescents and is accompanied by reduced intrinsic factor secretion (Abosamra *et al*., 2023). Diseases of the small intestine – coeliac disease, Crohn's disease, ileal resection – impair absorption of the B12-intrinsic factor complex (Vici *et al*., 2016; Germani *et al*., 2021; Akbulut, 2022).

Competitive Consumption

A separate group of causes consists of conditions in which vitamin B12 is consumed by other organisms or microorganisms within the host's gastrointestinal tract. The best-known example is infestation with the fish tapeworm (Diphyllobothrium latum), although this parasitosis is less common in school-aged children than in adults and is associated with the consumption of raw or insufficiently cooked freshwater fish (Yoshida *et al*., 2025). Much more relevant for school-aged children is massive colonisation of the small intestine by bacteria in small intestinal bacterial overgrowth syndrome, which can develop after intestinal surgery, in the presence of diverticula, strictures, or in certain diseases that impair intestinal motility. Bacteria actively take up B12, making it unavailable for absorption by the host (Guardiola-Arévalo *et al*., 2025). Furthermore, competitive consumption may be observed in severe forms of dysbiosis, although the clinical significance of this

mechanism in school-aged children has not been definitively established (Pelc *et al*., 2025).

Drug-Induced Deficiency

Drug-related causes of B12 deficiency are less common in children than in adults, but their importance is growing with the increasing duration of therapy for chronic diseases in childhood. Long-term use of proton pump inhibitors (omeprazole, lansoprazole, esomeprazole and others), which are often prescribed to children with gastro-oesophageal reflux disease or chronic gastritis, can reduce gastric acid secretion. This impairs the release of B12 from food proteins, and with prolonged therapy (more than one year) can lead to decreased vitamin levels (Bell, 2022; Swarnakari *et al*., 2022; Ito *et al*., 2024). Another medication associated with B12 deficiency is metformin, used in children with type 2 diabetes mellitus, obesity, or polycystic ovary syndrome. Metformin impairs B12 absorption in the terminal ileum, and this effect is dose-dependent and increases with treatment duration. Although clinically significant deficiency due to metformin develops less frequently in children than in adults, regular monitoring of B12 levels is recommended during long-term therapy (Infante *et al*., 2021; Khattab *et al*., 2023; Sayedali *et al*., 2023; Ramzan *et al*., 2024). Antiepileptic drugs (phenytoin, carbamazepine, phenobarbital) may also affect B12 metabolism, although the mechanism of this interaction is not fully understood (Kathiravan *et al*., 2021; Youness *et al*., 2022).

Hereditary Metabolic Disorders

A rare but critically important cause of B12 deficiency in children, for timely diagnosis, is genetic defects affecting vitamin transport or intracellular utilisation. These include transcobalamin II deficiency (the protein responsible for transporting B12 in the blood), congenital selective B12 malabsorption (Imerslund-Gräsbeck syndrome, in which absorption of the B12-intrinsic factor complex in the ileum is impaired despite preserved intrinsic factor production), and various intracellular cobalamin metabolism defects leading to methylmalonic acidaemia and homocystinuria. These conditions usually present in early childhood, but mild forms may first manifest in school-aged children and adolescents. Therefore, in cases of unexplained B12 deficiency, especially in the absence of obvious alimentary causes and with refractoriness to standard therapy, hereditary disorders must be excluded. Treatment of such patients often requires lifelong parenteral administration of high-dose vitamin B12 (Kvezereli-Kopadze & Mtvarelidze, 2019; Ünal *et al*., 2019; Kose *et al*., 2020).

The diversity of causes of vitamin B12 deficiency in school-aged children requires an individualised diagnostic approach. In most children with vegetarian or unbalanced diets, the cause will be alimentary deficiency. However, in the absence of obvious dietary factors, or in recurrent or treatment-refractory deficiency, it is necessary to sequentially exclude malabsorption (coeliac disease, Crohn's disease), competitive consumption (small intestinal bacterial overgrowth, parasitic infestations), drug-related causes and, although rare, hereditary disorders of B12 metabolism. **Figure 1** summarises the key aetiological categories and their place in the diagnostic work-up of school-aged children.

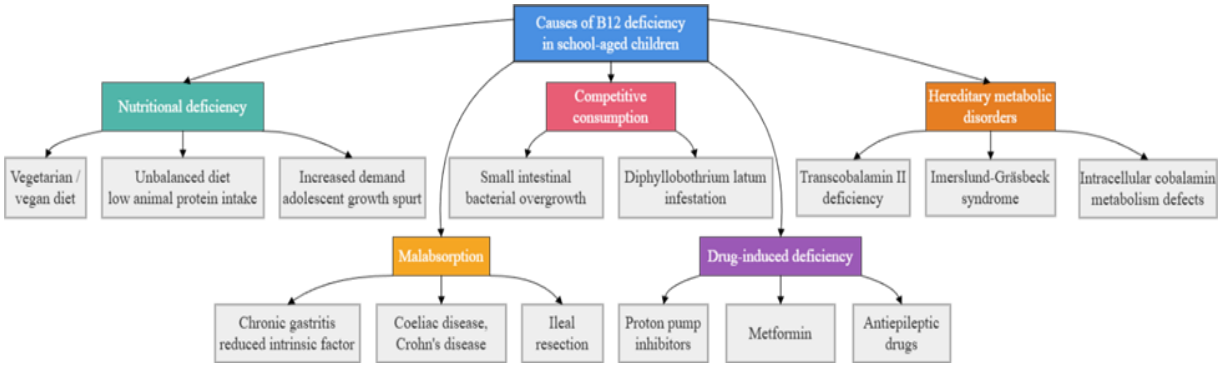

Figure 1. Aetiological categories of vitamin B12 deficiency in school-aged children

Consequences of Vitamin B12 Deficiency in School-Aged Children

Vitamin B12 deficiency in school-aged children can lead to a wide spectrum of clinical manifestations affecting the haematological, neurological and gastrointestinal systems. An important feature of childhood is that the consequences of B12 deficiency often extend beyond the classical picture of megaloblastic anaemia described in adults (De la Cruz-Góngora *et al*., 2021; Karabayir *et al*., 2022; Awasthi *et al*., 2023). In school-aged children, cognitive and behavioural disturbances frequently come to the forefront, remaining unrecognised for a long time or being mistakenly interpreted as pedagogical neglect, attention deficit hyperactivity disorder (ADHD) or emotional disorders (Venkatramanan *et al*., 2016; Lu & Paterson, 2025). Moreover, unlike in adults, neurological changes due to B12 deficiency in children can be reversible with timely diagnosis, but with prolonged deficiency, the risk of irreversible damage increases substantially (Kvestad *et al*., 2017; Cruz-Rodríguez *et al*., 2024).

Haematological Consequences

The classical manifestation of B12 deficiency is megaloblastic anaemia, the pathogenesis of which is based on impaired DNA synthesis in erythroid precursors in the bone marrow (Devi *et al*., 2026). In B12 deficiency, cell division is slowed while cytoplasmic synthesis continues, leading to the formation of large but functionally defective red blood cells – macrocytes and

megaloblasts. Complete blood count reveals decreased haemoglobin and red blood cell counts, elevated mean corpuscular volume (MCV > 100 fL), as well as characteristic morphological changes – hypersegmentation of neutrophils (more than five lobes) and the presence of Howell-Jolly bodies in erythrocytes (Katar *et al*., 2006; Mandal & Chandra, 2025). In school-aged children, anaemia may develop gradually, and in the initial stages the only manifestations may be pallor, increased fatigue, reduced exercise tolerance, dizziness and tachycardia upon mild exertion. With prolonged untreated deficiency, pancytopenia may develop, a decrease in all three haematopoietic lineages (erythrocytes, leukocytes, and platelets), which is accompanied by increased susceptibility to infections and bleeding manifestations (Verma *et al*., 2017; Sawada *et al*., 2024; Belhaj *et al*., 2025). It is important to emphasise that the severity of anaemia correlates not so much with the duration of deficiency as with its depth; however, subclinical B12 deficiency without anaemia is much more common and can also be accompanied by neurological disturbances.

Neurological Consequences

The nervous system is the most sensitive to B12 deficiency, as this vitamin plays a critical role in the synthesis of myelin – the sheath that ensures rapid and insulated transmission of nerve impulses. In school-aged children, neurological manifestations may precede anaemia or develop in the absence of overt haematological changes, which creates serious diagnostic difficulties (Gupta *et al*., 2017; Umasanker *et al*., 2020; Kumar *et al*., 2026).

Cognitive impairment occupies a central place in the clinical picture of B12 deficiency in schoolchildren. Parents and teachers may note decreased academic performance, difficulties with concentration, forgetfulness, and slowing of thought processes (Gupta *et al*., 2017). Children with subclinical B12 deficiency are often characterised as "able but lazy" or "absent-minded". In some cases, a so-called "brain fog" develops – a condition in which the child cannot articulate thoughts clearly, confuses words, and loses the thread of a conversation. These changes directly affect learning ability and social adaptation.

Peripheral neuropathy manifests as paraesthesias – sensations of tingling, prickling, and burning in the distal parts of the limbs (Etminan *et al*., 2019; Rathore *et al*., 2024). In school-aged children, these complaints are often not verbalised or are described as "strange sensations" in the legs or arms. Physical examination may reveal reduced vibratory sensation (especially in the lower extremities) and diminished deep tendon reflexes. In the early stages, neuropathy is fully reversible with replacement therapy.

Spinal cord involvement (subacute combined degeneration of the spinal cord) is one of the most severe neurological consequences of prolonged B12 deficiency, characterised by demyelination of the posterior and lateral columns of the spinal cord. In schoolchildren, this syndrome presents with progressive ataxia (impaired coordination of movements), gait unsteadiness, particularly in the dark (sensory ataxia), and weakness in the legs. In severe cases, spastic paraparesis may occur (Kasinathan *et al*., 2019; Lallemant-Dudek & Durr, 2021; Panda *et al*., 2021). It is important to note that subacute combined degeneration in children, unlike in adults, is potentially reversible with early initiation of therapy; however, with prolonged deficiency, the changes may become irreversible.

Psychiatric manifestations of B12 deficiency in adolescents deserve special attention. Cases of depressive disorders, anxiety, irritability, apathy, and, in rare cases, psychotic symptoms with hallucinations and delusions have been described (Ralapanawa *et al*., 2015; Stredny *et al*., 2016). These conditions can be mistakenly interpreted as primary psychiatric disorders, and patients may be referred to a psychiatrist without evaluation of vitamin status. Timely B12 correction in such cases leads to complete regression of psychiatric symptoms.

Gastrointestinal Consequences

B12 deficiency affects the rapidly renewing cells of the gastrointestinal mucosa. The most characteristic manifestation is atrophic glossitis — inflammation of the tongue, which becomes smooth, "lacquered", bright red and often painful (Wu *et al*., 2018). Cheilitis (lip involvement) and angular stomatitis ("perlèche") may also be observed (Boukssim & Chbicheb, 2024). Children often complain of decreased appetite, which, in combination with dyspeptic disorders (nausea, diarrhoea or constipation), can lead to secondary nutrient deficiency and delayed physical development (Manabe *et al*., 2025). Changes in the gastric mucosa, in turn, may exacerbate malabsorption of other vitamins and minerals, creating a vicious cycle (Sun *et al*., 2016).

Impact on Growth and Nutritional Status

Although the direct link between B12 deficiency and growth retardation is less obvious than with protein or energy deficiency, research data indicate that in children with severe malnutrition, B12 levels are significantly lower than in children with normal nutritional status. Decreased appetite due to B12 deficiency and associated gastrointestinal disorders contributes to the progression of protein-energy malnutrition, especially under conditions of limited access to animal products (Ng'eno *et al*., 2017; Desmond *et al*., 2024). Thus, B12 deficiency acts not only as an independent pathology but also as a factor that aggravates overall nutritional disadvantage.

Figure 2 summarises the key clinical consequences of vitamin B12 deficiency in school-aged children, highlighting their diversity and potential reversibility with timely diagnosis and treatment.

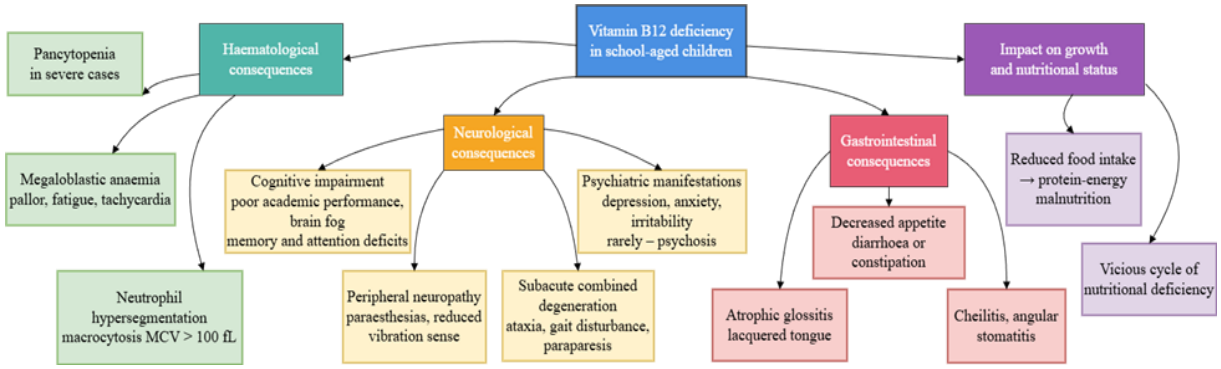

Figure 2. Clinical consequences of vitamin B12 deficiency in school-aged children

Socio-Economic Burden of Vitamin B12 Deficiency in School-Aged Children

From an individual case to a systemic problem. A school-aged child without prior academic difficulties begins to demonstrate declining academic performance, impaired concentration, deterioration of short-term memory, and increased fatigue. These symptoms correspond to the cognitive impairment associated with subclinical vitamin B12 deficiency (Iskra & Trufanov, 2023). However, neither the child nor the parents attributes them to a vitamin deficiency. The child interprets his or her condition as personal inability, the parents as lack of motivation or pedagogical neglect. A family conflict arises, with mutual accusations and deterioration of parent-child relationships. Psychological trauma may persist even after correction of the biochemical deficiency.

Information Vacuum as a Barrier

Medical consultation regarding declining academic performance does not occur because this problem is not positioned as a medical one either within the healthcare system or within education. Public information campaigns on the association between cognitive impairment in children and B12 deficiency are absent (Green & Plattel, 2024; National Institute for & Care, 2024). Unlike HIV, tick-borne encephalitis, hepatitis, or vaccination prophylaxis, the problem of vitamin-dependent cognitive disorders is not represented in health education materials at the level of outpatient facilities and schools. Parents do not know that the symptom complex of "declining academic performance + fatigue + cognitive 'brain fog'" has a potentially remediable biochemical cause.

Family Economics

Diagnosis of subclinical B12 deficiency requires determination of holotranscobalamin levels and, in some cases, homocysteine. The cost in commercial laboratories is 40-70 USD. For many families, this is a significant sum, but it is comparable to one month's payment for extracurricular developmental activities or 2-3 hours of tutoring. Lacking information about the true cause, the family invests in pedagogical correction, psychological support, over-the-counter sedatives, and nootropic drugs. The total costs over several months can many times exceed the cost of a one-time diagnostic work-up, with minimal effectiveness because the aetiological factor has not been addressed.

Systemic Defects

When consulting a physician (for example, during a routine check-up), in the absence of anaemia and overt neurological symptoms, testing of B12 levels is not ordered (Bruttin *et al*., 2021). The reasons are: (1) subclinical B12 deficiency as a cause of isolated cognitive impairment is not included in clinical guidelines for the management of school-aged children; (2) postgraduate medical education programmes do not systematically address this issue; (3) active B12 testing is not part of routine screening and is not covered by public health insurance in the absence of anaemia or neurological symptoms. Thus, systemic failure is documented at all levels: lack of public awareness, lack of diagnostic algorithms for physicians, and lack of financial coverage for the test.

Magnitude of Losses for the State

In populations with a high prevalence of vegetarianism, the prevalence of subclinical B12 deficiency among school-aged children reaches 20-40% (Groufh-Jacobsen *et al*., 2024). On a global scale, this represents tens of millions of children with unrealised cognitive potential. With timely diagnosis, each of them could learn better, achieve higher academic results, and enter prestigious professions (engineering, medical, scientific, IT specialties). At the national level, this means the loss of thousands of highly qualified specialists and a reduction in long-term economic competitiveness. The technical solution to the problem is simple and economically accessible: screening and correction of B12 deficiency require costs that are orders of magnitude lower than the annual expenditure on a child's additional education or on the rehabilitation of the consequences of prolonged undiagnosed deficiency. Realising this potential requires bridging the information gap and systemic changes in the organisation of medical care (Thazha *et al*., 2023; Al-Twaijri *et al*., 2024; Ansari *et al*., 2024; Feng *et al*., 2024; Jeung, 2024; Landry *et al*., 2024; Suchy & Jurkowski, 2024).

Figure 3 illustrates the cascade of consequences of vitamin B12 deficiency in school-aged children: from an individual case through the family and systemic levels to macroeconomic losses.

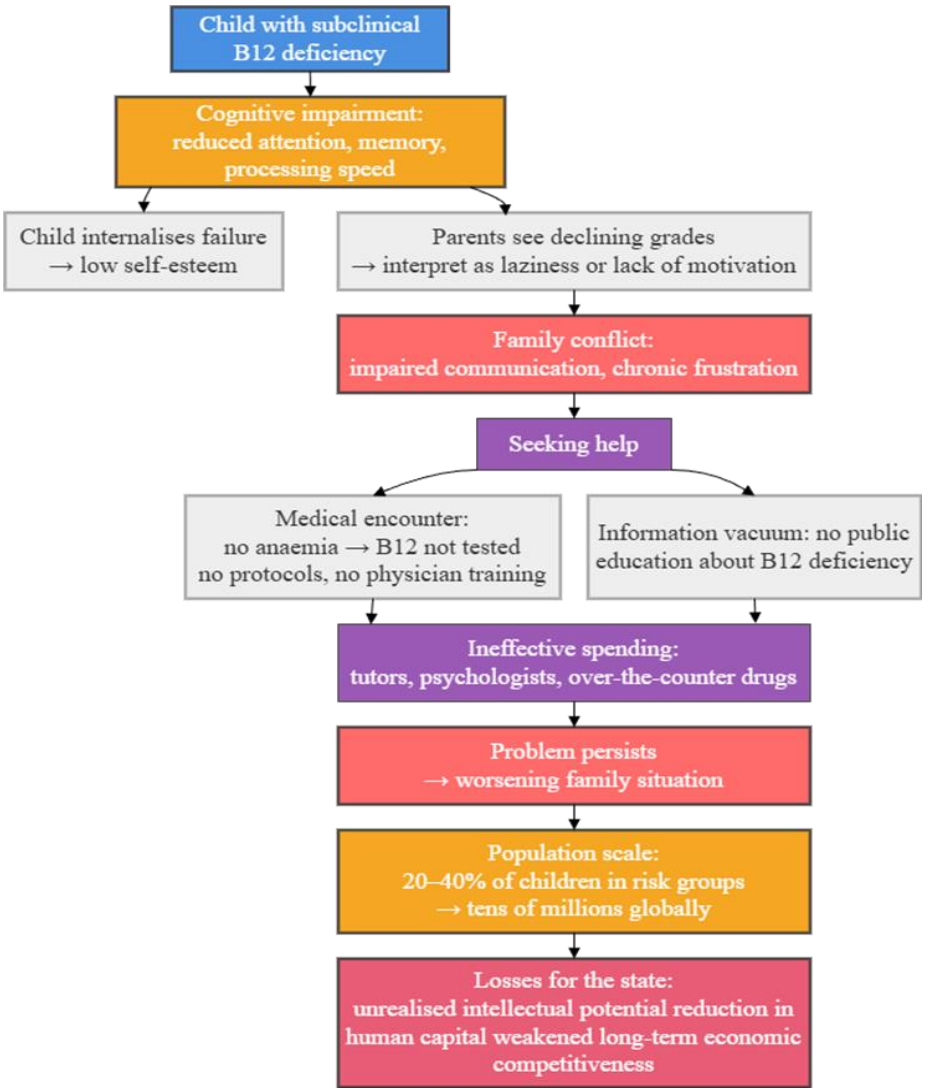

Figure 3. Cascade of consequences: from an individual case to macroeconomic losses

Recommendations for the Treatment of Vitamin B12 Deficiency in School-Aged Children

Laboratory Criteria for Deficiency and Indications for Treatment

The decision to initiate replacement therapy is based on active B12 (holotranscobalamin, HoloTC) or, when unavailable, total serum B12, as well as homocysteine levels. Subclinical B12 deficiency can exist without anaemia and without a decrease in total B12, manifesting as isolated homocysteine elevation and cognitive impairment. Relying solely on total B12 and complete blood count leads to both overdiagnosis (falsely normal results) and underdiagnosis of functional deficiency. **Table 2** presents laboratory criteria for interpreting results in school-aged children (Constantin *et al*., 2022; Mojsak *et al*., 2022; Bandi *et al*., 2024; Delcea *et al*., 2024; Essah *et al*., 2024; Frost *et al*., 2024; Kajanova & Badrov, 2024; Landry *et al*., 2024; Lee & Ferreira, 2024; Rosellini *et al*., 2024; Umarova *et al*., 2024).

Table 2. Laboratory criteria for vitamin B12 deficiency in school-aged children

Parameter	Normal (optimal level)	Grey zone (borderline values, require attention)	Deficiency (treatment indicated)	Critical deficiency (urgent treatment)
Total serum B12 (pg/mL)	> 400-500	200-400	150-200	< 150
Active B12, HoloTC (pmol/L)	> 50	25-50	15-25	< 15
Homocysteine (µmol/L)	< 8-10	10-15	15-30	> 30
Note	No deficiency	Functional deficiency possible. If symptoms	Treatment mandatory regardless of symptoms	Intramuscular treatment, high doses, urgent

present – trial treatment.

A special situation deserves attention when homocysteine is elevated (>10-15 μmol/L) while total and active B12 are within the normal range (>400 pg/mL and >50 pmol/L, respectively) (Kaye *et al*., 2020). The most likely cause is folate (vitamin B9) deficiency or, less commonly, vitamin B6 deficiency (Pusceddu *et al*., 2020). Determination of serum folate levels is recommended. Combined B12 and folate deficiency is common, especially in alimentary causes (veganism, unbalanced diet) (Genc *et al*., 2023; Ku *et al*., 2023; Simonyan *et al*., 2023; Tsiganock *et al*., 2023; Delcea *et al*., 2024; Ribeiro *et al*., 2024; Sanlier & Yasan, 2024; Uneno *et al*., 2024).

Primary Evaluation before Therapy

Before starting treatment, the following are mandatory: complete blood count with reticulocytes and red cell morphology (macrocytosis, neutrophil hypersegmentation); ferritin level (iron deficiency often coexists and masks response); folate level (differential diagnosis from isolated folate deficiency). Additional tests (as indicated): antibodies to parietal cells and intrinsic factor (autoimmune gastritis), antibodies to tissue transglutaminase (coeliac disease), stool examination for helminth eggs (Diphyllobothrium latum) (Socha *et al*., 2020; Wolffenbuttel *et al*., 2024).

Choice of Administration Route and Dosage

The choice depends on deficiency severity, neurological symptoms, and suspected malabsorption. Parenteral route (cyanocobalamin or methylcobalamin IM) is preferred in: critical deficiency (active B12 <15 pmol/L or total B12 <150 pg/mL); neurological symptoms or anaemia; confirmed or suspected malabsorption. Starting dose in school-aged children: 500-1000 μg IM daily or every other day for 1-2 weeks (induction), then weekly for 4-8 weeks (saturation), then monthly (maintenance, often lifelong).

Oral/sublingual route (methylcobalamin or hydroxocobalamin tablets or drops) is possible in mild or moderate deficiency (active B12 15-50 pmol/L, total B12 150-400 pg/mL), absence of neurological symptoms, and preserved absorption (alimentary origin). Daily dose: 500-1000 μg. Control of B12 and homocysteine at 1-2 months is mandatory; lack of normalisation indicates a switch to the parenteral route (Tandon *et al*., 2022; Saxena *et al*., 2023; Sachdeva *et al*., 2025).

Monitoring of Treatment Efficacy

Haematological response (if anaemia is present) is assessed at 5-7 days (reticulocyte crisis) and at 4-8 weeks (normalisation of haemoglobin and MCV). Neurological response (cognitive function, paraesthesias, ataxia) is assessed at 1-3 months. If no improvement occurs despite stable normalisation of B12 and homocysteine within 3-4 months, the diagnosis should be reconsidered. Laboratory monitoring: B12 and homocysteine at 1-2 months, then every 3-6 months until stabilisation, then annually (Al Abadie *et al*., 2023; Guzek *et al*., 2023; Lee *et al*., 2023; Ncube *et al*., 2023; Oran & Azer, 2023; Szklener *et al*., 2023; Tsvetkova *et al*., 2023).

Elimination of the Cause of Deficiency

Concurrently with replacement therapy, the aetiological factor should be addressed: alimentary deficiency – dietary correction or, in vegans, lifelong prophylactic B12 (10-50 μg/day); coeliac disease – gluten-free diet; Crohn's disease and other enteropathies – treatment of the underlying disease; parasitic infestations – antiparasitic treatment; drug-induced deficiency (PPIs, metformin) – if possible discontinue or reduce dose; if not possible – regular monitoring and prophylaxis; hereditary disorders – lifelong parenteral high-dose B12 (Bensky *et al*., 2019; Rossignol & Frye, 2021; Abdelwahab *et al*., 2024).

Prevention in Risk Groups

Prophylactic B12 without prior testing is indicated for: vegan and strict vegetarian children; children on long-term (>1 year) PPI or metformin therapy; children with established malabsorption (coeliac disease, Crohn's disease, after ileal resection); children with a family history of hereditary B12 metabolism disorders. Prophylactic dose for school-aged children: 10-50 μg/day oral cyanocobalamin or 500-1000 μg 1-2 times per week (Melina *et al*., 2016; Tardy *et al*., 2020).

The management algorithm for a child with suspected vitamin B12 deficiency is summarised in **Figure 4**.

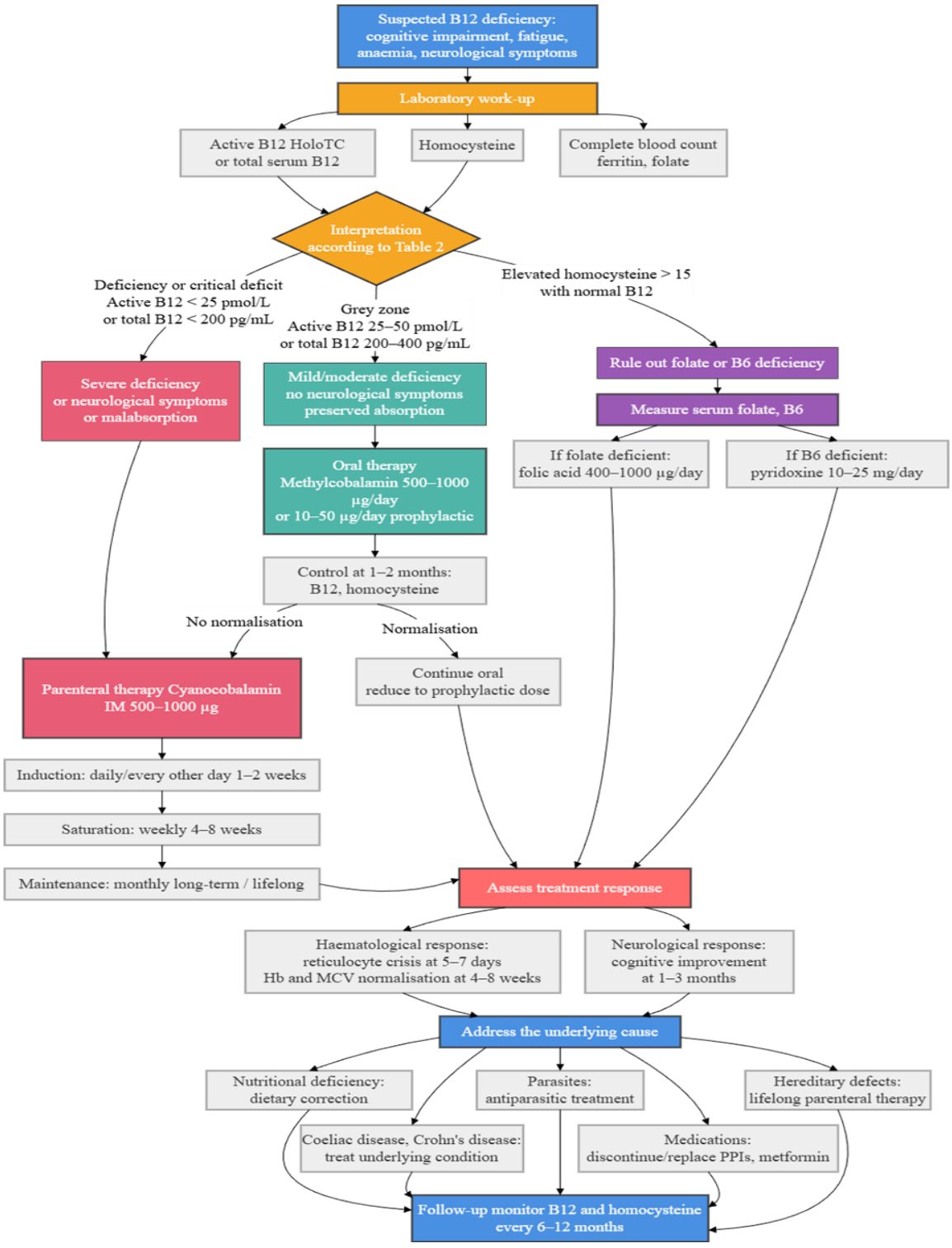

Figure 4. Management algorithm for suspected vitamin B12 deficiency in school-aged children

Conclusion

Vitamin B12 deficiency in school-aged children is a common but underdiagnosed condition. Depending on the region and dietary habits, the prevalence of subclinical deficiency (without anaemia but with cognitive impairment) in at-risk groups reaches 20-40%, which globally corresponds to tens of millions of children.

The key clinical feature of B12 deficiency in this age group is its ability to present as isolated cognitive impairment (reduced

attention, working memory, and information processing speed) in the absence of anaemia and with normal total serum B12 levels. These symptoms lack pathognomonic signs and are often misinterpreted in routine practice as behavioural or pedagogical problems. This leads to delayed diagnosis, chronic academic failure, and the development of secondary psychoemotional disorders. At the population level, the consequence is loss of intellectual potential and a reduction in human capital.

The most sensitive diagnostic method is the measurement of active B12 (holotranscobalamin) in combination with homocysteine. The cost of a full work-up is 40-70 USD, comparable to the cost of alternative but less effective interventions, including pedagogical correction and psychological support. Treatment of confirmed deficiency is technically simple and economically accessible. Parenteral administration of cyanocobalamin or methylcobalamin at a dose of 500-1000 μg, or in mild cases with preserved absorption, high oral doses (500-1000 μg/day), achieve clinical and laboratory response within 1-3 months. Most neurological manifestations are reversible with timely therapy.

The main obstacle to timely detection of B12 deficiency is a systemic information gap: lack of public awareness campaigns, insufficient knowledge among primary care physicians, and absence of screening protocols for at-risk groups. Overcoming this gap requires including information on the cognitive manifestations of B12 deficiency in postgraduate medical education programmes, developing clinical guidelines for screening (vegetarian diets, chronic gastrointestinal diseases, long-term PPI or metformin therapy, unexplained decline in academic performance), and conducting targeted public information campaigns.

Vitamin B12 is one of those micronutrients whose deficiency can be completely corrected by simple and affordable means. Timely diagnosis and adequate therapy can restore cognitive function, prevent secondary psychoemotional disorders, and preserve academic performance. Ignoring this problem is unjustifiable from clinical, economic, and ethical perspectives.

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