J Biochem Technol (2026) 17(2): 78-91
https://doi.org/10.51847/nWmH2IFNYi
ISSN: 0974-2328

# Iron and Vitamin D Deficiency and Cognitive Impairment in Working-Age Adults: From Neurobiological Mechanisms to Practical Correction

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Received: 28 January 2026 / Received in revised form: 12 April 2026, Accepted: 19 April 2026, Published online: 25 May 2026

## Abstract

Iron and vitamin D deficiencies are among the most common micronutrient disorders worldwide, yet their impact on cognitive function in working-age adults remains underestimated. This narrative review synthesises current evidence on neurobiological mechanisms linking these deficiencies to cognitive decline, psycho emotional disturbances, and reduced work productivity, focusing on latent deficiency states that escape routine diagnosis. Latent iron deficiency (low ferritin, normal haemoglobin) affects 40-55% of women of reproductive age, while vitamin D insufficiency (25(OH)D <20 ng/mL) is present in 30-50% of adults in northern latitudes and also in sunny regions where clothing limits skin exposure. Vegetarians, office workers, and residents of high-latitude countries are at the highest risk. Iron deficiency impairs dopaminergic signalling, myelin synthesis, and mitochondrial energy metabolism, leading to cognitive deficits. Vitamin D deficiency reduces neurotrophin expression, promotes neuroinflammation, and weakens antioxidant defence, resulting in executive dysfunction and slower processing. Both deficiencies contribute to anxiety, depression, apathy, and sleep disturbances. Cognitive decline begins at the stage of latent deficiency, before anaemia or osteomalacia develop, and is often misattributed to stress or ageing. Partial reversibility following iron repletion has been demonstrated, whereas the cognitive response to vitamin D supplementation is more modest and inconsistent. Screening for ferritin (target >50 µg/L) and 25(OH)D (target 30-50 ng/mL) is indicated in patients complaining of "brain fog", particularly in risk groups. Hidden hunger is a socially significant challenge that reduces economic productivity and quality of life.

**Keywords:** Iron deficiency, Vitamin D deficiency, latent deficiency, Cognitive function, "Brain fog", Screening

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## Introduction

Modern life demands high cognitive performance (concentration, working memory, decision-making speed, and emotional stability) as essential tools for work, learning, and social functioning. The decline of these functions is particularly acutely perceived by working-age adults, who bear responsibility for themselves and their families. Complaints of "brain fog", memory lapses, increased anxiety, and chronic fatigue are becoming increasingly common in this age group, yet they often remain unexplained (Jones *et al*., 2024).

These symptoms are frequently attributed to chronic stress, overwork, or age-related changes. However, modern science points to an often-ignored cause: "hidden hunger" – deficiencies of micronutrients essential for neuronal function and nerve impulse

conduction. Even a diet balanced in proteins, fats, and carbohydrates may leave the brain energy-deficient, directly impairing cognition (Clemente-Suárez *et al*., 2024; Das *et al*., 2024).

Among essential micronutrients, iron and vitamin D deserve special attention for two reasons. First, their deficiency is pandemic. Globally, iron deficiency affects up to a quarter of the population; among women of reproductive age, latent iron deficiency (low ferritin with normal haemoglobin) reaches 40-55% (Bellad *et al*., 2024; Kolarš *et al*., 2025). Vitamin D insufficiency (25(OH)D <20 ng/mL) affects 30-50% of adults in northern latitudes and, paradoxically, is also highly prevalent in sunny regions where clothing limits skin exposure (Silva *et al*., 2022; Chen *et al*., 2024). Thus, two of the most common and seemingly accessible micronutrients are insufficient for a large proportion of the world's population.

Second, both iron and vitamin D play critical roles in the central nervous system, far beyond their classical functions (oxygen transport and calcium-phosphorus metabolism). Iron is a cofactor for dopamine and serotonin synthesis, supports axonal myelination, and maintains mitochondrial energy metabolism in neurons (Shah *et al*., 2021; Liu *et al*., 2025). Even latent iron deficiency is associated with reduced working memory, psychomotor speed, and cognitive flexibility, as well as increased anxiety and apathy (Kung *et al*., 2021; Tsiglopoulos *et al*., 2021; McManus *et al*., 2025). Vitamin D, acting as a neurosteroid, regulates BDNF expression, protects against neuroinflammation and oxidative stress, and modulates neurotransmitter systems (Schildroth *et al*., 2022; Liu *et al*., 2025). Its deficiency is linked to executive dysfunction, slower thinking, and sleep disturbances (van den Berg *et al*., 2021; Hung *et al*., 2022; Tamanna, 2025). Notably, these negative effects appear before anaemia or rickets develop, making early diagnosis and correction an urgent task (Rosenberg *et al*., 2024; Varthaliti *et al*., 2025).

The high prevalence of these deficiencies among the economically active population and their proven impact on cognitive function call for a systematisation of current evidence. This review therefore aims to analyse and summarise the effects of iron and vitamin D deficiency on cognition and psycho-emotional health in adults, focusing on clinical manifestations, underlying mechanisms, and the potential reversibility of these disorders.

This narrative review synthesises current evidence on the neurobiological links between iron and vitamin D deficiency and cognitive function in adults of working age. In recent years, increasing scientific interest has also been directed toward plant-derived bioactive compounds and their potential biological and systemic effects in human health (Hamad *et al*., 2018; Ahmed *et al*., 2020; Ahmed *et al*., 2023). Literature was identified through targeted searches of PubMed, Scopus, and Web of Science, supplemented by manual screening of reference lists. Priority was given to systematic reviews, meta-analyses, randomised controlled trials, and large observational studies published between 2020 and 2026. No formal meta-analysis or exhaustive systematic search was conducted.

## *Iron Deficiency*

### *Epidemiology and risk Groups*

Iron deficiency is recognized as the most common micronutrient deficiency in the world, affecting up to a quarter of the global population (Leung *et al*., 2024). According to a systematic review by Kolarš *et al*., the prevalence of latent iron deficiency (low ferritin with normal hemoglobin) among women of reproductive age reaches 40-55%, and in some regions of South Asia exceeds 60%. Bellad *et al*. report that iron deficiency anemia occurs in 30-40% of women in low- and middle-income countries. Additional risk factors include vegetarian and vegan diets: in Western populations, up to 30% of vegans have laboratory signs of iron deficiency, and in regions with traditionally plant-based diets, this figure reaches 60% (Auerbach *et al*., 2025). McManus *et al*. note that 43% of women in the United Kingdom consume less than 40 g of red meat per day – below the level required to maintain adequate iron status (McManus *et al*., 2025). In the Russian Federation, epidemiological studies estimate the prevalence of latent iron deficiency among women of reproductive age at 20-30%, and among pregnant women at over 40% (Gvozdenko *et al*., 2024; Mareev *et al*., 2024a, 2024b).

### *Why is Iron Poorly Absorbed and Rapidly Consumed?*

Dietary iron exists in two forms: heme iron (from animal sources, absorption 25-30%) and non-heme iron (from plants, absorption only 5-12%) (Piskin *et al*., 2022; Moustarah & Daley, 2024; Frazer *et al*., 2025). Absorption of non-heme iron critically depends on accompanying food components. Phytic acid (cereals, legumes), tannins (tea, coffee, red wine), oxalates (spinach, beetroot), and calcium (dairy products) form insoluble complexes with iron, reducing its bioavailability by 2-5 times (Bhoot *et al*., 2023; López-Moreno *et al*., 2025; López-Moreno *et al*., 2025). In contrast, vitamin C (ascorbic acid) reduces ferric iron to ferrous iron, increasing absorption 3-6-fold when taken simultaneously (Skolmowska & Głąbska, 2022; von Siebenthal *et al*., 2023). This is why the recommendation «take the tablet with orange juice» has a biochemical basis, while «take it with tea» directly counteracts absorption. In women of reproductive age, iron is additionally lost during menstruation (on average 15-30 mg of iron per cycle) and is also consumed for fetal and placental growth during pregnancy and for lactation (Cappellini *et al*., 2022; Petraglia & Dolmans, 2022; Li *et al*., 2023; Bellad *et al*., 2024). Thus, even with normal dietary iron intake, many women develop a negative iron balance that, without correction, leads to depletion of iron stores.

### *Neurobiological Mechanisms*

The effect of iron deficiency on the brain is mediated through several pathways. Iron serves as a cofactor for tyrosine hydroxylase and tryptophan hydroxylase – the key enzymes for dopamine and serotonin synthesis. Reduced activity of these enzymes leads to decreased dopamine concentrations in the striatum and serotonin in limbic structures, which underlies anxiety, depressive disorders, and, in severe cases, psychotic symptoms (Bani-Ahmad *et al*., 2022; Newbolds & Wenger, 2024). In addition, iron is necessary for myelination: oligodendrocytes accumulate iron for myelin sheath synthesis, and its deficiency reduces nerve impulse conduction velocity (Fiani *et al*., 2025; Hod *et al*., 2025). Das *et al*. add that iron participates in the functioning

of hippocampal neuronal mitochondria, supporting the energy metabolism required for synaptic plasticity and long-term potentiation. Schildroth *et al*. show that iron deficiency enhances the absorption of neurotoxic heavy metals (lead, manganese, cadmium) through the competitive transporter DMT 1, thereby potentiating toxic damage to the dopaminergic system.

*Cognitive and Psycho Emotional Manifestations*

A systematic review by Shah *et al*. identifies key phenotypes associated with iron deficiency: in adults, anxiety disorders, depressive episodes, sleep disturbances, and negative symptoms of psychotic disorders come to the fore. These effects can develop already at the stage of latent deficiency, before the onset of anemia. A meta-analysis by Kung *et al*., including 20 studies with 6,558 participants, showed that anemia is associated with a significantly increased risk of cognitive impairment – the adjusted relative risk was 1.39 (95% CI 1.25-1.55). Bellad *et al*. cite a study by Bruner in which 81 adolescent girls with ferritin ≤12 μg/L received either iron supplements or placebo: the placebo group performed significantly worse on verbal memory tests.

*Correction and Reversibility*

A meta-analysis by McManus *et al*. showed that increasing red meat consumption (a source of heme iron) leads to a significant increase in ferritin levels when the intervention lasts more than 16 weeks – the mean difference was +5.62 μg/L. Hemoglobin levels increased by an average of 2.36 g/L. The effect on ferritin was more pronounced in women, which is explained by lower baseline ferritin levels and more efficient iron absorption due to lower hepcidin levels. However, dietary correction requires time and discipline, and for vegetarians – careful meal planning with attention to food combinations (legumes + vitamin C, avoidance of tea and coffee during meals) (Slywitch *et al*., 2021; Koeder & Perez-Cueto, 2024; Malhotra & Lakade, 2025; Luna *et al*., 2026).

An important clinical conclusion by Kolarš *et al*. is that cognitive impairments caused by iron deficiency in adults are partially reversible: normalization of ferritin is accompanied by improvement in cognitive performance. However, Shah *et al*. point out that when deficiency occurs in early development, the consequences may be persistent even after correction (Shah *et al*., 2021). In practice, about 50-70% of patients discontinue oral iron supplements due to gastrointestinal side effects: nausea, constipation, diarrhea, metallic taste (Schaefer *et al*., 2022; Benson *et al*., 2025). The key reason is not the drug itself, but the dosing regimen. The traditional regimen (high doses daily) leads to a sharp rise in hepcidin – a peptide hormone that blocks iron absorption for 24-48 hours. As a result, most of the ingested iron is not absorbed, remains in the intestinal lumen, and causes irritation (Lewkowitz *et al*., 2022). Current guidelines recommend an alternative strategy: low doses (50-100 mg of elemental iron) every other day, preferably in the morning on an empty stomach, taken with a glass of water and vitamin C, avoiding tea, coffee, and dairy products for one hour before and after intake (Pai *et al*., 2023). This regimen does not cause a prolonged hepcidin rise, increases the fraction absorbed, and significantly reduces side effects, thereby improving treatment adherence (Chaudhry *et al*., 2023; Chaudhry *et al*., 2023; Szklener *et al*., 2023; Thazha *et al*., 2023; Tsvetkova *et al*., 2023; Vogel *et al*., 2023; Weerasinghe *et al*., 2023; Bandi *et al*., 2024; Wolderslund *et al*., 2024).

**Figure 1** schematically presents the pathogenetic links of iron deficiency effects on cognitive functions and mental health, as well as the key factors influencing iron absorption and losses.

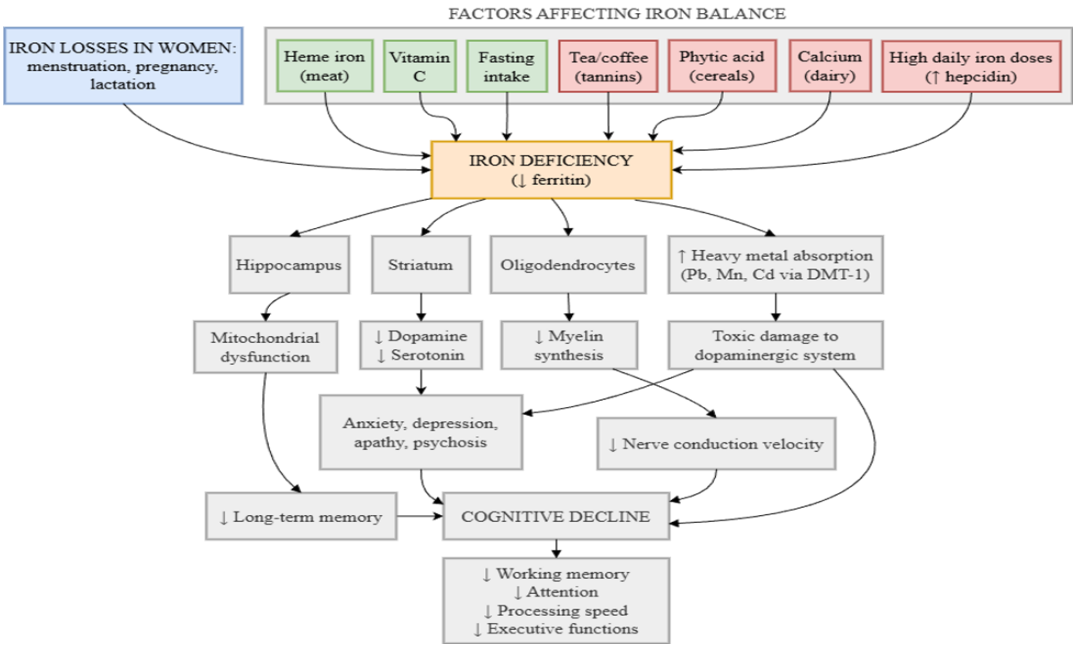


**Figure 1.** Pathogenetic mechanisms of iron deficiency effects on cognitive function and mental health, with factors influencing iron absorption and losses.

*Vitamin D Deficiency*

*Epidemiology and the Paradox of Accessibility*

Vitamin D insufficiency is a pandemic of modern urbanized society. According to a meta-analysis by Chen *et al*., the prevalence of 25(OH)D deficiency (less than 20 ng/mL) reaches 30-50% in populations of northern latitudes (Chen *et al*., 2024). In Russia, depending on the estimate, vitamin D deficiency is detected in 40-60% of the adult population, and in Arctic regions – in 70-80% (Suplotova *et al*., 2021; Smirnova *et al*., 2022; Karonova *et al*., 2025). Silva *et al*. confirm that risk groups include office workers, people with dark skin, and residents of megacities (Silva *et al*., 2022). Varthaliti *et al*. report that among pregnant women, deficiency can reach 47.9% in the general population and up to 96% in some regions of developing countries. It would seem that vitamin D is easy to replenish: it is synthesized in the skin under the influence of ultraviolet light and also comes from food (fatty fish, eggs, fortified products). However, the reality is that the two most accessible sources (sunlight and diet) turn out to be unreliable in practice. In northern latitudes (above the 42nd parallel) from October to March, the angle of the sun's rays is such that ultraviolet B (UVB) practically does not reach the Earth's surface, and endogenous vitamin D synthesis stops (Bezuglov *et al*., 2023; Boldyreva *et al*., 2023; Vilms *et al*., 2024). In southern latitudes, despite an abundance of sun, vitamin D deficiency is widespread among populations that, for cultural or religious reasons, wear clothing that completely covers the skin, reducing synthesis to almost zero (Aghapour *et al*., 2023; Vergara-Maldonado & Urdaneta-Machado, 2023). In addition, the use of sunscreens (SPF ≥30) blocks up to 95% of vitamin D synthesis. Even dietary intake rarely meets requirements: to obtain 1000 IU of vitamin D, one would need to eat about 200 g of salmon or 10 egg yolks per day, which is economically and physically unattainable for most people (Janoušek *et al*., 2022; Bennett & Khachemoune, 2022; Weaver & Wallace, 2024). Thus, despite its apparent "accessibility", vitamin D turns out to be one of the most deficient micronutrients.

*Neurobiological Mechanisms*

The most detailed mechanisms of the neuroprotective action of vitamin D are presented in the review by Liu *et al*. Vitamin D receptors (VDR) are widely expressed in the central nervous system, including the hippocampus, prefrontal cortex, amygdala, and hypothalamus (Oczkowicz *et al*., 2021). Activation of VDR by vitamin D stimulates the synthesis of brain-derived neurotrophic factor (BDNF), nerve growth factor (NGF), and neurotrophins NT-3 and NT-4, which are critically important for neuroplasticity, neuronal survival, and synaptogenesis. In addition, vitamin D modulates microglial activity: its deficiency leads to chronic microglial activation with increased production of pro-inflammatory cytokines (IL-6, TNF-α) through the TLR4/MyD88/NF-κB pathway, contributing to neuroinflammation (Galoppin *et al*., 2022). Liu *et al*. also describe the activation by vitamin D of the antioxidant Nrf2/HO-1 pathway, which protects neurons from ferroptosis and other forms of oxidative damage (Liu *et al*., 2025).

*Cognitive Manifestations in Healthy Adults*

A systematic review by Silva *et al*., focused specifically on healthy adults, showed a positive effect of vitamin D on verbal memory, verbal working memory, attention, and executive functions (Silva *et al*., 2022). A crucial practical observation by the authors was that low doses of vitamin D (400-600 IU per day) were more effective in terms of cognitive improvement than high doses (2400-5000 IU per day) (Silva *et al*., 2022). This U-shaped effect is explained by the fact that very high doses may not provide additional increases in the active form and may potentially cause adverse effects (hypercalcemia, suppression of active metabolite synthesis) (Mojsak *et al*., 2022; Lee *et al*., 2023; Ncube *et al*., 2023; Oran & Azer, 2023; Oran & Azer, 2023; Szklener *et al*., 2023; Tam *et al*., 2023; Lee & Ferreira, 2024; Kajanova & Badrov, 2024). A meta-analysis of 24 randomized controlled trials by Chen *et al*. demonstrated a small but statistically significant effect of vitamin D on global cognition: Hedges' g was 0.128 (p=0.008). In the subgroup of participants with baseline vitamin D deficiency, the effect was substantially stronger – g=0.480 (Chen *et al*., 2024).

*Impact on Mental Health and Sleep*

Tsiglopoulos *et al*. showed that in patients with first-episode psychosis and schizophrenia, 25(OH)D levels are significantly lower than in the control population (Tsiglopoulos *et al*., 2021). In 57% of observational studies, an inverse relationship was found between vitamin D levels and the severity of negative symptoms, and in 63% of studies, a positive relationship with cognitive function (Tsiglopoulos *et al*., 2021). Low 25(OH)D levels are associated with poor sleep quality, reduced sleep duration, and increased time to fall asleep (Rouhani *et al*., 2023). At the same time, van den Berg *et al*., in a systematic review of 31 RCTs, found no convincing evidence for an antidepressant effect of vitamin D, indicating the need to take baseline vitamin D status into account when interpreting results (van den Berg *et al*., 2021). A meta-analysis by Hung *et al*. showed that preoperative vitamin D deficiency is associated with an increased risk of postoperative delirium and postoperative cognitive dysfunction: the odds ratio was 1.54 (95% CI 1.21-1.97) (Hung *et al*., 2022).

*Practical Recommendations for Correction*

In practice, the main problem is not the lack of drugs, but low patient adherence. People stop taking vitamin D because they do not see a rapid effect ("I have been taking it for a month, but the brain fog is still there"). However, normalization of 25(OH)D levels takes time: with intake of 800-2000 IU/day, levels increase by approximately 0.7-1.0 ng/mL per week, meaning that correcting deficiency (<20 ng/mL) to optimal values (30-50 ng/mL) may take 3-6 months (Takacs *et al*., 2023; Babalyan *et al*., 2026). It is also important to understand that low doses (400-600 IU/day) may be no less effective for cognitive outcomes than high doses and are less likely to cause hypercalcemia (Cianferotti *et al*., 2026). Recommended regimen: cholecalciferol (vitamin D3) at a dose of 800-2000 IU/day (depending on baseline level), preferably with food for better absorption (fat-soluble vitamin), with monitoring of 25(OH)D after 3-6 months. For patients with obesity or malabsorption, doses may be increased to 2-3 times above standard levels (Pludowski, 2023).

**Figure 2** schematically presents the pathogenetic links of vitamin D deficiency effects on cognitive functions and mental health, as well as the geographical and sociocultural barriers that prevent its synthesis (Constantin *et al*., 2022; Genc *et al*., 2023; Ku *et al*., 2023; Essah *et al*., 2024; Frost *et al*., 2024; Rosellini *et al*., 2024; Ribeiro *et al*., 2024; Uneno *et al*., 2024; Umarova *et al*., 2024; Umarova *et al*., 2024).

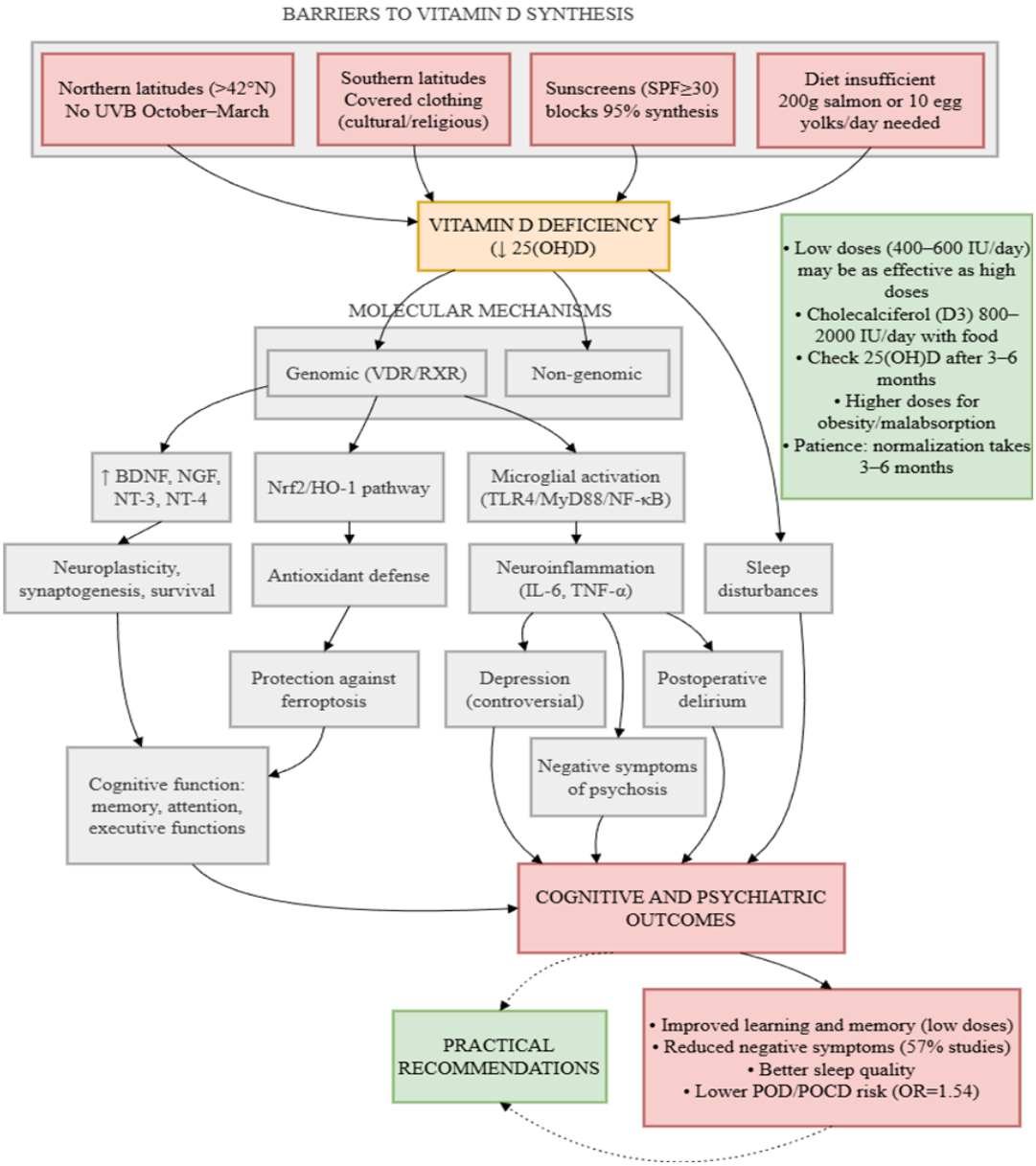


**Figure 2.** Pathogenetic mechanisms of vitamin D deficiency effects on cognitive function and mental health, with geographical and sociocultural barriers to synthesis.

*Comparative Analysis*

The parallel analysis of iron and vitamin D deficiencies reveals a striking picture: two completely different micronutrients, with distinct sources, metabolism, and molecular targets, converge on a common clinical outcome – cognitive decline and psycho-emotional disorders. This convergence is not coincidental but reflects the fundamental dependence of the central nervous system on a narrow set of essential elements that support energy metabolism, neurotransmission, synaptic plasticity, and myelin integrity (Al Abadie *et al*., 2023; Guzek *et al*., 2023; Simonyan *et al*., 2023; Tsiganock *et al*., 2023; Sanlier & Yasan, 2024).

*From a clinical perspective*, both deficiencies are extremely common, often coexist, and both produce nonspecific symptoms (fatigue, "brain fog", mood disturbances) that are easily attributed to stress or overwork. However, their mechanisms differ fundamentally, and this difference has direct therapeutic implications. Iron deficiency primarily disrupts monoaminergic systems (dopamine, serotonin) through impaired synthesis,

reduces myelin production, and compromises mitochondrial energy supply (Shah *et al*., 2021; Das *et al*., 2024; Kolarš *et al*., 2025). Vitamin D deficiency, in contrast, acts as a neurosteroid hormone deficiency: it downregulates the transcription of neurotrophins (BDNF, NGF), unleashes microglial neuroinflammation, and weakens antioxidant defenses (Liu *et al*., 2025; Liu *et al*., 2025). Understanding these distinctions allows the clinician to recognize why an iron-deficient patient may present with apathy and executive dysfunction (striatal dopamine deficit), while a vitamin D-deficient patient may present with slower thinking and poorer verbal memory (hippocampal BDNF deficit and neuroinflammation) (Wang & Li, 2023; Barnett *et al*., 2025).

Key *Similarities*

Both deficiencies share several important features. First, cognitive impairment can develop at the stage of latent deficiency, before overt anemia (iron) or osteomalacia (vitamin D) appears. Serum ferritin levels below 30 µg/L and 25(OH)D levels below 20 ng/mL are already associated with measurable cognitive decline, even when routine blood counts and calcium metabolism are normal (Silva *et al*., 2022; Bellad *et al*., 2024; Chen *et al*., 2024; Kolarš *et al*., 2025). Second, both deficiencies are more prevalent in women of reproductive age (iron due to menstrual losses, vitamin D due to lifestyle factors and darker skin in some ethnic groups) and in residents of northern latitudes (Rogozińska *et al*., 2021; Skoracka *et al*., 2021; Lucchetta *et al*., 2022; Amzajerdi *et al*., 2023). Third, dietary sources alone are often insufficient to correct established deficiency, making supplementation necessary in most cases (Benedik, 2022; Bopape *et al*., 2023; Feng *et al*., 2024; Pantopoulos, 2024). Fourth, the cognitive effects are at least partially reversible in adults if the deficiency is recognized and treated early, although permanent damage may occur when the deficiency occurs during critical developmental windows (fetal life, infancy) (Elahi *et al*., 2023; Ciobanu *et al*., 2023; Theola & Andriastuti, 2025).

*Key Differences*

The most clinically relevant differences relate to mechanisms, cognitive profiles, and treatment response. Iron deficiency affects the striatum and hippocampus via dopamine depletion and mitochondrial dysfunction, leading to impaired working memory, attention, and psychomotor slowing (Kung *et al*., 2021; Shah *et al*., 2021; Li & Finberg, 2025). Vitamin D deficiency affects the hippocampus and prefrontal cortex via BDNF downregulation and neuroinflammation, leading to executive dysfunction, slower thinking, and verbal memory deficits (Silva *et al*., 2022; Stoica & Mărginean, 2023; Chen *et al*., 2024). From a therapeutic standpoint, iron repletion produces measurable cognitive improvement within weeks to months, especially when using an every other day regimen that avoids hepcidin blockade (Tang & Sholzberg, 2024; Kolarš *et al*., 2025). In contrast, vitamin D repletion requires 3-6 months to normalize 25(OH)D levels, and the cognitive response is more modest and inconsistent across studies (Chen *et al*., 2024; Fraser, 2025; Liu *et al*., 2025). Importantly, low doses of vitamin D (400-600 IU/day) may be as effective for cognitive outcomes as high doses (2000-5000 IU/day), suggesting a U-shaped or threshold effect (Silva *et al*., 2022).

*Practical Implications for the Clinician*

The comparative analysis directly informs clinical decision-making. In a patient with "brain fog" and fatigue, measurement of both ferritin (not just hemoglobin) and 25(OH)D is warranted, as these deficiencies are easily missed when relying on standard screening panels (Silva *et al*., 2022; Bellad *et al*., 2024). If ferritin is below 50 µg/L, oral iron supplementation (50-100 mg elemental iron every other day, with vitamin C, away from tea/coffee and dairy) should be initiated, with expected cognitive improvement within 2-3 months (Kolarš *et al*., 2025; McManus *et al*., 2025). If 25(OH)D is below 30 ng/mL, cholecalciferol (800-2000 IU/day with food) should be prescribed, with reassessment after 3-6 months and patience regarding cognitive effects (Wylenzek *et al*., 2024). Importantly, the presence of one deficiency does not exclude the other; combined deficiencies are common and may have additive or synergistic effects on brain function (Shoemaker *et al*., 2022; Al Hinai *et al*., 2024; Chen *et al*., 2025).

*Limitations of the Comparative Framework*

Most studies on iron deficiency have focused on anemia rather than latent deficiency, and most vitamin D trials have been conducted in older adults with comorbidities, making extrapolation to healthy working-age adults tentative (Kung *et al*., 2021; Silva *et al*., 2022). The two deficiencies often coexist with other micronutrient deficits (zinc, B12, magnesium), and few studies control for these confounders (Schildroth *et al*., 2022; Clemente-Suárez *et al*., 2024; Liu *et al*., 2025). Finally, the effect sizes for vitamin D on global cognition are small (Hedges' g=0.128), and the clinical relevance of such improvements in otherwise healthy individuals remains debated (Chen *et al*., 2024).

*Summary of Comparative Findings*

**Table 1** summarizes the key similarities and differences between iron and vitamin D deficiency with respect to their effects on cognitive function. The table is structured to allow quick reference for clinicians and researchers, highlighting the distinct mechanisms, affected brain regions, cognitive domains, psychiatric manifestations, reversibility, and evidence levels for each deficiency.

**Table 1.** Comparative characteristics of the effects of iron and vitamin D deficiency on cognitive functions

| Parameter | Iron Deficiency | Vitamin D Deficiency |
|---|---|---|
| **Main mechanisms of action** | Cofactor for dopamine and serotonin synthesis, myelination, and mitochondrial energy metabolism | Regulation of BDNF and other neurotrophins, modulation of neuroinflammation, antioxidant defense (Nrf2/HO-1) |
| **Key brain structures** | Hippocampus, striatum, oligodendrocytes | Hippocampus, prefrontal cortex, amygdala, hypothalamus |

| | | |
|---|---|---|
| **Most sensitive cognitive domains** | Working memory, attention, psychomotor speed | Executive functions, processing speed, and verbal memory |
| **Psychiatric phenotypes** | Anxiety, depression, apathy, psychosis | Depression (controversial), negative symptoms of psychosis, sleep disorders |
| **Effect on global cognition** | aRR = 1.39 (anemia - cognitive impairment) | Hedges' g = 0.128 |
| **Enhancement of effect with baseline deficiency** | Yes, pronounced | Yes, g = 0.480 vs. 0.128 |
| **Reversibility of cognitive impairment after correction in adults** | Partial (documented) | Insufficient data, results conflicting |
| **Effective doses for correction (cognitive outcomes)** | 50-100 mg/day elemental iron | 400-600 IU/day (low doses more effective than high doses) |
| **Level of evidence (GRADE)** | Moderate (B) | Low to moderate (C-B) |

*Social and Psychological Significance of Hidden Hunger*

The discourse on iron and vitamin D deficiency is typically confined to haematological and bone health – anaemia and rickets, respectively. This clinical framing, while correct, obscures a far more pervasive and socially disruptive consequence: the slow, creeping erosion of cognitive capacity in otherwise healthy, working-age adults. A person can have a normal haemoglobin level, normal calcium metabolism and yet struggle to remember a phone number, follow a complex conversation, or make a quick decision under stress. This is not a character flaw, not laziness, and not early dementia. It is the brain running on insufficient fuel.

*The Executive Function Collapses*

The frontal lobes, particularly the prefrontal cortex, are exquisitely sensitive to energy deficits and neurotransmitter imbalances. Iron deficiency reduces dopamine signalling in the frontostriatal circuits that govern working memory, attentional set shifting, and inhibitory control (Greer & Baker, 2022; Gingoyon *et al*., 2022; Ding *et al*., 2026). A person with latent iron deficiency (normal haemoglobin, low ferritin) may take 20-30% longer to complete tasks requiring sustained attention, make more errors in verbal fluency tests, and report a subjective feeling of «mental fog» that is indistinguishable from sleep deprivation (Raz *et al*., 2022; Dimas-Benedicto *et al*., 2024; Fiani *et al*., 2025). Vitamin D deficiency, acting through reduced BDNF and increased neuroinflammation, impairs the hippocampus and prefrontal cortex, leading to slower processing speed, poorer verbal memory, and reduced cognitive flexibility (Doğan *et al*., 2022; Imerbsin *et al*., 2025; Hua *et al*., 2026). Together, these deficits produce a clinical picture that mimics mild cognitive impairment but without the neurodegenerative pathology.

*The Working Memory Bottleneck*

Working memory (the ability to hold and manipulate information online for seconds to minutes) is the rate-limiting step for complex cognition. It allows you to keep a shopping list in mind while calculating a budget, or to remember the beginning of a sentence as you read to its end. Iron deficiency directly impairs working memory capacity, as shown by performance on digit span and N-back tasks (Kung *et al*., 2021; Xu *et al*., 2021; Zachariou *et al*., 2021). Vitamin D deficiency adds an independent hit by slowing the speed of information processing, effectively narrowing the bottleneck even further (Silva *et al*., 2022; Bailey & Pettersen, 2024; Dalibalta *et al*., 2025). A person with both deficiencies may find themselves unable to follow multi-step instructions, lose track of conversations, and avoid tasks that require simultaneous handling of several pieces of information. This is not simply «being distracted» – it is a measurable reduction in cognitive firepower.

*Emotional Dysregulation and Social Withdrawal*

Beyond cold cognition (memory, attention, reasoning), both deficiencies impair hot cognition – the emotional and social aspects of decision-making. Iron deficiency is consistently associated with increased anxiety, irritability, and depressive symptoms, even in the absence of anaemia (Leung & Kyung, 2023; Lim *et al*., 2024). Vitamin D deficiency has been linked to negative symptoms of psychosis (apathy, anhedonia, social withdrawal), as well as to poor sleep quality and fatigue (Gaughran *et al*., 2021; Guirgis *et al*., 2023; Tsiglopoulos *et al*., 2024). A person who is tired, apathetic, anxious, and sleep-deprived is not going to perform well at work, maintain strong relationships, or engage in complex social negotiations. The cumulative effect is a gradual withdrawal from social and professional life, often mislabelled as «burnout» or «personality change».

*The Generational Burden: Why This Matters Now*

The prevalence of latent iron deficiency among women of reproductive age reaches 40-55% in many populations, and vitamin D deficiency affects 30-50% of adults in urbanised and northern regions (Silva *et al*., 2022; Bellad *et al*., 2024; Chen *et al*., 2024; Kolarš *et al*., 2025). These are not rare conditions affecting marginalised groups – they are the statistical norm for large segments of the working-age population. A young professional who cannot sustain attention, a parent who cannot manage the mental load of family logistics, a manager who makes slower, more error-prone decisions – these are not isolated tragedies but a systemic drain on human capital. The economic cost of micronutrient deficiency is measured not only in healthcare expenditures but in lost productivity, reduced innovation, and impaired social cohesion.

*The Hidden Epidemic*

Unlike anaemia or rickets, latent deficiency produces no dramatic clinical signs. The patient does not appear pale, does not have bone

pain, and does not complain of anything specific. They say «I am just tired» or «I cannot concentrate». These complaints are easily dismissed by physicians who are trained to look for disease, not for suboptimal function. And yet, the gap between «normal» (ferritin >15 μg/L, 25(OH)D >12 ng/mL) and «optimal» (ferritin >50 μg/L, 25(OH)D >30 ng/mL) is precisely where cognitive impairment emerges (Chen *et al*., 2024; Kolarš *et al*., 2025; McManus *et al*., 2025). This gap is invisible to standard screening panels, but it is devastating for the individuals who live in it. They are not sick by laboratory criteria, but they are also not well. They are functioning at 70-80% of their cognitive potential, often without realising that improvement is possible.

*A Call for Awareness*

The social significance of hidden hunger lies in its invisibility. No one blames a person for having anaemia or rickets. But a person with "brain fog" is often blamed for being lazy, inattentive, or poorly motivated. The stigma is internalised: they begin to doubt their own abilities, accept suboptimal performance as their «normal», and lose confidence in their professional and personal lives. Breaking this cycle requires, first, awareness among clinicians that latent deficiency is real and clinically important; second, routine screening of ferritin and 25(OH)D in patients with nonspecific cognitive complaints; and third, a shift in cultural expectations – from viewing cognitive decline as an inevitable consequence of ageing or stress, to recognising it as a potentially reversible metabolic condition.

## Results and Discussion

The present analysis confirms that both deficiencies are significant modifiable risk factors for cognitive decline, but their clinical trajectories differ substantially. The most important practical insight is that cognitive impairment begins at the stage of latent deficiency – before anaemia or osteomalacia become detectable by routine screening. This state is systematically overlooked in primary care, yet it impairs work performance, emotional well-being, and quality of life in working-age adults.

The most striking inconsistency in the evidence concerns vitamin D. Observational studies consistently link low 25(OH)D with cognitive deficits, yet randomised controlled trials (RCTs) often fail to show significant improvement after supplementation (van den Berg *et al*., 2021; Chen *et al*., 2024). This paradox likely arises from three factors: most RCTs enrol cognitively healthy adults with only mild deficiency (a ceiling effect); supplementation durations of 6-12 months may be too short to restore neuroplasticity; and early-life vitamin D deficiency may cause irreversible structural changes through developmental programming. For iron, the evidence is more coherent: both observational and interventional studies show cognitive improvement after correction, especially in individuals with low baseline ferritin. Nevertheless, whether long-standing iron deficiency from early childhood is fully reversible in adulthood remains uncertain.

Interpreting the available data requires caution due to heterogeneity in cognitive assessment tools, deficiency thresholds, and follow-up durations. Moreover, the two deficiencies rarely occur in isolation; they often coexist with B12, zinc, calcium, or magnesium deficits, and most studies do not control for these confounders. Most vitamin D research has been conducted in older adults or patients with comorbidities, leaving a critical gap for healthy adults aged 25-45 years.

From a clinical perspective, screening for ferritin (not just haemoglobin) and 25(OH)D is indicated in patients complaining of "brain fog", especially women of reproductive age, vegetarians, and office workers. The conventional laboratory “normal” ranges (ferritin 15-150 μg/L; 25(OH)D >12 ng/mL) are suboptimal for cognitive health; target levels should be at least 50 μg/L and 30-50 ng/mL, respectively (Chen *et al*., 2024; Liu *et al*., 2025; McManus *et al*., 2025). For iron deficiency, oral supplements should be taken every other day (rather than daily) to avoid hepcidin blockade and reduce gastrointestinal side effects. For vitamin D deficiency, cholecalciferol 800-2000 IU/day with food is recommended; low doses (400-600 IU/day) may be as effective for cognitive outcomes as higher doses. Importantly, micronutrient replacement does not replace a full diagnostic work-up for other causes of cognitive decline (depression, sleep apnoea, hypothyroidism, neurodegenerative disease) (Hung *et al*., 2022; Jones *et al*., 2024).

Several research priorities emerge. First, high-quality RCTs of iron and vitamin D supplementation (alone and in combination) are needed in healthy adults aged 25-45 years with confirmed latent deficiency, using validated neuropsychological test batteries. Second, long-term cohort studies should determine whether a critical window of irreversibility exists. Third, the potential synergistic effects of combined repletion (dopamine + BDNF pathways) warrant investigation. Fourth, social-epidemiological research is required to identify barriers to screening and correction in different socio-economic groups.

In summary, hidden hunger is not merely a medical problem but a socially significant factor that directly affects the cognitive health and economic productivity of the working-age population. Early detection and appropriate correction are accessible, effective strategies for preventing "brain fog", anxiety, depressive symptoms, and reduced quality of life.

## Conclusion

Iron and vitamin D deficiency is a highly prevalent, yet potentially modifiable risk factor for cognitive decline and psycho-emotional disorders in adults. The most vulnerable groups include women of reproductive age, vegetarians, office workers, and residents of northern latitudes.

Cognitive decline (reduced attention, working memory, processing speed, and executive function) can develop already at the stage of latent deficiency, i.e., with normal haemoglobin but low ferritin, or with 25(OH)D levels below 20 ng/mL. This condition is routinely missed in clinical practice, yet it substantially impairs quality of life and work productivity.

The mechanisms of iron and vitamin D action differ but are complementary. Iron is critical for dopamine and serotonin

synthesis, myelination, and mitochondrial energy metabolism. Vitamin D regulates the expression of neurotrophins (BDNF, NGF), modulates neuroinflammation, and supports antioxidant defence. Partial reversibility of cognitive impairment following iron correction has been well documented in adults; for vitamin D, the evidence on reversibility remains conflicting and requires further study.

Screening for ferritin (target level at least 50 μg/L) and 25(OH)D (target range 30-50 ng/mL) is indicated in adults complaining of "brain fog", particularly in the presence of risk factors, regardless of whether classical signs of anaemia or osteomalacia are absent. Correction should be personalised. For iron, low doses (50-100 mg of elemental iron every other day) are preferred to enhance absorption and reduce side effects. For vitamin D3, a daily dose of 800-2000 IU is recommended; notably, low doses (400-600 IU/day) may be as effective for cognitive outcomes as higher doses.

Hidden hunger is no longer a purely medical problem but a socially significant challenge, especially for the economically active population. Timely diagnosis and correction of iron and vitamin D deficiency are accessible and effective tools for preserving cognitive health, preventing anxiety and depressive disorders, and improving quality of life. Future research should focus on large randomised controlled trials in the 25-45-year age group, investigation of synergistic effects of combined iron and vitamin D repletion, and the development of population-based screening and prevention strategies.

**Acknowledgments:** The authors would like to thank the medical librarians at Ingush State University (Magas, Republic of Ingushetia, Russia) for their assistance with the literature search.

**Conflict of interest:** None

**Financial support:** None

**Ethics statement:** As this article is a narrative literature review and does not involve original data collection from human participants or animals, ethical approval was not required. All cited studies were conducted in accordance with their respective ethical standards and the principles of the Declaration of Helsinki.

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