

BIOCHEMICAL SIGNIFICANCE OF VITAMINS AND MINERALS

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**Abstract:** Vitamins and minerals are vital micronutrients that do not provide energy to the human body directly, yet they ensure energy metabolism, anabolic (plastic) processes, antioxidant protection, hormonal regulation, and immune-metabolic stability. This article systematically presents the fat-soluble and water-soluble groups of vitamins, the biochemical functions of macro- and microelements, their synergistic and antagonistic interactions, and the influence of the food matrix and technological processing on micronutrient bioavailability. In addition, metabolic risks associated with micronutrient deficiency and excess, as well as practical aspects of laboratory assessment based on biomarkers, are discussed. From a research perspective, viewing micronutrients as an integrative “enzyme-signal-tissue” network contributes to evidence-based dietary optimization and the development of functional food strategies.

**Keywords:** vitamins, minerals, coenzyme, cofactor, antioxidant system, bioavailability, homeostasis, trace elements, hypovitaminosis, metabolic regulation.

**INTRODUCTION**

In modern nutrition science and medical biochemistry, micronutrients are interpreted not merely as “additional substances,” but as regulators that sustain the architecture of metabolism. While proteins, fats, and carbohydrates supply energy and building substrates to the organism, vitamins and minerals ensure the correct breakdown, synthesis, transport, and coordination of these substrates at the cellular and organ levels. From a biochemical viewpoint, many vitamins determine the direction and rate of reactions in their coenzyme forms, whereas minerals function as structural components of enzymes (Zn, Fe, Cu, Mn), as signaling ions ( $\text{Ca}^{2+}$ ), as regulators of osmotic balance and membrane potential ( $\text{Na}^+$ ,  $\text{K}^+$ ,  $\text{Cl}^-$ ), or as substrates for hormone synthesis (I). Therefore, disruption of vitamin-mineral balance may lead to the breakdown of “key links” in biochemical pathways, manifesting clinically from clearly defined syndromes to subclinical metabolic shifts. In today’s dietary model, the growing share of refined products, seasonality, intensified technological processing, and individualized needs require an evidence-based reassessment of micronutrient sufficiency. The aim of this article is to summarize-at an advanced academic level-the principal biochemical functions of vitamins and minerals, the regularities of their interactions, and the determinants of bioavailability in foods, thereby presenting scientific conclusions applicable to dietary optimization and to educational and practical contexts.

**MAIN PART**

Most water-soluble vitamins (the B group) serve as coenzymes or their precursors for enzymes. For example, thiamine (B1) supports decarboxylation reactions in the pyruvate dehydrogenase and alpha-ketoglutarate dehydrogenase complexes, thereby contributing to aerobic energy production.

Riboflavin (B2), as part of FMN/FAD, participates in electron transport chains and dehydrogenase activity.

Niacin (B3), in the forms NAD<sup>+</sup>/NADP<sup>+</sup>, is a central coenzyme of redox metabolism. Folate (B9) and cobalamin (B12) participate in one-carbon fragment metabolism and are essential for DNA synthesis and methylation processes; their deficiency is associated with megaloblastic anemia and neurological disorders. Ascorbic acid (vitamin C) acts as a reductant for prolyl/lysyl hydroxylases in collagen synthesis and enhances iron absorption.

Fat-soluble vitamins are distinguished by their regulatory functions. Vitamin A (retinoids) influences cell differentiation and the stability of epithelial tissue and participates in the visual cycle (rhodopsin). Vitamin D (calcitriol) alters gene expression via nuclear receptors and regulates calcium-phosphorus homeostasis. Vitamin E (tocopherols) is a chain-breaking antioxidant that halts lipid peroxidation in membranes. Vitamin K is required for gamma-carboxylation of Gla-proteins such as clotting factors and osteocalcin.

Redox homeostasis is important for cellular signaling and membrane stability. Antioxidant defense is multicomponent and consists of enzymatic elements (superoxide dismutase, catalase, glutathione peroxidase) and non-enzymatic elements (vitamins E and C, carotenoids, glutathione).

Minerals are decisive in this system: Zn, Cu, and Mn function in superoxide dismutase isoforms; Se is required for glutathione peroxidase and thioredoxin reductase; and Fe acts as a cofactor in catalase and peroxidases. Therefore, evaluating antioxidant status comprehensively-considering not only vitamins but also trace elements-is appropriate.

Calcium and phosphorus form the basis of the mineral component of bone tissue; Ca<sup>2+</sup> also functions as a second messenger in muscle contraction, secretion, and neuronal transmission. Magnesium forms complexes with ATP and is an obligatory component for many kinase and ATPase reactions. Iron, beyond oxygen transport in hemoglobin/myoglobin, ensures mitochondrial energy metabolism as part of cytochromes and reductases. Iodine is necessary for thyroid hormone synthesis (T3, T4), influencing basal metabolic rate and growth and development. Zinc participates in the activity of hundreds of enzymes (dehydrogenases, DNA/RNA polymerases) and transcription factors and is crucial for immunity, skin health, and wound healing. Selenium is required for selenoproteins linked to antioxidant protection and thyroid hormone metabolism.

Iron deficiency is characterized by decreased ferritin, reduced transferrin saturation, and limited erythropoiesis; clinically, it manifests as anemia, fatigue, and cognitive decline. Iodine deficiency reduces thyroid hormone synthesis and increases the risk of delayed neurodevelopment in children. Vitamin D deficiency may lead to disturbances in Ca/P metabolism and decreased bone mineral density.

Excess intake is also dangerous. High doses of vitamin A may cause hepatotoxicity and teratogenicity; excess vitamin D may lead to hypercalcemia and nephrocalcinosis; and uncontrolled iron supplementation may intensify oxidative stress. Therefore, prevention requires identifying risk groups, individualizing dosing, and monitoring through biomarkers.

Thus, to ... the biochemical essence of vitamins, their functional classification is crucial:

- those serving as coenzymes/cofactors,
- those participating in antioxidant and membrane protection,
- hormone-like / gene-expression regulators,
- those supporting specific tissue functions.

A shared biochemical feature of fat-soluble vitamins (A, D, E, K) is transport within a lipid matrix, storage in the liver/adipose tissue, and a relatively higher toxic potential when consumed excessively. For instance, vitamin A, via retinoids, participates in epithelial differentiation and transcriptional regulation; its deficiency weakens epithelial barriers and causes shifts in the visual cycle. Vitamin D, rather than being a classic “vitamin,” is a steroid-like prohormone that, in the forms 25(OH)D and 1,25(OH)<sub>2</sub>D (calcitriol), regulates calcium-phosphorus homeostasis, osteoblast-osteoclast balance, and certain immune-metabolic signals. Vitamin E (tocopherols) preserves membranes as a “chain-breaking” antioxidant that interrupts lipid peroxidation; its effect is expressed in functional synergy with selenium-dependent glutathione peroxidase systems. Vitamin K is required for  $\gamma$ -carboxylation reactions and participates in the activation of coagulation factors and bone matrix proteins (osteocalcin).

Water-soluble vitamins (C and the B group) are more often the “starting key” of metabolic reactions and, because they do not form large reserves in the body, require regular intake. B<sub>1</sub> (thiamine), as TPP, participates in pyruvate dehydrogenase,  $\alpha$ -ketoglutarate dehydrogenase, and transketolase reactions, forming an integral part of carbohydrate metabolism and the pentose phosphate pathway. B<sub>2</sub> (riboflavin) supports oxidation-reduction chains via FMN/FAD, while B<sub>3</sub> (niacin) ensures glycolysis, the TCA cycle,  $\beta$ -oxidation, and biosynthetic reactions via NAD<sup>+</sup>/NADP<sup>+</sup>. B<sub>5</sub> (pantothenate) is part of coenzyme A; therefore, it is central to acetylation, lipid metabolism, and energy generation. B<sub>6</sub> (pyridoxal phosphate) is important in amino acid transamination/decarboxylation and in neurotransmitter synthesis. Folate (B<sub>9</sub>) and cobalamin (B<sub>12</sub>) work together in one-carbon fragment metabolism, DNA synthesis, and erythropoiesis; consequently, their deficiency can lead to conditions such as megaloblastic anemia. Vitamin C (ascorbate) supports hydroxylation reactions in collagen biosynthesis, enhances absorption of non-heme iron, and may also participate in restoring vitamin E within the antioxidant network.

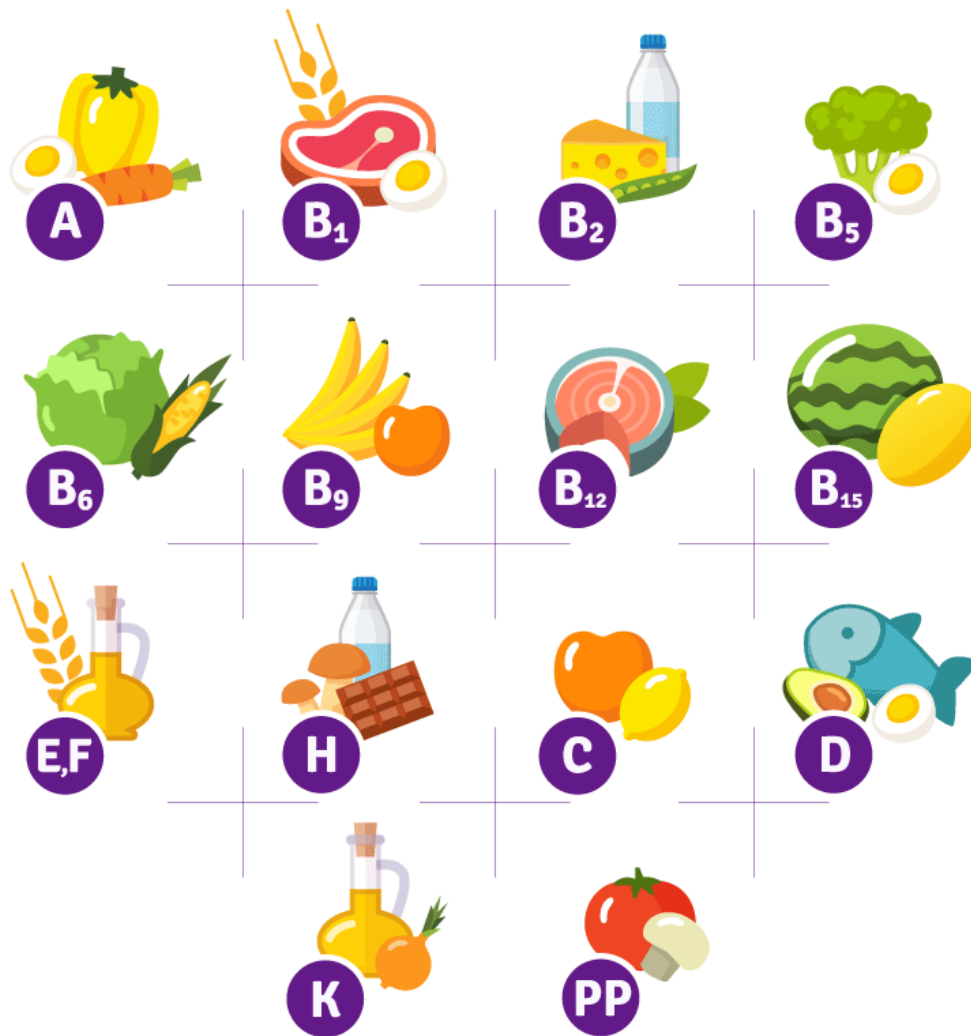


Figure 1. Biochemical significance of vitamins and minerals

In mineral biochemistry, macronutrients (Ca, P, Mg, Na, K, Cl, S) and micronutrients/trace elements (Fe, Zn, Cu, Mn, Se, I, Mo, Cr, Co, and others) are distinguished. Macronutrients often ensure structural and physicochemical conditions:  $\text{Ca}^{2+}$  is a universal ion of intracellular signaling and participates in muscle contraction, secretion, and apoptosis regulation; P determines energy and membrane architecture as a component of ATP, nucleic acids, and phospholipids;  $\text{Mg}^{2+}$  stabilizes the biologically active ATP complex (Mg-ATP) and creates conditions for many kinase and synthetase reactions;  $\text{Na}^+/\text{K}^+$  underlie membrane potential, osmotic pressure, and transport processes via  $\text{Na}^+/\text{K}^+$ -ATPase;  $\text{Cl}^-$  is linked to acid-base balance and gastric HCl synthesis.

Trace elements are extremely important from the standpoint of enzymology: Fe ensures oxygen transport and electron transport as a component of hemoglobin/myoglobin, cytochromes, and oxidoreductases; Zn plays catalytic or structural roles in more than 300 enzymes and influences transcriptional regulation via “zinc-finger” motifs; Cu is required for enzymes such as cytochrome c oxidase and lysyl oxidase; Mn participates in mitochondrial superoxide dismutase and a number of metabolic enzymes; Se is essential for selenoproteins, including glutathione peroxidase and deiodinases linked to thyroid hormone metabolism; I is an integral component of thyroxine ( $\text{T}_4$ ) and

triiodothyronine ( $T_3$ ) synthesis. In this way, minerals operate in the organism within a “structure-enzyme-signal” triad.

Vitamin-mineral interactions represent one of the most “delicate points” in nutrition science. For example, vitamin D deficiency reduces intestinal calcium absorption and may enhance mineral mobilization from the bone matrix via secondary hyperparathyroidism, while phosphorus balance also shifts during this process. Vitamin C supports the reduction of non-heme iron ( $Fe^{3+} \rightarrow Fe^{2+}$ ), increasing its absorption; however, phytates, oxalates, and certain polyphenols (tea/coffee) reduce the bioavailability of iron and zinc.

Competition between zinc and copper (at the transporter level) may increase the risk of copper deficiency during long-term high zinc intake; therefore, ratios must be considered in complex supplementation strategies. The antioxidant effect of vitamin E is complemented by selenium-dependent enzyme systems: one acts within membranes, the other neutralizes peroxides. Thus, micronutrients function not in isolation but as an interconnected metabolic network.

In foods, the practical value of micronutrients is determined not only by their amount but also by bioavailability. Bioavailability is influenced by the food matrix, technological processing, and storage conditions. Heat treatment may reduce labile compounds such as vitamin C and folates; fat-soluble vitamins, especially vitamin E, are sensitive to oxidation, and losses increase with prolonged exposure to light and air.

Minerals do not “disappear” with heat, but their bioavailability may change: for instance, phytates in grains bind iron/zinc and reduce absorption; fermentation, soaking, and flour-processing technologies can partially degrade phytates and thereby increase bioavailability. From this perspective, the development of functional foods requires scientifically grounded technologies that preserve vitamin-mineral content (additional antioxidant protection, appropriate packaging, and rational thermal regimes).

Laboratory and clinical biochemistry rely on biomarker systems to assess micronutrient sufficiency. In iron metabolism, indicators such as ferritin and transferrin saturation are used; for vitamin D, 25(OH)D; for  $B_{12}$ /folate, specific biochemical markers alongside blood cell morphology; for calcium, albumin-corrected total calcium and ionized calcium; for magnesium, serum magnesium, and similar approaches. Importantly, interpretation of biomarkers must consider factors such as inflammation, kidney/liver function, hormonal status, and dietary habits; otherwise, subclinical deficiency or functional insufficiency may remain hidden behind apparently “normal” values.

## DISCUSSION

Rather than listing vitamins and minerals separately, it is scientifically appropriate to explain them as a network of metabolic regulation:

- enzyme activity (coenzymes/cofactors),
- signaling ( $Ca^{2+}$ , phosphorylation reactions, thyroid hormones),
- tissue remodeling (bone, blood, epithelium) are integrally connected.

Therefore, dietary approaches focused on “one vitamin” or “one mineral” may fail to produce the expected outcome; a complex balance is essential.

A second aspect is the need to clarify food composition data under local conditions: the variety/cultivar of a product, the soil in which it is grown, and storage and preparation methods can significantly alter the micronutrient profile.

A third aspect is individualization of needs: during adolescence, pregnancy, lactation, high physical workload, gastroenterological disorders, and conditions such as metabolic syndrome, micronutrient requirements and absorption may change.

Thus, nutritional strategies based on biochemical knowledge (rational diet, and monitoring/correction when necessary) provide high effectiveness in prevention and health improvement. At the same time, because excessive micronutrient intake can also generate risk factors, making decisions within the triad of “norm-biomarker-clinical state” is scientifically justified.

### **CONCLUSION**

Vitamins and minerals, as the regulatory “core” of the biochemical system, ensure enzymatic reactions, antioxidant protection, hormonal control, and tissue homeostasis. Differences between fat- and water-soluble vitamins are expressed in storage, transport, and toxic potential, while differences between macro- and microelements are reflected in their contributions to structure, signaling, and enzymology. Micronutrients operate through synergy and competition; therefore, in dietary optimization it is necessary to consider bioavailability, technological processing, and individual needs in addition to quantity. In practice, biomarker-based assessment helps to detect micronutrient deficiency or functional insufficiency early. From a scientific standpoint, enriching local food composition data, evaluating micronutrient profiles and bioavailability across the technological chain (raw material-storage-preparation), and developing integrated nutritional strategies remain relevant directions for future research.

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