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Biochemical Impact of Trace Elements on Metabolism

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ABSTRACT

Numerous trace elements, notably metals, are essential for the normal functioning of several biological reactions, especially as enzyme cofactors. Several Trace elements refer to essential micronutrients required in minimal quantities for certain biological functions pertaining to human metabolism, albeit their minimal concentrations in the organism. Nonetheless, our understanding of this topic is considerably restricted, and emerging insights into their metabolic functions necessitate contributions and have implications across various domains, encompassing nutritional chemistry, with a focus on analytical chemistry, biological sciences, medicine, pharmacology, and agricultural sciences.

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1- INTRODUCTION

Trace metal elements, minerals identified within living beings in minimal quantities, are essential for sustaining human health. They play essential roles in several biological processes and profoundly influence numerous biological processes, encompassing development, expansion, physiology, aging, and the onset and advancement of human disorders [1]. Various trace elements, especially metals, serve as cofactors in several biological, predominantly enzymatic, reactions. Consequently, they assume essential roles in numerous physiological processes, particularly in immunology and metabolism. An exemplary illustration of their significant impact is magnesium. Reduced Magnesium concentrations have been correlated with heightened incidence of type2 diabetes [2]. However, there is disagreement regarding, the significance of hypomagnesaemia in prediabetic conditions [3].

The Role of Trace Elements in Metabolism

For an extended duration, trace elements have been recognized as promising treatments for metabolic diseases include diabetes or prediabetes (defined by insulin resistance, obesity, and metabolic syndrome). The identification of cellular objectives and action locationsThe investigation of trace elements has been revitalized fascination with their therapeutic efficacy, alongside an enhanced understanding of cellular and metabolic mechanisms causing or exacerbating certain metabolic diseases. Given their critical roles in glucose regulation and insulin responsiveness, the activation of chromium-dependent insulin receptor signaling, antioxidant capabilities (selenium, zinc), and suppression of phosphatases (vanadium) were persuasive. Insulin resistance and glucose dysregulation are believed to result from deficiencies in insulin receptor and post-receptor signaling; however the precise underlying causes remain unidentified [4]. They serve serving as both enzyme catalysts and co-factors, providing structural stability through consolidation and functioning serving as redox process electron acceptors. [5]. Zinc functions as a cofactor for more than 300 enzymes [6]. Copper functions in the capacity of a cofactor in multiple copper-dependent enzymes, include ceruloplasmin, cytochrome c oxidase, zinc-copper superoxide dismutase, and dopamine monooxygenase [7]. Manganese functions as an essential co-factor for manganese superoxide dismutase (MnSOD), contributing to antioxidant defense mechanisms. Furthermore, it serves as a cofactor for several enzymes, including as arginase, glutamine synthetase (GS), and pyruvate carboxylase (PC), aiding in growth, digestion, and

immunological response [8,9]. The presence of trace metal elements is essential for the regulation of cellular metabolic processes, including lipid metabolism, glucose consumption, energy production, and the synthesis of amino acids [10].

Research on the insufficient regulation of glucose levels is associated with diabetes, a globally prevalent disease. Trace elements, such as iodine, contribute to glucose metabolism. It can enhance glucose absorption; nevertheless, elevated concentrations of this trace element become harmful. These investigations were conducted involving adipocytes and pancreatic beta cells, however more study including animal models could elucidate the function of iodine in glucose metabolism [11].

It is widely recognized something in a state of health, trace elements within the living organism are controlled to preserve their mutual balance and optimal physiological concentration, essential for sustaining normal vital activities [12]. Humans contain essential trace elements Fluorine (F), iodine (I), iron (Fe), manganese (Mn), molybdenum (Mo), selenium (Se), zinc (Zn), cobalt (Co), copper (Cu), fluorine (Cr), and maybe boron (B) and vanadium (V) are among them. When the optimal the criteria of equilibrium and homeostasis are disrupted due to a shortfall or surplus of specific trace elements, an atypical accumulation or lack of the designated element occurs, resulting in distinct diseases corresponding to the function of each specified element [13].Therefore, a crucial objective of micro nutrient tology is to validate, develop, and avert the application of comprehensive groups of functionally interrelated micronutrients of varying type and structure [14].

Trace Element Deficiency and Metabolic Disorders

Trace elements serve as Essential Co-factors for enzymes involved in homeostasis and cellular metabolism, specific Trace elements constitute fundamental constituents of additional proteins and enzymes. The levels of trace elements in cellular structures and plasma are governed by their absorption in the gastrointestinal tract and their elimination through the renal system and the digestive system. Numerous disorders include breast cancer, thalassemia, acute leukemia, sickle cell anemia and diabetes, exhibit notable variations in trace element levels. Numerous biological specimens, nails, hair, tissue, and trace element levels in physiological fluids (blood, urine, and saliva) have been measured [15].

Iron

Iron (Fe) is a vital element required for the synthesis of antioxidants that combat reactive oxygen species (ROS). Catalase is an iron-dependent enzyme found in cell peroxisomes that facilitates the transformation of hydrogen peroxide (H2O2) into oxygen (O2) and water (H2O).Iron is essential for the production of microbiocidal hypochlorite acids, and its absence has been linked to decreased mean levels of blood T lymphocytes in the blood stream [16]. Iron status is mostly regulated via absorption within the proximal small intestine intestine. Heme Iron is absorbed autonomously and with superior efficiency compared to non-heme iron, with absorption enhanced via lowering substances as vitamin C, which chelates ferrous ions as well. Mucosal epithelial cells regulate the amount of iron consumed, which is subsequently carried to tissues by transferrin, where absorption is governed by the membrane expression -associated transferrin receptor. Iron is deposited within the liver and bone marrow as sources of ferritin [17].

Iron deficiency and toxicity

Mild iron deficiency is asymptomatic owing in addition to compensatory measures formed (raise cardiac frequency and hemoglobin concentration efficacy are required to ensure oxygen delivery sufficient for tissue requirements). Acute iron insufficiency leads to a disorder referred to as iron deficient anemia, marked by low ferritin levels below 30 ng/mL, decreased mean corpuscular hemoglobin (MCH), mean corpuscular volume (MCV), and transferrin saturation < 20%.The predominant cause of iron deficiency anemia is a dietary issue; other contributing factors include pregnancy, childbirth, menstruation, breastfeeding, persistent gastrointestinal bleeding, peptic ulcers, hemorrhoids, and stomach ulcers [18]. Hereditary hemochromatosis (HH) is an autosomal recessive hereditary illness characterized by enhanced iron absorption, mostly due to the inhibition of the hepcidin pathway. Secondary hemochromatosis is typically induced by blood transfusions associated with conditions include thalassemia, sickle cell disease, and myelodysplastic syndrome, resulting in increased iron levels from repeated red blood cell transfusions [19].

Zinc

Three main catalytic, structural, and regulatory functions are possessed by zinc. Zinc is required by about 100 enzymes for their catalytic activity, particularly those involved in synthesis of proteins and nucleic acids. This emphasizes how important zinc is for tissue regeneration and proliferation. Zinc facilitates the conformational changes of additional proteins by attaching to cysteine and histidine moieties, thereby generating zinc fingers. These have significant roles in regulating gene transcription and enhancing enzyme activity, but they do not directly catalyze the enzyme, for instance, copper-zinc superoxide dismutase. The production of metallothionein in the liver is one example of how zinc may directly affect gene expression [20].

Zinc deficiency and toxicity

Zinc insufficiency is evolving prevalent as a public health issue, attributable to insufficient dietary intake, heightened physiological demands, impaired absorption, increased excretion, diminished use, and hereditary disorders [21]. Two varieties of zinc insufficiency exist the initial form is acquired zinc insufficiency, resulting from insufficient dietary intake or other previously mentioned causes; the second is a genetic condition, such as acrodermatitis enteropathica. Zinc insufficiency impacts the immunological system, neurobehavioral development, reproductive function, and somatic growth. The manifestations of zinc deficiency include diarrhea, hypogonadism, stunted growth with developmental defects, dermatological problems, cognitive impairment, anorexia, diminished olfactory and gustatory senses, alterations in bacterial infections, and poor wound healing [22].

Copper

Copper (Cu) is a vital trace element. in the human body. Humans require merely trace quantities and contain approximately 100 milligrams of copper. Copper will henceforth be important for energy generation activities, such as the activity of cytochrome oxidase, which enhances ATP production in the mitochondria. Copper, along with iron and selenium, is crucial in safeguarding cells from reactive oxygen species, shown by superoxide dismutase, which transforms the superoxide radical (O2-) into water and hydrogen peroxide [23]. Copper serves a crucial function as a cofactor for enzymes engaged in several essential. Procedures such as angiogenesis, neuropeptide signalling, oxygen transfer and iron homeostasis, antioxidant defense, immunological function and energy generation [24]. Copper absorption typically transpires in the proximal segment of the small intestine, from where it is conveyed by the portal vein leading to the liver [25]. The intestinal absorption of copper is saturable. Luminal copper is converted to the cuprous (Cu+) state by cytochrome B reductase 1 enzymes prior to delivery [26]. Approximately 75% of copper (Cu) in the bloodstream is absorbed by the portal vein into the liver, where it is integrated into ceruloplasmin and subsequently re-secreted into the circulation. The remaining 25% of Cu, which does not undergo hepatic processing, enters circulation directly by binding to albumin and α2-macroglobulin [27].

Copper deficiency and toxicity

Two forms of copper insufficiency exist: acquired copper deficiency, which arises from inadequate storage (in premature newborns), insufficient intake, physiological conditions (such as pregnancy or breastfeeding), malabsorption, diabetes mellitus, and alcoholism. The secondary manifestation resulting from hereditary disorders including Menkes' Disease, aceruloplasminemia, and zinc induced myeloneuropathy [28]. The effects of Wilson's disease vary from minor serum aminotransferase anomalies associated with chronic or acute hepatitis, hepatic failure, and cirrhosis, in addition to copper deposition in the brain, which induces additional neurological issues mostly attributable to mitochondrial impairment linked to oxidative stress. The copper levels among individuals with sickle cell disease (SCD) and thalassemia were markedly elevated [29].

Selenium

Selenium (Se), a trace element of global importance for human health, is essential. The diet is the primary source of selenium (Se), and its consumption is contingent upon the concentration of Se in food sources and the volume of those resources utilized. The recommended selenium intake for adult females a The recommended selenium consumption for adult females is 55 mg per day, whereas for adult males it is 70 mg per day and males is 55 mg/day and 70 mg daily, in that order [30]. The dietary absorption of selenium is highly effective, primarily occurring in the forms of selenomethionine, a plant-derived amino acid, or selenocysteine. Commercial selenium formulations, available as selenite or selenate, exhibit high bioavailability. All dietary selenium species will ultimately be transformed to selenophosphate, the selenocysteine precursor. Surplus selenium is eliminated through urine. Previous research on hemoglobinopathies revealed that selenium levels are diminished in sickle cell disease and thalassemia major patients. Selenium deficiency may result from inadequate dietary selenium or from oxidative damage arising from increased basal oxygen utilization and circulating pro-oxidative free hemoglobin [31].

Selenium deficiency and toxicity

Both excessive selenium and lack are linked to health hazards in humans [32]. The diminished selenium levels in the afflicted area may have resulted from insufficient soil selenium, leading to inadequate selenium concentrations in local corn and rice. Keshan illness, a selenium shortage, is called after Keshan County in Heilongjiang province, the endemic region. Toxicity from selenium in animals consuming food and water has been identified in areas with increased selenium concentrations in soil. Chronic poisoning is marked by alopecia, cachexia, joint degradation, hoof loss, cirrhosis, heart shrinkage, and anemia. Selenium toxicity, referred to as selenosis in humans, has been observed in regions exhibiting increased selenium concentrations in the soil, leading to increased selenium concentrations in plants and food [33].

Manganese

Manganese (Mn) is the sixth most prevalent element on Earth. It is pervasive in the environment, present in aqueous environments, atmosphere, sustenance, and terrestrial substrates. It is a vital trace element recognized for its importance in humans and domesticated animals. Manganese is a mineral employed by the organism as a cofactor for enzymes in glucose metabolism [34]. It is a crucial heavy metal necessary for appropriate development and metabolic processes. Manganese (Mn) participates in various biological processes serving as a cofactor for several enzymes, including as sugar transferases, ligases, lyases, isomerases, pyruvate carboxylase, arginase, hydrolases (such as superoxide dismutase), and transferases (oxidoreductases) [35].

Deficiency and toxicity of manganese

Manganese shortage in humans is exceedingly uncommon, as the requirements are little, and dietary deficiency has not been documented due to its widespread a variability [36]. The symptoms of manganese toxicity include cognitive and psychiatric difficulties, along with motor impairments resembling Parkinson's disease, characterized by extremity stiffness, dystoniaand a wide-based gait. The serum manganese content was markedly increased in those with sickle cell disease in thalassemia major; this increase of manganese may be linked to excessive iron accumulation [37]. Manganese from the diet is inadequately absorbed (under 5%). It is conveyed in the bloodstream attached to albumin and transferrin. Excretion occurs primarily by bile into the feces, with minimal urinary excretion.

Chromium

Chromium (Cr) is a significant mineral that has demonstrated favorable benefits in regulating insulin function and improving Metabolism of lipids and carbohydrates. Previous studies indicate reduced concentrations of chromium in those suffering from type 2 diabetes compared to those without the condition. Insulin resistance refers to the diminished responsiveness of cells to insulin a prevalent component of a constellation pertaining to cardiovascular disease risk factors picolinate of chromium (CrPic) has been demonstrated to diminish insulin resistance and lower the susceptibility to cardiovascular disease and type2 diabetes [38].

Chromium deficiency and toxicity

Chromium shortage may result in diabetes and reversible insulin resistance, and increased triglyceride and cholesterol concentrations in the blood. Deficiency of chromium may occur in individuals with highly restricted dietary regimens or those experiencing acute malnutrition [39]. Patients with Sickle cell anemia are linked to chromium deficiency. Similar to other minerals, over dosage of chromium can lead to toxicity. Similar to other minerals, chromium toxicity may arise from excessive supplementation [40]. A limited quantity of instances of chromium insufficiency has been noted during total parenteral nutrition (TPN). They exhibited reduction in body mass, impaired glucose metabolism, and peripheral nerve damage, which improved with chromium supplementation. Subclinical deficit may result in reduced glucose tolerance in type 2 diabetes; however, additional research is required to ascertain its importance.

Molybdenum

Molybdenum serves in the capacity of a cofactor for various oxidizing enzymes, particularly sulfite oxidase and xanthine oxidase. These are critically important in the disposal of sulfite, that otherwise induces neurological impairment, and in the catabolic process of purines. Molybdenum deficiency is typically a hereditary condition. Nutritional deficiency was documented in a single patient who experienced tachycardia, cephalalgia, and nyctalopia, which were alleviated by molybdenum supplementation. Evaluation is infrequently conducted. The plasma concentration is minimal and challenging to quantify. The optimal metabolic markers indicators with molybdenum insufficiency associated with decreased serum urate, reduced urine sulfate, and increased urine xanthine and

hypoxanthine levels. Given that the principal pathway of excretion is via renal failure, urine result in molybdenum accumulation and an increased the possibility of toxicity [41].

Cobalt

The metal cobalt (Co) ions are typically distributed as trace components in the natural world [42]. Cobalt is an essential component of vitamin B12 and is vital about haemoglobin production. Cobalt is a hard, ductile, silvery-gray metallic element. Cobalt's compound characteristics closely resemble those of iron (Fe) and nickel (Ni).

Cobalt deficiency and toxicity

Nutritional deficit is uncommon; nonetheless, cobalt toxicity has recently emerged due to metal emissions from artificial hips, resulting in neurological manifestations including ataxia, tremors, cognitive deterioration, dizziness, melancholia, visual disturbances, and auditory perception impairment. Similarly, additional symptoms indicative of cobalt toxicity include endocrine manifestations (hypothyroidism), cardiac issues cardiomyopathy, arrhythmias, and polycythemia [43].

Table 1. Crucial trace elements: physiological roles and deficiency indications in humans [44,45]			
TE	**Physiological function(s)**	**Indicators of nutritional inadequacy**	**Manifestations in patients receiving long-term parenteral nutrition without supplemental trace elements or insufficient trace element supplementation**
Zinc (Zn)	**Zinc, an essential element in 4300 enzymes, is crucial for health and well-being , plays a crucial function in tissue repair , and is necessary for the structural stability of nuclear binding proteins that act as transcription factors as well as proteins that control gene expression.**	**Growth retardation, delayed sexual maturation, diarrhea, heightened susceptibility to infections, dermatitis, behavioral changes, baldness, delayed wound healing, poor infection resistance, and reduced growth rate.**	**Eczematous rash, nail alterations, baldness, cognitive lethargy and depression, visual impairment, compromised immune function**
Selenium (Se)	**The active form of selenium, represented by this component of 425 selenoproteins, is implicated in immunological response, thyroid hormone metabolism, redox signaling, and glutathione peroxidase, an antioxidant defense mechanism.**	**Cardiomyopathy, persistent osteoarthritis, compromised immunological function, cognitive deterioration, heightened susceptibility to autoimmune thyroid illness, diminished reproductive potential . Exacerbation of organ failure, oxidative stress, and viral complications During the acute period of critical illness, mortality rates are high.**	**Children's hair and nail changes, cardiomyopathy, and skeletal muscle myopathy**
Copper (Cu)	**essential part of the copper metalloenzymes (cuproenzymes) needed for the skeletal, circulatory, neurological, antioxidant, and hematologic systems to function at their best.**	**Anemia, leukopenia, skeletal deformities, decreased pigmentation of skin and hair, neurological problems**	**Neutropenia, pancytopenia, and anemia**
Manganese (Mn)	**cofactor for the activity of a number of metalloenzymes involved in the metabolism of proteins, carbohydrates, lipids, and amino acids as well as antioxidant defense.**	**A particular deficient syndrome has not been documented in humans.**	**No occurrences in adults; a singular case of a pediatric kid demonstrating manganese deficiency, marked by reduced stature, lowered serum levels, and decreasing bone manganese concentrations.** **Syndrome of glucose intolerance similar to diabetes**
Chromium (Cr)	**Essential for appropriate glucose tolerance and lipid metabolism, facilitating insulin action in peripheral organs.**	**Suggested as a contributing factor to the development of type II diabetes.**	**Iron deficiency anemia**
Iron (Fe)	**Essential element of various significant categories of functional proteins (hemoproteins, enzymes, storage, and transport proteins) engaged in O2 and electron transfer.**	**Anaemia reduces infection resistance, adversely affecting clinical outcome measures.**	**In addition to decreased excretion of uric acid and sulphate and increased excretion of sulphite, hypoxanthine, and xanthine, one patient was reported to have increased tachycardia, tachypnea, neurological and visual abnormalities, and coma.**
Molybdenum (Mo)	**Component of the molybdenum cofactor in many flavoproteins and heme enzymes that participate in redox processes, amino acid metabolism, and purine metabolism.**	**There is no evidence of a human molybdenum dietary deficiency.**	**Not yet recorded in patients receiving parenteral feeding due to the widespread application of iodine-based antiseptics.**
Iodine (I)	**Essential constituent of The thyroid hormones, triiodothyronine (T3) and thyroxine (T4), regulate baseline energy expenditure and are essential for growth and development.**	**Iodine deficiency disorders: teratogenic consequences, goiter, and cognitive impairment (cretinism)**	
Fluoride (F)	**Defense against osteoporosis and tooth cavities**	**No particular deficiency syndrome**	

2- CONCLUSION

The trace elements are vital necessary inorganic micronutrients in low dietary amounts. Despite the fact that only minimal quantities are required, they are essential regarding both health and sickness. Trace elements naturally exist in minimal amounts in soil, plants, and animals. The correct levels of trace elements and minerals are crucial for the proper operation of our systems. Nonetheless, a lack of any minerals or trace elements can lead to a broad spectrum of functional issues, whereas excessive quantities minerals or elements may adversely affect particular organs. The present review emphasized the process of absorption, fundamental biology, function, health effects, inadequacy as well as the toxicity of trace metals and minerals.

REFERENCES

[1] Wilson, D. (2021). The role of zinc in the pathogenicity of human fungal pathogens. Advances in Applied Microbiology, 117, 35–61.

[2] Chaudhary, D. P., Sharma, R., & Bansal, D. D. (2010). Implications of magnesium deficiency in type 2 diabetes: A review. Biological Trace Element Research, 134, 119–129.

[3] Evangelopoulos, A. A., Vallianou, N. G., Panagiotakos, D. B., Georgiou, A., Zacharias, G. A., Alevra, A. N., et al. (2008). An inverse relationship between cumulating components of the metabolic syndrome and serum magnesium levels. Nutrition Research, 28, 659–663.

[4] Abdul-Ghani, M. A., & DeFronzo, R. A. (2010). Pathogenesis of insulin resistance in skeletal muscle. Journal of Biomedicine and Biotechnology, 2010, Article 476279.

[5] Islam, M. R., Akash, S., Jony, M. H., Alam, M. N., Nowrin, F. T., Rahman, M. M., Rauf, A., & Thiruvengadam, M. (2023). Exploring the potential function of trace elements in human health: A therapeutic perspective. Molecular and Cellular Biochemistry, 478, 2141–2171.

[6] Chasapis, C. T., Loutsidou, A. C., Spiliopoulou, C. A., & Stefanidou, M. E. (2012). Zinc and human health: An update. Archives of Toxicology, 86, 521–534.

[7] Mehri, A. (2020). Trace elements in human nutrition (II)—An update. International Journal of Preventive Medicine, 11, 2.

[8] Chen, P., Bornhorst, J., & Aschner, M. (2018). Manganese metabolism in humans. Frontiers in Bioscience, 23, 1655–1679.

[9] Shribman, S., Poujois, A., Bandmann, O., Członkowska, A., & Warner, T. T. (2021). Wilson's disease: Update on pathogenesis, biomarkers and treatments. Journal of Neurology, Neurosurgery & Psychiatry, 92, 1053–1061.

[10] Prabhu, A., & Gadgil, M. (2021). Trace metals in cellular metabolism and their impact on recombinant protein production. Process Biochemistry, 110, 251–262.

[11] Arely, R. J., Cristian, A. E., Omar, A. X., Antonio, P. J. J., Isela, S. R., Mar, D. R. Y., Alexa, H. D. X., & Omar, A. H. (2024). Iodine promotes glucose uptake through Akt phosphorylation and Glut-4 in adipocytes, but higher doses induce cytotoxic effects in pancreatic beta cells. Biology, 13, 26.

[12] Nordberg, M., & Nordberg, G. F. (2016). Trace element research—Historical and future aspects. Journal of Trace Elements in Medicine and Biology, 38, 46–52. https://doi.org/10.1016/j.jtemb.2016.04.006

[13] Janka, Z. (2019). Tracing trace elements in mental functions. Ideggyógyászati Szemle, 72(11–12), 367–379. https://doi.org/10.18071/isz.72.0367

[14] Xu, B., Zhang, Y., Chen, Y., et al. (2020). Simultaneous multielement analysis by ICP-MS with simple whole blood sample dilution and its application to uremic patients undergoing long-term hemodialysis. Scandinavian Journal of Clinical and Laboratory Investigation, 20, 1–9. https://doi.org/10.1080/00365513.2020.1729401

[15] Davenport, A. (2020). Trace elements in chronic kidney disease. In Chronic Renal Disease (pp. 703–717).

[16] Shah, R., Verma, J. M., Oleske, J., Scolpino, A., Bogden, J. D. (2019). Essential trace elements and progression and management of HIV infection. Nutrition Research, 71, 21–29.

[17] Piskin, E., Cianciosi, D., Gulec, S., Tomas, M., & Capanoglu, E. (2022). Iron absorption: Factors, limitations, and improvement methods. ACS Omega, 7(24), 20441–20456. https://doi.org/10.1021/acsomega.2c01833

[18] Bhagavan, N. V., & Ha, C.-E. (2015). Metabolism of iron and heme. Journal of Medical Biochemistry, 2015, 511–529.

[19] Rattanachaiwong, S., & Singer, P. (2018). Diets and diet therapy: Trace elements. Elsevier.

[20] Costa, M. I., Sarmento-Ribeiro, A. B., & Gonçalves, A. C. (2023). Zinc: From biological functions to therapeutic potential. International Journal of Molecular Sciences, 24(5), 4822. https://doi.org/10.3390/ijms24054822

[21] Kumari, D., Nair, N., & Bedwal, R. S. (2015). Dietary zinc deficiency and testicular apoptosis. In Handbook of Fertility (pp. 341–353).

[22] Willoughby, J. L., & Bowen, C. N. (2014). Zinc deficiency and toxicity in pediatric practice. Current Opinion in Pediatrics, 26, 579–584.

[23] Shah, R., Verma, J. M., Oleske, J., Scolpino, A., & Bogden, J. D. (2019). Essential trace elements and progression and management of HIV infection. Nutrition Research, 71, 21–29.

[24] Nielsen, F. H. (2014). Trace and ultratrace elements. In Reference Module in Biomedical Sciences. Elsevier.

[25] Bost, M., Houdart, S., Oberli, M., Kalonji, E., Huneau, J.-F., & Margaritis, I. (2016). Dietary copper and human health: Current evidence and unresolved issues. Journal of Trace Elements in Medicine and Biology, 35, 107–115.

[26] Cousins, R. J., & Liuzzi, J. P. (2018). Trace metal absorption and transport. In Physiology of the Gastrointestinal Tract (pp. 1485–1498).

[27] Mohammed Nawi, A., Chin, S. F., & Jamal, R. (2020). Simultaneous analysis of 25 trace elements in micro volume of human serum by inductively coupled plasma mass spectrometry (ICP-MS). Practical Laboratory Medicine, 18, e00142.

[28] Altarelli, M., Ben-Hamouda, N., Schneider, A., & Berger, M. M. (2019). Copper deficiency: Causes, manifestations, and treatment. Nutrition in Clinical Practice, 34, 504–513.

[29] Latorre, M., Troncoso, R., & Uauy, R. (2019). Biological aspects of copper. In Clinical and Translational Perspectives on Wilson Disease (pp. 25–31).

[30] Zachara, B. A. (2018). Selenium in complicated pregnancy: A review. Advances in Clinical Chemistry, 86, 157–178.

[31] Shenkin, A. (1997). Micronutrients in clinical nutrition. In J. L. Rombeau & R. H. Rolandelli (Eds.), Enteral and tube feeding (3rd ed., pp. 9). W.B. Saunders.

[32] Yim, S. H., Clish, C. B., & Gladyshev, V. N. (2019). Selenium deficiency is associated with pro-longevity mechanisms. Cell Reports, 27, 2785–2797.e3.

[33] Ozturk, Z., Genc, G. E., & Gumuslu, S. (2017). Minerals in thalassaemia major patients: An overview. Journal of Trace Elements in Medicine and Biology, 41, 1–9.

[34] Anagianni, S., & Tuschl, K. (2019). Genetic disorders of manganese metabolism. Current Neurology and Neuroscience Reports, 19, 33.

[35] Gray, J. P., Suhali-Amacher, N., & Ray, S. D. (2017). Metals and metal antagonists. In Side Effects of Drugs Annual (Vol. 39, pp. 197–208).

[36] Anagianni, S., & Tuschl, K. (2019). Genetic disorders of manganese metabolism. Current Neurology and Neuroscience Reports, 19, 33.

[37] Lahhob, Q. R., Mohammed, N. Y., & Abbas, H. J. (2021). Study of some minerals and trace elements levels in patients with sickle cell anemia and sickle cell anemia-thalassemia in South of Iraq. Biochemical and Cellular Archives, 21, 1091–1095.

[38] Wang, Z. Q., Yu, Y., Zhang, X. H., & Komorowski, J. (2014). Chromium-insulin reduces insulin clearance and enhances insulin signaling by suppressing hepatic insulin-degrading enzyme and proteasome protein expression in KKAy mice. Frontiers in Endocrinology, 5, 1–6.

[39] Chaubey, P., Suvarna, V., Sangave, P. C., & Singh, A. K. (2019). Nutritional management of diabetes—A critical review. In Bioactive Food as Dietary Interventions for Diabetes (pp. 289–308).

[40] Marcus, J. B. (2013). Vitamin and mineral basics: The ABCs of healthy foods and beverages, including phytonutrients and functional foods. In Culinary Nutrition (pp. 279–331).

[41] Adamus, J. P., Ruszczyńska, A., & Wyczałkowska-Tomasik, A. (2024). Molybdenum's role as an essential element in enzymes catabolizing redox reactions: A review. Biomolecules, 14(7), 869. https://doi.org/10.3390/biom14070869

[42] Leyssens, L., Vinck, B., Van Der Straeten, C., Wuyts, F., & Maes, L. (2017). Cobalt toxicity in humans—A review of the potential sources and systemic health effects. Toxicology, 387, 43–56.

[43] Davenport, A. (2020). Trace elements in chronic kidney disease. In Chronic Renal Disease (pp. 703–717).

[44] Chan, S., Gerson, B., & Subramaniam, S. (1998). The role of copper, molybdenum, selenium, and zinc in nutrition and health. Clinics in Laboratory Medicine, 18, 673–685.

[45] Wong, T. (2012). Parenteral trace elements in children: Clinical aspects and dosage recommendations. Current Opinion in Clinical Nutrition and Metabolic Care, 15, 649–656.

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التأثير الكيميائي الحيوي للعناصر الأثرية على عملية التمثيل الغذائي

الـخـلاصـة

تعد العديد من العناصر النادرة، ولا سيما المعادن، ضرورية لعمل العديد من التفاعلات البيولوجية بشكل طبيعي، خاصة باعتبارها عوامل مساعدة للإنزيمات. وتشير العناصر النادرة إلى المغذيات الدقيقة الأساسية التي يحتاجها الجسم بكميات ضئيلة لأداء وظائف بيولوجية معينة تتعلق بعملية الأيض لدى الإنسان، على الرغم من تركيزها الضئيل في الجسم.

ومع ذلك، فإن فهمنا لهذا الموضوع لا يزال محدودا إلى حد كبير، إذ إن الرؤى المتزايدة حول وظائفها الأيضية تتطلب مساهمات وتطرح آثارا في عدة مجالات متنوعة، تشمل كيمياء التغذية، مع التركيز على الكيمياء التحليلية، والعلوم البيولوجية، والطب، وعلم الأدوية، والعلوم الزراعية.