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Impact of Genetic Polymorphisms on Micronutrient Metabolism: A Systematic Review

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Introduction

Micronutrients such as vitamins and minerals play crucial roles in human health, acting as cofactors for enzymatic reactions, antioxidants, structural components, and signaling molecules. While recommended dietary intakes provide a baseline for population-level nutritional adequacy, individual responses to micronutrient intake can vary significantly. This variation is increasingly attributed to genetic polymorphisms, or common variations in DNA sequence, that affect the absorption, transport, utilization, and metabolism of micronutrients. As the field of nutritional genomics advances, evidence is accumulating to show that these genetic differences can influence an individual's risk for nutrient deficiencies, toxicity, and related chronic diseases. This systematic review synthesizes current knowledge on how genetic polymorphisms impact micronutrient metabolism, focusing on key vitamins and minerals with well-established gene-nutrient interactions, including folate, vitamin D, vitamin A, iron, and zinc. The implications of these findings are far-reaching for clinical nutrition, public health policy, and the emerging practice of personalized nutrition [1].

Genetic variants and folate metabolism

Folate, a B-vitamin essential for DNA synthesis and methylation, is one of the most well-studied nutrients in the context of genetic variation. The MTHFR (methylenetetrahydrofolate reductase) gene is critical for converting 5,10-methylenetetrahydrofolate to 5-methyltetrahydrofolate—the biologically active form used in homocysteine remethylation. The C677T polymorphism in the MTHFR gene leads to a thermolabile enzyme with reduced activity, particularly in individuals homozygous for the T allele. This results in elevated plasma homocysteine levels, which are associated with increased risk of cardiovascular disease, neural tube defects, and certain cancers. Individuals with the TT genotype may require higher folate intake or supplementation with methylated folate forms to maintain normal metabolic function. Studies also show gene-diet interactions, where folate status modifies the impact of the polymorphism on health outcomes, emphasizing the need for genotype-informed dietary recommendations [2].

Vitamin D pathway polymorphisms

Vitamin D is essential for calcium homeostasis, bone health, immune function, and gene regulation. Its bioavailability and activity are influenced by genetic variants in several genes, including VDR (vitamin D receptor), GC (group-specific component), and CYP2R1. Polymorphisms in the VDR gene, such as FokI, BsmI, and TaqI, can alter receptor expression or function, influencing vitamin D's biological effects. For instance, the FokI polymorphism is associated with differences in bone mineral density and susceptibility to autoimmune conditions. Similarly, SNPs in the GC gene, which encodes vitamin D-binding protein, affect circulating levels of 25(OH)D and may alter tissue availability. Variants in CYP2R1, responsible for hydroxylating vitamin D in the liver, have also been linked to inter-individual differences in serum vitamin D levels. These findings help explain why some individuals with adequate sun exposure or intake may still experience deficiency and point to the potential for genotype-guided vitamin D supplementation strategies [3].

Vitamin A and genetic susceptibility

Vitamin A, vital for vision, immune function, and cellular differentiation, is obtained through preformed retinoids or provitamin A carotenoids like beta-carotene. The conversion of beta-carotene to active retinol is mediated by the BCMO1 (beta-carotene monooxygenase 1) gene. Common polymorphisms in BCMO1, such as R267S and A379V, are associated with reduced enzymatic activity and a diminished ability to convert carotenoids to retinol. As a result, individuals carrying these variants may have lower plasma retinol levels despite adequate intake of plant-based carotenoids. This has implications for populations relying heavily on carotenoid sources rather than preformed vitamin A from animal products. Personalized dietary recommendations or direct retinol supplementation may be necessary to prevent deficiency in genetically susceptible individuals [4].

Iron metabolism and genetic variants

Iron metabolism is tightly regulated, as both deficiency and overload can have severe health consequences. Genetic polymorphisms in genes involved in iron absorption, transport, and storage such as HFE, TMPRSS6, and TF (transferrin) can significantly influence iron status. The C282Y and H63D mutations in the HFE gene are associated with hereditary hemochromatosis, a disorder characterized by excessive iron absorption and accumulation. Individuals with these mutations are at risk of iron overload even at normal dietary intake levels. On the other hand, polymorphisms in the TMPRSS6 gene, which encodes matriptase-2 (a regulator of hepcidin), are associated with iron-refractory iron deficiency anemia (IRIDA). Variants in the transferrin gene can also affect iron transport efficiency and influence serum iron levels. These genetic insights are critical for guiding iron supplementation strategies and screening for iron-related disorders [5].

Zinc and genetic regulation

Zinc is a trace mineral involved in immune function, DNA synthesis, and cellular repair. Genetic polymorphisms in zinc transporter genes such as SLC30A8 (ZnT8) and SLC39A8 (ZIP8) influence zinc homeostasis and are associated with chronic disease risk.

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For example, a missense variant in SLC30A8 has been linked to altered insulin secretion and increased risk of type 2 diabetes. Moreover, SNPs in SLC39A8 have been associated with differences in zinc uptake and have been implicated in inflammatory and neurodevelopmental conditions. Although research on zinc-related polymorphisms is still emerging, current findings underscore the importance of genetic regulation in micronutrient metabolism and its potential impact on disease susceptibility [6].

Clinical and public health implications

Understanding the impact of genetic polymorphisms on micronutrient metabolism offers a foundation for precision nutrition the practice of tailoring dietary interventions based on individual genetic profiles. In clinical settings, this could enhance the diagnosis and management of micronutrient-related disorders, ensuring more effective treatment and prevention. For instance, identifying individuals with MTHFR polymorphisms can guide folate supplementation in pregnancy to reduce the risk of birth defects. Screening for HFE mutations could prevent iron overload through dietary management or phlebotomy in affected individuals [7].

On a population level, integrating genomic data into public health nutrition strategies can help refine dietary reference intakes (DRIs) and fortification policies. Traditional one-size-fits-all recommendations may not account for the nutritional needs of genetically diverse populations. For example, vitamin A fortification strategies may need adjustment in regions where BCMO1 polymorphisms are prevalent. Moreover, genetic screening could identify subgroups at risk for deficiency despite standard interventions, allowing for targeted support.

However, challenges remain in translating this knowledge into practice. The polygenic nature of nutrient metabolism, gene-environment interactions, and limited accessibility to genetic testing are significant barriers. Ethical considerations regarding genetic data privacy, equity in healthcare access, and the potential for discrimination must also be addressed. Further research, including large-scale, multi-ethnic cohort studies, is needed to validate these associations and develop robust, evidence-based guidelines for personalized micronutrient recommendations [8].

Conclusion

The interplay between genetic polymorphisms and micronutrient metabolism represents a pivotal frontier in nutrition science and preventive medicine. Variants in genes involved in folate, vitamin D, vitamin A, iron, and zinc pathways can significantly alter individual nutrient requirements and influence disease risk. These findings underscore the limitations of generalized dietary recommendations and highlight the need for more personalized approaches that consider genetic variability. As scientific understanding deepens and genetic testing becomes more accessible, the integration of genomic information into dietary planning promises to revolutionize the prevention and management of micronutrient-related health conditions. Ultimately, acknowledging and addressing genetic diversity in nutrient metabolism will be key to achieving optimal nutrition and long-term health for all individuals.

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Conflict of Interest

None

References

1. Wiklund P, Toss F, Weinehall L, Hallmans G, Franks PW, et al. (2008) Abdominal and gynoid fat mass are associated with cardiovascular risk factors in men and women. J Clin Endocrinol Metab 93: 4360-4366.
2. Piché ME, Poirier P, Lemieux I, Després JP (2018) Overview of epidemiology and contribution of obesity and body fat distribution to cardiovascular disease: an update. Prog Cardiovasc Dis 61: 103-113.
3. Fock KM, Ang TL (2010) Epidemiology of Helicobacter pylori infection and gastric cancer in Asia. J Gastroenterol Hepatol 25: 479-486.
4. Thrift AP (2020) Global burden and epidemiology of Barrett esophagus and oesophageal cancer. Nat Rev Gastroenterol Hepatol 18: 432-443.
5. Dalamaga M, Diakopoulos KN, Mantzoros CS (2012) The role of adiponectin in cancer: a review of current evidence. Endocr Rev 33: 547-594.
6. Anuurad E, Shiwaku K, Nogi A, Kitajima K, Enkhmaa B, et al. (2003) The new BMI criteria for asians by the regional office for the western pacific region of WHO are suitable for screening of overweight to prevent metabolic syndrome in elder Japanese workers. J Occup Health 45: 335-343.
7. Wiklund P, Toss F, Weinehall L, Göran Hallmans, Franks PW, et al. (2008) Abdominal and gynoid fat mass are associated with cardiovascular risk factors in men and women. J Clin Endocrinol Metab 93: 4360-4366.
8. Chooi YC, Ding C, Magkos F (2019) The epidemiology of obesity. Metabolism 92: 6-10.