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Advancements in Personalized Nutrition: Integrating Genetic and Metabolic Profiles for Tailored Dietary Recommendations in Chronic Disease Prevention and Management

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Abstract

Background: Personalized nutrition (PN) is an emerging field that leverages individual genetic and metabolic profiles to tailor dietary recommendations for optimizing health outcomes. This approach shifts the focus from generalized dietary guidelines to specific interventions that account for genetic variability and metabolic responses.

Methods: This review synthesizes recent advancements in the integration of genetic, phenotypic, and biochemical data to inform personalized dietary strategies. It examines methodologies employed in nutrigenomics, including genome-wide association studies and bioinformatics tools, to analyze gene-diet interactions. Literature on the impact of specific genetic polymorphisms on nutrient metabolism and disease susceptibility is also evaluated.

Results: Evidence suggests that genetic variations significantly influence individual responses to dietary patterns, with implications for chronic disease management, including obesity and type 2 diabetes. Studies demonstrate that tailored dietary interventions based on genetic profiling can enhance the efficacy of nutritional therapies, ultimately leading to improved health outcomes. However, challenges remain regarding the scalability and ethical implementation of these personalized strategies in diverse populations.

Conclusion: The potential of personalized nutrition to revolutionize dietary practices is substantial. By integrating genetic insights into nutritional guidance, healthcare providers can offer more effective, individualized interventions. Continued research is essential to validate these approaches and address ethical considerations, ensuring that personalized nutrition is both scientifically robust and widely accessible.

Keywords: Personalized nutrition, nutrigenomics, genetic polymorphisms, dietary interventions, chronic disease management.

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1. Introduction

Personalized nutrition (PN) is a field that use individual characteristics to develop dietary strategies for illness prevention, management, treatment, and general health enhancement. The American Nutrition Association delineates three interrelated components of this field: the scientific foundation and data pertaining to personalized nutrition (PN), professional education and training in PN, and the implementation of PN in advisory and therapeutic practices [1]. Personalized nutrition (PN) involves the use of genetic, phenotypic, biochemical, and nutritional information to assess their impact on an individual's health. It also equips healthcare practitioners to execute PN strategies in varied settings and tailor treatments to address individual needs [1]. The International Society of Nutrigenetics/Nutrigenomics (ISNN) elucidates personalized nutrition (PN), emphasizing how an individual's genetic composition and many biological and cultural factors, including dietary intolerances, preferences, and allergies, affect their nutritional response [2]. PN works on the premise that individual genetic variants may affect how certain diets or nutritional quantities alter illness risk. The scope of PN is expanded by integrating diverse phenotypic data, such as body composition metrics, physical activity levels, clinical indicators, and biochemical markers evaluating nutritional status, in conjunction with genomic information, to provide more tailored dietary recommendations [3]. This methodology is seen to operate at several levels, ranging from internet-based services to the use of genetic data in formulating tailored nutritional recommendations [3].

The historical perspective on dietary guidelines indicates a transition from managing nutritional shortages to mitigating chronic disorders linked to food excesses. Initially, dietary standards were established to guarantee nutritional sufficiency and avert deficits, especially in situations of food shortage. As cultures evolved from scarcity to plenty, the incidence of chronic illnesses, including heart disease, obesity, and diabetes, escalated, necessitating a shift in dietary recommendations aimed at preventing these ailments [4-6]. Initial dietary guidelines prioritized the intake of animal products and exhibited less focus on the prevention of chronic diseases. As data mounted connecting nutrition to chronic illness risk, a consensus emerged about the advantages of plant-based diets, such as vegetarian, Mediterranean, and Asian diets, which were correlated with reduced incidences of chronic diseases [7]. The constraints of historical dietary recommendations stem from a dependence on research that may lack robustness or comprehensiveness, resulting in guidance that may not be entirely substantiated by the existing knowledge. The first Dietary Goals for Americans suggested alterations in macronutrient intake without enough data to definitively endorse these adjustments [5]. The implementation of these recommendations for various groups, including children, sometimes relied on assumptions instead of direct proof of efficacy [4]. Moreover, the techniques used to evaluate dietary consumption, such Food Frequency Questionnaires, have been criticism for their inaccuracy and susceptibility to memory bias, which may compromise the validity of diet-disease associations identified in epidemiological research [8].

The potential of genomics to improve dietary interventions is rooted on the capacity to customize nutrition according to individual genetic differences. Nutrigenomics investigates the relationship between nutrition and genetics, analyzing how individual genetic differences affect human reactions to dietary nutrients. This domain offers possibilities for customizing dietary recommendations to meet individual health requirements, hence perhaps improving health outcomes. Genome-wide single nucleotide polymorphism (SNP) data may be used to formulate tailored food recommendations, considering an

individual's genetic diversity across different SNPs [9]. Integrating genetics with nutritional sciences may improve the effectiveness of nutritional therapies. This method enables a comprehensive study of the complex interactions between dietary elements and the human genome in different health and disease states [10]. This method may facilitate the development of dietary recommendations that are highly predictive, minimizing the risk of unexpected consequences and accounting for the influence of human genetic variations [10]. Furthermore, including genetic data into dietary treatments has shown an improvement in the precision of weight reduction models, highlighting the effectiveness of coaching based on participants' genomic risk [11]. This indicates that genetics may contribute to implementing changes beyond general healthy eating recommendations, possibly reducing the incidence of obesity and other chronic illnesses [12]. Nonetheless, it is crucial to acknowledge that while nutritional genomics shows significant promise, it remains in its nascent stages, necessitating more study to properly harness its potential in clinical applications. The ISNN acknowledges the ethical, practical, and scientific challenges that must be addressed to effectively implement the results from gene-nutrient interaction research into dependable practice recommendations for PN [12,13].

2. Genetic Variation and Nutrient Metabolism

Human genetic diversity includes the variances in DNA sequences across people, which contribute to the varied phenotypic traits found in the human population. Each human genome has around 3 million single nucleotide variations (SNVs) relative to the reference genome, with approximately 1% of an individual's genome differing from this reference sequence [14]. This category of genomic variants includes single nucleotide polymorphisms (SNPs), insertions and deletions (indels), copy number variations (CNVs), and structural alterations such as inversions and complex rearrangements [14-16]. The typical human gene has many biallelic polymorphisms, with a portion located in coding areas that may influence protein function [17]. The prevalence of these genetic variations differs between groups, with some variants being widespread and others infrequent. Rare variations often exhibit significant spatial heterogeneity, shaped by mechanisms including evolutionary conservation, coding impact, and purifying selection [15]. The 1000 Genomes Project has developed a comprehensive map of human genetic variability, including SNPs, small insertions and deletions, and bigger structural deletions. It encompasses up to 98% of available SNPs with a frequency of at least 1% among closely related populations [15]. This variance illustrates the history of human migration, demographic changes, and adaption to diverse settings [18,19]. Comprehending human genetic diversity is essential for investigating genetic disorders, advancing customized therapy, and executing genomic-informed food strategies (Figure 1). It facilitates the identification of genetic determinants that affect illness susceptibility and responses to therapies, including dietary modifications.

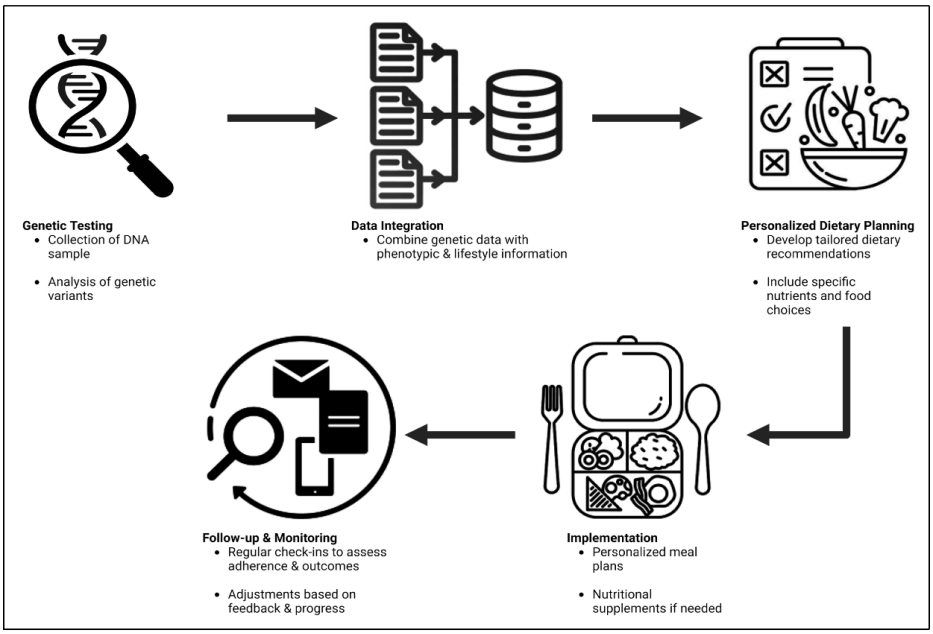

Figure 1. Procedure of a customized dietary regimen.

Genetic variables may profoundly affect nutrition metabolism, with several instances documented in the literature. Polymorphisms in the methylenetetrahydrofolate reductase (MTHFR) gene may influence folate metabolism and are associated with cardiovascular disease and diabetes. Individuals with certain SNPs in this gene may exhibit modified responses to folate consumption and might get advantages from personalized folate supplementation [20-22]. Genetic variation may also alter selenium metabolism, since SNPs change the expression or activity of selenoproteins in response to selenium supplementation [20]. Genetic polymorphisms in the beta-carotene oxygenase 1 (BCMO1) gene, essential for beta-carotene metabolism, may result in fluctuations in plasma carotenoid levels and may have clinical implications, including the onset of liver steatosis independent of dietary vitamin A [23,24]. Additionally, genetic differences in genes involved in lipid metabolism, such as cholesteryl ester transfer protein, lipoprotein lipase, low-density lipoprotein receptor, and apolipoprotein E, may affect the risk of coronary artery disease [25-27]. Tailored dietary suggestions that account for these genetic variations may be advantageous [12]. Moreover, SNPs in genes associated with choline and folate metabolism might influence the dietary need for choline, essential for hepatic function and fetal development [28]. These examples highlight the need of accounting for genetic characteristics in the assessment of food metabolism and the possibility for parenteral nutrition therapies to enhance health outcomes tailored to individual genetic profiles.

Literature case studies demonstrate the influence of genetics on dietary requirements using the framework of nutrigenetics. Individuals with the homozygous mutation (TT) in the MTHFR gene (MTHFRAla222Val, C > T polymorphism) exhibit heightened folate needs owing to modified folate metabolism. These people may need a higher consumption of folate than the recommended dietary allowances (RDAs) to mitigate their vulnerability to disorders linked to folate deficiency [12]. Individuals with Down syndrome, characterized by an extra copy of chromosome 21, have increased requirements for zinc and folate due to the elevated production of cystathionine-β-synthase, a crucial enzyme in folate metabolism [12]. A separate case study investigates the APOA1 polymorphism (G > A). Individuals with the A allele have heightened levels of HDL cholesterol in response to increased consumption of long-chain omega-3 polyunsaturated fatty acids (PUFAs), but those with the GG genotype do not show this benefit [12]. Moreover, individuals with high-risk alleles for serum- and glucocorticoid-inducible kinase 1 (SGK1) may exhibit elevated systolic blood pressure while adhering to a sodium-rich diet [12].

3. Nutrigenomics

Significant research has shown gene-diet connections that affect the likelihood of acquiring illnesses including obesity and type 2 diabetic mellitus (T2DM). Dietary patterns that prioritize whole grains, vegetables, and fruits while restricting total and saturated fats, such as the Mediterranean and DASH diets, have shown the capacity to reduce obesity risk in individuals with elevated genetic predisposition scores, particularly among those possessing risk alleles of FTO rs9939609, rs1121980, and rs1421085 [29]. Nevertheless, the results of additional SNPs in genes such as MC4R, APOA5, and PPARG were ambiguous [29]. In the realm of T2DM, investigations into gene–macronutrient interactions have shown that several genetic variations associated with genes such as TCF7L2, GIPR, CAV2, and PEPD may interact with macronutrients including carbs, lipids, saturated fats, dietary fiber, and glycemic load. Nonetheless, the extensive EPIC-InterAct investigation failed to reproduce these associations [30]. Investigations into gene-diet interactions have progressed into maternal-child health, addressing concerns such as gestational diabetes, pregnancy-induced hypertension, recurrent miscarriages, iron deficiency anemia, and excessive weight gain during gestation. Research findings suggest that understanding gene-diet connections may enhance personalized nutritional interventions for mothers and their children [31-33]. Additional study is required to enhance comprehension of these interactions and to develop personalized nutrition regimens based in genetic profiles [29,30,33].

Technological developments in genetic testing and bioinformatics have markedly improved the identification of genetic biomarkers that indicate disease susceptibility, progression, and therapeutic response. The advancement of molecular point-of-care tests (POCTs) is significant, since microfluidic technology and innovative amplification techniques provide fast genetic testing at the point of care, likely promoting the integration of personalized medical approaches [34]. Advancements in next-generation

sequencing (NGS) technology, including massively parallel sequencing, have substantially reduced prices and expedited the DNA sequencing process. This development facilitates both whole-genome sequencing (WGS) and the targeted sequencing of specific genomic regions. Consequently, NGS has started integration into clinical environments, enhancing diagnosis, prognosis, and therapy selection for several illnesses [35-37]. Artificial intelligence (AI) is being used into clinical laboratory genomics to handle the extensive data produced by these technologies. Artificial intelligence augments genetic research by facilitating the identification of variations in DNA sequences, forecasting the potential impact of these variants on protein structure and function, and correlating genomic data with clinical findings. This assistance enhances geneticists' capacity to transform complex data into actionable information for patient care management [38]. Improvements in long-read sequencing and long-range mapping technologies are augmenting genomic diagnostics by facilitating the detection of a wider array of variations and offering a more comprehensive view of transcriptomes and epigenomes. To fully use the unique characteristics of these technologies and address their intricate error profiles, novel bioinformatics methodologies are essential [39].

Numerous genetic indicators have been recognized in nutritional research as important for comprehending gene–diet interactions. Variants of the fat mass and obesity-related (FTO) gene, such as rs9939609, rs1121980, and rs1421085, are associated with a heightened risk of obesity. These genetic markers have shown associations with dietary patterns characterized by high consumption of whole grains, vegetables, and fruits, and reduced intake of total and saturated fats [29]. Additional genes often examined include MC4R, PPARG, and APOA5; however, the results concerning their interaction with food in relation to overnutrition have been ambiguous [29]. The gene associated with fibroblast growth factor 21 (FGF21) has been linked to macronutrient consumption. A variant at the chromosome 19 locus (rs838145) is linked to heightened carbohydrate consumption and reduced fat intake [39,40]. The FTO variation (rs1421085) is linked to increased protein consumption, irrespective of body mass index [40]. The ISNN underscores the importance of offering dietary recommendations tailored to genetic variations, particularly in genes essential for lipid metabolism, such as cholesteryl ester transfer protein, lipoprotein lipase, low-density lipoprotein receptor, and apolipoprotein E, which may influence susceptibility to coronary artery disease [2]. Moreover, some SNPs have shown interactions with food consumption, affecting obesity and abdominal obesity. The presence of the minor allele (A) of the Ca binding protein 39 (CAB39) rs6722579 gene variation is associated with a heightened risk of abdominal obesity in individuals who surpass the Dietary Reference Intakes (DRIs) for fat intake [41]. In contrast, persons with the minor allele (T) of the carboxypeptidase Q (CPQ) rs59465035 gene often have less vulnerability to abdominal obesity, especially with increased vitamin C intake [41]. These genetic indicators are crucial in progressing the field of PN, enabling dietary advice to be customized according to an individual's genetic composition to enhance health results.

4. Personalized Nutrition Research

The approaches used in nutrigenomics research include many high-throughput omics technologies. Transcriptomics, proteomics, and metabolomics are essential methodologies used to evaluate biological systems' reactions to dietary treatments and to comprehend the interplay between nutrition and genetics [42-45]. Transcriptomics examines genome-wide alterations in gene expression and is the most often used method in nutrigenomics research [45]. Proteomics investigates the whole spectrum of proteins generated by a genome, cell, tissue, or organism; a profile subject to modification by dietary consumption [43]. Metabolomics examines the unique chemical signatures produced by biological processes, concentrating on the investigation of small-molecule metabolite profiles [43,44]. Microarray technology is a potent instrument used in nutrigenomics to assess global gene expression patterns and comprehend the regulation of gene transcription by nutrients or dietary bioactive substances [46]. Furthermore, advancements in bioinformatics are essential for the synthesis and analysis of the extensive data produced by these omics technologies, facilitating the exploration of intricate gene-nutrient connections and the formulation of personalized nutrition plans [43,47]. These approaches jointly enhance the comprehension of how dietary components affect gene regulation, protein expression, and metabolite formation, which are fundamental to the domain of nutrigenomics and the goal of personalized nutrition.

Designing research to assess genetic and nutritional relationships poses several problems. A significant challenge is the intricacy of gene–nutrient interactions, which amplifies the problem's dimensionality, complicating the examination of these interactions at the population level [48]. Moreover, the precise evaluation of dietary intake in population research is intricate owing to the constraints of existing nutritional assessment instruments such as food frequency surveys, 24-hour meal recalls, and dietary diaries. These instruments may lack reliability or sensitivity to reliably measure long-term consumption, which is essential for establishing gene-nutrient relationships [49]. A further problem is the little impact size of prevalent genetic polymorphisms and the intricacy of determining connections between lifestyle variables and the probability of future obesity development. This necessitates an analytical approach that depends on explicitly established prior probabilities to mitigate the danger of inaccurate results [50]. Furthermore, investigations on gene-environment interactions must confront design and analytical problems, including confounding and selection bias, the precision of exposure and genotype measurements, and the assumptions related to biological components [51]. The genetic architecture of nutrition-related disorders is complex, including several genes and interactions that cannot be elucidated by individual polymorphisms. This intricacy necessitates whole-genome study to comprehend gene connections and pathways affecting nutritional metabolism [12]. The use of Mendelian randomization might enhance causal inference in nutrition research; nevertheless, it requires meticulous identification of genetic markers to mitigate biases inherent in observational studies [52].

5. Conclusions

The capacity of PN to transform dietary guidelines is significant and complex. By synthesizing genetic, phenotypic, and environmental data, PN provides a sophisticated methodology for nutrition and health, enhancing results via personalized dietary recommendations based on individual biological profiles. This personalized strategy is particularly relevant in managing chronic conditions like obesity, cardiovascular disease, and diabetes, when universal dietary recommendations have proven inadequate. The results indicate that PN improves the accuracy of dietary treatments and meets the growing customer desire for personalized health solutions.

Nonetheless, the actualization of PN's whole potential requires ongoing cooperative endeavors across all sectors. There is an urgent need for more study to enhance our comprehension of gene-diet interactions and to substantiate the effectiveness of personalized nutrition therapies across varied populations. This study must be supported by advancements in genetics, bioinformatics, and biotechnology, which are continually progressing swiftly. Collaboration among scientists for data and insight sharing, physicians for responsible use in patient care, and lawmakers for the establishment of supporting regulatory frameworks is essential. These collaborative endeavors will guarantee that PN advances scientifically while also being morally and practically applicable on a broad scale.

Upon considering the equilibrium between the advantages and hazards of individualized dietary guidance, it is evident that while the benefits have considerable potential, the concerns must not be disregarded. The ethical, legal, and societal ramifications, especially with data privacy, informed permission, and the risk of worsening health inequities, need stringent management. It is crucial to rigorously assess the clinical significance of genetic testing in nutrition and to guarantee that such interventions do not trivialize the intricacies of human nutrition and health. In conclusion, as we approach a dietary revolution driven by personalized nutrition, it is vital to traverse this burgeoning field with a measured viewpoint, recognizing its transformative potential while diligently confronting the inherent risks. The future of PN will largely depend on our capacity to merge scientific innovation with ethical accountability and inclusion.

References

1. Bush, C.L.; Blumberg, J.B.; El-Sohemy, A.; Minich, D.M.; Ordovás, J.M.; Reed, D.G.; Behm, V.A.Y. Toward the Definition of Personalized Nutrition: A Proposal by the American Nutrition Association. J. Am. Coll. Nutr. 2020, 39, 5–15.

2. Ferguson, L.R.; De Caterina, R.; Görman, U.; Allayee, H.; Kohlmeier, M.; Prasad, C.; Choi, M.S.; Curi, R.; De Luis, D.A.; Gil, Á. Guide and Position of the International Society of Nutrigenetics/Nutrigenomics on Personalised Nutrition: Part 1-Fields of Precision Nutrition. Lifestyle Genom. 2016, 9, 12–27.
3. Gibney, M.J.; Walsh, M.C. The Future Direction of Personalised Nutrition: My Diet, My Phenotype, My Genes. Proc. Nutr. Soc. 2013, 72, 219–225.
4. Harper, A.E. Dietary Guidelines in Perspective. J. Nutr. 1996, 126, 1042S–1048S.
5. Hite, A.H.; Feinman, R.D.; Guzman, G.E.; Satin, M.; Schoenfeld, P.A.; Wood, R.J. In the Face of Contradictory Evidence: Report of the Dietary Guidelines for Americans Committee. Nutrition 2010, 26, 915–924.
6. Byers, T. The Role of Epidemiology in Developing Nutritional Recommendations: Past, Present, and Future. Am. J. Clin. Nutr. 1999, 69, 1304S–1308S.
7. Nestle, M. Animal v. Plant Foods in Human Diets and Health: Is the Historical Record Unequivocal? Proc. Nutr. Soc. 1999, 58, 211–218.
8. Archer, E.; Marlow, M.L.; Lavie, C.J. Controversy and Debate: Memory-Based Methods Paper 1: The Fatal Flaws of Food Frequency Questionnaires and Other Memory-Based Dietary Assessment Methods. J. Clin. Epidemiol. 2018, 104, 113–124.
9. Mullins, V.A.; Bresette, W.; Johnstone, L.; Hallmark, B.; Chilton, F.H. Genomics in Personalized Nutrition: Can You “Eat for Your Genes”? Nutrients 2020, 12, 3118.
10. Stover, P.J. Nutritional Genomics. Physiol. Genom. 2004, 16, 161–165.
11. Sinha, R.; Kachru, D.; Ricchetti, R.R.; Singh-Rambiritch, S.; Muthukumar, K.M.; Singaravel, V.; Irudayanathan, C.; Reddy-Sinha, C.; Junaid, I.; Sharma, G. Leveraging Genomic Associations in Precision Digital Care for Weight Loss: Cohort Study. J. Med. Internet Res. 2021, 23, e25401.
12. Kohlmeier, M.; De Caterina, R.; Ferguson, L.R.; Görman, U.; Allayee, H.; Prasad, C.; Kang, J.X.; Nicoletti, C.F.; Martinez, J.A. Guide and Position of the International Society of Nutrigenetics/Nutrigenomics on Personalized Nutrition: Part 2-Ethics, Challenges and Endeavors of Precision Nutrition. Lifestyle Genom. 2016, 9, 28–46.
13. Murgia, C.; Adamski, M.M. Translation of Nutritional Genomics into Nutrition Practice: The next Step. Nutrients 2017, 9, 366.
14. Trost, B.; Loureiro, L.O.; Scherer, S.W. Discovery of Genomic Variation across a Generation. Hum. Mol. Genet. 2021, 30, R174–R186.
15. 1000 Genomes Project Consortium. An Integrated Map of Genetic Variation from 1,092 Human Genomes. Nature 2012, 491, 56.
16. 1000 Genomes Project Consortium. A Map of Human Genome Variation from Population Scale Sequencing. Nature 2010, 467, 1061.
17. Crawford, D.C.; Akey, D.T.; Nickerson, D.A. The Patterns of Natural Variation in Human Genes. Annu. Rev. Genom. Hum. Genet. 2005, 6, 287–312.
18. Li, J.Z.; Absher, D.M.; Tang, H.; Southwick, A.M.; Casto, A.M.; Ramachandran, S.; Cann, H.M.; Barsh, G.S.; Feldman, M.; Cavalli-Sforza, L.L. Worldwide Human Relationships Inferred from Genome-Wide Patterns of Variation. Science 2008, 319, 1100–1104.
19. Hinds, D.A.; Stuve, L.L.; Nilsen, G.B.; Halperin, E.; Eskin, E.; Ballinger, D.G.; Frazer, K.A.; Cox, D.R. Whole-Genome Patterns of Common DNA Variation in Three Human Populations. Science 2005, 307, 1072–1079.
20. Lietz, G.; Hesketh, J. A Network Approach to Micronutrient Genetics: Interactions with Lipid Metabolism. Curr. Opin. Lipidol. 2009, 20, 112–120.
21. Loktionov, A. Common Gene Polymorphisms and Nutrition: Emerging Links with Pathogenesis of Multifactorial Chronic Diseases. J. Nutr. Biochem. 2003, 14, 426–451.
22. Haggarty, P. B-Vitamins, Genotype and Disease Causality. Proc. Nutr. Soc. 2007, 66, 539–547.
23. Zumaraga, M.P.P.; Arquiza, J.M.R.A.; Concepcion, M.A.; Perlas, L.; Alcudia-Catalma, M.N.; Rodriguez, M. Genotype Effects on β-Carotene Conversion to Vitamin A: Implications on Reducing Vitamin A Deficiency in the Philippines. Food Nutr. Bull. 2022, 43, 25–34.
24. Suzuki, M.; Tomita, M. Genetic Variations of Vitamin A-Absorption and Storage-Related Genes, and Their Potential Contribution to Vitamin A Deficiency Risks among Different Ethnic Groups. Front. Nutr. 2022, 9, 861619.

25. Xu, M.; Zhao, J.; Zhang, Y.; Ma, X.; Dai, Q.; Zhi, H.; Wang, B.; Wang, L. Apolipoprotein E Gene Variants and Risk of Coronary Heart Disease: A Meta-analysis. BioMed Res. Int. 2016, 2016, 3912175.
26. Wilson, P.W.; Schaefer, E.J.; Larson, M.G.; Ordovas, J.M. Apolipoprotein E Alleles and Risk of Coronary Disease: A Meta-Analysis. Arterioscler. Thromb. Vasc. Biol. 1996, 16, 1250–1255.
27. Lumsden, A.L.; Mulugeta, A.; Zhou, A.; Hyppönen, E. Apolipoprotein E (APOE) Genotype-Associated Disease Risks: A Phenome-Wide, Registry-Based, Case-Control Study Utilising the UK Biobank. EBioMedicine 2020, 59, 102954.
28. Zeisel, S.H. Diet-Gene Interactions Underlie Metabolic Individuality and Influence Brain Development: Implications for Clinical Practice Derived from Studies on Choline Metabolism. Ann. Nutr. Metab. 2012, 60, 19–25.
29. Tan, P.Y.; Moore, J.B.; Bai, L.; Tang, G.; Gong, Y.Y. In the Context of the Triple Burden of Malnutrition: A Systematic Review of Gene-Diet Interactions and Nutritional Status. Crit. Rev. Food Sci. Nutr. 2024, 64, 3235–3263.
30. Li, S.X.; Imamura, F.; Ye, Z.; Schulze, M.B.; Zheng, J.; Ardanaz, E.; Arriola, L.; Boeing, H.; Dow, C.; Fagherazzi, G. Interaction between Genes and Macronutrient Intake on the Risk of Developing Type 2 Diabetes: Systematic Review and Findings from European Prospective Investigation into Cancer (EPIC)-InterAct. Am. J. Clin. Nutr. 2017, 106, 263–275.
31. Drabsch, T.; Holzapfel, C. A Scientific Perspective of Personalised Gene-Based Dietary Recommendations for Weight Management. Nutrients 2019, 11, 617.
32. Rozga, M.; Latulippe, M.E.; Steiber, A. Advancements in Personalized Nutrition Technologies: Guiding Principles for Registered Dietitian Nutritionists. J. Acad. Nutr. Diet. 2020, 120, 1074–1085.
33. Favara, G.; Maugeri, A.; Magnano San Lio, R.; Barchitta, M.; Agodi, A. Exploring Gene–Diet Interactions for Mother–Child Health: A Systematic Review of Epidemiological Studies. Nutrients 2024, 16, 994.
34. De Olazarra, A.; Wang, S. Advances in Point-of-Care Genetic Testing for Personalized Medicine Applications. Biomicrofluidics 2023, 17, 031501.
35. Plichta, J.K.; Griffin, M.; Thakuria, J.; Hughes, K.S. What's New in Genetic Testing for Cancer Susceptibility? Oncology 2016, 30, 787.
36. Korf, B.R.; Rehm, H.L. New Approaches to Molecular Diagnosis. JAMA 2013, 309, 1511–1521.
37. Boyd, S.D. Diagnostic Applications of High-Throughput DNA Sequencing. Annu. Rev. Pathol. Mech. Dis. 2013, 8, 381–410.
38. Aradhya, S.; Facio, F.M.; Metz, H.; Manders, T.; Colavin, A.; Kobayashi, Y.; Nykamp, K.; Johnson, B.; Nussbaum, R.L. Applications of Artificial Intelligence in Clinical Laboratory Genomics; Wiley Online Library: Hoboken, NJ, USA, 2023; Volume 193, p. e32057.
39. Sedlazeck, F.J.; Lee, H.; Darby, C.A.; Schatz, M.C. Piercing the Dark Matter: Bioinformatics of Long-Range Sequencing and Mapping. Nat. Rev. Genet. 2018, 19, 329–346.
40. Tanaka, T.; Ngwa, J.S.; Van Rooij, F.J.; Zillikens, M.C.; Wojczynski, M.K.; Frazier-Wood, A.C.; Houston, D.K.; Kanoni, S.; Lemaitre, R.N.; Luan, J. Genome-Wide Meta-Analysis of Observational Studies Shows Common Genetic Variants Associated with Macronutrient Intake. Am. J. Clin. Nutr. 2013, 97, 1395–1402.
41. Kwon, Y.-J.; Park, D.H.; Choi, J.-E.; Lee, D.; Hong, K.-W.; Lee, J.-W. Identification of the Interactions between Specific Genetic Polymorphisms and Nutrient Intake Associated with General and Abdominal Obesity in Middle-Aged Adults. Clin. Nutr. 2022, 41, 543–551.
42. Wittwer, J.; Rubio-Aliaga, I.; Hoeft, B.; Bendik, I.; Weber, P.; Daniel, H. Nutrigenomics in Human Intervention Studies: Current Status, Lessons Learned and Future Perspectives. Mol. Nutr. Food Res. 2011, 55, 341–358.
43. García-Cañas, V.; Simó, C.; León, C.; Cifuentes, A. Advances in Nutrigenomics Research: Novel and Future Analytical Approaches to Investigate the Biological Activity of Natural Compounds and Food Functions. J. Pharm. Biomed. Anal. 2010, 51, 290–304.
44. Kussmann, M.; Krause, L.; Siffert, W. Nutrigenomics: Where Are We with Genetic and Epigenetic Markers for Disposition and Susceptibility? Nutr. Rev. 2010, 68, S38–S47.
45. Afman, L.A.; Müller, M. Human Nutrigenomics of Gene Regulation by Dietary Fatty Acids. Prog. Lipid Res. 2012, 51, 63–70.

46. Masotti, A.; Da Sacco, L.; Bottazzo, G.F.; Alisi, A. Microarray Technology: A Promising Tool in Nutrigenomics. Crit. Rev. Food Sci. Nutr. 2010, 50, 693–698. [Google Scholar] [CrossRef]
47. Zhang, X.; Yap, Y.; Wei, D.; Chen, G.; Chen, F. Novel Omics Technologies in Nutrition Research. Biotechnol. Adv. 2008, 26, 169–176.
48. Cocozza, S. Methodological Aspects of the Assessment of Gene–Nutrient Interactions at the Population Level. Nutr. Metab. Cardiovasc. Dis. 2007, 17, 82–88.
49. Tucker, K.L. Assessment of Usual Dietary Intake in Population Studies of Gene–Diet Interaction. Nutr. Metab. Cardiovasc. Dis. 2007, 17, 74–81.
50. Wareham, N.J.; Young, E.H.; Loos, R.J. Epidemiological Study Designs to Investigate Gene–Behavior Interactions in the Context of Human Obesity. Obesity 2008, 16, S66–S71.
51. Liu, C.; Maity, A.; Lin, X.; Wright, R.O.; Christiani, D.C. Design and Analysis Issues in Gene and Environment Studies. Environ. Health 2012, 11, 93.
52. Pirastu, N.; McDonnell, C.; Grzeszkowiak, E.J.; Mounier, N.; Imamura, F.; Merino, J.; Day, F.R.; Zheng, J.; Taba, N.; Concas, M.P. Using Genetic Variation to Disentangle the Complex Relationship between Food Intake and Health Outcomes. PLoS Genet. 2022, 18, e1010162

التقدم في التغذية الشخصية: دمج الملفات الجينية والتمثيل الغذائي لتوصيات غذائية مخصصة للوقاية من الأمراض المزمنة وإدارتها

المستخلص

الخلفية :التغذية الشخصية (PN) هي مجال ناشئ يعتمد على الملفات الجينية والتمثيل الغذائي الفردية لتخصيص التوصيات الغذائية بهدف تحسين النتائج الصحية. يركز هذا النهج على الانتقال من الإرشادات الغذائية العامة إلى تدخلات محددة تأخذ في الاعتبار التباين الجيني والاستجابات الأيضية.

الطرق :يستعرض هذا البحث التطورات الحديثة في دمج البيانات الجينية، الظاهرية، والكيميائية الحيوية لتوجيه استراتيجيات غذائية مخصصة. كما يسلط الضوء على المنهجيات المستخدمة في علم الجينوم الغذائي، بما في ذلك دراسات الارتباط على نطاق الجينوم وأدوات المعلوماتية الحيوية لتحليل تفاعلات الجين والنظام الغذائي. كما يتم تقييم الأدبيات المتعلقة بتأثير تعدد الأشكال الجينية على استقلاب المغذيات وقابلية التعرض للأمراض.

النتائج :تشير الأدلة إلى أن التباينات الجينية تؤثر بشكل كبير على استجابات الأفراد للأنماط الغذائية، مما ينعكس على إدارة الأمراض المزمنة مثل السمنة وداء السكري من النوع الثاني. تظهر الدراسات أن التدخلات الغذائية المخصصة بناءً على الملف الجيني يمكن أن تعزز فعالية العلاجات الغذائية، مما يؤدي في النهاية إلى تحسين النتائج الصحية. ومع ذلك، لا تزال هناك تحديات تتعلق بقابلية تطبيق هذه الاستراتيجيات الشخصية على نطاق واسع وتنفيذها بشكل أخلاقي في المجتمعات المتنوعة.

الخلاصة :تمتلك التغذية الشخصية إمكانات هائلة لإحداث ثورة في الممارسات الغذائية. من خلال دمج الرؤى الجينية في التوجيه الغذائي، يمكن لمقدمي الرعاية الصحية تقديم تدخلات أكثر فعالية وملاءمة للأفراد. يتطلب الأمر إجراء المزيد من الأبحاث للتحقق من صحة هذه الأساليب ومعالجة الاعتبارات الأخلاقية، مما يضمن أن تكون التغذية الشخصية قوية علميًا ومتاحة على نطاق واسع.

الكلمات المفتاحية :التغذية الشخصية، علم الجينوم الغذائي، تعدد الأشكال الجينية، التدخلات الغذائية، إدارة الأمراض المزمنة.