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The Role of Personalized Nutrition in Preventing Chronic Diseases: A Comprehensive Review

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Abstract

Background: The link between nutrition and reproductive health is increasingly recognized, particularly in the context of chronic diseases. Personalized nutrition, informed by genetic and epigenetic factors, may offer targeted interventions for improving reproductive outcomes.

Methods: This comprehensive review synthesizes contemporary literature on nutrigenetics, nutrigenomics, and epigenetics as they relate to human reproductive health. Studies were selected based on their focus on dietary impacts on fertility and the genetic variability influencing nutrient metabolism.

Results: Evidence indicates that specific dietary patterns, such as the Mediterranean diet, positively correlate with improved semen quality and fertility treatment outcomes. Genetic variants, particularly single nucleotide polymorphisms (SNPs), significantly affect individual responses to dietary components. For instance, variations in genes such as FTO and TCF7L2 modulate obesity risk and metabolic responses to nutrition. Additionally, maternal and paternal nutritional experiences can lead to epigenetic modifications that influence offspring health, highlighting the potential for transgenerational effects.

Conclusion: Personalized nutrition strategies that consider genetic predispositions may enhance reproductive health and reduce chronic disease risk. Further research is needed to develop nutrigenetic programs that tailor dietary recommendations based on individual genetic profiles, ultimately fostering improved health outcomes for future generations.

Keywords: Personalized nutrition, reproductive health, nutrigenetics, epigenetics, chronic diseases.

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1. Introduction

Recent years have seen an increased focus on the influence of diet on human reproduction, primarily due to infertility being a significant health issue in Western nations and emerging studies supporting the "developmental origins of health and disease (DOHaD)" hypothesis, which emphasizes the effects of prenatal and preconception environmental exposures on the long-term health of offspring [1,2]. Despite extensive media coverage on optimal nutritional strategies for enhancing human health and reproductive fitness, the prevailing approach remains rooted in a "one size fits all" model, neglecting the genetically influenced interindividual variability in food metabolism. This review summarizes contemporary literature and the newest research results in nutrigenetics, nutrigenomics, and epigenetics.

2. Nutrition and Human Reproductive Health

Numerous studies have shown the correlation between diet and male fertility, detailing the impact of several foods on semen quality [3]. Specifically, it has been proposed that fruits and vegetables, vitamins A, C, and E, folates, organ meats, and fish positively influence semen quality. Conversely, trans fats, total fats, processed meats, dairy products, and soy phytoestrogens are regarded as detrimental to spermatogenesis [3]. Additional dietary practices, including caffeine and tea consumption, have been proposed to affect male reproductive health regarding conception, pregnancy, and miscarriage rates [4].

Research indicates a correlation between diet and reproductive fitness, highlighting the beneficial impact of the Mediterranean diet on the semen quality of male partners in couples undergoing fertility treatment, as well as on in vitro fertilization success rates in non-obese women [5,6]. Given the contemporary significance of reproductive concerns, it has been proposed that the impact of food on fertility may serve as a focused subject for public health nutrition initiatives, particularly since the relationship between diet and other risk factors manifests as a detrimental cycle [7]. Dietary risk factors, including elevated consumption of saturated fat and sugar, are significantly associated with obesity, which subsequently poses a risk for infertility in both males and females. Obesity may be associated with psychological risk factors, including depression, anxiety, and stress, which are shown to diminish fertility. Moreover, sociodemographic risk factors may contribute to an unhealthy diet. In this perspective, nutrition seems to be a pivotal element in the array of environmental influences influencing human health and fertility.

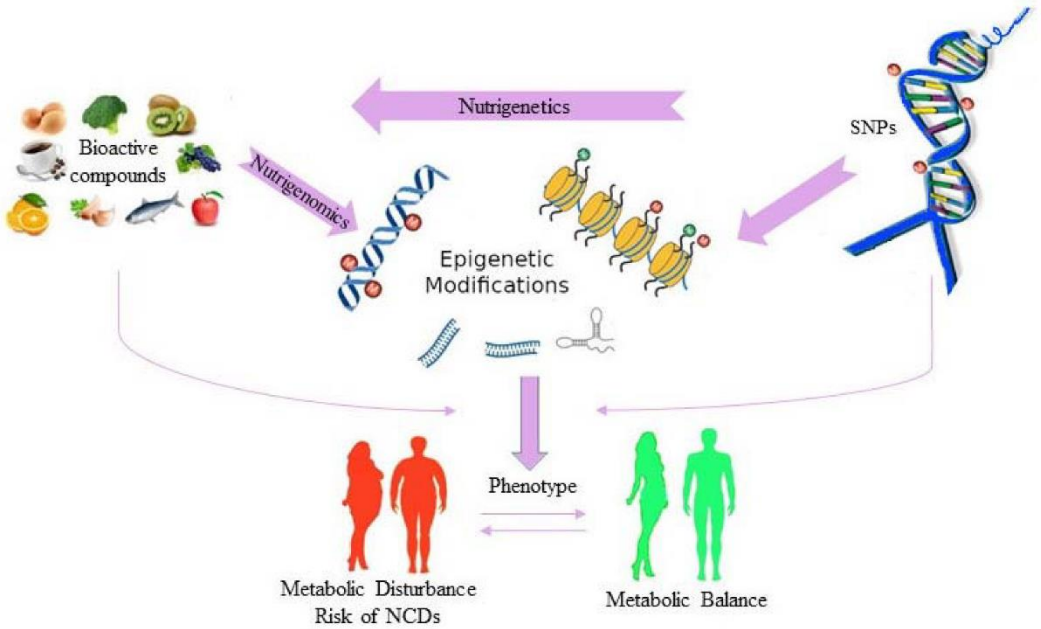
Notwithstanding the substantial evidence indicating the significant impact of nutrition on a couple's reproductive fitness, differences are evident across various research. Gaskins and Chavarro [8] investigated inconsistencies in the claimed effects of several diets, revealing that the impact of some components on human fertility, including vitamin D, antioxidants, long-chain omega-3 fatty acids, and dairy products, remains ambiguous. Recent investigations on folic acid and zinc supplementation in male partners revealed no enhancement in semen quality or live birth rates in couples receiving infertility therapy [9]. A potential reason for these disparities is that individuals are not genetically similar, and the existence of certain genetic variants might affect metabolism; hence, the impact of nutrition may vary. A particular vitamin may provide advantages to some people while being ineffective for others, depending on their distinct genotypes. This necessitates the development of individualized dietary regimens tailored to the distinct genetic profiles of individuals, referred to as nutrigenetic programs [10].

3. Nutrigenetics and Human Well-being

Nutrigenetics is a branch of nutritional genomics that examines (i) the impact of specific genetic variants, particularly single nucleotide polymorphisms (SNPs), on the response to dietary components, and (ii) the consequences of this interaction, including its effects on health and susceptibility to nutrition-related diseases [11,12] (Figure 1). The main objective of nutrigenetics is to design effective, individualized

dietary strategies that promote weight reduction and avoid metabolic disorders, including Type 2 diabetes (T2DM), hypertension, dyslipidaemias, and cardiovascular disease (CVD).

Figure 1. Relationships between genes, food, and human health.



Numerous genes in our genome are recognized to affect food metabolism [13]. The primary genes associated with body weight reduction in adults due to hypocaloric diets and/or physical activity programs are implicated in lipid metabolism and adipogenesis; additional genes pertain to carbohydrate metabolism, energy intake and expenditure, and the circadian system [13]. It has been shown that variations in genes associated with taste, olfaction, and texture might change food perception and preferences, hence influencing susceptibility to nutrition-related diseases [14].

Clinically significant nutrigenetic interactions are well-documented, including (i) the relationship between saturated fat intake, the APOA2 2265T > C variant, and BMI; (ii) coffee consumption, gene variants primarily related to adrenergic receptors, and hypertensive response; and (iii) folic acid supplementation, MTHFR gene variants, homocysteine levels, and cardiovascular disease risk.

Single nucleotide polymorphisms (SNPs) in the FTO gene significantly influence body weight and composition among genetic variations associated with diet [15, 16]. Individuals with the FTO rs9939609 AA genotype are predisposed to greater obesity compared to non-carriers of the A-risk allele [15-17]. This variation is regarded as one of the most significant risk factors for polygenic obesity. However, it has been shown that the heightened vulnerability to obesity caused by the A risk gene may be altered with either physical activity or a decrease in caloric consumption. This illustrates how genetic predisposition to certain non-communicable diseases may be influenced by beneficial lifestyle modifications [18]. Separate research identified a gene-diet interaction involving the Mediterranean Diet (MedDiet) for both the FTO rs9939609 and MC4R rs17782313, revealing an increased risk of Type 2 Diabetes Mellitus (T2DM) among carriers of the variant alleles relative to wild-type individuals when adherence to the MedDiet was lacking. These relationships vanish with significant devotion to the Mediterranean Diet [19]. Numerous studies have shown the associations between TCF7L2 rs7903146 (C > T) and dietary factors in influencing T2DM risk [20-23]. Wholegrain consumption was negatively correlated with the incidence of T2DM in CC carriers; however, this protective impact was diminished by the presence of the T-allele. These examples illustrate the intricacy of nutrigenetics regarding impacts, including the varying genetic predispositions among populations and the environmental variables that might affect the gene-nutrient relationship.

Consequently, people with distinct genotypes metabolize fats, carbohydrates, and folates variably and exhibit unique responses to the same diets. Nutrigenetic tests are now used in some situations to determine

suitable diets for people predisposed to various illnesses. Our research established an elevated risk of gestational diabetes mellitus (GDM) in women with the TT genotype of the TCF7L2 gene, using a straightforward panel of nine nutrigenetic variations (OR 2.5) [24,25]. Additionally, a correlation has been identified between polymorphisms in the PPARG2, APOA5, MC4R, LDLR, and FTO genes and lipid markers [24-26]. The diagnosis of gestational diabetes mellitus (GDM) facilitates the identification of a population more susceptible to type 2 diabetes mellitus (T2DM) and metabolic syndrome, serving as a convenient and optimal technique that aligns with regular anthropometric and biochemical parameters, dietary evaluations, and genetic predispositions in clinical practice [25,26]. The incorporation of precision nutrition into standard clinical practice is an escalating challenge, and this proven strategy might enhance tailored nutrient consumption as well as the early detection and classification of women at heightened risk. This exemplifies a particular circumstance where daily food consumption and its impact on body size and clinical parameters may be readily evaluated. Furthermore, compliance with a particular diet among women impacted by a disease that may harm fetal health is anticipated to be significantly elevated. Conversely, accurate information on food habits is more challenging to get when engaging with the general community and couples attempting to conceive.

Research indicates that people may exhibit inaccuracies and modify their food habits when requested to disclose their dietary consumption [27,28]. Our observations indicate that Italian guys experiencing reproductive issues often claim near-complete adherence to a Mediterranean diet, despite their BMI and clinical indicators indicating harmful dietary habits [unpublished data]. This signifies a genuine deficiency in any dietary plan aimed at minimizing the consumption of harmful foods, since patients often lack complete awareness of their dietary practices.

Over the last decade, more than 2 million direct-to-consumer tests have been offered by various firms. These tests may be procured online without a medical prescription and are often marketed as “lifestyle” genetic testing, circumventing the more rigorous regulations governing clinical and medical devices/services [29,30]. These techniques include ethical and legal considerations, including the intricacies of data interpretation, uncertainties over the therapeutic relevance of outcomes, and the management of information. Determining the target population and the degree to which a nutrigenetic test is deemed therapeutically beneficial might be contentious. Moreover, the ambiguity around the interpretation and implications of their prospective outcomes for consumers and patients has been critically scrutinized, as such assessments may foster unreasonable expectations or induce a false feeling of security or excessive worry [30,31]. Despite the continuous nature of this discussion, we emphasize that both pre-test and post-test genetic counseling must be offered for nutrigenetic panels. Numerous factors must be considered before and after nutrigenetic testing, including (i) the characteristics of the test and its outcomes, (ii) the test's usefulness, (iii) the interpretation and implications of the findings, and (iv) associated hazards [30].

Moreover, a significant subject in this domain is the new finding that a certain genetic variation may affect the metabolism of certain foods, while diet itself can alter gene expression. This prompts a discussion on a significant topic in this domain: the epigenetic effects of nutrition on human reproduction.

4. Epigenetics, Nutrition, and Human Health

Epigenetics examines the biological mechanisms that regulate gene expression without altering the DNA sequence, including DNA methylation, histone modification, and microRNA (miRNA) control [32]. Currently, nutritional epigenetics, which examines alterations in gene expression caused by bioactive food substances, is developing as a unique area of research exploring the influence of nutrition on health [33] (Figure 1).

Nutrition is a highly adjustable element that may influence DNA methylation processes. Research has shown that nutrition can affect the epigenetic regulation of DNA methylation through various mechanisms, including modifying the substrates and cofactors required for this process, altering the activity of enzymes involved in the one-carbon cycle, or influencing DNA demethylation activity [34].

The primary impact of diet on the epigenetic modulation of gene expression is exemplified by early life nutritional experiences, which can induce lasting metabolic and physiological alterations via modified epigenetic profiles, resulting in varying susceptibility to chronic diseases in later life [35]. In this perspective, prenatal exposure to many factors is crucial. Maternal malnutrition and/or over-nutrition throughout the prenatal and postnatal periods are primary stressors that might affect child outcomes and adult phenotypic repercussions by increasing the risk of metabolic disorders [2,36].

A consistent epigenetic association exists between an unbalanced maternal diet during pregnancy, fetal growth, and cardiovascular disease risk in adulthood. Specifically, DNA methylation levels at genes that regulate cortisol levels, tissue glucocorticoid action, and blood pressure have been linked to both early-life factors and cardiometabolic risk factors [37]. Substantial seasonal fluctuations in maternal consumption of methyl-donor nutrients throughout the periconceptional period affect many maternal plasma indicators that forecast alterations in methylation at metastable epialleles in lymphocytes and hair follicles in offspring postnatally [38]. Moreover, maternal obesity may induce alterations in DNA methylation that are evident at birth and persist beyond birth. The investigation of maternal obesity, irrespective of gestational diabetes mellitus (GDM), has shown several differentially methylated sites in the DNA of umbilical cord blood from kids, as well as in samples from children aged 4–5 years and 9–16 years [39,40].

Maternal nutrition significantly influences the reprogramming of the epigenome in the early embryo, impacting offspring phenotype by modifying oocyte maturation, provisioning, and the reserves of mitochondria and metabolites inside oocytes. The cytoplasmic contents specifically react to maternal nutrition: dietary fat enhances lipid droplet size and composition, micronutrients impact DNA methylation, and variations in dietary lipids and carbohydrates influence mitochondrial function [41]. Caloric restriction or an omega-3-enriched diet may restore oocyte quality, mitochondrial function, and fertility in animal models, indicating that these changes may be amenable to dietary or focused therapeutic therapies [42,43]. Furthermore, maternal supplementation with methyl donors has been shown to counteract DNA hypomethylation caused by endocrine-disrupting drugs during early development [44]. Recent studies have shown that fathers pass epigenetic alterations to their kids, therefore affecting both embryonic development and lifelong health [44,45].

Epigenetic modifications may influence male germ cell development at various stages. Environmental or lifestyle factors, including pollutants, endocrine disruptors, smoking, and obesity, may influence sperm during their growth in the testes or maturation in the epididymis [46,47]. Research indicates that sperm from obese, glucose-intolerant guys have unique short noncoding RNA (sncRNA) expression and DNA methylation patterns in contrast to lean, glucose-tolerant individuals. Changes in DNA methylation influence differential methylation clusters within genes associated with SNPs linked to obesity, including FTO, MC4R, and others. Furthermore, alterations in the sperm DNA methylation pattern linked to obesity have been noted in a distinct sample of males after bariatric surgery. This particular remodeling included gene regulators associated with appetite regulation (e.g., MC4R, BDNF, NPY, CR1) and metabolism (e.g., FTO, CHST8, SH2B1) [48]. The findings were validated by Soubry et al. [49], who established that male overweight and obesity are reflected in the sperm epigenome. These authors evidenced reduced methylation levels at the MEG3, NDN, SNRPN, and SGCE/PEG10 differentially methylated regions (DMRs), and a marginal increase in DNA methylation at the MEG3-IG DMR and H19 DMR in the sperm of overweight or obese individuals.

The aforementioned investigations do not explain whether changed sperm methylation results from obesity itself or the patients' eating choices; yet, together, these discoveries indicate a correlation between nutrition and sperm epigenetic patterns. Consequently, sperm epigenetic alterations may be inherited by children, potentially resulting in paternal epigenetic transmission of metabolic disorders (paternal origins of health and diseases (POHaD)) [50]. It has been suggested that a nutrition-related mechanism transmitted through the paternal lineage can affect longevity and the risk of mortality from cardiovascular diseases and diabetes mellitus when either the father or paternal grandfather experiences food excess between the ages of 9 and 12 years. Consequently, alterations in the epigenetic landscape induced by dietary compounds can influence overall health as well as the reproductive health of both

genders, along with the stressors experienced by both parents before conception, thereby shaping the developmental and life-course trajectory of the embryo and fetus.

Consequently, the "epigenetic diet" may serve as an effective strategy to mitigate epigenomic anomalies induced by environmental pollutants. Growing data indicates that bioactive dietary substances, including isothiocyanates in broccoli, genistein in soybean, epigallocatechin-3-gallate in green tea, resveratrol in grapes, and ascorbic acid in fruits, provide advantageous health consequences via altering the epigenome.

5. Alternative Perspectives

While existing data increasingly supports the relationship between parental food, genetics, and child health, other studies conversely highlight the shortcomings of the gene-centric paradigm (i.e., DNA and epigenetics) in therapeutically relevant research [51]. Bonduriansky et al. [52] proposed that non-genetic inheritance, including epigenetic, environmental, behavioral, and cultural factors, may significantly influence evolution by imposing transgenerational effects and producing heritable variations across a wide range of traits in all organisms, following an analysis of the gene-centrism perspective and a reassessment of the fundamentals of evolutionary theory.

The maternal resources hypothesis presents a new framework for understanding inheritance and evolution, positing that non-genetic factors, such as cumulative maternal effects (including maternal prenatal energy metabolism and maternal postnatal physical activity), socio-environmental influences, and phenotypic evolution, are the primary determinants of the health of subsequent generations [53]. In this evolutionary framework, the "overconsumption" resulting in metabolic disease is attributed not to dietary factors alone, but to physical inactivity stemming from increased energy intake and non-genetic evolutionary mechanisms related to the adipogenic partitioning of nutrient-energy.

This theory posits that although nutrition is a fundamental aspect of health, it may represent a minor risk factor for chronic illnesses [54,55], indicating that macronutrients might exert metabolic effects contingent upon the individual's physiological setting (e.g., physical activity level) [54]. The influence of nutrition on chronic illnesses has been a subject of much debate [55]. The dispute was initiated by the release of epidemiological publications endorsing memory-based approaches for assessing food consumption, despite the potential for these methods to provide faulty dietary data, leading to erroneous connections and consequences. Given these conflicting perspectives, more interventional and longitudinal studies using a rigorous methodology are necessary to investigate the potential involvement of epigenetic pathways and food as mediators of impacts on future generations.

6. Conclusions

There are significant anticipations for nutrigenetics, particularly with research on its prospective advantageous use in health enhancement and tailored nutrition for the avoidance of chronic diseases. While research aimed at elucidating the interplay between nutrition, genetics, and human health requires more investigation, the discovery of effective therapies may mitigate inherent genetic risks, hence decreasing vulnerability to lifestyle-related disorders. The concepts of nutrigenetics and nutrigenomics should be seen not as separate systems linking diet and gene expression, but as two interrelated patterns.

Recent suggestions indicate that lifestyle alterations, such as individualized dietary and physical activity interventions, may influence obesity by altering the expression levels of the FTO and IRX3 genes. This effect is influenced by the FTO genotype, which may alter the effect of lifestyle modifications on its expression. Conversely, it is essential to consider nutrigenomic research that examines the impact of diet on the health of people and their progeny.

Dietary patterns, nutrients, and bioactive chemicals influence metabolic characteristics via epigenetic pathways, making them promising therapeutic targets. Recent studies have proposed that bioactive food components may have a protective function in counteracting epigenetic abnormalities caused by various environmental variables [56]. Nutrigenetics, nutrigenomics, and epigenetic diets are

increasingly being investigated for their potential adoption as creative and effective strategies to safeguard human health, particularly for future generations.

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دور التغذية الشخصية في الوقاية من الأمراض المزمنة: مراجعة شاملة

الملخص

الخلفية :يتم التعرف بشكل متزايد على الرابط بين التغذية والصحة الإنجابية، خاصة في سياق الأمراض المزمنة. قد توفر التغذية الشخصية، المستندة إلى العوامل الجينية والوراثية اللاجينية(epigenetics) ، تدخلات مستهدفة لتحسين النتائج الإنجابية.

الطرق :تقوم هذه المراجعة الشاملة بتجميع الأدبيات المعاصرة حول علم الوراثة التغذوي(nutrigenetics) ، وعلم الجينوم التغذوي (nutrigenomics)، وعلم الوراثة اللاجينية فيما يتعلق بصحة الإنسان الإنجابية. تم اختيار الدراسات بناءً على تركيزها على تأثير التغذية على الخصوبة والتنوع الجيني الذي يؤثر على استقلاب العناصر الغذائية.

النتائج :تشير الأدلة إلى أن أنماط التغذية المحددة، مثل حمية البحر الأبيض المتوسط، ترتبط بشكل إيجابي بتحسين جودة الحيوانات المنوية ونتائج علاجات الخصوبة. تؤثر المتغيرات الجينية، لا سيما تعدد أشكال النوكليوتيدات المفردة(SNPs) ، بشكل كبير على استجابات الأفراد لمكونات التغذية. على سبيل المثال، تؤثر التباينات في جينات مثل **FTO** و**TCF7L2** على خطر السمنة والاستجابات الأيضية للتغذية. بالإضافة إلى ذلك، يمكن أن تؤدي التجارب التغذوية للأمهات والآباء إلى تعديلات وراثية لاجينية تؤثر على صحة الأبناء، مما يبرز إمكانية وجود تأثيرات عبر الأجيال.

الخلاصة :قد تعزز استراتيجيات التغذية الشخصية التي تأخذ في الاعتبار الاستعدادات الجينية الصحة الإنجابية وتقلل من خطر الأمراض المزمنة. هناك حاجة إلى مزيد من الأبحاث لتطوير برامج وراثية تغذوية تهدف إلى تقديم توصيات غذائية مخصصة بناءً على ملفات جينية فردية، مما يعزز في النهاية تحسين النتائج الصحية للأجيال القادمة.

الكلمات المفتاحية :التغذية الشخصية، الصحة الإنجابية، علم الوراثة التغذوي، علم الوراثة اللاجينية، الأمراض المزمنة.