

Effects of Nutrigenetic Interventions on Glycemic Outcomes (HbA1c, Fasting Glucose, Insulin Resistance)

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ABSTRACT

Background: Type 2 diabetes mellitus (T2DM) and prediabetes are serious global health problems with a variety of patient responses to the current traditional dietary approaches. The rapidly expanding discipline of precision nutrition focuses on how each person reacts to food based on their genetic composition. In particular, FPG and HbA1c blood markers, and insulin sensitivity traits based on dietary carbohydrate, fat, and energy contents are strongly associated with single nucleotide polymorphisms (SNPs). The genes associated with fat mass and obesity (FTO), insulin receptor substrate 1 (IRS1) and transcription factor 7-like 2 (TCF7L2).

Objective: In order to gather and compile the clinical and translational data about the impact of nutrigenetic variations linked to reactions to dietary treatments aimed at insulin resistance, FPG, and HbA1c levels, we carried out a systematic review.

Methods: We searched for PubMed, EMBASE, Medline, and Cochrane databases for relevant studies reporting the effects of meal tests, prospective cohorts, randomized controlled trials (RCTs), and systematic reviews. The dietary interventions included standardized medical nutrition therapy (MNT), low-calorie, reduced-carbohydrate, carbohydrate/fat-modified, and multi-marker tailored dietary approaches.

Results: The reviewed clinical studies demonstrate that individual responses to dietary treatments are determined by genetic variants. For instance, TT carriers of TCF7L2 (rs7903146) exhibit attenuated glycaemic response to standard MNT resulting in smaller reductions in HbA1c levels (-0.6% vs -1.2% , $P < 0.001$) and FPG changes compared to wild-type counterparts (-14.3 mg/dL vs -28.5 mg/dL; $p < 0.001$). Patients with TCF7L2 risk alleles experience impaired insulin sensitivity and dysregulated glucose homeostasis on high-fat diets whereas non-risk carriers demonstrate improved HOMA-IR during a 12-month period of weight loss maintenance following a very low-calorie diet. By contrast, TCF7L2 variants interact with whole-grain and fibre-rich diets to improve glucose tolerance and reduce the risk of developing T2DM.

Furthermore, compared to non-risk carriers, IRS1 (rs2943641) CC homozygotes exhibit larger drops in weight and HOMA-IR on high-carb, low-fat diets. However, the same genetic variant is associated with higher risks of impaired insulin action when placed on low-carbohydrate diets, thus highlighting the clinical relevance of optimized carbohydrate intake for better metabolic health. The impact of dietary lipid content on variations in insulin sensitivity was found to depend on FTO (rs1121980/rs9939609) variants with partial meal replacement hypocaloric diets eliciting significant decreases in HOMA-IR across genotypes.

Finally, systematic reviews of multi-omic individualized dietary interventions involving microbiota, genotype, and continuous glucose monitoring (CGM) suggest better clinical outcomes using tailored approaches compared to standard RCTs. Specifically, an analysis of individual patient data (IPD)-RCTs of precision nutrition revealed beneficial effects on postprandial glucose regulation with a median reduction in HbA1c of -0.925% . Compared to standardized low-fat and Mediterranean diets, AI-driven Digital Twin and CGM-based individualized postprandial glucose management resulted in significant reductions in HbA1c, FPG, and HOMA-IR levels.

Conclusion: Individual responses to macronutrient distribution are substantially determined by genetic variants, particularly in TCF7L2, IRS1, and FTO loci. As such, a more effective approach to improving HbA1c, reducing FPG, and managing insulin resistance in patients with T2DM and prediabetes involves the use of personalized medical nutrition therapy based on individual genotype and employing multi-marker tailored dietary interventions.

KEYWORDS: Personalized Nutrition, Nutrigenetics, Type 2 diabetes, HbA1c, insulin resistance, TCF7L2, IRS1.

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INTRODUCTION

Type 2 diabetes mellitus (T2DM) and its pre-diabetic stage have become a worldwide health challenge, with a large clinical and economic burden on health care systems.¹ An estimated 537 million people have diabetes in 2021. By 2045, this is anticipated to increase to 783 million.² In the

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past, conventional dietary guidelines for obesity and T2DM have focused on population-wide averages, emphasizing general caloric restriction, reduction of saturated fats and added sugars, and increased dietary fiber intake.³ However, clinical experience points to substantial phenotypic heterogeneity, with individuals with similar BMIs and lifestyle habits frequently showing very different metabolic risks and responses to therapy with these 'one-size-fits-all' standard medical nutrition therapies (MNT).

This inter-individual variability is driven by complex interactions between genetic variants, gut microbiota dynamics, lipid metabolism, and environmental exposures, which are grouped together as "diabesity". Precision nutrition is a revolutionary paradigm that has emerged to tackle this heterogeneity. Using multi-layer omics data (genomics, lipidomics, metabolomics, and microbiomics) for customized dietary treatments to optimize weight, glycemic control, and overall cardiometabolic risk is the next step in the evolution of precision nutrition.³ One of the most basic of the concepts in precision nutrition is nutrigenetics, the science of understanding the role of a single nucleotide polymorphism (SNP) in the human genome that affects the body's response to certain food components.^{3,4} More than 140 genetic loci have been associated with predisposition to T2DM and to individual postprandial responses to meals by genome-wide association studies (GWAS). Variations in candidate genes, especially Transcription factor-7-like 2 (TCF7L2), Insulin receptor substrate 1 (IRS1), and Fat mass and obesity-associated (FTO), have been linked to metabolic pathways related to insulin secretion, beta-cell function, macronutrient oxidation and hypothalamic control of food intake^{4,6}. Furthermore, nutrigenomics and epigenetics are examining the direct effects that bioactive dietary compounds (phenolics, omega-3 fatty acids and one-carbon nutrients like folate) have on gene expression.^{4, 5} DNA methylation, and chromatin states to reverse negative epigenetic marks associated with insulin resistance and high-fat diets.^{2,3} With mounting evidence, the transition from general dietary recommendations to genotype-informed and microbiome-guided medical nutrition therapy is increasingly recognized as an essential strategy to enhance therapeutic efficacy in diabetes management.^{3,5,6}

OBJECTIVE

The goal of this review is to thoroughly summarize the available clinical and translational data about the impact of nutrigenetic therapies on the three primary glycemic outcomes: insulin resistance (measured by HOMA-IR), fasting plasma glucose (FPG), and glycated hemoglobin (HbA1c). By combining data from recent randomized controlled trials (RCTs), prospective cohort studies, systematic reviews, and multi-omics research, this article seeks to elucidate the role of particular gene-diet interactions (such as TCF7L2, IRS1, FTO, PPARG, etc.) and microbiome dynamics in

modulating the efficacy of dietary treatments in adults with T2DM, prediabetes, and obesity. Additionally, employing cutting-edge technologies like Digital Twin platforms and Continuous Glucose Monitoring (CGM) algorithms, this paper addresses the translational application of such discoveries.

METHODS

A literature search was performed on the precision nutrition approach to the management of type 2 diabetes and prediabetes utilizing online databases such as PubMed, EMBASE, Medline, Web of Science and Cochrane Library^{7,8}, examining the effectiveness of precision nutrition based on individual genetic, metabolic, and microbiome factors. The following free-text terms and Medical Subject Headings (MeSH) were used as search terms: "Precision Nutrition," "Personalized Nutrition," "Nutrigenomics," "Type 2 Diabetes Mellitus," "Glycemic Control," "HbA1c," "Genetic Variants," "Metabolomics," and "Gut Microbiota".⁹

Human studies involving adults (>18 years old) with T2DM, prediabetes, or obesity who received a customized diet intervention met the inclusion criteria.^{8,9}

Randomized controlled trials (RCTs), open-label clinical trials, n-of-1 cross-over superiority trials, prospective cohorts, meal test studies, and systematic reviews with meta-analyses were among the study designs that qualified.^{5,7,8} Interventions that tracked changes in HbA1c, FPG, postprandial glucose response (PPGR), and the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) were explicitly identified by the study.^{8,10} Patient demographics, particular SNP variants (e.g., rs7903146, rs2943641), dietary intervention types (e.g., low-fat, low-carbohydrate, Mediterranean, high-fiber), follow-up duration (ranging from acute meal tests to 2-year interventions), and the resulting magnitude of glycemic change were all methodically assessed during data extraction.^{4,7}

RESULTS

The reviewed literature provides compelling evidence that individual genetic and microbiome profiles are important determinants of the success of dietary interventions for glycemic control. Results are grouped according to the major candidate genes and the use of multi-omics technologies.

TCF7L2 Gene-Diet Interaction and Glycemic Outcomes

Hepatic glucose production, insulin secretion kinetics, and beta-cell maturation are all significantly impacted by transcription factor 7-like 2 (TCF7L2), which is thought to be the most potent gene for T2DM susceptibility to date.^{4,7} The most well studied variant rs7903146 shows a dramatic interaction with dietary macronutrients, particularly fat and carbohydrates.⁴

A prospective cohort study was conducted on 112 Saudi Arabian adults with T2DM, stratified by TCF7L2 rs7903146

genotypes (homozygous risk allele TT, heterozygous CT, and wild-type CC), and prescribed a standardized carbohydrate-moderate Medical Nutrition Therapy (MNT) for 6 months. All groups exhibited high dietary adherence and weight loss, but degrees of glycemic improvement varied dramatically by genotype. The wild-type (CC) group had the greatest reductions in HbA1c (−1.2%) and fasting plasma glucose (−28.5 mg/dL). Conversely, homozygous risk allele (TT) carriers exhibited significantly blunted responses with HbA1c of only −0.6% and FPG of −14.3 mg/dL ($p < 0.001$). Additionally, TT carriers showed a significantly less improvement in insulin resistance (HOMA-IR reduction −1.0 vs. −1.9 in wild-type). Multivariable regression confirmed that the TCF7L2 genotype is an independent predictor of glycemic change after adjusting for baseline covariates⁴.

Numerous systematic reviews have verified these conclusions. TCF7L2 alters the relationship between food (particularly fatty acids and fiber) and insulin resistance, according to an analysis of 38 research (meal tests and RCTs). In meal tolerance tests, risk-allele carriers with healthy diets high in total dietary fat had worse insulin sensitivity and higher glucose concentrations than non-risk carriers. Furthermore, long-term weight loss RCTs (>1-year) also show that non-risk allele subjects with overweight or obesity have significant decreases in serum glucose and insulin levels, while risk carriers have impaired responses to dietary fat⁷.

In contrast, some dietary components may reduce the risk of TCF7L2. Whole grain and dietary fiber intake have been associated with protective interactions. Increasing fiber intake and whole-grain consumption significantly improved glucose tolerance and decreased T2DM incidence in Swedish and German cohort studies, in particular counteracting the genetic risk conferred by the TCF7L2 variants^{6,7,11}. Moreover, beneficial effects of low total energy intake (TEI) have been reported on the insulin levels of the carriers of the TT risk allele¹².

IRS1 polymorphisms and Carbohydrate/Fat Modification

The Insulin Receptor Substrate 1 (IRS1) gene is a key component in the insulin signaling pathway.⁶ The variant IRS1, which is closely linked to the gene associated with insulin resistance and hyperinsulinemia, has been well correlated with the former condition. The gene–diet interaction of this locus has been thoroughly evaluated in a 2-year randomized weight-loss study of overweight adults called the Preventing Overweight Using Novel Dietary Strategies (POUNDS LOST) study.¹³

The effect of the IRS1 rs2943641 mutation on long-term changes in insulin resistance in various macronutrient diets was examined in this trial¹³. The results showed a strong gene-by-diet interaction, with individuals homozygous for CC exhibiting considerably higher reductions in fasting

insulin and insulin resistance (HOMA-IR) when placed on a diet heavy in carbohydrates (65%) and low in fat (20%) than those with the T-allele. In contrast to T-allele carriers, CC genotype patients on the high-carb diet sustained improvements in metabolic indicators over the course of the two-year intervention¹³. On the other hand, other research has shown that the T-allele of rs2943641 is linked to a lower risk of T2DM in populations with low carbohydrate intake. This suggests that optimizing dietary fat and carbohydrate ratios is crucial for reducing genetic risks related to impairments in insulin signaling^{6,9}.

FTO Variants and Low-Calorie Diets

Energy intake, appetite control, and obesity risk are all highly correlated with the fat mass and obesity associated (FTO) gene^{3,6}. Specific variants, including rs9939609 and rs1121980, have been shown to interact significantly with dietary intervention to affect weight loss and glycemic parameters.

In a 12-week clinical study of Caucasian obese subjects on a partial meal-replacement hypocaloric diet, the metabolic responses were modulated by the FTO variant¹⁰. The study found significant associations between the FTO genotype and dietary fat intake in modifying changes in insulin sensitivity. However, both sets of genotypes (wild-type versus mutant allele carriers) achieved weight loss⁶. FTO risk allele carriers consuming high saturated fat diets exhibit a greater risk for insulin resistance and T2DM⁶. Furthermore, studies in Indian and Spanish populations reveal that FTO risk carriers (A allele for rs9939609) experience higher fasting plasma glucose concentrations when adhering poorly to Mediterranean dietary patterns or consuming low folate⁶. However, providing personalized, high-protein or modified-fat diets to these risk carriers often yields more favorable outcomes in weight loss, body composition, and HOMA-IR reduction than standard diets¹⁴.

Other Gene-Diet Interactions in Glycemia

Precision nutrition studies identify important gene-diet interactions in addition to the above-mentioned three major genes (TCF7L2, IRS1, and FTO):

PPARG (Peroxisome proliferator-activated receptor gamma): The PPARG Pro12Ala polymorphism (rs1801282) has been strongly associated with fatty acid storage and glucose metabolism⁶. Studies show that the Pro12 allele interacts adversely with high fat consumption to increase T2DM risk⁶. Nutrigenetic diets specifically restricting saturated fats for these carriers have shown improvements in insulin sensitivity indices¹⁵.

MTNR1B: Melatonin's circadian effects are controlled by this gene. The time of meal consumption is influenced by the rs10830963 variation ("chrononutrition"). Compared to early eaters, carriers of the risk allele (G) who eat late or have late dinners have considerably worse insulin secretion

and glucose tolerance. High dietary iron intake further exacerbates elevated fasting glucose and HbA1c levels in these carriers⁶.

AMY1 & CDKAL1: Genetic variants in AMY1 (salivary amylase) influence carbohydrate digestion. Individuals with certain AMY1 variants with a dietary carbohydrate content of more than 65% of total energy have an increased incidence of T2DM. Likewise, CDKAL1 variants interact with low intakes of protein and fat for T2DM risk⁶.

Personalized Diets by Multi-Omics, Microbiome and Algorithms

Isolated nutrigenetic interactions are impactful, but the state-of-the-art of precision nutrition combines genomics with microbiome profiling, clinical data, and CGM (Continuous Glucose Monitoring) through machine learning algorithms.

The microbiota is a key part of precision nutrition. The gut microbiota balance of *Prevotella*, *Bacteroides*, and *Bifidobacterium* significantly impacts the gut production of short-chain fatty acids as well as the metabolism of bile acids and systemic inflammation. These factors all influence the host's glycemic reactions³. The Personalized Postprandial-Targeting (PPT) diet is one of the most innovative advances in this area. The PPGRs were predicted for each participant in this randomized crossover study by a machine learning technique, combining clinical features, CGM data, and stool sample metagenomic sequence^{8,16}. When compared to the gold-standard Mediterranean (MED) diet, the PPT diet yielded considerably lower CGM-based parameters, including a significant reduction in the amount of time each day when glucose levels were above 140 mg/dL (-2.42 hours/day; $p < 0.001$). After a 6-month long-term intervention, the PPT diet significantly improved HbA1c (-0.39%), fasting glucose (-16.4 mg/dL), and triglycerides, with a 61% prediabetes remission rate¹⁷.

A systematic review of RCTs on how effective personalized nutrition for adults with T2DM and prediabetes also confirmed these better outcomes. The synthesis of high-quality trials showed that personalized nutrition resulted in a median mean difference in HbA1c of -0.925% ($p < 0.01$). It also led to important reductions in total PPGR (-14.85 mg/dL*h), carbohydrate consumption (-10.8% of total energy) and increased gut microbiome richness and diversity⁸.

"Digital Twin" technology is further validated by multi-omic approaches. A 1-year RCT assessed a digital twin-enabled personalized nutrition program that used AI to integrate real-time CGM data, diet logs, and biometric inputs. The dynamic model produced personalized and real-time dietary recommendations to reverse metabolic-associated fatty liver disease (MAFLD) and T2DM. When compared to standard of care on the digital twin platform, active manipulation of specific PPGRs with exact macronutrient timing and combination led to significant reductions in

HbA1c, ASCVD risk, and hyperinsulinemia¹⁸.

Furthermore, the ASPIRE-DNA pilot study evaluated DNA-based dietary advice in subjects with non-diabetic hyperglycaemia. At 26-weeks, participants who received DNA-personalized guidance, matching 11 SNPs (such as TCF7L2 and FTO) to particular macronutrient intolerances (such as sensitivity to salt or saturated fat) had significantly lower FPG and HbA1c (-0.038%) than the conventional care control arm ($p=0.04$)¹⁹. This genetic-guided intervention importantly showed early evidence of prevention of progression to T2DM ($p=0.06$), independent of major weight loss, thereby demonstrating that macronutrient distribution based on DNA optimization directly improves glucose regulation¹⁹.

Finally, clinical success with the use of n-of-1 trial methodologies demonstrates the superiority of genetically guided MNT. In cross-over trials, switching from standard MNT to precision MNT resulted in significant improvements in body weight, fasting plasma glucose, and blood pressure in Caucasian adults with T2DM and facilitated remission of prediabetes based on individual genetic risk scores (GRS) for T2DM, fasting glucose, and specific SNPs (e.g., MTHFR and TCF7L2)⁵.

CONCLUSION

The incorporation of nutrigenetic and multi-omics information into diabetes management is an absolutely necessary and extremely efficient change of paradigm when compared to classical dietary recommendations. Existing evidence clearly proves that individual gene polymorphisms (TCF7L2, IRS1, and FTO genes, in particular) significantly affect the body's response to diet intake in terms of carbohydrates, fats, and fibers consumption. So, for instance, a moderate carbohydrate-based diet helps individuals with normal alleles of TCF7L2, while those carrying risk alleles need to implement more specific changes to their macronutrients.

Moreover, the combination of genomics information, microbiota, lipidomics, and continuous glucose monitoring using machine learning (Digital Twins and PPT diets, in particular) provides exceptional clinical efficiency. Personalized nutrition approaches always beat classic control diets in terms of HbA1c decline (median -0.925% reduction), lowering of postprandial glucose levels, overcoming insulin resistance, and higher rates of T2DM remission. With the prevalence of "diabesity" increasing steadily around the world, future recommendations in medicine need to focus on applying these models based on genomics and microbiomics to the development of feasible healthcare solutions. The move towards genotype-specific and microbiome-based medical nutritional therapy will not only ensure better compliance but also ensure glycemic control and reduce the serious complications that come with Type 2 diabetes.

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