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Precision Nutrition in Diabesity: Integrating Genomics, Lipidomics, and Microbiome Data for Personalized Intervention

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ABSTRACT

Obesity and type 2 diabetes increasingly co-occur as "diabesity," yet individuals with similar BMI and lifestyle can show strikingly different metabolic risk and treatment responses. This heterogeneity reflects complex interactions among genetic variants, lipid metabolism and gut microbiota, superimposed on diet and environment. Precision nutrition seeks to harness this variability by using multi-layer omics data to design individualized dietary interventions that optimize weight, glycemic control and cardiometabolic risk rather than relying on one-size-fits-all guidelines. Large nutrigenetic and microbiome-informed nutrition trials demonstrate that inter-individual variation in postprandial glycemia and lipemia can be partially predicted from genomic, clinical and microbiome features, and that diets tailored using these predictors can improve glycemic profiles beyond standard advice. Parallel advances in lipidomics and metabolomics have identified lipid signatures that better capture diabesity risk than traditional lipids and may serve as targets and readouts for tailored diets. Integrative multiomics frameworks and machine learning now provide tools to combine genomics, lipidomics and microbiome data into clinically usable models. This review summarizes the genomic, lipidomic and microbiome foundations of precision nutrition in diabesity, outlines emerging multiomics integration strategies and discusses how these can be translated into personalized interventions. We highlight current limitations in evidence, equity, data integration and implementation, and propose research priorities for moving from proof-of-concept algorithms to scalable precision nutrition services in obesity-related diabetes care.

Keywords: diabesity; precision nutrition; nutrigenomics; lipidomics; gut microbiome

INTRODUCTION

Conventional dietary guidelines for obesity and type 2 diabetes focus on population averages. Calorie restriction, reduced saturated fat and added sugars and increased fiber and whole foods reduce incident diabetes and cardiovascular events at the group level. Yet within any trial, a substantial fraction of participants exhibits minimal improvement or even deterioration on "optimal" diets, while others show profound benefit with similar adherence[1]. This response heterogeneity undermines long-term effectiveness and adherence in real-world settings where patients quickly abandon regimens that do not "work for them."

Diabesity exemplifies this problem. People with similar BMI and lifestyle can display very different patterns of visceral fat, ectopic hepatic and pancreatic fat, dyslipidemia, beta-cell reserve and microvascular risk. General advice to "eat less and move more" fails to address underlying biological differences that shape appetite regulation, postprandial glycemic excursions, lipid handling and microbiome–host interactions[2, 3]. Precision nutrition has emerged as a strategy to move beyond this population-level approach by tailoring dietary recommendations to an individual's genomic, metabolic and microbiome profile and their clinical and behavioral context[4]. Evidence for the feasibility of precision nutrition in glycemic control came from the Personalized Nutrition Project, which showed that individuals eating identical meals had highly variable postprandial glucose responses that could not be explained by meal composition alone. A machine-learning model integrating clinical variables, blood parameters, lifestyle and gut microbiome features predicted glycemic responses better than calorie or carbohydrate content, and personalized diets constructed using this model significantly improved glycemic profiles compared with standard advice[5]. Subsequent microbiome-based personalized diet trials in

hyperglycemic and hyperlipidemic individuals reported improvements in glycemia and lipids when diet was tailored to microbiome and clinical data[5].

Parallel work in nutrigenetics reveals that common variants in obesity and T2D genes modify responses to macronutrient distribution. Variants in FTO, MC4R and especially TCF7L2, the strongest T2D locus, influence the impact of dietary carbohydrate and fat on glycemic control and diabetes risk[6]. For example, carriers of the rs7903146 TCF7L2 risk allele appear more susceptible to high glycemic load and respond differently to low-carbohydrate or high-fiber diets in terms of insulin secretion, HbA1c and incident T2D[7]. Nutrigenomic studies further show that specific nutrients and bioactive compounds can modulate the expression of genes involved in insulin signaling and inflammation, suggesting that diet–gene interactions are bidirectional[7].

Lipidomics adds another layer. Detailed profiling of serum lipid species reveals distinct "lipotypes" that reflect hepatic de novo lipogenesis, adipose lipolysis, mitochondrial function and inflammation more precisely than traditional lipid panels. Certain ceramides, acylcarnitines and triglyceride species strongly predict T2D risk and cardiovascular events, sometimes independently of LDL cholesterol and triglycerides[8]. These lipid signatures are modifiable by diet and weight loss and may help identify which individuals will benefit most from specific dietary fat and carbohydrate patterns.

The gut microbiome represents a third pillar of precision nutrition. Microbiome composition and functional capacity influence short-chain fatty acid production, bile acid metabolism, choline handling and endotoxin exposure, which in turn affect appetite, insulin sensitivity, hepatic lipid metabolism and low-grade inflammation[9–11]. Inter-individual differences in microbiome structure explain part of the variation in glycemic and lipemic responses to standardized meals and predict weight-loss responses to specific diets[8].

Multiomics frameworks now allow simultaneous measurement of host genomics, transcriptomics, epigenomics, lipidomics, metabolomics and metagenomics. Integrative analyses have begun to map how diet interacts with these layers to shape diabesity phenotypes and treatment responses[12, 13]. When coupled with machine learning and digital health tools such as continuous glucose monitoring, these data can be turned into predictive models that generate personalized nutrition prescriptions.

In this context, diabesity is an ideal testbed for precision nutrition: the disease burden is high, diet is central to pathogenesis and therapy, traditional approaches leave many "non-responders," and biomarkers that capture genomic, lipidomic and microbiome diversity are increasingly accessible. The challenge is to convert complex omics data into clinically actionable dietary advice that is safe, equitable and cost-effective. The following sections examine the contribution of genomics, lipidomics and microbiome science to this goal and discuss strategies for integrating them into personalized interventions for diabesity.

2. Genomic Foundations of Precision Nutrition in Obesity and Type 2 Diabetes

Genome-wide association studies have identified hundreds of loci associated with BMI, adiposity distribution and T2D risk, many in pathways related to appetite regulation, adipogenesis, insulin secretion and hepatic glucose metabolism[14]. Most variants have modest effect sizes, but collectively they shape susceptibility to diabesity and modulate responses to dietary exposures. Nutrigenetics focuses on how such variants influence the impact of diet on metabolic outcomes[15]. TCF7L2 exemplifies this interaction. The rs7903146 risk allele strongly increases T2D risk, largely by impairing beta-cell function and incretin signaling[16, 17]. Systematic reviews indicate that TCF7L2 variants modify the relationship between carbohydrate intake, glycemic load and glycemic outcomes, with risk allele carriers often experiencing greater deterioration in glucose tolerance on high-carbohydrate diets and variable benefit from intensive lifestyle or Mediterranean-style interventions[18].

Other loci such as FTO and MC4R influence appetite and satiety signaling. Carriers of risk alleles may have higher energy intake and stronger hedonic responses to high-fat or sugary foods, but may also respond particularly well to structured dietary counseling that targets these tendencies[19]. Variants in genes related to lipid metabolism, such as APOA5 or PNPLA3, can affect postprandial triglyceride handling and hepatic steatosis risk, potentially guiding fat quality and carbohydrate recommendations[19]. Nutrigenomics extends beyond static variants to examine how diet influences gene expression, DNA methylation and chromatin state in pathways relevant to diabesity. Polyphenols, long-chain omega-3 fatty acids, fiber-derived short-chain fatty acids and caloric restriction can modulate expression of genes involved in insulin signaling, inflammation and oxidative stress, sometimes reversing adverse epigenetic marks associated with high-fat diets or hyperglycemia[20–23]. Such findings suggest that genotype-informed diets might be combined with nutrients that actively reprogram pathogenic transcriptional networks.

While some commercial nutrigenetic tests already provide macronutrient advice based on panels of risk variants, robust evidence that genotype-guided diets outperform standard approaches in diabesity remains limited. However, accumulating data linking specific variants such as TCF7L2, FTO and PNPLA3 to diet responsiveness provides a mechanistic foundation for integrating genomics into multiomics precision nutrition models rather than using genetics in isolation[24].

3. Lipidomics and Metabolic Phenotyping for Personalized Dietary Targeting

Traditional lipid measures such as triglycerides, HDL and LDL cholesterol only partially capture the complex lipid disturbances that accompany diabesity. High-resolution lipidomics reveals hundreds of lipid species differing in chain length, saturation and headgroup, which reflect distinct metabolic pathways and confer

differing cardiometabolic risks[25]. Ceramides have received particular attention. Elevated plasma ceramides and specific ceramide ratios predict incident T2D and cardiovascular events and correlate with hepatic steatosis and insulin resistance, sometimes independent of BMI and conventional lipids[26]. Diet influences ceramide metabolism: saturated fat and excess fructose promote ceramide synthesis, whereas weight loss, Mediterranean-style diets and increased unsaturated fat intake tend to reduce ceramide levels. This positions ceramides as both biomarkers and potential therapeutic targets for precision nutrition in diabesity. Beyond ceramides, triglyceride species that are rich in de novo lipogenesis–derived fatty acids, such as palmitate-containing TGs, reflect high hepatic carbohydrate load and lipogenesis, suggesting that individuals with such signatures may benefit especially from carbohydrate restriction or low-glycemic-load diets[27]. Conversely, lipidomic patterns indicative of impaired mitochondrial beta-oxidation or elevated acylcarnitines may call for interventions that enhance mitochondrial function, including tailored exercise and specific micronutrients[27].

Multiomics obesity biomarker studies integrate lipidomics with genomics and microbiome data to define metabolic subtypes that differ in pathways such as lipogenesis, inflammation and bile acid metabolism. These subtypes show differing risks for progression from obesity to T2D and cardiovascular disease, and may respond differently to weight loss and dietary composition[28]. For example, individuals with an "inflammatory lipotype" characterized by certain sphingolipids and oxidized lipids may require more aggressive anti-inflammatory dietary strategies than those with primarily lipogenesis-driven profiles. Lipidomics can also serve as a dynamic readout of dietary adherence and effect. Changes in specific lipid species can confirm whether a low-carbohydrate or low-saturated-fat diet is effectively shifting underlying metabolic fluxes, supplementing weight and glucose monitoring[29].

Overall, lipidomics refines metabolic phenotyping in diabesity, identifies high-risk patterns invisible to standard lipids and provides mechanistic anchors for individualized dietary prescriptions focused on fat quality, carbohydrate load and energy balance.

4. Gut Microbiome Signatures and Microbiome-Informed Precision Nutrition

The gut microbiome shapes host energy harvest, bile acid pools, SCFA production, intestinal permeability, and immune tone, all of which influence diabesity risk and progression[9, 10, 30, 31]. Obesity and T2D are associated with shifts in microbial diversity, depletion of SCFA-producing taxa, expansion of pathobionts, and altered microbial metabolic pathways[32, 33].

Microbiome-based personalized nutrition gained prominence after studies showed that microbial composition and function markedly improved the prediction of postprandial glucose responses beyond nutrient content. In the Personalized Nutrition Project, models that integrated microbiome features with clinical and dietary data outperformed calorie-based predictions and enabled the design of personalized diets that reduced postprandial glycemia[34]. More recent trials in hyperglycemic and hyperlipidemic individuals demonstrated that microbiome-guided personalized diets improve glycemic control and lipid profiles over months, with changes in microbial pathways related to carbohydrate metabolism and SCFA production correlating with clinical benefit[34].

Microbiome-informed precision nutrition leverages both compositional and functional readouts. The presence or absence of specific taxa, such as Akkermansia or Prevotella, can influence responses to high-fiber or whole-grain–rich diets. Functional metagenomic profiles, including carbohydrate-active enzymes, bile salt hydrolases and SCFA synthesis pathways, may better capture the capacity to metabolize specific fiber types, polyphenols or emulsifiers[35]. Deep learning approaches trained on microbiome plus metabolomic data further enhance the prediction of individual food responses and weight-loss outcomes[35]. These models can, in principle, suggest personalized adjustments in fiber type, fat quality, fermented foods and probiotic or prebiotic supplementation to shift microbiome function in a direction that supports better glycemic and lipid control.

However, microbiome-based precision nutrition must contend with substantial intra-individual temporal variability, context dependence and the influence of medications such as metformin, GLP-1 analogues and SGLT2 inhibitors on microbial composition[36–38]. Robust translation will require repeated sampling, standardized analytical pipelines and clinical trials that test microbiome-informed diets against conventional diets in well-characterized diabesity cohorts.

5. Integrating Genomics, Lipidomics and Microbiome Data: Multiomics Frameworks for Precision Nutrition

Although genomics, lipidomics and microbiome science each provide valuable insights, diabesity is a systems-level disorder in which these layers interact. Multiomics frameworks seek to integrate them into coherent models that explain metabolic heterogeneity and guide personalized interventions[39]. Systems biology studies have combined genotype, transcriptome, metabolome and microbiome data to map diet–gene–microbe networks that underlie obesity and T2D phenotypes[40]. For example, host variants in TCF7L2 and related pathways may influence bile acid metabolism and intestinal hormone secretion, which interact with microbiome-derived metabolites and dietary patterns to shape hepatic lipidomics and glycemic control[40]. Recent reviews outline methodological strategies for multiomics integration, including network-based approaches, factor analysis and machine learning methods that identify latent components capturing shared variance across omics layers[41].

These components can define metabolic subtypes that differ in gene–diet interactions, lipid pathways and microbiome configurations, providing a more nuanced basis for precision nutrition than single-omics clustering.
In parallel, large-scale precision nutrition initiatives such as Food4Me, PREDICT and related studies have started to integrate genetic, metabolomic and microbiome data with continuous glucose monitoring and digital diet tracking to build prediction models for postprandial glycemia, lipemia and weight change[42]. These models are moving toward clinical usability as algorithms that generate individualized dietary recommendations through apps or clinician dashboards. Artificial intelligence, including deep learning and large language models, is increasingly used to analyze complex multiomics datasets and incorporate contextual variables such as medications, physical activity and circadian patterns[42, 43]. In diabesity, such models could, in theory, calculate for each person a set of dietary patterns, meal timing and macronutrient distributions that optimally lower glycemic variability and harmful lipotypes while supporting weight loss and microbiome health.

However, data harmonization, computational transparency and clinical validation remain major challenges. Multiomics models must be robust across diverse ancestries, environments and healthcare settings, and their predictions must be tested against standard-of-care diets in randomized diabesity trials before widespread adoption.

6. From Prediction to Prescription: Designing Personalized Nutrition Interventions for Diabesity

Translating multiomics insights into care requires workflows that start with accessible measurements and end with realistic diets that patients can follow. In a diabesity clinic, a precision nutrition pathway might begin with baseline phenotyping, including anthropometrics, clinical labs, continuous glucose monitoring or standardized meal tests, targeted genomics focused on well-validated loci such as TCF7L2 and FTO, lipidomic panels emphasizing ceramides and key triglyceride species and gut microbiome profiling[44].

These data can be fed into trained prediction models that output individualized targets for macronutrient distribution, food choices and meal timing. For example, a patient with high ceramides, TCF7L2 risk alleles and microbiome features associated with poor carbohydrate handling might receive a diet emphasizing low-glycemic-index carbohydrates, higher monounsaturated fat, specific fibers and polyphenol-rich foods that support SCFA production and reduce ceramide synthesis. Another patient with a lipidomic profile dominated by de novo lipogenesis signatures but favorable microbiome diversity might be guided toward a moderate-carbohydrate, high-fiber Mediterranean pattern coupled with time-restricted eating to align with circadian glucose rhythms[44].. Importantly, precision nutrition does not imply designing a unique diet from scratch for every individual. Rather, it means selecting from a repertoire of evidence-based dietary patterns the one most compatible with the patient's omics profile and personal preferences and then fine-tuning elements such as carbohydrate quality, fat subtype, fermentation, and timing[45]. Behavioral support, digital tools and iterative adjustment based on follow-up glycemic and lipidomic responses are essential to maintain adherence[44].

Early precision nutrition trials demonstrate that personalized advice based on baseline diet, phenotype and sometimes genotype improves dietary behaviors and risk markers more than generic guidelines, but the incremental benefit of adding genomic, lipidomic and microbiome data in diabesity-specific outcomes still needs rigorous testing. Implementation will require simplified, cost-effective omics panels, user-friendly interfaces for clinicians and patients and integration with existing diabetes care pathways.

7. Challenges, Ethics and Future Directions in Precision Nutrition for Diabesity

Despite rapid advances, multiomics precision nutrition for diabesity remains largely in the translational research phase. Several challenges must be addressed before it can become standard care. First, the evidence base is still emerging. Many nutrigenetic associations are modest, context-dependent or inconsistent across populations, and microbiome–diet response models are often trained in relatively homogeneous cohorts[46]. Large, diverse diabesity trials that directly compare multiomics-guided diets with high-quality standardized diets on hard outcomes such as HbA1c, weight, liver fat and cardiovascular events are needed. Second, data integration and interpretability remain difficult. Multiomics models risk becoming black boxes, which can undermine clinician trust and complicate regulatory approval. There is a tension between predictive performance and mechanistic transparency. Methods that link model features back to interpretable biological pathways will be important for gaining clinical acceptance and for refining interventions[46].

Third, equity and accessibility are major concerns. High-throughput sequencing and lipidomics remain expensive and concentrated in well-resourced settings. There is a risk that precision nutrition could widen disparities by being available only to affluent patients, while many people with diabesity lack access to basic healthy foods. Research should prioritize scalable, low-cost omics panels, validate models in low- and middle-income populations and ensure that personalized recommendations are compatible with local food systems and cultural patterns[47]. Fourth, ethical and privacy issues arise from the collection and storage of genomic and microbiome data. Clear consent processes, data governance frameworks and patient education are essential, especially if artificial intelligence systems are used to generate recommendations[47].

Looking ahead, the most promising path may be tiered precision nutrition. Basic phenotype-driven personalization using clinical and CGM data could be offered broadly, with genomics, lipidomics and microbiome layers added for individuals with refractory diabesity, early complications or strong family history.

Continuous learning systems could update prediction models as more outcome data accumulate, gradually improving performance and enabling finer stratification of diabesity subtypes.

CONCLUSION

In summary, integrating genomics, lipidomics and microbiome data into precision nutrition frameworks offers a path to more effective, individualized management of diabesity. While clinical translation is incomplete, the conceptual and technological groundwork is in place. As evidence grows and tools become more accessible, precision nutrition has the potential to shift diabesity care from generic dietary prescriptions toward targeted nutritional strategies that reflect each person's unique biological and environmental context.

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