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Review Article

A Nutriomic Approach for Day-to-Day Blood Sugar Control

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Abstract: Rising sugar sets off health! How much and how soon, our genes can say and our lifestyle can accomplish. Beyond treating diabetes, we have set foot in a new era of reversing hyperglycemia to normoglycemia through nutriomic approaches which enhance treatment. Diabetes is a metabolic disorder. Hence its metabolic markers like hypo/hyper glycemia, insulin resistance, altered adiposity, and lipid abnormalities should be mapped to their underlying genetic cause. And genetic insights should be used as cues for precise lifestyle modifications to achieve glycemia control. The precise lifestyle changes suggested through nutriomics are more effective and perpetual in reversing blood sugar levels. Nutriomics indulges genetic insights to provide information on nutrient metabolism relevant to glycemia. Blood sugar reduction and its consistent maintenance within a desirable range can assure a betterment in the quality of life through symptom management, co-morbidity reduction & improved organ health. This review research outlines the importance of nutriomics in efficient, every day blood sugar management illustrating practical scenarios. It also summarizes the scope and vitality of nutriomics in enhancing treatment approaches for glycemia control.

Keywords: Nutriomics, Pharmacogenomics, Dysglycemia, Xerostomia, Precise lifestyle modifications.

Introduction

Diabetes mellitus is a major metabolic disorder characterized by impaired glucose metabolism, insulin resistance, and associated metabolic abnormalities. The increasing global prevalence of diabetes has highlighted the need for more effective and personalized approaches for glycemic control. In recent years, nutriomics, including nutrigenomics and nutrigenetics, has emerged as an innovative field that studies the interaction between nutrients and genes and their influence on metabolic health.

Nutriomic approaches help identify gene nutrient interactions involved in blood sugar regulation, insulin signaling, lipid metabolism, and diabetic complications. Personalized nutritional interventions based on genetic insights may improve glycemic control, enhance treatment response, and reduce diabetes-associated symptoms and co-morbidities. This review summarizes the role of nutriomics in day-to-day blood sugar management and highlights its potential in supporting precision-based diabetic care.

Nutriomics- An Innovative approach in Glycemia control

Diabetes is a disorder which associates with altered nutrient metabolism. Genetic insights impacting the nutrient metabolism are crucial in recommending precise dietary patterns. And every day dietary practices contribute to an effective glycemia control. Hence this review has considered research articles which portray the molecular mechanics of gene-nutrient interactions in diabetes and its symptom management. Additionally, there is a section on integrating nutriomics with pharmacogenomics, wherein scientific articles have been explored for nutritional influence on 'diabetic' drug-target pathways. The following examples illustrate the necessity of precise nutrition in coping with unfavorable genetic changes (which underlie the metabolic abnormalities of dysglycemia) [1,2,3].

Nutritional genomics can aid in identifying molecular events that underlie a post-meal blood glucose spike

For instance, caffeine can increase the blood sugar levels when consumed along with a main meal. The ideal time gap between the intake of caffeinated food/beverage and a main meal differs between individuals based on their genetic changes.

The Cytochrome P450 1A2 or CYP1A2 gene, is the prime controller of caffeine metabolism, through its enzymatic activity. A polymorphism in the CYP1A2 gene, namely, rs762551 (−163C > A) makes its 'C' allele carriers slow caffeine metabolizers. Blood glucose concentrations an-hour after meal were significantly higher in AC/CC genotypes, when caffeine was consumed along with a main meal [4,5]. Hence nutrigenetic recommendations suggest refraining from caffeine an hour before- and after- a meal. Additionally, inclusion of cruciferous vegetables like broccoli, cabbage, cauliflower, kale, radish, turnip and garlic can lower blood sugar by enhancing the pace of caffeine metabolism [6]. While vegetables like carrot, celery, parsley and parsnip can slow down caffeine metabolism, hence do not consume these vegetables for at least an hour after caffeine intake.

Gene-based nutritional recommendations can alleviate diabetes' symptoms

Xerostomia or dry mouth is a common symptom of diabetes, especially in people with blood sugar spikes. It can be caused by changes in the amount or composition of saliva. Hyposalivation in diabetes-induced xerostomia can increase the risk of dental caries. Genes determine salivary health as they stimulate oral buffers, regulate oral pH and accelerate acid neutralization; notable ones include salivary amylase/AMY1, aquaporin 5/AQP5, carbonic anhydrase/CA [7,8,9]. Considering gene-suitable recommendations for salivary health, fruits like watermelon and vegetables like cucumber are great choices owing to their high-water content. Capsicum, and spices like pepper and oregano contain a component called capsaicin which improves salivary health [10,11,12]. The following table (Table 1) summarizes the scope and vitality of nutriomics in enhancing treatment approaches

Table 1: Scope and Vitality of Nutriomics in Enhancing Diabetic Treatment Approaches.

Diabetic drug	Drug target pathway and potential genetic role (pharmacogenomics)	Nutriomics
Rapamycin	Rapamycin inhibits mTOR pathway. Insulin initiates the action of Mammalian target of rapamycin (mTOR). The mTOR phosphorylates insulin receptor substrate (IRS) to limit insulin signaling, and thus its overstimulation leads to insulin resistance/IR and cellular senescence [13].	epigallocatechin gallate and isoliquiritigenin are mimetics of rapamycin [14].
Metformin	Metformin, a biguanide, corrects hyperglycemia mainly by lowering hepatic gluconeogenesis. It also increases insulin sensitivity and lowers circulating lipid levels primarily by activation of AMPK pathway. The Adenosine Monophos-phate activated Protein Kinase (AMPK) is a major cellular regulator of lipid and glucose metabolism	allantoin and ginseno-side are mimetics of metformin [14].
Repaglinide	Repaglinide is a meglitinide category of antidiabetic drug that lowers blood glu-cose by blocking ATP-dependent potassium channels in pancreatic beta cells. This consequently stimulates insulin secretion. The 'potassium voltage-gated channel subfamily Q member 1' or KCNQ1 gene encodes the KCNQ1 protein, and its in-creased expression limits insulin secretion from pancreatic β-cells by regulating the potassium channel current [15]. Following repaglinide treatment, type 2 diabetics with the T allele at rs2237892 and C allele at rs2237895 of the KCNQ1 gene were more likely to have a positive response to repaglinide as compared to the rs2237892 CC and rs2237895 AA geno-types [16].	The amino acid L-arginine is dually beneficial as a secretagogue as well as an essential synergic compound for nutrient-dependent insulin secretion [17].
	The transcription factor 7-like 2 (TCF7L2) regulates glucagon-like peptide 1 (GLP-1) and glucose-mediated insulin secretion. In Type 2 diabetics, the 'T' allele at rs290487 of TCF7L2 gene might confer 1.85 times risk of suffering Diabetic Nephropathy compared to C allele (OR = 1.85, 95% CI = 1.02–3.10). TT genotype may have better response to repaglinide [18,19].	Chromium improves insulin sensitivity by up-regulating insulin-stimulated insulin signal transduction [20].
	The solute carrier family 30 (zinc transporter), member 8 (SLC30A8) encodes a protein, zinc efflux transporter, which accumulates zinc in intracellular vesicles. This gene is highly expressed in the pancreatic islets. The encoded protein colocalizes with insulin in the secretory pathway granules of insulin-secreting INS-1 cells. The C allele of SLC30A8 rs13266634 was associated with higher odds of type 2 diabetes. A better response to repaglinide was seen amongst CT and TT genotypes compared with CC genotype [21].	In 'C' allele carriers, higher plasma zinc inversely correlated with fasting glucose levels [22].
Pioglitazone	Pioglitazone, a thiazolidinedione, targets the peroxisome proliferator-activated receptor-gamma (PPARγ) pathway. Activation of PPARγ increases the transcription of insulin-responsive genes. In the PPARγ gene, a polymorphism, rs1801282, de-creases pioglitazone response in CC genotypes [23].	Naringin and hesperidin, the two major flavanones of citrus fruits are functional in activation of the fat and liver PPARG expres-sion [24,25].
Fenofibrate	Fenofibrate is a fibrate that activates peroxisome proliferator activated receptor alpha (PPARα) to favorably alter lipid metabolism in diabetes. Fenofibrate increas-es lipoprotein lipolysis and hepatic fatty acid uptake, and it reduces hepatic tri-glyceride production. In type 2 diabetes with hypertriglyceridemia, the presence of 'G' allele at rs4253778 of PPARα associates with an effective triglyceride reduction after fenofibrate treatment [26].	Anthocyanins (especially Bilberry anthocyanins) can reduce hyperglycemia, improve insulin sensitivity and aid in obesity (co-morbidity) management. They down-regulate the expression of gluconeogenic and lipogenic enzymes; and up-regulate the expression of PPARα, L-carnitine palmetoyl phosphatase 1/Cpt-1, Glut4 and aconitase (Aco) in the liver [27,28].

Conclusion

Nutriomics indulges genetic insights to provide information on nutrient metabolism relevant to glycemia. The precise lifestyle changes suggested through nutriomics are more effective and perpetual in reversing blood sugar levels. Blood sugar reduction and its consistent maintenance within a desirable range can assure a betterment in the quality of life through symptom management, co-morbidity reduction & improved organ health.

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