International Journal of Food Science and Nutrition
www.foodsciencejournal.com
ISSN: 2455-4898
Received: 21-12-2022, Accepted: 06-01-2023, Published: 23-01-2023
Volume 8, Issue 1, 2023, Page No. 23-26

Role of nutrigenomics in diabetes mellitus

Tarandeep Kaur
Assistant Professor Department of Home Science, Kanya Maha Vidyalaya, Jalandhar, Punjab, India

Abstract
The "silent killer," diabetes mellitus, has indeed been identified as a significant worldwide medical problem. Dietary management has been shown to be a significant strategy for achieving adequate glycaemic control in diabetes, and nutrition/diet continues to be an important factor in both the management and prevention of the disease. Nutritional genomics, also known as nutrigenomics, is the study of how diets influence how genetic information is expressed in an individual as well as how a person's genetic make-up influences how their body processes nutrients and other bioactive ingredients in food. Given that both diet and genes influence health and illness susceptibility, discovering genes that are influenced by diet and that cause or contribute to chronic diseases could lead to the development of diagnostic tools, tailored intervention, and eventually techniques for preserving health. Studies on nutritional genomics typically focus on eating practices based on gene variations, the importance of gene-nutrient interactions, the links between genes, diet, and phenotypes, and changes in epigenetic state brought on by nutrients. Individual genetic variations and how they react to these functional foods are a key problem. It is now necessary to understand whether nutrients work at the cellular level as a result, which calls for a series of interactions involving nutrients at the protein, metabolic levels and genes. A nutrigenomic approach provides a tool to evaluate the effect of foods on gene/protein expression as well as a snapshot of the genes that are on/off at any one time (the genetic potential). In light of the high prevalence of diabetes mellitus, it is crucial to encourage nutrigenomics research. As a result, the current analysis offers insight into the significance of nutrient-gene interactions in diabetes, as well as their treatment and prevention.

Keywords: nutrigenomics, genes, diabetes mellitus, nutrient & nutrient- gene interaction

Introduction

The metabolic disorder known as diabetes mellitus (DM) is primarily caused by the pancreas' inability to release the hormone insulin, which regulates blood sugar levels, or by a concomitant condition characterised by problems with insulin sensitivity, insulin resistance, and excessive glucagon secretion *(Nwozo SO et al.*, 2019) [16]. DM is regarded as a worldwide burden since it is linked to the malfunction and failure of numerous organs, including the blood vessels, heart, and kidneys. The prevalence of diabetes mellitus (DM), as previously reported, is predicted to rise from 171 million cases in 2010 to 366 million cases in 2030 *(Nwawuba et al.*, 2019) [17]. DM is regarded as a silent killer because both the prevalence and the number of cases have been shown to be rapidly increasing over time. Numerous strategies, including dietary therapy, have been tried and implemented for the prevention and treatment of diabetes mellitus in an effort to lessen its threat. Genetic variability, on the other hand, is a significant issue that requires consideration. Therefore, it has become imperative to adopt a strategy that acknowledges the interaction between nutritional therapy and hereditary factors. Nutrigenomics is thrust into the spotlight by this necessity. In order to treat and prevent a variety of medical disorders, including diabetes mellitus (DM), nutrigenomics provides tailored nutritional counselling and produces specialised diets and nutrients for communities or for individuals *(Barnett MPG et al.*, 2017) [1]. The study of human health and disease can greatly benefit from a nutrigenomic approach, which is also essential to comprehending the intricate interactions between normal metabolic processes and outside factors in disease processes. Nutrigenomics has been shown to be essential for the development of more specialised approaches to the diagnosis, treatment, and prevention of disease. *(Barnett and Ferguson 2016).*

Due to the fact that food-derived bioactive components significantly impact genomic, proteomic, and metabolic changes, nutrigenomics research is currently the cornerstone *(Ravindra PV et al., 2009)* [21]. Nutrigenomics explains how a particular diet's bioactive ingredients affect a gene's expression, resulting in either activation or suppression *(Sales NMR et al., 2014)* [22]. The understanding of the connection between nutrition, genes, and diseases provided by nutrigenomics is backed up by a wealth of information. The Mediterranean diet has been proven to reduce heart disease risk factors and the incidence of stroke in persons with type 2 DM, a condition with which polymorphisms are strongly associated *(Berna G et al., 2014).* Through modulation of cell CLL/lymphoma 2 (BCL-2) expression, the flavanol epigallocatechin gallate (EGCG) has been shown to protect insulin secretory cells against pro-inflammatory cytokine-induced damage *(Zhang Z et al., 2011)* [26]. Spinach, eggs, sweet potatoes, and almonds are nutritional sources of biotin, which improves insulin secretion and islet function by signalling an increase in the genes FOXA2 (Fork head Box A2), HNF-4, and Calcium Channel Neuroendocrine/Brain-Type, Alpha-1 Subunit (CACNA1D) *(Lazo ML et al., 2013)* [14]. In order to combat the increase in diabetes mellitus, it is necessary to promote nutrigenomics research. Since nutrition and genes interact in the development, regulation, and control of diabetes, this current studies shed light on this interaction.

Diabetes mellitus and nutrigenomics

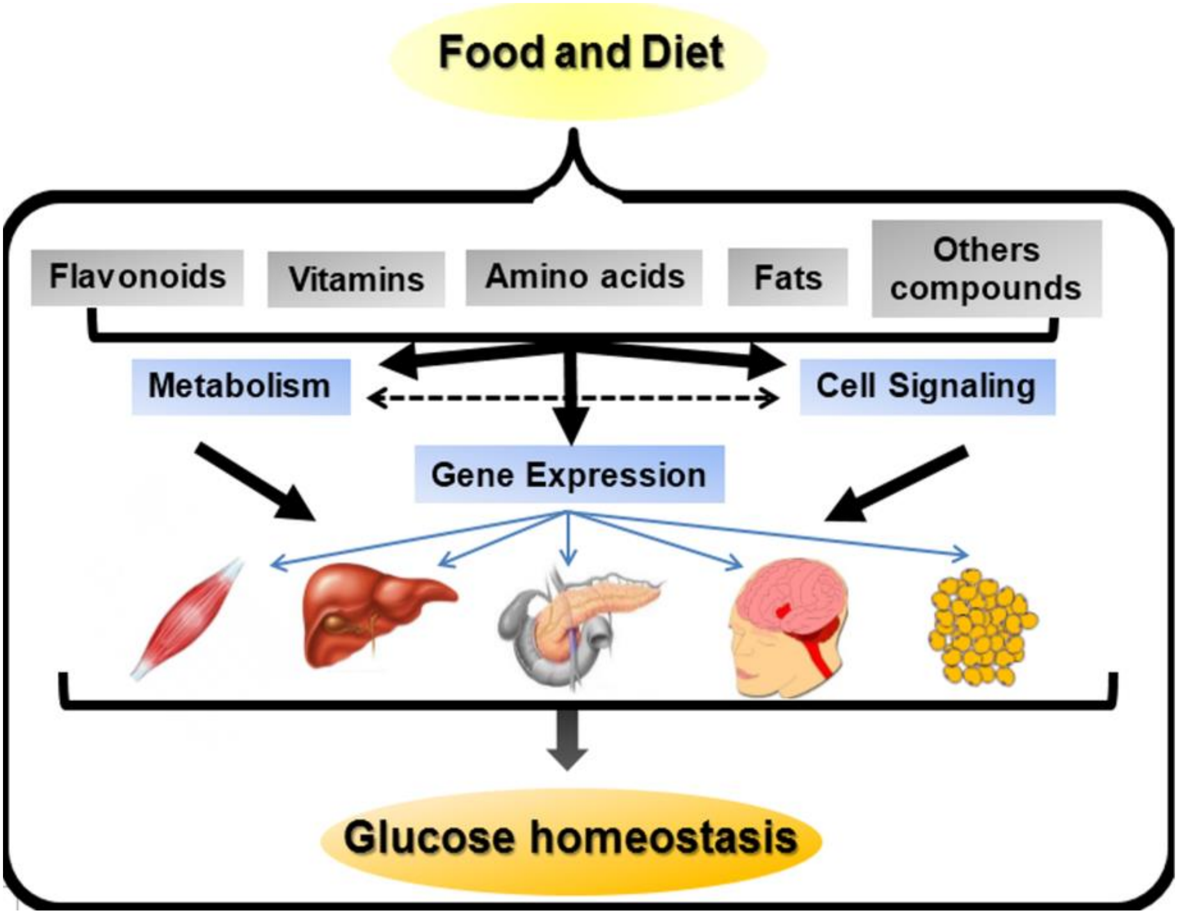
Type 1 diabetes (T1DM) and type 2 diabetes (T2DM), which are each classified according to their etiopathogenetic causes, are both recognised as dangerous chronic diseases. Insulin-dependent diabetes is often referred to as T1DM *(Dib SA et al.,* 2009) [6]. Diabetic patients must use exogenous insulin as a result of pancreatic cell failure, often known as juvenile-onset diabetes. Genetic predisposition caused by environmental factors in the form of dietary components is one of the major contributors to the development of T1DM *(Nwozo SO et al., 2019)* [16]. T2DM, on the other hand, is a result of circumstances brought on by the combination of insulin resistance, insulin insensitivity, and increased glucagon secretion. Diet, environmental variables, and the interactions between these genes and environmental factors may all play a role in its aetiology. DM is one of the top four non-communicable diseases (NCD) that need to be addressed by world leaders and is still seen as a serious public health issue. Therefore, T1DM develops in genetically predisposed people as a result of intricate interactions between cells, the immune system, and environmental variables *(Dib and Gomes* 2009) [6]. T1DM affects 5%–10% of diabetic individuals and is most common in children and young people. T2DM is a chronic condition brought on by inadequate insulin production and insulin resistance. T2DM is a complex trait caused by several genes, a variety of environmental factors, including nutrition, and the interactions between these genes and environmental factors *(Hansen and Pedersen 2005)* [9].

sbeen shown that the prevalence of diabetes has been steadily increasing over time, and as a result, it is referred to as a silent killer disease that affects a sizable population of people worldwide. DM is undoubtedly a significant worldwide health issue that has reached epidemic proportions and disproportionately affects developing nations compared to industrialised nations *(Nwozo SO et al., 2018)* [18]. Due in part to this, the medical community, particularly the American Diabetes Association, began to recognise the critical role that diet and nutrition play in the management and prevention of diabetes. As a result, new recommendations were made that centred on creating individualised eating plans that were aimed at achieving particular biomarkers *(Ordovas JM et al., 2016)* [19].

Nutrigenomics, also known as nutritional genomics, is a young field of study that has attracted a lot of attention recently. It focuses on nutrition research that uses molecular methods to clarify reactions to particular diets exposed to either individuals or population groupings (*Sharma and Dwivedi, 2017)* [24] *(Pavlidis C et al., 2015)* [20]. There are various definitions of nutrigenomics, but they all tend to mean the same thing: nutrigenomics is the study of the effects of nutrition and nutrients on a person's genome. Accordingly, Barnett and Ferguson's concept of nutrigenomics is the interaction of a person's diet and nutrition with their genome and the subsequent impacts on their phenotype. It provides individualised nutritional counselling and generates specific diets and nutrients for communities or for individuals. Nutrigenomics is crucial in the creation of a scientific framework to better understand the genes that contribute to these choices, demands and reactions to diet and nutrition as future consumer assessments of nutritional and well-being status become more sophisticated *(Kussmann and Daniel 2008)* [12]. It has been shown that nutrigenomics can identify genetic variables that are affected by nutrition in relation to disease states. much as its analogue, pharmacogenomics, and its relevance in the context of individualised nutrition have gained widespread appeal. It recognises that a certain diet may not be appropriate for another person based on their preferences.

Interplay between of nutrient/gene interactions

The interaction of numerous genes in response to diet has been demonstrated and proven in numerous studies to have a significant involvement in the occurrence and causation of illness problems, such as diabetes. In order to understand the etiopathogenesis, prevention, and management of diabetes, it is crucial to identify and analyse how diet, nutrition, and genes interact (nutrigenomics). Nutrigenomics heavily relies on high-throughput technologies and methodologies in genomics, transcriptomics, proteomics, and metabolomics for the assessment of genetic alterations and metabolic products which respond to the eating plan, in addition to the interpretation of bioactive molecules found in foods and also how they affect the human metabolic activity, nutrient homeostasis, and cellular provides the context in dietary ailments. From the viewpoint of molecular nutrition, nutrients can be viewed as signalling molecules that, when in contact with the right cellular sensing systems, can either transmit or translate dietary signals into changes in gene expression as well as the production of proteins and metabolites. According to Genoveva *et al*., diet/nutrients can generally influence gene expression through the following mechanisms: I directly; (ii) through their metabolites; and (iii) through signal transduction molecules. Figure 1 illustrates this, demonstrating how food, nutrition, and nutrients affect gene expression, resulting in the prevention and management of diabetes. Precision nutritional treatment methods to the study of health and disease have been established thanks to our growing understanding of how diet/nutrients and the human genome interact *(Mead MN. ,2015)*. It is obvious that one of the fundamental objectives of healthcare is the potential that evidence-based tailored or precision nutritional therapy techniques offer for the preservation of health as well as the treatment of several disease conditions, such as diabetes. Several dietary-bioactive compounds have been shown to have the power to regulate gene expression both explicitly or implicitly by triggering metabolites or chemical messengers that alter the intricate pathways that contribute to the onset of diabetes.



www.mdpi.com

Fig 1

Flavonoids-Gene Interactions in DM Pathogenesis

Numerous investigations using both living experimental animals and cell culture have shown that dietary flavonoids have a positive impact on glucose homeostasis *(Babu P.V. et al., 2013)* [3]. Additionally, human studies have shown that consuming more anthocyanins-especially those found in blueberries, apples, and pears-was consistently linked to a lower risk of developing diabetes *(Wedick N.M. et al., 2012)* [25]. Through diverse intracellular signalling pathways, it has been demonstrated that these substances control insulin secretion, glucose uptake, insulin signalling, and carbohydrate digestion in insulin-sensitive tissues *(Hanhineva K et al., 2010)* [10]. Fascinatingly, flavan-3-ols, particularly epigallocatechin gallate (EGCG), which are found in many fruits, teas, cocoa, and chocolate, have been demonstrated to enhance insulin secretory activity and the survivability of beta cells under conditions of glucotoxicity.

Bioactive Compounds-Gene Interactions during DM Etiology

In addition to flavonoids, other bioactive substances, like triterpenoids, have the potential to treat diabetes. Oleanolic acid is one of the members of this family that has been the subject of the most research *(Castellano J.M., et al., 2013)* [4]. More than 120 different plants contain this chemical, although the olive leaf has the most of it *(Jäger S., et al., 2009)* [11]. In 2010, *(Guinda A et al.2010)* [8] has been shown that oleanolic acid enhances insulin responsiveness and maintains the life and activity of pancreatic cells. These routes influence the expression of essential defence genes.

Conclusion

Nutrigenomic research reveals how food affects alterations to the specific gene and the way individual responds to them. In T1DM, pancreas cells are specifically targeted for autoimmune destruction. There is scant evidence that other nutrients may influence the T1DM onset except vitamin D, early consumption of gluten and cow's milk, and the protective benefits of breastfeeding. Additionally, the significance of the gut microbiota in the aetiology of T1DM has grown. Identifying polymorphisms that connect dietary factors with the drivers of T1DM development as well as understanding the potential impact of nutritional factors in the onset and progression of T1DM are equally crucial. To provide dietary approaches for the control and prevention of diabetes mellitus, genotype-dependent dietary healthy lifestyle methods, scientific proof individualised or personalized healthcare via dietary changes, and it is essential to advance the field of nutrigenomics research.

References

1. Barnett MPG, Ferguson LR. Nutrigenomics: integrating genomic approaches into nutrition research. Molecular Diagnostics, 2017, 305-326.
2. Berná G, Oliveras-López MJ, Jurado-Ruíz E, *et al*. Nutrigenetics and nutrigenomics insights into diabetes etiopathogenesis. *Nutrients*,2014:6(11):5338-5369.
3. Babu PV, Liu D, Gilbert ER. Recent advances in understanding the anti-diabetic actions of dietary flavonoids. *J. Nutr. Biochem*,2013:11:1777-1789. doi: 10.1016/j.jnutbio.2013.06.003.

4. Castellano J, Guinda A, Delgado T, Rada M, Cayuela JA. Biochemical basis of the antidiabetic activity of oleanolic acid and related pentacyclic triterpenes. *Diabetes*,2013:62:1791-1799. doi: 10.2337/db12-1215
5. Dauncey MJ. Recent advances in nutrition, genes and brain health. Proceedings of the Nutrition Society, 2012:71(4):581-591.
6. Dib SA, Gomes MB. Etiopathogenesis of type 1 diabetes mellitus: prognostic factors for the evolution of residual beta cell function. *Diabetol Metab Syndr*,2009:1(25):1-8.
7. Ferguson JF, Allayee H, Gerszten RE *et al*. Nutrigenomics, the microbiome, and gene-environment interactions: new directions in cardiovascular disease research, prevention, and treatment. a scientific statement from the American heart association. *Circ Cardiovasc Genet*,2016:9(3):291-313.
8. Guinda A, Rada M, Delgado T, Gutierrez-Adane P, Castellano JM. Pentacyclic triterpenoids from olive fruit and leaf. *J. Agric. Food Chem*,2010:58:9685-9691. doi: 10.1021/jf102039t.
9. Hansen L, Pedersen O. Genetics of type 2 diabetes mellitus: Status and perspectives. *Diabetes Obes. Metab*,2005:7:122-135. doi: 10.1111/j.1463-1326.2004.00396.x.
10. Hanhineva K, Torronen R, Bondia-Pons I, Pekkinen J, Kolehmainen M, Mykkanen H *et al*. Impact of dietary polyphenols on carbohydrate metabolism. *Int. J. Mol. Sci*,2010:11:1365-1402. doi: 10.3390/ijms11041365.
11. Jäger S, Trojan H, Kopp T, Laszczky MN, Scheffler A. Pentacyclic triterpene distribution in various plant-rich sources for a new group of multi-potent plant extracts. *Molecules*,2009:14:2016-2031. doi: 10.3390/molecules14062016.
12. Kussmann M, Daniel H. Editorial overview. *Curr Opin Biotechnol*,2008:19(2):63-65.
13. Kussmann M, Van Bladeren PJ. The extended nutrigenomics understanding the interplay between the genomes of food, gut microbes, and human host. Frontiers in Genetics,2011:2(21):1-13.
14. Lazo ML, Larrieta E, German MS *et al*. Effects of biotin supplementation in the diet on insulin secretion, islet gene expression, glucose homeostasis and beta-cell proportion. J Nutr Biochem,2013:24(1):169-177.
15. Mead MN. Nutrigenomics: the genome–food interface. *Environ Health Perspect*, 2007:115(12):A582-A589.
16. Nwozo SO, Ikpeme GE, Nwawuba SU. Functional dietary supplementation of Okara (soybeans residue) on streptozotocin induced diabetes mellitus in male wistar rats. Global Journal of Medical Research: F Diseases,2019:19(5):17-28.
17. Nwawuba SU, Nwozo SO, Mohammed KA. Dietary management of diabetes mellitus with focus on Nigeria. International Journal of Diabetes Research,2019:2(1):26-32.
18. Nwozo SO, Modeme TE, Nwawuba SU. Evaluation of momordica charantia, boerhaavia diffusa and cotreatment on streptozotocin induced diabetes in male wistar rats. International Journal of Biological Sciences,2018:14(2):66-73.
19. Ordovas JM, Berciano S, Mico V *et al*. Diet–Gene interactions in the development of diabetes. Molecular Nutrition and Diabetes, 2016, 41-50.
20. Pavlidis C, Patrinos GP, Katsila T. Nutrigenomics: a controversy. *Appl Transl Genom*,2015:4:50-53.
21. Ravindra PV, Jaiswal N, Janhavi P. Nutrigenomics: diabetes perspective. Asian Journal of Emerging Research,2019:1(1):8-11.
22. Sales NMR, Pelegrini PB, Goersch MC. Nutrigenomics: definitions and advances of this new science. Journal of Nutrition and Metabolism,2014:202759:1-6.
23. Schulze MB, Hu FB. Primary prevention of diabetes: What can be done and how much can be prevented? *Annu. Rev. Public Health*,2005:26:445-467. doi: 10.1146/annurev.publhealth.26.021304.144532.
24. Sharma P, Dwivedi S. Nutrigenomics and nutrigenetics: new insight in disease prevention and cure. *Indian J Clin Biochem*,2017:32(4):371-373.
25. Wedick NM, Pan A, Cassidy A, Rimm EB, Sampson L, Rosner B *et al*. Dietary flavonoid intakes and risk of type 2 diabetes in US men and women. *Am. J. Clin. Nutr*,2012:95:925-933. doi: 10.3945/ajcn.111.028894.
26. Zhang Z, Ding Y, Dai X, *et al*. Epigallocatechin-3-gallate protects pro-inflammatory cytokine induced injuries in insulin-producing cells through the mitochondrial pathway. Eur J Pharmacol,2011:670(1):311-316.