Review Article

Nutrigenomics: A Narrative Review of Diet's Influence on Periodontal Health

Ashneet Kaur[1], Anchal Sood[1], Swantika Chaudhry[1], Aditya Khichy[1], Rachita Arora[1]

[1] Department of Periodontology, Baba Jaswant Singh Dental College & Hospital, Ludhiana, Punjab, India

ABSTRACT

Nutrigenomics is a methodical study of the impact of nutrition input on the natural environment and genetic factors and their application to the health of the periodontium will offer valuable insights in the pathogenic processes of the disease. The relevance of the interface between nutrigenomics and prophylaxis/therapeutic approaches to periodontal disease is currently interrogated in the present review with respect to the relationship between periodontal status and the variety of nutrients and dietary compounds, the macronutrients, antioxidants, phytochemicals, and micronutrients. A review is also given on recent nutrigenomic discoveries that refer to gingival health. All of this evidence represents that genetic heterogeneity adjusts digestive efficiency and predisposition to periodontal pathology. The connection between nutrition and periodontal health is mused upon, and special consideration has been given to the effect that components of the diet have on the periodontal gene expression and inflammatory reactions. Despite the significant efforts undertaken, it is difficult to apply the nutrigenomic strategies in the practice of clinical work. Therefore, it is crucial that further research of the mechanisms peculiar to periodontal disease is continued to provide specific recommendations regarding diets, depending on the individual phenotype. In this way, this review underlines the promise of nutrigenomic technologies in supporting the field of periodontal care as well as in optimising the oral health of clinically heterogeneous populations.

Keywords: Disease prevention, Gene-diet interactions, Nutrigenomics, Periodontal health, Personalized nutrition.

BACKGROUND

Nutrigenomics is a field in the scientific nature that deals with how the choices of diet affect the way genes are expressed and hence the human wellbeing. Its arrival has been able to reshape the mainstream notions on the molecular processes by which nutrition has a moderating effect on overall good health. In the context of oral medicine, the discipline has attracted special prominence when it comes to periodontal disease, namely maintenance of the periodontal connective tissues [1]. The essence of periodontal disease is chronic inflammation of

the structures supporting the teeth; colonisation of microbes acts as one of the leading factors of pathogenesis. However, host genetic predispositions, immune reactions and lifestyle factors, especially nutritional status, influence the course of the disorder as well [2]. The nutritional habits can adjust the activity of these genes that control inflammatory pathway, immune homeostasis, and tissue repair, and the antioxidant. Therefore, food constituents can have negative or positive effects on the periodontal condition usually on the basis of their effects on gene expression [3]. Saturated fat-high sugar diet comprised of poor micronutrient intake seems to increase the expression of the inflammatory-associated genes leading to an acceleration of the tissue destruction process [5]. Nutrigenomic solutions, such as altering the composition of the diet in order to overcome the pro-inflammatory cascade induced, on the other hand, can potentially ameliorate the periodontal risk [6]. Salivary or gingival tissue molecular profiling of patients with periodontal disease has identified the existence of genetic variations which effect the efficacy of nutrient uptake and immune competence. These revelations are hoped to be used to come up with precision nutritional measures that would either supplement or replace traditional periodontal treatments [7]. The body of evidence has now confirmed that oral health is linked to the systemic physiology and periodontal disease can be managed using nutrition-based intervention approach. Nutrigenomic point of view hence renders innovative understanding in the associations between diet, microbial colonisation and inflammation processes thereby giving rise to personalised treatment methods [8]. A nutrigenomic regimen is not just a plan of specific eating habits but also behavioural lifestyle changes that would encourage a healthier lifestyle in general. Calcium, vitamin C, vitamin D and long-chain omega-3 fatty acids are the key nutritional determinants of periodontal resilience, though all presented a unique body of evidence of mechanisms underlying those effects explaining tissue integrity, collagen production, and immune response [11]. An example is vitamin C that acts as an antioxidant and also collagen precursor, hence helping in homeostasis of periodontal connective tissue [12]. Nevertheless, different sensitivity to periodontal breakdown may be obtained with genetic variability in the enzymatic pathway genes that determine nutrient bioavailability despite sufficient nutrition acquisition [13]. To conclude, nutrigenomics offers the significant plausibility that dietary influences overlap with genetic host factors in the process of defining the vulnerability to and course of periodontal disease. An intensive nutritional approach is better able to optimise these interactions, which could include preventative implications to precision biotherapeutics. Nutrigenomic testing reveals these genetic variations, enabling us to provide personalized dietary recommendations [14].

If we often consume sugary drinks, plaque builds up in our teeth and harmful germs begins to grow. This throws off the balance of bacteria in your mouth and increases the chance that we will suffer from periodontal diseases [16]. If we don't consume sufficient nutrients that your body requires, our immune system will weaken. This makes us more susceptible to illness and inflammation [17]. Rather than consuming processed carbs, we can maintain our gingiva's health by consuming more whole grains, fruit, vegetables, and lean proteins [18]. Scientists are finding ways to prevent and treat some periodontal diseases better if they understand how nutrition influences gingival health [19]. Physicians can utilize nutrigenomic concepts to treat periodontal disease and provide every patient with a diet that is established on their DNA. This ensures individuals

receive the right amount of minerals and vitamins to maintain healthy mouths [20].

Receiving tailored nutrition recommendations may make our gums more robust. Nutrigenomics can assist with conditions that involve the entire body. There is a relationship between gingival disease and inflammation. If we know what foods cause you to become inflamed, we may be able to reduce your risk of getting diabetes or heart disease. This emphasizes how important it is to consider your overall health. We can become healthy by modifying what we eat and taking care of our teeth. Therefore, this review is essential to know about the Diet's Influence on periodontal health.

REVIEW

Gum health requires proper nutrient intake in the diet to help immune competence, normal tissue homeostasis and lessening inflammation. In order to realize this, people need calcium, phosphorus, omega-3 fatty acids and both vitamins D and C [22]. Vitamin C contains strong anti-oxidant activity, reduces oxidative stress, increases collagen production and free radical scavenging; therefore, it can reduce gum inflammatory diseases and accelerate repair of periodontal ulcers. Vitamin D leads to the formation of bone minerals through its calcium absorption ability, in addition to having immunomodulatory effects that cushion inflammatory activities in the periodontum and maintains tissue balance [23]. Two omega-3 fatty acids, docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA) also contribute to maintaining the health of periodontium because they reduce proinflammatory processes. Thus, fatty fish, walnuts, and flaxseeds comprise fatty food items rich in these fatty acids, therefore making them desirable dietary products [24]. Calcium and phosphorus together strengthen the integrity of the teeth and the periodontal tissue as well as compact the alveolar bones, promote remineralisation and reduce bone resorption in the periodontal tissues [25]. Collectively, these nutrients result in the supporting of the overall tissue health and guarding against gum disease progressing. There are also salutary effects of supplementing with antioxidant vitamins and phytochemicals. Coenzyme Q10, vitamin E and C, and selenium have anti-oxidant activities and bind reactive oxygen species (ROS); carotenoids, flavonoids, and polyphenols are also ant-inflammatory in their effects on the periodontal tissue [26]. The increased use of probiotic and prebiotic compounds alters the microbial ecology of the oral cavity and involves a decrease in swelling due to the reinstatement of a balanced microbiome [27]. Nutrigenomics and other such constructs are summaries of biological processes involved in how nutrition affects gum health. Nutrient gene relations show that the elements of the diet affect the expression of genes either directly or indirectly through epigenetic variations, such as DNA modifications, adjustments to histones, and regulatory non-coding RNA; and therefore, the phenotypic effects vary between individuals [28]. Personalised nutrition programs are designed with such factors in mind, using them as a basis to influence diet on the basis of genomic information, lifestyle, and clinical condition, in order to best optimise health and reduce disease risk. It can be summed up that, balanced dietary pattern with high micronutrients and antioxidants and following necessary supplementation is crucial to gum health and prevention of disease. Daily eating of nutrition-rich meals and specific dietary interventions are consistent with biological processes to develop homeostasis, reduce inflammation, and preserve tissue health in the gum tissues [29].

DISCUSSION

The impact of nutrigenomics in the complex relationship between diet habits and periodontal health has been unveiled in modern research. Nutrigenomics sheds light on how diet may be used as an intervention to regulate the risk of periodontal disease by understanding the correlation between genes and diet [30]. Gentle knowledge of these two-way relationships is invaluable in the generation of evidence-based preventive and therapeutic regimens that can stimulate oral health. The eating habits are directly related to the development and functional integrity of periodontium; the wellbeing of the gums thus also determines nutritional action and systemic physiology, which is a two- way process [31]. This mutuality highlights how challenging it is to determine the implications of foods on gum status. Nutrigenomics studies suggest that some genes influence susceptibility to gum disease in different individuals and determine efficacy rates of the treatment procedures. Several mechanisms by which an anti-inflammatory effect can be produced via nutrition have been outlined. Discrete genes and regulatory elements respond to the bioactive compounds found in fruit and vegetable as well as spicy foods and adjust the level of gene expression resulting in a change in tissue structure, inflammatory condition, and oxidative stress resistance [32]. In addition, epigenetic changes such as DNA methylation and histone acetylation caused by nutritional exposure may initiate the occurrence of gum inflammation due to the altering of gene expressions. There is a lot of hope in the possibility of personalised nutritional advice through genotype. The combination of genetic variation understanding, metabolic and lifestyle-related data with clinical practice provides clinicians with the capability to prescribe preventive and therapeutic regimens in order to minimize the occurrence of periodontal pathology [33]. Some of the strategies could be specific micronutrient supplementation, alterations of dietary bioactive molecules, minimization of pro-oxidative and pro-inflammatory dietary constituents, and lifestyle intervention to dampen the gum inflammation. However, there are several methodological issues that need to be resolved prior to large scale implementation in the clinical setting. The nature of gene x diet interactions is highly mappable; strong relationships that exist between particular genetic variants, dietary intake profiles, and periodontal outcomes necessitate large size data sets [34]. Thus, standardisation of gene sequencing procedures, dietary evaluation technique and measures of periodontal health are critical to the validity of future research. It is highly important to jointly collaborate among geneticists, nutritional scientists, bioinformaticians, and practitioners in health care are essential in order to convert nutrition-genomic knowledge into clinical practice guidelines. More so, ethical issues-informed consent and the safeguards of genetic testing to preserve privacy of data will be of utmost consideration [35]. The best way in which causal association between the contents of gene variants, exposure to diet and periodontal status can be made is the longitudinal cohort designs. Application of genomics, epigenomics, transcriptomics, and metabolomics will improve the molecular understanding of the diet periodontal balance, the pathway of the tissue remodelling, immune regulation, and inflammatory dysregulation. The recent developments have resulted in nutrigenetic markers where genetic polymorphism has been related to periodontal susceptibility and metabolism. The efficiency of dietary antioxidants and presence of gum diseases have been linked to differences in the genes, which code the antioxidant enzymes like superoxide dismutase and glutathione peroxidase [36]. A series of complementary genome-wide association studies have identified new loci linked with dietary metabolism and periodontal outcomes and have therefore provided a platform upon which future personalised dietary interventions may be based. Selective mutations in cytokine signalling and antioxidant metabolism can regulate the periodontal inflammatory reaction. These inflammatory phenotypes can be decreased with dietary regimens high in omega-3 fatty acids, polyphenols, and other antioxidants highlighting the need to prescribe diets on an individualised basis [37]. Regenerative processes, such as folate metabolism and vitamin D receptor activity, are also key regulators of inflammatory homeostasis as well as nutrient absorption; SNPs in both genes may both dull antioxidant defences and inhibit inflammatory resolution. To sum up, because of supplying personalised dietary recommendations, nutrigenomic studies stand to reform gum-disease prevention and treatment. The next step will be validation and optimisation of the methodological standards as well as systematic cohort studies and molecular omics analyses necessary to bring current insights into the routine clinical practice [38].

CONCLUSION

Diet and genes affect gene expression and periodontal inflammation, personalizing treatment. Nutrigenomics shows how nutrition and genetics affect gingival health. Nutritional genomics is needed for periodontal treatment. This knowledge is needed for prevention. Doctors can utilize genetic data to prevent periodontal disease, change diets, and detect risk factors. Nutrigenomics may cure gingival disease. Nutrigenomics research needs more funding from academics, healthcare professionals, and legislators in coming future.

REFERENCES

1. Taylor H et al. Journal of dental research ; 2020, 100:253-260. [DOI:10.1177/0022034520965958]
2. Sedghi LM et al. Frontiers in Cellular and Infection Microbiology; 2021, 11:766944. [DOI:10.3389/fcimb.2021.766944].
3. Bui F et al. Biomedical Journal, 2019, 42:27-35. [DOI:10.1016/j.bj.2018.12.001]
4. Bulgin D. Journal of Oral Hygiene and Health, 2014, 2:1-1. [DOI:10.4172/2332-0702.1000E109]
5. Hajishengallis G. Nature Reviews Immunology, 2014, 15:30-44. [DOI:10.1038/nri3785]
6. Jeffcoat M et al. American Journal of Preventive Medicine, 2014, 47(2):166-174. [DOI:10.1016/j.amepre.2014.04.001]
7. Orlandi M et al. Journal of Clinical Periodontology, 2021. [DOI:10.1111/jcpe.13554]
8. Arigbede A et al. Journal of the Indian Society of Periodontology, 2012, 16:487-491. [DOI:10.4103/0972-124X.106878].
9. Beck J et al. Journal of Dental Research, 2019, 98:1053-1062. [DOI:10.1177/0022034519846113].
10. Meghil M et al. Oral Diseases, 2019, 25(5):1403-1413. [DOI:10.1111/odi.1397].
11. Kapila Y. Periodontol 2000, 2021, 87:11-16. [DOI:10.1111/prd.12398].
12. Montebugnoli L et al. Journal of Clinical Periodontology, 2005, 32(2):188-192. [DOI:10.1111/J.1600-051X.2005.00641.X]
13. Chapple et al. Journal of Nutrition, 2007, 137(3):657-664. [DOI:/10.1093/JN/137.3.657]
14. Liccardo D et al. International Journal of Molecular Sciences, 2019, 20. [DOI:10.3390/ijms20061414].
15. Iacopino A & Cutler C. Journal of Periodontology, 2000, 71(8):1375-1384. [DOI:10.1902/JOP.2000.71.8.1375]
16. Iwasaki M et al. Nutrition, 2010, 26(11-12):1105-1109. [DOI:10.1016/j.nut.2009.09.010]

17. Lu E. Journal of Periodontal Research, 2022 [DOI:10.1111/jre.13083].
18. Dommisch H et al. Periodontol 2000, 2018, 78(1):129-153. [DOI:10.1111/prd.12233]
19. Brock G & Chapple I. Current Oral Health Reports, 2016, 3:337-346. [DOI:10.1007/s40496-016-0116-9]
20. Nishida M et al. Journal of Periodontology, 2000, 71(8):1215-1223. [DOI:10.1902/JOP.2000.71.8.1215].
21. Isola G. Issues in Developmental Health Research, 2021, 4. [DOI:10.9734/bpi/idhr/v4/11160d].
22. Bhargava A et al. Journal of Oral Biology and Craniofacial Research, 2019, 9(2):177-179. [DOI:10.1016/J.JOBCR.2018.07.001].
23. Mewes L et al. Nutrients, 2022, 14. [DOI:10.3390/nu14091939].
24. Najeeb S et al. Nutrients, 2016, 8. [DOI:10.3390/nu8090530].
25. Geethika M & Chava V. Annals of Essential Dentistry, 2016, 8:1-7.
26. Janciauskiene S et al. Chronic Obstr Pulm Dis. 2020 Jul;7(3):182-202. [PMID: 32558487]
27. Yu X et al. J Oral Microbiol. 2024 Jan 31;16(1):2307416. [PMID: 38304119]
28. Elsamanoudy AZ et al. J Microsc Ultrastruct. 2016 Jul-Sep;4(3):115-122. [PMID: 30023217]
29. Singar S et al. Nutrients. 2024 Aug 13;16(16):2673. [DOI: 10.3390/nu16162673]
30. Dang TS et al. Periodontol 2000. 2014 Feb;64(1):154-60. [PMID: 24320962]
31. Chamut S et al. Int J Equity Health. 2024 Dec 2;23(1):261. [PMID: 39623427]
32. Spahr A et al. Periodontol 2000. 2022 Oct;90(1):146-175. [PMID: 35916868]
33. Handy DE et al. Circulation. 2011 May 17;123(19):2145-56. [PMID: 21576679]
34. Martinon P, Fraticelli L, Giboreau A, Dussart C, Bourgeois D, Carrouel F. Nutrition as a Key Modifiable Factor for Periodontitis and Main Chronic Diseases. J Clin Med. 2021 Jan 7;10(2):197. [PMID: 33430519]
35. Espinosa-Salas S et al. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. [Available from: https://www.ncbi.nlm.nih.gov/books/NBK597352/]
36. Cho YD et al. Clin Epigenetics. 2021 Apr 26;13(1):92. [PMID: 33902683]
37. Nogawa S et al. BMC Oral Health. 2024 Aug 27;24(1):1003. [PMID: 39192244]
38. Voltan G et al. Genes (Basel). 2023 Aug 25;14(9):1691. [PMID: 37761831]

Ethical Approval: Institutional Review Board approval was not required.

Declaration of Patient Consent: Patient consent was not required as there are no patients in this study.

Use of Artificial Intelligence (AI) - Assisted Technology for Manuscript Preparation: The authors confirm that no artificial intelligence (AI)- assisted technology was used to assist in the writing or editing of the manuscript, and no images were manipulated using AI tools.

AUTHOR CONTRIBUTIONS:

Ashneet Kaur: Contributed to the conceptualization, writing of the original draft, and data analysis.

Anchal Sood: Led the study design and methodology. Supervised the overall research process and critically reviewed the manuscript.

Swantika Chaudhry: Contributed to data collection, analysis, and interpretation. She also provided substantial revisions to the manuscript.

Aditya Khichy: Assisted in writing and reviewing the manuscript, providing insights into the interpretation of the findings.

Rachita Arora: Contributed to the writing and reviewing process. Provided critical revisions and helped in the final drafting of the manuscript.

ABBREVIATIONS USED IN THE STUDY:

a) **DHA**: Docosahexaenoic acid
b) **EPA**: Eicosapentaenoic acid
c) **ROS**: Reactive oxygen species
d) **SNP**: Single nucleotide polymorphism
e) **DNA**: Deoxyribonucleic acid
f) **RNA**: Ribonucleic acid
g) **CoQ10**: Coenzyme Q10

DECLARATION ON PUBLICATION ETHICS:

The authors declare that they adhere to the COPE guidelines on publishing ethics, as described elsewhere at https://publicationethics.org/. The authors also undertake that they are not associated with any other third party (governmental or non-governmental agencies) linking with any form of unethical issues related to this publication. The authors also declare that they are not withholding any information that is misleading to the publisher regarding this article.

DECLARATION ON OFFICIAL E-MAIL:

The corresponding author declares that a lifetime official e-mail from their institution is not available for all authors.

COMMENTS FROM READERS:

Articles published in the ORAL SPHERE JOURNAL OF DENTAL AND HEALTH SCIENCES are open for relevant post-publication comments and criticisms, which will be published immediately, linking to the original article without open access charges. Comments should be concise, coherent, and critical in fewer than 1000 words.

DISCLAIMER: