REVIEW ARTICLE

Nutrition and Periodontium

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Abstract

Aim: With few literature providing a detailed knowledge of the role of nutrition on periodontal tissue, we aimed to provide an insight for the dentists on how nutrition impacts general and oral health and how dental treatment can impact the nutritional status of the patient.

Background: For human growth, development, and preservation of health, nutrition is crucial. The relation of oral and general health has been well known and well-studied in the past years.

Review results: Also, this review article explains how nutrition is linked to biofilm, host nutrition and immunity in periodontal disease, effects of nutrition on the immune response, role of nutrients in the wound-healing process. We have also explained the necessary periodontal screening parameters, the role of each nutritional aspect that affects periodontal tissues along with an emphasis on the newer concepts.

Conclusion: Nutrition plays an integral aspect that influences oral as well as periodontal health directly or indirectly.

Clinical significance: An in-depth knowledge of the influence of nutrition on periodontal aspect is a must for all dental professionals for a healthy clinical practice.

Keywords: Antioxidant, Diseases, Nutrition, Periodontal, Systemic health, Vitamin.

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Introduction

Nutrition is vital to human development, growth, and health maintenance. The present wellness and health promotion landscape includes nutritional concerns, which are prominent in popular culture. People are more concerned than ever today with maximizing their health through the acquisition of nutritional knowledge and its application to their everyday life. In the past, a dentist's nutritional advice could only consist of telling patients to eat less sweets and to snack less frequently; today, nutritional counseling entails much more. Dentists need to understand how diet affects oral and overall health as well as how dental procedures might affect a patient's nutritional state.

Dental practitioners must evaluate their patients' general health in addition to comprehending the consequences of underlying conditions that may affect oral health in order to provide comprehensive dental treatment. A fundamental element in periodontal disease is the interplay between the immune system's reaction to the bacterial onslaught and nutritional condition. Diseases like periodontal disease are more likely to occur and spread due to changes in the immune system. Environmental and host variables influence the severity of periodontal disorders, even if bacteria are the major cause of the illness.

An improved healing response after periodontal treatment may usually be achieved by modifying or modulating most of the systemic and local variables.[1] Yet, despite reports of potential consequences of nutrient deficiency and supplementation having surfaced early in periodontal literature, other potentially modifiable systemic factors, such as a balanced diet or nutrient supplementation, have not been fully examined in periodontal research.[2,3] Therefore, the aim of this paper is to review the available literature addressing the relationship between nutrition and periodontal disease onset, progression, and response to treatment.

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Review

Inflammation of the gingiva, loss of soft tissue attachment to tooth, and resorption of alveolar bone are all symptoms of periodontal disease, which eventually result in tooth loss. Though there is strong scientific evidence that anaerobic oral bacteria play a role in the pathogenesis of periodontitis and that tissue damage results from the intricate interaction between pathogenic bacteria and the host's immune response to infection, both systemic and local factors play a part.[4,5] Maintaining periodontal health is recognized to depend on nutrition, and a variety of dietary components have been proposed as potential aggravating factors.

To guarantee that periodontal therapy has a positive outcome, it is necessary to remove the complex community known as plaque biofilm, which is home to many of the microorganisms linked to the evolution of periodontal disease. The significance of the presence of microbial plaque in the etiology and pathogenesis of periodontal disease makes it a unique factor to associate it with periodontal health and disease.

Host Nutrition and Plaque Biofilm

Dietary factors influence the formation and makeup of plaque biofilm in both direct and indirect ways.

- The localized reduction of plaque development by an abrasive diet.[6,7]
- The direct provision of certain nutrients (such sucrose) to the bacteria as sources of carbon, nitrogen, or energy.[8]
- Affected organism's ability to produce metabolic byproducts that supply other organisms with nourishment.[8]
- Utilized by other bacteria as a means of producing certain polymers.[8]

Lastly, nutrition affects the plaque biofilm indirectly by changing the biofilm's environment, which in turn affects the bacteria that colonize it. This is done through the byproducts of bacterial metabolism of a nutrient.[8] Bacteria generate acids as byproducts of breaking down sucrose and glucose, which lowers pH and creates an environment more conducive to the growth of certain bacteria, including *Streptococcus mutans*.[8] After the biofilm formation reaches a stable state, it is believed that the host's nutrition has less of an impact on the plaque biofilm's maturation process.[9]

Host Nutrition and Immunity in Periodontal Disease

Nutrition is "critical determinant of immune responses"[9] due to fact that "nutrients derived from food sources, such as proteins, carbohydrates, and fats as well as micronutrients, vitamins, and minerals interact with immune cells in the blood stream, lymph nodes, and specialized immune system of the gastrointestinal tract."[10,11] Nutritional status is negatively impacted by infections, regardless of severity.[12] Numerous variables influence how these nutrients work:

- A nutrient's concentration and how it interacts with other important nutrients.
- Length of time the nutritional imbalance persists.
- Host's age.[10]

Most vitamin deficiencies, on the other hand, will weaken the immune system and make the person more vulnerable to illness.[12]

Not much is known about direct effect of nutrition on immune response in periodontal disease, despite the fact that there is a large body of literature about influence of diet on systemic immunity. Most of what is understood, therefore, regarding the relationship between diet and systemic immunity probably also applies to the problems associated with periodontal disease.

The host immune system reacts to a bacterial challenge in periodontal disease in a well-regulated manner. This response consists of three main components:

- Innate factors that trigger an inflammatory response by signaling the endothelium.
- Neutrophils that try to control pathogens in an acute inflammatory response to protect the periodontal tissues.
- Subsequent chronic inflammatory response is triggered by infection and is characterized by macrophages and lymphocytes trying to manage the local infection to keep it from spreading and becoming catastrophic.[13]

Vitamin A, C, D, E, B1, B2, B3, B5, B6, B7, B9, B12, calcium, phosphorus, magnesium, sodium, potassium, iron, fluoride, zinc and water have impact on periodontium in the case of deficiency.[14]

Dietary Influences on the Immune System (Table 1)

Nutritional deficits may modify immune response and raise infection risk, according to epidemiologic and clinical research. However, illness and numerous dietary deficits have hampered the majority of clinical investigations on the effect of nutrition on human immune system. Thus, information gathered from clinical and animal research has helped to establish a consensus regarding the impact of individual nutrients on immune system. Table 1 lists the effects of deficiency in key nutrients on immunological response.[9]

Nutrition's Function in the Healing of Wounds (Table 2)

Multilevel integration exists between the biology and physiology of wound healing. In order to restore the damaged organs, resources must be gathered and homeostasis must be maintained (Table 2).[15]

Table 1: Summary of impact of specific nutrient deficiencies on immune response

Nutrient	*Function*	*Deficiency impact on immune response (decreased)*
Protein energy intake	Energy metabolism	Salivary antimicrobial properties
	DNA/RNA synthesis	Immunoglobulin production lysozymes
		Bacterial adhesion
		Activation of lymphocytes
		Production of antibodies
Vitamin A	Cellular differentiation and proliferation	Immune cell differentiation
	integrity of the immune system	Response to antigens
		Antibody production
		Bacterial adhesion
		Immunoglobulin production
		Production of lymphocytes
Vitamin E	Antioxidant protecting lipid membranes from oxidation	Antibody synthesis
		Response of lymphocytes
		Phagocytic function
		Phagocytic function of neutrophils and macrophages
Vitamin C	Antioxidant that reduces free radicals that cause	Antibody response
	DNA damage to immune cells	Cytotoxic T-cell activity

(Contd...)

Table 1: *(Contd...)*

Nutrient	*Function*	*Deficiency impact on immune response (decreased)*
Riboflavin, vitamin B6, and pantothenic acid	Coenzymes in metabolic processes	Antibody synthesis Cytotoxic T-cell activity Lymphocyte response
Folic acid and vitamin B12	Involved in DNA/RNA synthesis	Production of lymphocytes Cytotoxic T-cell activity Phagocytic function of neutrophils
Zinc	More than 100 enzymes associated with carbohydrate and energy metabolism Protein catabolism and synthesis Nucleic acid synthesis	Antibody response Phagocytic function of macrophages B-cell and T-cell proliferation
Iron	Involved in hemoglobin, myoglobin, and cytochrome systems	Lymphocyte proliferation Neutrophil cytotoxic activity Antibody response

Table 2: Role of nutrients in the wound healing process

Nutrient	*Role/function*
Protein	Cell multiplication; enzyme synthesis; collagen and other connective tissue synthesis
Carbohydrate	Source of energy; part of ground substance
Fatty acids	Part of cell membrane; prostaglandin synthesis
Vitamin C	Cofactor in hydroxylation of proline and lysine; collagen synthesis
Vitamin A	Epithelial maintenance; glycoprotein and proteoglycan synthesis; stimulation of cellular differentiation in fibroblast and collagen formation
Vitamin D	Calcium homeostasis; calcium and phosphate absorption
Vitamin E	Maintenance of cell membrane
Calcium	Required by tissue collagenases; bone and tooth formation
Zinc	Transcription of RNA; cellular proliferation; protein synthesis
Iron	Involved in hydroxylation of proline and lysine
Copper	Component of lysyl oxidase; erythropoiesis

Risk Screening for Nutrition (Table 3)

Based on each person's needs, nutrition-risk screening in the dental environment can lead to the following outcomes:

- A basic diet assessment and education from dental professional to address nutritional and oral health concerns.
- Prescriptions for oral supplements (name, quantity, and frequency).
- Referrals to community resources for food and supplements through social services.
- Referrals to trained dieticians for medical nutrition treatment and nutrition consultations.

At the very least, nutrition screening comprises objective evaluation of anthropometric measures and oral cavity health in addition to subjective statements on food, dental health, and weight history. The type of dentistry practice, the patient's general health, and their medical history all have a role in how much laboratory data and other components are employed. Individuals who take several drugs and have complicated medical histories need a more thorough physical and laboratory evaluation (Table 3).[16]

While research on animals has demonstrated a connection between food and plaque development,[7] the relevance of this finding to humans is debatable due to variations in tooth structure. Furthermore, it is unknown if food influences the development of gingivitis and periodontitis in addition to the quantity of plaque that is present. The link between inadequate nutritional status and periodontal disease—wherein harmful metabolic alterations linked to a reduced nutritional status enhance susceptibility to periodontal problems—is a more significant factor in diet's impact on periodontal health. The importance of antioxidants, calcium, vitamin D, and vitamin C in various chronic phases of illness has received the most attention.

Vitamin C and Scurvy

One of the nutrients with the most research on its relationship to periodontal disease is vitamin C (ascorbic acid). A severe vitamin C shortage is known to cause scorbutic gingivitis, also known as scurvy, which is characterized by ulcerative gingivitis and a fast development of periodontal pockets with tooth exfoliation.[17] Histological investigations have revealed that with vitamin C shortage, there is a decrease in collagen production and an increase in endotoxin permeability from the oral mucosa.[18] Research has shown that vitamin C improves polymorphonuclear leukocytes' (PMNL) motility; thus, a deficit reduces the host's immunological responses.[19] After taking vitamin C supplements, there was a noticeable decrease in bleeding upon probing, which may indicate that changes in gingival indices are connected to vascular alterations or early inflammatory pathways.[20]

There is much debate regarding the optimal daily intake of vitamin C for good health, and conflicting findings have been found

Table 3: Nutrition risk factors to consider in physical examination

Body area	*Nutrition risk factors*	*Nutritional implications*
Hair	Dull, shedding, easily pluckable	Generalized protein calorie malnutrition
Face	Malar pigmentation (dark skin over cheeks and under eyes), bitemporal wasting, nasolabial seborrhea, edematous, moon face, lack of color	Niacin, B vitamins Malnutrition Niacin, riboflavin, B6 Protein deficiency Corticosteroid impact Inadequate Fe++, undernutrition
Eyes	Pale eye membranes	Inadequate Fe++
Lips	Cheilosis (red/swelling), angular fissures	Inadequate niacin, riboflavin, inadequate niacin, B6, riboflavin, Fe++
Gingivae	Spongy, bleeding, abnormal redness	Inadequate vitamin C
Tongue	Glossitis (red, raw, fissured), pale, atrophic, smooth/slick (filiform papillary atrophy), magenta color	Inadequate folate, niacin, riboflavin,Fe++, B6, B12 Inadequate Fe++, B12, niacin, folate Inadequate riboflavin
Nails	Spoon shaped, brittle, ridged	Inadequate Fe++
Back	Pony prominences along shoulder girdle	Malnutrition
Muscles	Tendons prominent to palpation	Malnutrition

in epidemiological research examining the link between periodontal disease and vitamin C. It is plausible that the quantities of vitamin C needed to prevent or treat scurvy may differ significantly from those required to get the maximum benefits of vitamin C. When clinical attachment was used to quantify periodontal disease in smokers, there was a statistically significant correlation between vitamin C consumption and the condition. Those who smoke and consume the least amount of vitamin C are likely to have the most clinically significant effects on the periodontal tissues, according to the findings. Thus, it was discovered that those with lower vitamin C intakes had a little but substantial dose–response increase in risk for periodontal disease.[17] Altered vitamin C impaired PMNL chemotaxis and is associated with periodontitis and necrotizing ulcerative gingivitis.[12]

Not much research has been done on how large dosages of vitamin C affect those who are not low in it. There is evidence linking low vitamin C levels to poor wound healing, and it has been proposed that vitamin C supplementation may be beneficial for gingival tissues healing. A single intravenous injection of 500 mg of ascorbic acid produced statistically significant relationships between gingival state and ascorbic acid levels in whole blood and urine in healthy young adult men categorized based on their periodontal condition.[21] On the other hand, a study by Woolfe et al.[22] assessed how vitamin C intake is related to gingival clinical parameters. Normal human subjects receiving 1 gram of vitamin C daily for 6 weeks did not see any change in the gingival reaction to the first therapy, and patients receiving vitamin C supplements and control groups showed the same gingival responses to periodontal therapy. The fact that the final blood levels of vitamin C seemed to have slightly risen indicates that excess vitamin C was eliminated by urination. The best current research suggests that there is no benefit to the periodontal patient from using vitamin C supplements, and that a healthy, balanced diet may easily meet the dietary reference intake (DRI).

Dietary Calcium and Periodontal Disease

Long regarded as a potential moderator of periodontal disease, dietary calcium deficiency has been linked to modifications in collagen synthesis and structure in the connective tissue of the mouth.[23] Research on humans and animals has shown a connection between tooth loss, bone mineral density (BMD), and calcium intake.[17] In animal studies, osteopenia or the resorption of alveolar bone has been linked to calcium deficiency.[23–25] Although the precise relationship between calcium intake and periodontal disease has not been determined, it is plausible that it influences alveolar BMD. The relationship between dietary calcium and periodontal disease has not been conclusively shown in human studies. Lower dietary calcium intake was found to be statistically significantly associated with periodontal disease in younger males and females as well as older males in a study by Nishida et al.[17] that included over 12,000 adults. Among the females, the lowest dietary calcium intake group was associated with a 54% increased risk of periodontal disease.

Other Nutrients and Periodontal Disease

Intake of calcium and vitamin D, kept at US DRI levels (to avoid osteoporosis), was found to have a favorable effect on tooth loss in a recent research of older persons.[26] The authors draw the conclusion that further research is necessary to validate these results in an intervention study, as the amount of teeth lost was dependent upon self-reports. While a large body of research has examined the effects of vitamin D insufficiency in humans, very few studies have examined calcium deficit in isolation. In a single study, young rats were given diets low in calcium, low in calcium with vitamin D, or high in vitamin D.[27] As long as there was a sufficient amount of calcium available, the rats were shown to be immune to vitamin D deprivation, in contrast to the preceding two diets which had notable impacts on the periodontal tissues. Periodontal disease was shown to be correlated with dietary protein, vitamin A, and calcium in one of the few studies that assessed the intake of a broad variety of nutrients.[28]

Antioxidant Nutrients and Periodontal Disease

Proteinases, oxidants, and other harmful substances are released by neutrophils in reaction to periodontal infections.[29] Antioxidants

Box 1: Following laboratory investigations can be carried out when necessary

• Hemoglobin	• Serum potassium
• Packed cell volume	• Serum urea
• Erythrocyte sedimentation rate	• Nitrogen
• Red blood cell count	• Creatinine
• White blood cell count	• Total protein
• Differential count	• Albumin
• Mean corpuscular Hb	• Cholesterol
• Mean corpuscular volume	• Blood glucose
• Mean corpuscular Hb concentration	• Fasting blood glucose
• Serum sodium	• Serum calcium

have the ability to prevent tissue damage and are found in all bodily fluids and tissues. Several components produced from nutrition, such as uric acid, non-protein thiols, and glutathione, as well as extracellular fluid-derived elements including ascorbic acid (vitamin C), tocopherol (vitamin E), and carotene are among them.[30]

It has been proposed that a better knowledge of the impact of food and nutrition exist on antioxidant status during periodontal health and disease that might result in the development of a potential nutritional therapy plan for periodontal disease.[30] Nonetheless, there is no proof to back up taking massive amounts of antioxidant vitamin supplements; eating a nutritious, balanced diet can provide an appropriate intake of these vitamins.

Tests to Assess Nutritional Risk in Laboratory

An understanding of the potential causes of systemic and oral diseases can be gained by diagnostic testing, such as hematologic assessments. For evaluating immunological reaction, anemia due to iron deficiency and others, the complete blood count offers vital information. Organ function, glucose, lipids, serum proteins, electrolytes, and trace elements are all represented by values in the laboratory data as shown in Box 1.

Medication Methods to Improve Immune System and Attire Infection

In order to maintain the immune response's optimal functioning, nutrition is crucial. Undernourished people have compromised immunological responses, including anomalies in phagocytosis, antibody function, and adaptive immunity among other immune responses.[9] According to research on animals, giving certain nutrients—like protein—at appropriate quantities is linked to increased immune function and decreased mortality following an infectious challenge.[31] According to these results, dietary interventions that might lower the risk of opportunistic infections in people with weakened immune systems should be started.[31]

Increased dietary intake of fiber, fish, fish oil, vegetables, fruits, and berries is advised with restriction on refined sugar, saturated fat, caloric intake, salt no more than 6 g daily.[12,32–34]

Newer Concepts

Phytochemicals

In addition to the usual nutrients known to be involved in normal metabolism, plant foods contain many other natural, nonnutritive compounds, some of which have newly recognized health effects. These compounds are called phytochemicals. There are hundreds or even thousands of phytochemicals found in fruits, vegetables, and some herbs. Phytochemicals are biologically active substances in plants that give plants their color, odor, flavor, and defense systems. The majority of active phytochemicals in grain products are in the bran and germ, so whole grains should be consumed. The protection against various systemic diseases like cancer, heart diseases, diabetes, hypertension and others have been provided by diet rich in fruits, whole grains and vegetables.

Functional Foods

A term generically presented for any type of food as it is or modified or with any food content that provide health benefits beyond traditional food. They are referred as designer or pharma food or nutraceuticals. For example, broccoli, carrots, and tomatoes are rich in certain components that are physiologically active agents, such as lycopene, sulforaphane, beta-carotene, respectively. Herbals and foods that have been modified or fortified with additional nutrients, phytochemicals, would also be considered functional foods. Functional foods may be categorized as:

- Whole foods that have been associated with reduced risk of disease.
- Food components for which there is evidence of a diet–disease relationship.
- To prevent or treat a clinical condition, a specific nutrient level is increased to fortify the food.

A *probiotic* is defined as "a live microbial food ingredient that is beneficial to health." For example, lactobacilli and bifidobacteria. At present, probiotics are almost exclusively consumed as fermented dairy products such as yogurt and ice creams. A *prebiotic* is defined as "a nondigestible food ingredient that beneficially affects the host by selectively stimulating the growth and/or activity of one or a limited number of bacteria in the colon". For example, inulin-type fructans, which include native inulin, enzymatically hydrolyzed inulin, or oligofructose, and synthetic fructooligosaccharides.[35]

A scientific data showed that probiotics and prebiotics may positively affect the various physiologic functions in ways that will permit them now or in the future to be classified as functional foods for which health claims (of enhanced function or of reduction in disease risk) will be authorized.[35]

Nutritional Genomics: The New Frontier[36]

Nutritional genetics, also known as nutrigenetics, is the area of research focused on the effects of individual gene variations on individual nutritional needs. Nutritional genomics research is focusing upon identification of gene diet interactions, determination of underlying mechanisms and validation of developed tools. The following concepts provide the basis for nutritional genomic research:[37]

- For certain diseases, diet can be a rich risk factor for certain individuals and circumstances. For example, gluten intolerance, lactose intolerance.
- Common dietary chemicals can act on the human genome (directly or indirectly) to alter gene expression or structure.
- The degree to which diet influences the balance between healthy and disease states may depend upon a person's genetic

make-up. For example, polymorphism in the gene for the hormone leptin which results in gross obesity.
- The chronic diseases are influenced by certain dietary genes which would influence its incidence, onset, progression and severity. For example, for foliate metabolism, genetic polymorphism that encodes methylenetetrahydrofolate reductase reductase has been identified.
- Chronic diseases are attempted to prevent and cure based on dietary intervention by considering the knowledge about nutritional status and requirement.
- Multiple elevation of glucose was found to have led to increased inflammatory pathologies and along with altered lipid levels they generated increased reactive oxygen species and its results triggering NF-kB activation.[34]

Role of Trace Elements on Periodontal Health

Few trace elements such as boron, cobalt, copper, chromium, manganese, molybdenum, nickel, and selenium are known to influence systemic health of an individual. One of such elements that have been documented to influence periodontal health is fluoride.

The trace element fluoride is naturally found in many of food components. Fluoride role in periodontal health and disease has been extensively studied in two review articles by Vandana KL et al.[38,39] Effect of other trace elements on periodontal health is not well defined and is yet to be studied.

Conclusion

The periodontal disease initiation and progression depends on optimum nutritional status. However, the disease stimulation by dental plaque is more effective than nutritional status. For better periodontal treatment outcomes, previous history of infection, nutritional status and dietary habits play a vital role. Prior to any extensive dental treatment, nutritional status assessment and poor dietary habits will be benefited by nutritional rehabilitation. A balanced diet is necessary to maintain optimal oral and periodontal health. Further researches are required to modulate periodontal disease in terms of dietary modification and/or supplementation.

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References

1. Caton JG, Lowenguth RA. Classification and diagnosis of periodontal diseases. J Calif Dent Assoc 1993;21(11):23–34. PMID: 8598519.
2. Glickman N, Mitchell HH. The total specific dynamic action of high-protein and high-carbohydrate diets on human subjects. J Nutr 1948;36(1):41–57. DOI: 10.1093/jn/36.1.41.
3. Lovdal A, Arno A, Waerhaug J. Incidence of clinical manifestations of periodontal disease in light of oral hygiene and calculus formation. J Am Dent Assoc 1958;56(1):21–33. DOI: 10.14219/jada.archive.1958.0021.
4. Genco RJ. Current view of risk factors for periodontal disease. J Periodontol 1996;67(10 Suppl):1041–1049. DOI: 10.1902/jop.1996.67.10.1041.
5. Kornman KS, Löe H. The role of local factors in the etiology of periodontal diseases. Periodontology 1993;2:83–97. DOI: 10.1111/j.1600-0757.1993.tb00222.x.
6. Jensen L, Logan E, Finney O, et al. Reduction in accumulation of plaque, stain and calculus in dogs by dietary means. J Vet Dent 1995;12(4):161–163. PMID: 9693645.
7. Logan EI, Finney O, Hefferren JJ. Effects of a dental food on plaque accumulation and gingival health in dogs. J Vet Dent 2002;19(1):15–18. DOI: 10.1177/089875640201900102.
8. Bowden GH, Li YH. Nutritional influences on biofilm development. Adv Dent Res 1997;11(1):81–99. DOI: 10.1177/08959374970110012101.
9. Chandra RK. Nutrition and the immune system: An introduction. Am J ClinNutr 1997;66:440S–443S. DOI: 10.1093/ajcn/66.2.460S.
10. Cunningham-Rundles S. Analytical methods for evaluation of immune response in nutrient intervention. Nutr Rev 1998;56:S27–37. DOI: 10.1111/j.1753–4887.1998.tb01641.x.
11. Raindi D. Nutrition and Periodontal Disease. Dent Update 2016;43(1):66–68, 71–72. DOI: 10.12968/denu.2016.43.1.66.
12. Scrimshaw NS, SanGiovanni JP. Synergism of nutrition, infection, and immunity: An overview. Am J ClinNutr 1997;66:464S–477S. DOI: 10.1093/ajcn/66.2.464S.
13. Haake SK, Nisengard RJ, Newman MG, et al. Microbial interactions with the host in periodontal diseases. In: Newman MG, Takei HH, Carranza FA, editors. Carranza's clinical periodontology. 9th ed. Philadelphia: W.B. Saunders; 2002. pp. 132–152.
14. Karmakar, Shaswata. Nutrition and Periodontium: A connecting link. IntJ Curr Res 2019;11(4):2913–2917. DOI: 10.24941/ijcr.35050.04.2019.
15. Nutrition and Oral Medicine. Edited by: Touger-Decker R, Sirois DA, and Mobley CC, Totowa, NJ: Humana Press Inc.
16. Palmer CA. Diet and Nutrition in Oral Health. 2nd ed. New Jersey: Prentice hall.
17. Nishida M, Grossi SG, Dunford RG, et al. Dietary vitamin C and the risk for periodontal disease. J Periodontol 2000;71(8):1215–1223. DOI: 10.1902/jop.2000.71.8.1215.
18. Alfano MC, Miller SA, Drummond JF. Effect of ascorbic acid deficiency on the permeability and collagen biosynthesis of oral mucosal epithelium. Ann NY Acad Sci 1975;258:253–263. DOI: 10.1111/j.1749-6632.1975.tb29286.x.
19. Goetzl EJ, Wasserman SI, Gigli I, et al. Enhancement of random migration and chemotactic response of human leukocytes by ascorbic acid. J Clin Invest 1974;53:813–818. DOI: 10.1172/JCI107620.
20. Leggott PJ, Robertson PB, Rothman DL, et al. The effect of controlled ascorbic acid depletion and supplementation on periodontal health. J Periodontol 1986;57(8):480–485. DOI: 10.1902/jop.1986.57.8.480.
21. Shannon IL, Gibson WA. Intravenous ascorbic acid loading in subjects classified as to periodontal status. J Dent Res 1965;44:355–361.104. DOI: 10.1177/00220345650440020801.
22. Woolfe SN, Kenney EB, Hume WR, et al. Relationship of ascorbic acid levels of blood and gingival tissue with response to periodontal therapy. J Clin Periodontol 1984;11:159–165. DOI: 10.1111/j.1600-051x.1984.tb01319.x.
23. Oliver WM, Leaver AG, Scott PG. The effect of deficiencies of calcium or of calcium and vitamin D on the rate of oral collagen synthesis in the rat. J Period Res 1972;7:29–34. DOI: 10.1111/j.1600-0765.1972.tb00628.x.
24. Henrikson PA. Periodontal disease and calcium deficiency: An experimental study in the dog. Acta Odontol Scand 1968; 26(Suppl. 50):1–132. PMID: 5248894.
25. Lundgren S, Rosenquist JB. Short term bone healing in calcium deficiency osteopenia and disuse osteopenia: Experimental studies in adult rats. Scand J Dent Res 1992;100:337–339. DOI: 10.1111/j.1600-0722.1992.tb01083.x.
26. Krall EA, Wehler C, Garcia RI, et al. Calcium and vitamin D supplements reduce tooth loss in the elderly. Am J Med 2001;111:452–456. DOI: 10.1016/s0002-9343(01)00899-3.
27. Oliver WM. The effect of deficiencies of calcium, vitamin D or calcium and vitamin and of variations in the source of dietary protein on the supporting tissues of the rat molar. J Peridont 1969;4:56–59. DOI: 10.1111/j.1600-0765.1969.tb01946.x.
28. Freeland JH, Cousins RJ, Schwartz R. Am J ClinNutr 1976;29:745–749. DOI: 10.1093/ajcn/29.7.745.
29. Enwonwu CO. Interface of malnutrition and periodontal diseases. Am J Clin Nutr1995;61(Suppl.):430S–436S. DOI: 10.1093/ajcn/61.2.430S.

30. Sculley DV, Langley-Evans SC. Salivary antioxidants and periodontal disease status. Proc NutrSoc 2002;61:137–143. DOI: 10.1079/pns2001141.
31. Chandra RK. Nutrition and immunoregulation. Significance for host resistance to tumors and infectious disease in human and rodents. J Nutr 1992;122:754–757. DOI: 10.1093/jn/122.suppl_3.754.
32. Tonetti MS, Chapple IL; Working group 3 of Seventh European Workshop on Periodontology. Biological approaches to the development of novel periodontal therapies – Consensus of Seventh European Workshop on Periodontology. J Clin Periodontol 2011;38(Suppl 11):114–118. DOI: 10.1111/j.1600-051X.2010.01675.x.
33. Department of Health. Delivering better oral health: An evidence-Based toolkit for prevention 3rd edn. Public Health England: Wellington House, London. June 2014.
34. Tomar N, Gupta C, Kaushik M, et al. Nutrigenomics: A perio-nutrition interrelationships. J Oral Res Rev 2017;9:32–36.
35. Roberfroid MB. Prebiotics and probiotics: are they functional foods? Am J Clin Nutr 2000;71(6):1682S–1687S. DOI: 10.1093/ajcn/71.6.1682S.
36. Jackson MJ, Dillon SA, Broome CS, et al. Are there functional consequences of a reduction in selenium intake in UK subjects? Proc Nutr Soc 2004;63(4):513–517. DOI: 10.1079/pns2004382.
37. DeBusk RM, Fogarty CP, Ordovas JM, et al. Nutritional genomics in practice: where do we begin? J Am Diet Assoc 2005;105(4):589–598. DOI: 10.1016/j.jada.2005.01.002.
38. Vandana KL. Fluorosis and periodontium: A report of our institutional studies. J Clin Dent Res Organ 2014;6:7–15. DOI: 10.4103/2231-0754.139084.
39. Vandana KL, Srishti Raj B, Desai R. Dental fluorosis and periodontium: An original research report of In vitro and in vivo institutional studies. Biol Trace Elem Res 2021;199(10):3579–3592. DOI: 10.1007/s12011-020-02494-0.