

Review Article

Vitamin Deficiency And Periodontal Disease – A Tie- in RelationshipDr. R.S. Pavithra¹, Dr. G. Ramaprabha², Dr. S. Rajasekar³, Dr. S. Lakshmi sree⁴¹Senior Lecturer, Division of Periodontia, Ragas Dental College and Hospital, ECR Uthandi, Chennai, Tamilnadu.²Ph.D, Division of Periodontia, RMDCH, Annamalai University, Chidambaram, Tamilnadu³Professor, Division of Periodontia, RMDCH, Annamalai University, Chidambaram, Tamilnadu.⁴Reader, Division of Periodontia, RMDCH, Annamalai University, Chidambaram, Tamilnadu***Corresponding author**

Dr. R.S. Pavithra

Email: pavisounder@gmail.com

Abstract: Periodontal disease is a multifactorial disease, the origin of which remains incomprehensible. Various researches have shown that many systemic diseases either cause or exaggerate the course of certain periodontal conditions. Periodontal tissue integrity basically depends upon proper intake of proteins, carbohydrates, fats, vitamins and mineral salts. Nutritional imbalance provokes more destruction in the periodontium of which vitamins, which are the main constituent in maintaining the integrity of the periodontal tissues share a pivotal role in the deficient state predisposing an individual to periodontitis. Therefore, balancing the vitamin requirement status in an individual can prevent periodontal tissue breakdown with respect to other systemic illness. Accordingly, a synchronised interdisciplinary approach can be emphasized with precise diagnosis of the nutritional status of an individual, creating a therapeutic approach in the management of periodontal destruction. Therefore, nutritional deficiencies adversely affect the periodontal health status. This paper reviews the relationship between the nutrition especially vitamins in periodontal health and diseases.

Keywords: Nutrition, Periodontitis, Vitamin C, Vitamin D, Deficiency, Oral bioassay

INTRODUCTION

Periodontitis is caused by an increase in the virulence factors of the infecting organisms and the decreased resistance of the host [1]. Vitamin deficiencies predispose an individual to periodontal destruction, but their exact mechanisms have not been precisely defined. Deficiency of vitamins may affect any of the basic periodontal defence factors such as the integrity of the dentogingival barrier and the turnover of its constituent cells. Therefore, nutritional imbalance throughout the life cycle can impair tissue regeneration and healing, and can increase susceptibility to oral infections [2].

Nutrients can be subdivided into two broad categories,

1. Macronutrients (fats, carbohydrates and proteins) which are required in large quantities from the diet.
2. Micronutrients (minerals, vitamins, trace elements, and amino-acids) which are only

required in small quantities in the diet and which are essential for a range of biological processes important in supporting optimal health. (TABLE I) [3].

The majority of opinions and research findings on the effects of nutrition on oral and periodontal tissues point to the following:

1. There are nutritional deficiencies that produce changes in the oral cavity. These changes include alterations of the lips, oral mucosa, and bone, as well as the periodontal tissues. These changes are considered to be periodontal or oral manifestations of nutritional disease.
2. There are no nutritional deficiencies that by themselves cause gingivitis or periodontal pockets. However, nutritional deficiencies can affect the condition of the periodontium and thereby aggravate the injurious effects of local factors and excessive occlusal forces [4].

Table 1: Role of Vitamins in Health [3]

Nutrient	RDA or DRI	Functions in the Body/Benefits	Dietary Sources
FAT-SOLUBLE VITAMINS			
Vitamin A Retinol, beta-carotene and various other carotenoids	RDA Males: 1000 ug RE Females: 800 UG RE	Helps maintain good vision (necessary for night vision), resistance to infections, and supports growth and repair of body tissues. Also maintains integrity of white and red blood cells, assists in immune reactions, helps maintain the stability of cell membranes.	Milk, eggs, meat, fish liver oils. Beta-carotene and other carotenoids are found in: Green leafy vegetables - kale, spinach, broccoli, collard greens, parsley, turnip greens, escarole. Yellow vegetables - carrots, sweet potatoes, winter squash, pumpkin. Yellow and orange fruits - mango, cantaloupe, papaya, and apricots.
Vitamin D Cholecalciferol, ergocalciferol	DRI Males: 5.0 ug Females: 5.0 ug	Member of a large and cooperative bone-making and bone maintenance team. Regulates absorption of calcium and phosphorus for bone health.	Formed in skin when exposed to sunlight. Also found in dairy products, egg yolk, fish liver oils, tuna, mackerel, herring, sardines, oysters, yeast.
Vitamin E Tocopherol, Tocotrienols	RDA Males: 10 mg α-TE Females: 8 mg α-TE	Fat-soluble antioxidant. Helps maintain cell membranes, red blood cell integrity, protects vitamin A and fatty acids from oxidation.	Found primarily in plant oils, green, leafy vegetables, wheat germ, whole grains, egg yolk, nuts, seeds, and liver.
Vitamin K	RDA Males: 80 ug Females: 65 ug	Helps make factors that promote blood clotting.	Bacterial synthesis in the digestive tract. Diet generally supplies remaining need. Green, leafy vegetables, cabbage-type vegetables and milk.
WATER-SOLUBLE VITAMINS			
Vitamin B1 Thiamine	DRI Males: 1.2 mg Females: 1.1 mg	Helps metabolize carbohydrates, maintain appetite and normal digestion. Part of a coenzyme used in energy metabolism. Supports normal appetite and nervous system function.	Found in many foods: whole grain cereals, legumes, beans, nuts, brewer's yeast, wheat germ, pork, ham, and liver.
Vitamin B2 Riboflavin	DRI Males: 1.3 mg Females: 1.1 mg	Part of coenzymes used in energy metabolism, supports normal vision and skin health.	Milk, yogurt, other dairy, meat, leafy greens, whole grains.
Vitamin B3 Niacin, nicotinic acid, niacinamide	DRI Males: 16 mg NE Females: 14 mg NE	Part of a coenzyme used in energy metabolism, supports health of skin, nervous system and digestive system. High (pharmacological) doses may help manage cholesterol.	Tuna, dairy, meat, whole grains, nuts and all protein containing foods.
Vitamin B5 Pantothenic Acid	DRI Males: 5.0 mg Females: 5.0 mg	Part of Coenzyme A, which is used in energy metabolism	Widespread in foods.
Vitamin B6 Pyridoxine, pyridoxal, other forms	DRI Males: 1.3 mg Females: 1.3 mg	Part of a coenzyme that helps the body synthesize nonessential amino acids. Significant role in protein metabolism.	Green leafy vegetables, meats, fish, poultry, shellfish, legumes, fruits, whole grains.
Vitamin B12 Cobalamin	DRI Males: 2.4 ug Females: 2.4 ug	Part of coenzymes used in new cell synthesis; helps to maintain nerve cells.	Animal products (meat, fish, poultry, shellfish, eggs, cheese, milk).
Biotin	DRI Males: 30 ug Females: 30 ug	Part of a coenzyme used in energy metabolism, fat synthesis, amino acid metabolism and glycogen synthesis.	Widespread in foods.
Folic acid Folate, folacin	DRI Males: 400 ug Females: 400 ug	Part of coenzymes used in new cell synthesis. Essential for blood cell formation, protein metabolism, and prevention of neural tube defects.	Green leafy vegetables, liver, fortified grain products, legumes and seeds.
Vitamin C (ascorbic acid)	RDA Males: 60 mg Females: 60 mg	Essential element in collagen formation (strengthens blood vessels, forms scar tissue, is a matrix for bone growth); an antioxidant; strengthens resistance to infections; and improves absorption of iron.	Abundant in most fresh fruits (esp. citrus) and vegetables.

ROLE OF VITAMINS IN DISEASE
FAT-SOLUBLE VITAMIN DEFICIENCY
Vitamin A Deficiency

Deficiency of vitamin A results in dermatologic, mucosal, and ocular manifestations. In the absence of vitamin A, degenerative changes occur in epithelial tissues, resulting in keratinizing metaplasia. Since epithelial tissues provide a primary barrier

function to protect against invading microorganisms, vitamin A also plays an important role in maintaining the epithelium [5].

In experimental animals, vitamin A deficiency results in hyperkeratosis and hyperplasia of the gingiva with a tendency for increased periodontal pocket

formation. The following periodontal changes have been reported in vitamin A-deficient rats:

1. Hyperplasia and hyperkeratinisation of the gingival epithelium with proliferation of the junctional epithelium.
2. Retardation of gingival wound healing [6].

VITAMIN D DEFICIENCY

Vitamin D, or calciferol, is essential for the absorption of calcium from the gastrointestinal tract and the maintenance of the calcium phosphorus balance. There are 2 molecules that make up vitamin D: ergocalciferol (D2) and cholecalciferol (D3).

1. Deficiency in vitamin D and/or imbalance in calcium-phosphorus intake results in rickets in the children and osteomalacia in adults.
2. Clinical manifestations of rickets include irritability, growth retardation, prominence of costochondral junctions (rachitic rosary), bowing of long bones, developmental anomalies of dentin and enamel, delayed eruption, high caries rate, abnormally wide predentin zone, interglobular dentin.
3. Osteomalacia frequently results in diffuse skeletal pain and fracture with relatively mild injury. [4]
4. Vitamin D3 has been shown to have an important function as anticancer, immune modulatory and innate immunity effects through Vitamin D receptor activation (a transcription factor of nuclear receptor super family). 1,25(OH)₂ D₃-VDR system plays a role in oral homeostasis and its dysfunction may lead to periodontal disease.[7]
5. Immunomodulatory actions may include
 - Potent stimulator of innate immune system acting through Toll-like receptors on monocytes and macrophages
 - Decreased threshold for long-latency diseases such as cancers (including leukemia and colon, prostate and breast cancers), psoriasis, diabetes mellitus, and autoimmune diseases (eg, multiple sclerosis, rheumatoid arthritis, systemic lupus erythematosus) [8].

Stages of vitamin D deficiency

- **Stage I**
25-OH-D level decreases, resulting in hypocalcaemia and euphosphatemia; 1, 25-OH₂-D may increase or remain unchanged.
- **Stage II**
25-OH-D level continues to decrease; PTH acts to maintain calcium through demineralization of bone; the patient remains eucalcemic and hypophosphatemic and has a slight increase in the skeletal alkaline phosphatase level.
- **Stage III**
Severe 25-OH-D deficiency with hypocalcaemia, hypophosphatemia, and

increased alkaline phosphatase; bones have overt signs of demineralization [8].

- **F.S. Martelli *et al.*; in 2011** [9] investigated the relationship between a vitamin D receptor polymorphism and the diagnosis of periodontal disease in non-smoker Italian patients with aggressive and chronic periodontitis.
 - The results showed that the individuals with the TT genotype were more susceptible than individuals with Tt to chronic periodontitis and individuals with Tt to aggressive periodontitis and it was concluded that VDR TaqI polymorphism is differentially associated with development of chronic periodontitis and aggressive periodontitis in Italian population.
 - The determination of VDR polymorphisms may therefore be essential for the prevention of periodontitis through mass screening from a very early age and for pre-treatment periodontal and/or implant assessment.
- **Boggess *et al.*; in 2011** [10] examined the relationship between maternal vitamin D status and periodontal disease and concluded that Vitamin D insufficiency (serum 25[OH] D <75 nmol/l) is associated with maternal periodontal disease during pregnancy. Hence, Vitamin D supplementation represents a potential therapeutic strategy to improve maternal oral health.
- **Jessica *et al.*; in 2013** [11] examined the relationship between vitamin 25(OH) D and chronic periodontitis in patients with chronic kidney disease and not on dialysis and found that in patients with chronic kidney disease and not on dialysis, vitamin D deficiency was associated with CP.
- **Assad *et al.*; in 2014** [12] evaluated the level of 25-Hydroxy vitamin D₃ and osteocalcin in GCF and serum before and after scaling and root planing in chronic periodontitis patients.
 - The results showed improvement in all clinical periodontal parameters (GI, PI, BOP, PD and CAL) after SRP in chronic periodontitis patients (study group) and exhibited significant improvements of all values after therapy compared to baseline records.
 - A significant reduction in GCF osteocalcin level in the study group after SRP was also seen.
 - There was a statistically significant increase in the mean GCF 25-Hydroxy vitamin D₃ after performing SRP in the study group
 - Hence, concluded that SRP was effective in improving clinical parameters in patients with chronic periodontitis. 25-hydroxy vitamin D₃ could be used as adjunctive therapeutic modality for the

prevention and treatment of different types of periodontitis. Osteocalcin could be used as a potential diagnostic marker for periodontal disease activity in both serum and gingival crevicular fluid.

- **Xin Zhang *et al.*; in 2014** [13] measured Vitamin D binding protein levels in plasma and gingival crevicular fluid (GCF) of patients with generalized aggressive periodontitis (GAgP), in comparison to healthy controls, with the goal of elucidating the relationship between VitD BP and GAgP. From the results, GAgP patients had higher plasma DBP concentrations ($P < 0.001$) but lower GCF DBP concentrations ($P < 0.001$) than healthy controls. Hence, he concluded that decreased GCF DBP level and increased plasma DBP level are associated with periodontitis.
- **Jayachandran perayil *et al.*; in 2015** [14] evaluated the effect of vitamin D and calcium supplementation in management of periodontitis and assessed whether calcium and vitamin D oral supplementation influences alveolar bone density. The results showed significant change in the periodontal parameters and bone density after three months and intragroup comparison showed highly significant results for vitamin D group in relation to GI, OHI -S and bone density. Therefore it was concluded that Calcium and vitamin D supplementation has got a positive effect on periodontal health and it can be used as an adjunct to non-surgical periodontal therapy.

VITAMIN E DEFICIENCY

Vitamin E serves as an antioxidant to limit free-radical reactions and to protect cells from lipid peroxidation. Cell membranes, which are high in polyunsaturated lipids, are the major site of damage in vitamin E deficiency. Deficiency results in increased tendency for haemolysis. It affects cross linking of collagen [4].

- **Neha Singh *et al.*; in 2014** [15] investigated the levels of superoxide dismutase (SOD) activity in serum and saliva of patients with chronic periodontitis (CP). In addition, the outcome of scaling and root planing (SRP) with and without vitamin E supplementation is evaluated in terms of changes in periodontal parameters and SOD activity in patients with CP and concluded that systemic and local SOD levels are lowered in CP. Adjunctive vitamin E supplementation improves periodontal healing as well as antioxidant defense.

VITAMIN K DEFICIENCY

Vitamin K compounds have been found to be required for growth of *Bacteroides melaninogenicus*, an organism closely associated with periodontal disease

[16]. It is speculated that a suitable antimetabolite of Vitamin K might interfere with the growth of this organism, and consequently, prevent the occurrence of periodontal disease [17]. Deficiency leads to coagulopathy because of inadequate synthesis of prothrombin and other clotting factors. The most common oral manifestation is gingival bleeding, with chances of spontaneous bleeding at levels below 20% [18].

WATER SOLUBLE VITAMINS

B-COMPLEX DEFICIENCY

VITAMIN B1 (Thiamine)

It is also known as “antineurotic factor” due to its antagonistic pharmacologic action against acetylcholine. Deficiency results in a disease called “beri-beri”, a condition marked by multiple neuritis, edema, and serous effusion. Oral manifestations include:

- Hypersensitivity of the teeth and oral mucosa.
- Gingiva may become “dusty-rose” in colour.
- Loss of gingival stippling.
- Aphthous ulcerations [19].

VITAMIN B2 (Riboflavin)

The tissues affected due to B2 deficiency have a typical purplish magenta color. Marginal gingiva and oral mucosa have a purplish color and are edematous. Itching and burning of oral mucosa, ulceration of marginal gingiva and interdental papillae, marginal gingivitis and periodontitis may be seen. Deficiency also causes glossitis and angular cheilitis along with epithelial atrophy [20].

VITAMIN B3 (Nicotinic Acid)

Deficiency of niacin or tryptophan results in a condition known as pellagra. Gingivitis, attributable to deficiency of niacin, is characterized by extremely painful, wedge shaped, punched out ulcers involving the interdental papillae and marginal gingiva. The lesions in humans are necrotic, exudative, and foul smelling [21]. Epithelial changes particularly in the areas exposed to sunlight (neck region) result in a characteristic skin rash called Castle’s necklace [22].

VITAMIN B4 (Folic Acid)

Folic acid deficiency is characterized by lesions in cells with rapid rate of renewal, which demonstrates the importance of this vitamin in the synthesis of DNA. This results in macrocytic anemia with megaloblastic erythropoiesis, accompanied by oral changes, gastrointestinal lesions, diarrhea, and intestinal malabsorption. Marked chronic periodontitis with loosening of teeth may occur. Folic acid deficiency impairs immune responses and resistance of the oral mucosa to penetration by pathogenic organisms such as candida [22].

- Folic acid deficiency in animals demonstrate
 - Necrosis of gingiva
 - Necrosed periodontal ligament
 - Necrosed alveolar bone [23]
- In a series of human studies, a significant reduction of gingival inflammation has been reported after systemic or local use of folic acid, when compared with placebo [24].
- **Sumona *et al.*; in 2011** [25] compared the serum folic acid levels in patients with chronic periodontal disease in relation to the patient's smoking habits and concluded that among patients with periodontal disease the serum folic acid level is lower in smokers compared with non-smokers.

VITAMIN B6 (PYRIDOXINE)

A deficiency of vitamin B6 alone is uncommon because it usually occurs in association with a deficit in other B complex vitamins. Hypovitaminosis B6 may often occur with riboflavin deficiency, because riboflavin is needed for the formation of the coenzyme PLP. A decrease in the metabolism of glutamate in the brain, which is found in vitamin B6 insufficiency, reflects a nervous system dysfunction. As is the case with other micronutrient deficiencies, vitamin B6 deficiency results in an impairment of the immune system [26].

VITAMIN B12 (Cobalamin)

1. It is the only vitamin that contains a mineral. Vitamin B12 functions as a coenzyme in conjunction with folate metabolism in nucleic acid synthesis.

2. It also functions in the catabolism of certain amino acids and fatty acids. Vitamin B12 is essential for making red blood cells and for myelin synthesis.
3. An oral examination may reveal stomatitis or a pale or yellowish mucosa, xerostomia, cheilosis, hemorrhagic gingiva and bone loss. Deficiency symptoms are rapidly corrected with vitamin B12 injections.[26]
4. **Hasan Hatipoglu *et al.*; in 2012** [27] showed that severe periodontal destruction was observed in a patient with severe iron and B12 deficiency anemia.

VITAMIN C (ASCORBIC ACID) DEFICIENCY

1. Severe vitamin C deficiency in humans results in scurvy, a disease characterized by hemorrhagic diathesis and retardation of wound healing.
2. Clinical manifestations of scurvy include hemorrhagic lesions into the muscles of the extremities, the joints, and sometimes the nail beds; petechial hemorrhages, often around hair follicles; increased susceptibility to infections; and impaired wound healing. Bleeding, swollen gingiva and loosened teeth are also common features of scurvy.[28]
3. It results in defective formation and maintenance of collagen, retardation or cessation of osteoid formation, and impaired osteoblastic function
4. Vitamin C deficiency is also characterized by increased capillary permeability, susceptibility to traumatic hemorrhages, hypo reactivity of the contractile elements of the peripheral blood vessels, and sluggishness of blood flow.[29] (**TABLE II**) [30].

Table 2: Clinical Manifestations of Nutritional Deficiency [30]

NUTRIENT DEFICIENCY	CLINICAL MANIFESTATIONS
Vitamin A	Gingivitis, Periodontitis Hyperplasia of the gingiva.
Thiamine/ Vitamin B1	Cracked lips A satin looking gingiva and tongue Angular cheilosis
Vitamin B2/Riboflavin Niacin	Inflammation of the tongue. Fiery red inflammation of the tongue Angular cheilosis Ulcerative gingivitis
Vitamin B6	Teeth or bone decay Periodontal disease. Anemia Sore tongue Burning sensation in the oral cavity.
Vitamin B12	Angular cheilosis Halitosis Bone loss Hemorrhagic gingivitis Detachment of periodontal fibers Painful ulcers in the mouth
Vitamin C	Bleeding gums, Mobile teeth, Delayed wound healing
Vitamin D	Enamel hypoplasia Absence of lamina dura Abnormal alveolar bone patterns
Iron	Very red, painful tongue with a burning sensation Dysphagia Angular cheilosis

POSSIBLE ETIOLOGIC RELATIONSHIPS BETWEEN ASCORBIC ACID AND PERIODONTAL DISEASE

It has been suggested that ascorbic acid may play a role in periodontal disease by one or more of the following mechanisms:

- a) Low levels of ascorbic acid influence the metabolism of collagen within the periodontium, thereby affecting the ability of the tissue to regenerate and repair itself. No experimental evidence supports this view of the role of ascorbic acid; furthermore, it has been shown that collagen fibers in the periodontal ligament of scorbutic monkeys are the last affected before death of the animals.
- b) Ascorbic acid deficiency interferes with bone formation, leading to loss of periodontal bone. Changes that do occur in alveolar bone and other bones as a result of failure of the osteoblasts to form osteoid take place very late in the deficiency state.
- c) Ascorbic acid deficiency increases the permeability of the oral mucosa to titrated endotoxin and titrated inulin and of normal human crevicular epithelium to titrated dextran. Optimal levels of this vitamin, therefore, would maintain the epithelium's barrier function to bacterial products.
- d) Increasing levels of ascorbic acid enhance both the chemotactic and migratory action of leukocytes without influencing their phagocytic activity. Mega doses of vitamin C seem to impair the bactericidal activity of leukocytes.
- e) An optimal level of ascorbic acid is apparently required to maintain the integrity of the periodontal microvasculature, as well as the vascular response to bacterial irritation and wound healing.
- f) Depletion of vitamin C may interfere with the ecologic equilibrium of bacteria in plaque and thus increase its pathogenicity. However, there is no evidence that demonstrates this effect [31].

➤ **Kuzmanova *et al.*; in 2012** [32] tested the hypothesis that vitamin C concentrations in plasma, polymorphonuclear neutrophilic leucocytes (PMNs) and peripheral blood mononuclear cells (PBMCs) are lower in periodontitis patients compared with healthy controls and concluded that lower plasma vitamin C concentrations are associated with periodontitis but the disease cannot be explained by insufficient vitamin C storage capacity of leucocytes.

➤ **Pushparani *et al.*; in 2013** [33] assessed the serum level of vitamin C and zinc in type 2 diabetes mellitus with and without periodontitis, and elucidated whether increased or decreased serum vitamin C and zinc could be related to a risk factor for developing oxidative stress in type 2 diabetes mellitus with periodontitis. It was concluded that

the decreased level of antioxidants, vitamin C and zinc are associated with an increased risk for the development of oxidative stress in type 2 diabetes mellitus with periodontitis.

IMPLEMENTING NUTRITION IN PERIODONTAL PRACTICE [34]

A practitioner must know how to evaluate the nutritional status of a patient and perform a comprehensive nutritional analysis. A number of integrated systems for nutritional analysis and therapy in dental practice are available. None of these systems has been sufficiently tested to warrant comparisons. One system, however, does have the distinct advantage of offering several options or "phases" so that the clinical nutrition program can be designed to the needs of the patients. This system is called **triphasic nutritional analysis**. It consists of three phases:

The **first phase** includes the standard medical/social history, clinical examination and a qualitative dietary analysis.

- It is only necessary to include nutritional aspects to gain important insights about the status of the patient. In addition, a careful clinical examination may reveal some of the signs of nutritional deficiency.
- Particularly important in the clinical examination is the **oral bioassay**. This assay is the subjective comparison of the amount of plaque present around the teeth and the degree of periodontal destruction.
- The oral bioassay should be conducted at three different stages during periodontal therapy:
 - a) The primary evaluation should be conducted prior to any form of therapy, when the patient first presents himself for treatment. This analysis provides useful baseline data in terms of both oral hygiene and the response of the periodontal tissues to the existing irritants.
 - b) The response of the periodontal tissues should be evaluated second time after initial preparation when the local irritants have been removed. Poor tissue reactivity at this stage suggests that an inadequate healing potential exists which may compromise the effectiveness of any anticipated surgical procedures.
 - c) It is useful to include a third assay for tissue response after the first surgical procedure. As the surgical periodontal patient has potential nutritional and dietary requirements which may surpass those of the average dental patient, the clinician should analyze these requirements, counsel the patients and modify or supplement the diet as the needs of the patient dictate.
- Any exaggerated response of the periodontal tissues to the amount of local irritant should increase the practitioner's suspicion of systemic

involvement and suggest the need for nutritional diet diary.

- This transformation is difficult and should be conducted by constrained hygienist. If the clinician does not employ a trained auxiliary, the patient may be referred to a consulting nutritionist or a computer-assisted diet analysis may be performed.

The **second phase** of the triphasic nutritional analysis includes a semi quantitative dietary analysis and routine blood chemistry with differential blood count and glucose tolerance test. This second level of analysis is required when, based on the first phase, the following conditions are noted:

- (1) The diet appears to be inadequate, but no clinical signs of malnutrition are present;
- (2) The diet appears adequate, but the patient may have increased needs because of a stress factor
- (3) The clinician desires to correlate specific nutrient intakes with clinical evidence.

The **third phase** of analysis reserved for complex metabolic problems is usually conducted in consultation with a physician.

DIET BEFORE PERIODONTAL SURGERY [35]

When prescribing a diet before periodontal surgery, the goal is to enable the patient to meet the stress of surgery. Furthermore, a well-nourished state is optimal for wound healing. It also increases resistance to infection and hastens convalescence and recovery. If the periodontal patient is malnourished, a diet high in protein and enough carbohydrates and fat to provide about 2,500 K cal should be prescribed for 7-14 days before surgery. If the periodontal surgery is elective, it may be best to delay it until the patient's nutritional status is optimal. It may be necessary to prescribe a multivitamin capsule to be certain that adequate amounts of ascorbic acid are ingested.

POSTOPERATIVE DIETARY MANAGEMENT OF HOSPITALIZED PERIODONTAL SURGERY PATIENT [35]

During periodontal surgery under general anesthesia, intravenous levels of solution of 0.45% saline with 5% dextrose in water and 38.5 mill equivalents of sodium is given. The intravenous infusion is terminated in the recovery room if the patient is in good health. However, if the patient seems dehydrated, it may be wise to continue the infusion until the patient is fully reactive and can take fluids by mouth. When fully recovered from the anesthesia, the patient should be given clear fluid as tolerated. In addition to water, beverages such as cola drinks, ginger ale, apple juice, orange juice in addition to clear broths or clear tea or black coffee with sugar and flavoured gelatin are usually well tolerated.

- 1) On the first post-operative day, in addition to the beverages mentioned, sherbets, custards and ice

creams may be advised if the patient is hungry. Gruel or cereal topped with sugar and milk, as well as, egg or eggnog or strained chicken, pea or vegetable soup can be suggested. Frequent small feeds are tolerated well than a few large ones.

- 2) On the second post-operative day, the patient may supplement the diet with the following:
 - Vegetable fruit group: Citrus fruits such as oranges and grapes are recommended as are tomato and other fruit and vegetable juices.
 - Bread cereal group: Strained gruels can be given eg: wheat with milk.
 - Milk group: Milk in all forms can be given such as ice creams, milk shakes and malted milks.
 - Meat group: Eggs in the form of eggnogs may be given in a liquid diet.

A full liquid diet that furnishes 2000 calories and 80 gm of proteins per day is recommended.

CONCLUSION

Although periodontal disease is not a nutritional deficiency disease, malnutrition plays a role in predisposing the host to the progression of pre-existing periodontal lesions and influences the outcome of periodontal treatment. The concepts developed with respect to experimental design and the host defences approach to periodontal disease, will hopefully serve to encourage definitive studies, which may delineate a more precise role for nutrition in periodontal disease.

REFERENCES:

1. Grant D.A. Periodontics. 6th ed. St Louis (Washington): Mosby Company; 1988.
2. Apatzidou DA, Riggio MP, Kinane DF. Impact of smoking on the clinical, microbiological and immunological parameters of adult patients with periodontitis. *Journal of clinical periodontology*. 2005 Sep 1; 32(9):973-83.
3. Milward MR, Chapple IL. The role of diet in periodontal disease. *Clin Dent Health*. 2013; 52:18-21.
4. Klokkevold.P.R, Mealey.B.L, Carranza.F.A Influence of Systemic Disease and Disorders on the Periodontium. In: Carranza's Clinical Periodontology. 9th ed. Philadelphia: Saunders Elsevier; 2002: 204-228.
5. Boyle PE. Effects of vitamin A deficiency on the periodontal tissues. *American Journal of Orthodontics and Oral Surgery*. 1947 Oct 31; 33(10):744-8.
6. Frandsen AM. Periodontal tissue changes in vitamin A deficient young rats. *Acta Odontologica Scandinavica*. 1963; 21:19-34.
7. Amano Y, Komiyama K, Makishima M. Vitamin D and periodontal disease. *Journal of oral science*. 2009; 51(1):11-20.

8. Wagner CL, Greer FR. Prevention of rickets and vitamin D deficiency in infants, children, and adolescents. *Pediatrics*. 2008 Nov 1; 122(5):1142-52.
9. Martelli FS, Mengoni A, Martelli M, Rosati C, Fanti E. VDR TaqI polymorphism is associated with chronic periodontitis in Italian population. *Archives of oral biology*. 2011 Dec 31; 56(12):1494-8.
10. Boggess KA, Espinola JA, Moss K, Beck J, Offenbacher S, Camargo Jr CA. Vitamin D status and periodontal disease among pregnant women. *Journal of periodontology*. 2011 Feb; 82(2):195-200.
11. Bastos JD, Andrade LC, Ferreira AP, Barroso ED, Daibert PD, Barreto PL, Vilela EM, Marcaccini AM, Colugnati FA, Bastos MG. Serum levels of vitamin D and chronic periodontitis in patients with chronic kidney disease. *Jornal Brasileiro de Nefrologia*. 2013 Mar; 35(1):20-6.
12. Matouga.A.A, El-Shinnawi.U.M, AL-Sharkawy.H, El-Farahaty.R.M. Assessment of 25-Hydroxy Vitamin D3 and Osteocalcin in Chronic Periodontitis Patient (Clinical and Laboratory study). *Mansr J Dent*. 2014; 1: 34-41.
13. Zhang X, Meng H, Xu L, Zhang L, Shi D, Feng X, Lu R, Chen Z. Vitamin D-binding protein levels in plasma and gingival crevicular fluid of patients with generalized aggressive periodontitis. *International journal of endocrinology*. 2014 May 12; 2014.
14. Perayil J, MeNON KS, Kurup S, Thomas AE, Fenol A, VYIOP Pillil R, Bhaskar A, MeGHA S. Influence of Vitamin D & Calcium Supplementation in the Management of Periodontitis. *Journal of clinical and diagnostic research: JCDR*. 2015 Jun; 9(6):ZC35.
15. Singh N, Chander Narula S, Kumar Sharma R, Tewari S, Kumar Sehgal P. Vitamin E supplementation, superoxide dismutase status, and outcome of scaling and root planing in patients with chronic periodontitis: a randomized clinical trial. *Journal of periodontology*. 2014 Feb; 85(2):242-9.
16. Macdonald JB, Gibbons RJ. The relationship of indigenous bacteria to periodontal disease. *J Dent Res*. 1962 Jan 1; 41:320-6.
17. Nizel.A. Nutrition in preventive dentistry: Science and Practice. 2th ed. New York: W.B.Saunders; 1981.
18. Schifferle RE. Periodontal disease and nutrition: separating the evidence from current fads. *Periodontology 2000*. 2009 Jun 1; 50(1):78-89.
19. Goldman.H. Role of Nutrition in periodontal disease. 3rd ed. St. Louis: Mosby; 1990.
20. Moynihan PJ. The role of diet and nutrition in the etiology and prevention of oral diseases. *Bulletin of the World Health Organization*. 2005 Sep; 83(9):694-9.
21. Becks H, Wainwright WW, Morgan AF. Comparative study of oral changes in dogs due to deficiencies of pantothenic acid, nicotinic acid, and unknowns of the B vitamin complex. *American Journal of Orthodontics and Oral Surgery*. 1943 Apr 30; 29(4):183-207.
22. Mann. A.W, Spies. T.M, Springer. L.M. Oral manifestations of vitamin B complex deficiencies. *J Dent Res*. 1941; 20:269-72.
23. Shaw JH. The relation of nutrition to periodontal disease. *J Dent Res*. 1962 Jan 1; 41(1):264-74.
24. Vogel RI, Fink RA, Frank O, Baker H. The effect of topical application of folic acid on gingival health. *Journal of oral medicine*. 1977 Dec; 33(1):22-.
25. Sumona B, Sheetal S, Anil M, Suvarna P. Comparative evaluation of serum folic acid levels in smokers and non-smokers with chronic periodontitis. *Bangladesh Journal of Medical Science*. 2011 Jan 1; 10(2):83.
26. Ghosh A, Pallavi SK, Nagpal B, Hegde U, Archana S, Nagpal MJ. Role of Vitamins in Oral Health & Disease: an Overview. *Indian Journal of Applied Research*. 2016 May 7; 5(12).
27. Hatipoglu H, Hatipoglu MG, Cagrankaya LB, Caglayan F. Severe periodontal destruction in a patient with advanced anemia: a case report. *European journal of dentistry*. 2012 Jan; 6(1):95.
28. Cotran.R.S, Kumar.V, Robbins.S.R. Pathologic Basis of Disease. 4th ed. Philadelphia: W.B.Saunders; 1989.
29. Wolbach SB, Bessey OA. Tissue Changes in Vitamin Deficiencies. *Physiological Reviews*. 1942 Jul; 22(3):233-89.
30. Ehizele.A.O, Ojehanon.P.I, Akhionbare.O. Nutrition and Oral Health. *J Post Med*. 2009; 11:76-82.
31. Woolfe.S.N, Wyatt.H.R, Kennedy.E.B. Ascorbic acid and periodontal disease. *J West Soc Periodontal* .1980; 28:44-56.
32. Kuzmanova D, Jansen ID, Schoenmaker T, Nazmi K, Teeuw WJ, Bizzarro S, Loos BG, Velden van der U . Vitamin C in plasma and leucocytes in relation to periodontitis. *J Clin Periodontal* .2012; 39: 905-12.
33. Pushparani DS, Nirmala S, Theagarayan P. Low Serum Vitamin C and Zinc are associated with the Development of Oxidative Stress in Type 2 Diabetes Mellitus with Periodontitis.
34. Mahajan.A, Mahajan.P, Thakur.S, Kashyap.D, Kumar.A. Role of Nutrition in Periodontal Health. *J Dent*. 2014; 2: 35-43.
35. Kanaparthi.R, Kanaparthi.A, Niranjana.N.T. Nutritional Requirements of the Periodontal Patient. *Indian J Stomatol* .2011; 2:175-78.