NUTRITION AND PERIODONTAL DISEASE

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INTRODUCTION

Periodontal diseases are chronic inflammatory conditions that lead to destruction of the supporting structures of the teeth. They include: chronic periodontitis, aggressive periodontitis, and necrotizing ulcerative gingivitis and periodontitis. Specific microbial organisms, many of which derive their nutritional requirements from the host, cause these. They are characterized by infiltration of leukocytes, particularly the polymorphonuclears, loss of connective tissue, alveolar bone resorption, and formation of periodontal pockets.

The primary aetiology of periodontal disease is bacterial plaque biofilm but a susceptible host is important for disease initiation. A periodontal lesion is essentially a wound, and sufficient host resources must be available for optimal healing to take place. Decreased host immune response and nutrient deficiencies increase the risk and severity of the periodontal disease. Many nutrients play an important role in immune cell function and production. Nutritional status influences the production and release of cytokines and their action.

Malnutrition consistently impairs innate and adaptive defenses of the host, including phagocytic function, cell-mediated immunity, complement system, secretary antibody, and cytokine production and function. In protein energy malnutrition (PEM), there are marked changes in the oral microbial ecology resulting in a preponderance of pathogenic anaerobic organisms, increased propensity of bacteria to bind to oral mucosal cells, attenuation of acute phase protein response, and dysfunction of the cytokine system. Cellular depletion of antioxidant nutrients promotes immune-suppression, accelerated replication rate of ribonucleic acid viruses and increased disease progression.

The protein-energy-malnutrition enhances periodontal diseases by decreased resistance of mucosa to colonization and invasion by pathogens impaired salivary flow and antibacterial properties. Increased prevalence and potency of pathogenic oral microorganisms possibly due to altered bacterial profile. Cytokines involved in the healing process compromised reduced acute phase protein response.

It is a truism that the individual nutritional status affects the periodontium however no nutritional deficiencies of themselves cause i.e initiate gingivitis or periodontal pockets.

The dental health professional therefore has a responsibility not only to remove the local irritants by scaling, root planing and curettage accompanied by a meticulous daily home plaque control program but also to help the patient increase the systemic resistance of periodontal tissues by nutritional counseling.

NUTRITION IN PERIODONTAL HEALTH:

Periodontal health is influenced by a number of factors such as oral hygiene, systemic health, and nutrition. Many studies have observed that a balanced diet has an essential role in maintaining periodontal health. Nutrition is one of the bendable factors that impact host’s immune response and the integrity of soft and hard tissues of the oral cavity. The role of diet in the development and progression of periodontal disease, however, is less well understood. Hence the present review aimed at understanding the relationship between nutrition and periodontal disease.

KEYWORDS: Periodontitis, Malnutrition, nutrients, nutritional status.
the host, environmental, and bacterial factors. The primary etiology of periodontal disease is bacterial (plaque biofilm), but a susceptible host is also necessary for disease initiation. Nutrition is one of the modifiable factors that impact the host's immune response and the integrity of the hard and soft tissues of the oral cavity.

Nutrient effects on collagen healing and maintenance

Collagens constitute about 30% of the total body proteins, and it is involved in forming the matrix of dentin, cementum, alveolar bone, and the periodontal ligament. About 10 to 15% of the amino acids that make up collagen are hydroxyproline, which is essential in stabilizing the structure of collagen through cross-linking. Marginal or deficient nutrient intakes may be associated with changes in the connective tissue that result in altered growth and wound healing, but studies of these effects are difficult due to the "long half-life of connective tissue proteins and the extracellular nature of events. Animal models suggest that even short-term fasting (4 days) results in a 40% reduction in collagen production, and low intakes of protein result in a significant reduction in collagen synthesis. However, it is unclear how this finding might apply to collagen synthesis and wound healing in humans. Ascorbic acid (vitamin C) is involved in hydroxyproline formation and has been shown to stimulate collagen formation by fibroblast three- to four fold in vitro. The classic symptoms of scurvy (vitamin C deficiency) includes swollen and inflamed gingiva, loosening of the teeth, follicular hyperkeratosis, perifollicular hemorrhage, weakness, malaise, sore joints, bruising, and weight loss. The most recent epidemiologic data from the National Health and Nutrition Examination Survey (NHANES III) suggests the odds of having periodontal disease are 1.2 times greater in those with low dietary vitamin C intakes. In the same study, smokers and former smokers with low vitamin C intakes are at 1.6 time's greater risk of having periodontal disease. Even though overt deficiencies are seldom seen in the healthy population, marginal vitamin C intakes may be a conditioning factor in the development of gingivitis because of the under hydroxylation of the collagen and increase in intracellular permeability of blood vessels as well as the sulcular epithelium.

However, megadoses of vitamin C have not been shown to have a strong effect on the healing response in initial periodontal therapy and, therefore, pharmacologic doses should not be recommended to patients until further research confirms its utility. Vitamin A is involved in collagen metabolism by affecting cell differentiation. In animal studies, supplemental vitamin A increases the breaking strength of healing wounds, and in vivo it has enhanced collagen accumulation. The caveat to remember is excessive intakes of vitamin A result in an increase in collagen catabolism and resorption of bone so moderation is the key.

Boron, copper, manganese, silicon, and zinc are also important participants in collagen metabolism and wound healing. Copper in particular is an essential nutrient for the cross-linking of both collagen and elastin. A deficiency results in a decrease in the tensile strength of collagen and osteoporotic-like bone lesions. Zinc is important for its effect on protein synthesis including DNA and RNA synthesis. Zinc concentrations increase around the wound margin during formation of granulation tissue, scar formation, and re-epithelization. Zinc, copper, and iron all compete for absorption so they need to be kept in balance for optimum wound healing. Supplementation of minerals above the recommended daily allowance (RDA) is not recommended at this time.

Maintenance of calcified tissues:

Bone remodeling continues throughout life, but around age 40 osteoclastic activities begins to exceed osteoblastic activity with a net loss of bone on the average of 1.2% per year. The nutrients known to affect bone formation include vitamin A, C, D, K, zinc, magnesium, phosphorus and calcium. Vitamin D regulates the absorption of calcium from the gastrointestinal tract to maintain calcium balance. When calcium intake is inadequate, vitamin D stimulates osteoclasts to help mobilize the calcium stored in bone. Both collagen synthesis and accumulation of mineralized bone are dependent on adequate levels of vitamin D and calcium. A nutrient seldom discussed in terms of bone health is vitamin K. There is, however, an association between vitamin K insufficiency and reductions in bone density and possibly bone strength. This may be of particular concern among people taking vitamin K antagonists such as oral anticoagulants as a significant decrease in bone mineral density has been observed. Minerals involved in the calcified tissues include boron, calcium, copper, and magnesium. A recent epidemiologic study using the NHANES III data found the risk of periodontal disease was 56% greater in women with dietary intakes of calcium below 500 mg/day and 27% greater for those taking in less than 800 mg/day. The study goes on to suggest that low dietary calcium intake results in more severe periodontal disease. However, it remains unclear how calcium supplementation might impact the course of periodontal disease. Furthermore, evidence is accumulating that indicates a correlation between systemic osteoporosis, alveolar bone, and ultimately tooth loss in post-menopausal women.

Nutrition strategies to enhance immunity and prevent infection

Nutrition plays an important role in maintenance of the optimal functioning of the immune response. Individuals who are undernourished have impaired immune responses including abnormalities in adaptive immunity, phagocytosis, and antibody function. Animal studies suggest that providing adequate levels of specific nutrients...
such as protein is associated with improved immune-competence and reduced mortality after infectious challenge. These findings point to the need to initiate nutritional strategies that may help reduce the occurrence of opportunistic infections in immune-compromised patients.

The previous nutritional status of the patient, the nature and duration of the infection, and dietary intake during recovery are important aspects of nutrition that must be considered in order to improve the outcomes of periodontal treatment, as well as other invasive dental procedures. A nutritional assessment will help identify individuals with marginal nutritional status or poor dietary habits who will benefit from nutritional rehabilitation prior to extensive dental treatment.

The American Dental Association and the American Dental Hygiene Association recommend following nutrition recommendations such as the USDA Food Guide Pyramid and the Dietary Guidelines for Americans as basic guidelines for "educating and counseling their patients about proper nutrition and oral health." The average American diet contains more than adequate amounts of protein and calories; however, patients should also be encouraged to include nuts and legumes to meet some of their protein needs, which will also increase intakes of vitamin E, copper, boron.

The average intake of fruits and vegetables in the United States is around two servings per day, which is significantly below the recommendation of three to five servings of vegetables and two to four servings of fruit. Fruits and vegetables are excellent sources of vitamins A, C, and K, beta-carotene and magnesium.

Low-fat dairy products should also be encouraged as excellent sources of protein, calcium, vitamin A, and vitamin D. Two to four servings of dairy products per day are needed to meet the new recommended dietary allowance. Those who are lactose intolerant consume an average of 325 mg/day of calcium, which is only 25% of the current recommended dietary allowance of 1000 mg/day to 1300 mg/day, placing these patients at risk of poor healing following implants and regeneration procedures. Patients avoiding dairy products need to find alternative sources of calcium such as fortified soy milk, rice milk, or orange juice, to name a few. Supplements should be a last resort because they are often forgotten and are more expensive than using food as a nutrient source.

For anyone with marginal nutrient intakes, more than the average intakes of the USDA Food Guide Pyramid may be required to replete nutrient levels. In individuals with chronic disease and in the elderly, a multivitamin with minerals that has 100% of the recommended dietary allowance levels taken daily may be beneficial for the prevention of infection and to facilitate healing.

Supplementation with individual nutrients may need to be provided for individuals with documented nutrient deficiencies, but the use megadoses of nutrients should be discouraged until more research supports their long-term daily use. Any patient with complex nutritional needs should be referred to a registered dietitian for individualized nutritional advice.

**Building healthy periodontal tissues:**

Building and maintaining healthy oral tissue is the body’s first line of defense against periodontal disease. Health oral tissue is less susceptible to infection.

The epithelial lining of the gingival sulcus requires an adequate supply of nutrients because it has one of the fastest turnover rates in the body.(3 days). Vitamin A, B-complex and D and calcium and magnesium all work to build and maintain health soft tissue and bone.

Vitamin A assists in the formation of healthy epithelium and its vital for functioning of the immune system. The B-complex vitamins are important in helping the body to form new cells and in keeping the immune system healthy.

Vitamin D enhances the absorption of calcium and magnesium, principal minerals for bone development and regeneration.

**Repairing periodontal infection:**

Periodontal infection is a ‘wound’ and the body needs nutrients that will help repair tissues and covert tissues to healthy tissues. Inadequate nutrition may cause impairment in the repair process of the gingival sulcus.

Trace elements are needed for protein synthesis, and protein support growth and maintenance of healthy cells and play a role in resistance to infection.

Vitamin C aids in the formation of collagen, a cementing substance that helps wounds heal and assists the body’s resistance to infection and also promotes capillary integrity and enhances the body’s immune response. Iron, Zinc, Copper assist with collagen formation and therefore the healing of wounds. Selenium’s antioxidant qualities work to prevent harm cells and tissues.

**Role of nutrition in periodontal disease**

Periodontitis results from the loss of a delicate balance between microbial virulence factors and a proportionate host response. The role of nutrition in the pathogenesis of periodontal disease has been a topic of speculation and investigation since the beginning of the century.

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There is also a mounting evidence that periodontal disease (PD) is linked to low serum 25-hydroxyvitamin D \([25(OH) \text{D}]\) concentrations in addition to recognized risk factors like diet and smoking. Since oral bacteria are important risk factors for periodontal disease, and activated vitamin \(D\), effectively fights bacterial infections, through induction of cathelicidin (LL-37) and other defensins it is reasonable to expect that maintenance of adequate repletion with vitamin \(D\) could reduce the risk of periodontal disease.\(^{14}\)

Vitamin \(D\) plays a role in various physiologic processes including bone and calcium metabolism, cellular growth and differentiation, immunity and cardiovascular function. Calcium physiologic processes are such that a single low concentration of the vitamin \(D\) hormone stimulates enterocytes to absorb calcium and phosphate. If the plasma calcium concentration fails to respond, then the parathyroid glands continue to secrete parathyroid hormone, which increases production of the vitamin \(D\) hormone to mobilize bone calcium (acting with parathyroid hormone).

Krall et al (2001)\(^{15}\) found that a moderate benefit with respect to periodontal health for combined vitamin \(D\) and calcium supplementation. A cross-sectional study conducted by Dietrich T et al (2004)\(^{16}\) found a significant inverse correlation between periodontal disease and serum 25-hydroxyvitamin \(D\) \([25(\text{OH}) \text{D}]\) concentrations.

Hildebolt et al (2005)\(^{17}\) demonstrated that calcium and vitamin \(D\) are important adjuncts to standard treatments for preventing and treating periodontal disease. This is in accordance with Liu et al (2010)\(^{18}\) who observed reduction of 25-hydroxyvitamin \(D\) \((3)\) and interleukin-1beta levels in patients with GAgP by initial periodontal therapy. In contrast to the above findings, Bashutski J et al (2011)\(^{19}\) demonstrated that Vitamin \(D\) deficiency at the time of periodontal surgery negatively affects treatment outcomes for up to 1 yr. Analysis of these data suggests that vitamin \(D\) status may be critical for post-surgical healing.

Garcia et al (2011)\(^{20}\) determined whether the patients in periodontal maintenance programs taking vitamin \(D\) and calcium supplementation had a trend for better periodontal health compared to patients not taking supplementation and they found that measures of periodontitis were consistently higher in non-takers than in takers. So they concluded that there is a possibility that vitamin \(D\) may positively impact periodontal health.

Henning et al (1999)\(^{21}\) found that Specific vitamin \(D\)-receptor genotypes have been shown to be associated with localized aggressive periodontal disease, with oral bone loss, clinical attachment loss, and tooth loss. Amano et al 2009 1.25(OH)\(_2\text{D}_3\) plays a role in maintaining oral health through its effects on bone and mineral metabolism and innate immunity, and several VDR gene polymorphisms have been reported to be associated with periodontal disease. VDR ligands should prove to be useful in the treatment and prevention of periodontal disease.

A number of infectious diseases have been linked with low levels of vitamin \(D\), and it has been demonstrated that vitamin \(D\) can suppress cytokine production. Zitterman et al (2003)\(^{22}\) Because of periodontal disease’s unique periodontal-pathogen, hard-tissue environment, it may be that the effect of vitamin \(D\) and calcium on alveolar bone is more pronounced than its effects in the spine and hip.

Douglas Miley et al (2009)\(^{23}\) suggested that in patients receiving periodontal maintenance therapy, there was a trend for better periodontal health with vitamin \(D\) and calcium supplementation. Dietrich et al (2005)\(^{24}\) suggested that Vitamin \(D\) may reduce susceptibility to gingival inflammation through its anti-inflammatory effects. Gingivitis may be useful clinical model to evaluate the anti-inflammatory effects of vitamin \(D\).

Boggess et al (2011)\(^{25}\) conducted a case control study and concluded that Vitamin \(D\) insufficiency is associated with maternal periodontal disease during pregnancy and its supplementation represents a potential therapeutic strategy to improve maternal oral health. Hokugo et al (2010)\(^{26}\) suggested that vitamin \(D\) deficiency has been associated with compromised osseous healing in the oral cavity in bisphosphonate associated osteoradionecrosis of the jaw.

The possible role of vitamin \(E\) in the management of periodontal disease is based upon its ability to interfere with prostaglandin synthesis, which themselves are important in the development of inflammation.

Murakami et al (2013)\(^{27}\) reported that tocopherols exhibit anti-inflammatory activity. Beta, gamma and delta-tocopherols have particularly more potent anti-inflammatory activity than alpha-tocopherol, tocopherols may have potential utility for prevention of periodontal and chronic oral diseases. In line with Murakami, Singh et al (2014)\(^{28}\) suggested that adjunctive vitamin \(E\) supplementation improves periodontal healing as well as antioxidant defense. In contrast to above, Carvalho et al (2013)\(^{29}\) Vitamin \(E\) may have potential to reduce oxidative damage and inflammatory response in early onset periodontitis but does not prevent alveolar bone loss.

Among all nutrients necessary for normal physiologic functions, the vitamin-B complex may be important for periodontal wound healing.

Esaki M et al (2010)\(^{30}\) reported that dietary intake of folic acid, an important indicator of gingival bleeding in adults, may provide an important clinical target for intervention to promote gingival health. Neiva et al (2005)\(^{31}\) stated that Vitamin B-complex supplement in...
combination with access flap surgery resulted in statistically significant superior CAL gains. HasanHatipoglu et al (2012)\(^{31}\) reported that severe periodontal destruction was observed in a patient with severe iron and B\(_2\) deficiency anemia.

Additionally, other members of the vitamin-B complex, such as thiamine, may have also enhanced wound repair. Vitamin C or ascorbic acid is essential for the formation of collagen and intercellular material, bone and teeth, and for the healing of wounds. Vitamin C affects the response of periodontium by influencing the collagen turnover, epithelial barrier function and leucocyte function. It helps maintain elasticity of the skin, aids the absorption of iron, and improves resistance to infection. Vitamin C is one of the relatively few nutrients examined in relation to periodontal disease. Severe vitamin C deficiency is well known to lead to a severe periodontal syndrome called ‘scorbutic gingivitis’, which is characterized by ulcerative gingivitis and rapid periodontal pocket development with tooth exfoliation.

Vogel and Wechsler (1979)\(^{32}\) showed that the daily intake of vitamin C in a group of periodontitis patients was significantly less than in the control subjects. On the basis of the NHANES I study, Ismail et al (1983)\(^{33}\) found a weak but significant negative correlation between dietary vitamin C intake and periodontal disease after controlling for the potentially confounding variables of age, gender, race, education, income, and oral hygiene status.

On the basis of the NHANES III survey, Nishida et al (2000)\(^{34}\) found that the dietary intake of vitamin C showed a weak, but statistically significant inverse relationship to periodontal disease in current and former smokers. Smokers with the lowest intake of vitamin C were likely to have the worst periodontal condition. Using the same NHANES III data set Chapple et al (2007)\(^{35}\) found a strong and consistent inverse association between serum vitamin C concentrations and the prevalence of periodontitis in adjusted models (multiple logistic regression analysis adjusted for age, gender, race/ethnicity, BMI, cigarette smoking, oral contraceptives and hormone replacement therapy use, diabetes, poverty income ratio, and education). In contrary to this Mathias et al (2014)\(^{36}\) stated that SRP did not significantly change the levels of ascorbic acid in smokers.

Amaliya et al (2007)\(^{37}\) suggested that vitamin C deficiency may contribute to the severity of periodontal breakdown.

Ekuni et al (2009)\(^{38}\) suggested that vitamin C intake attenuates the degree of experimental atherosclerosis induced by periodontitis in the rat by decreasing oxidative stress.

Akman et al (2013)\(^{39}\) found that Alpha lipoic acid and Vitamin C treatment provides therapeutic effects on inhibition of alveolar bone resorption and periodontal tissue destruction.

Magnesium is an essential cation playing a crucial role in many physiological functions. Imbalances of magnesium are common and are associated with a great number of pathological situations responsible for human morbidity and mortality. Magnesium deficiency has been suggested to be involved in the aetiology of cardiovascular diseases, diabetes, pre-eclampsia, eclampsia, sickle cell disease and chronic alcoholism.

MeiselP et al (2005)\(^{40}\) suggested that the concentrations of serum magnesium and calcium were determined and related to periodontal parameters. Subjects taking Mg supplements showed less attachment loss and a higher number of remaining teeth than did their matched counterparts and stated that that nutritional magnesium supplementation may improve periodontal health and prevent or delay tooth loss.

Although relatively little attention has been given by periodontal research, the medical literature has studied the effects of nutritional supplementation among patients receiving various treatment modalities, and at present nutrition is considered to play an important role in wound healing processes. The effects of nutrition on periodontal disease status and response to treatment have been studied using different methods and study models. Studies by Kral et al have reported various degrees of association between nutritional elements/supplements and periodontal status, and others have reported possible positive influences of nutritional supplementation on periodontal therapeutic outcomes.

CONCLUSION

Periodontal disease is not due to nutritional deficiency per se, malnutrition is likely to play a role in either predisposing the host to the progression of preexisting periodontal lesions, influence the outcome of periodontal treatment, or both. There is an insufficient evidence to justify treatment with vitamin and mineral supplementation in the adequately nourished individual. Future research should focus on an evaluation of which foods and nutrients may help to prevent the onset and the progression of oral diseases.

References


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