

# You Are What You Eat: Nutrition and Periodontal Health



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**CE Credits:** 2 hours

**Intended Audience:** Dentists, Dental Hygienists, Dental Assistants, Dental Students, Dental Hygiene Students, Dental Assisting Students

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**Disclaimer:** Participants must always be aware of the hazards of using limited knowledge in integrating new techniques or procedures into their practice. Only sound evidence-based dentistry should be used in patient therapy.

## Conflict of Interest Disclosure Statement

- Dr. Geisinger reports no conflicts of interest associated with this course. She has no relevant financial relationships to disclose.

## Introduction – You Are What You Eat: Nutrition and Periodontal Health

This course seeks to review the association between micronutrient levels and periodontitis and the underlying physiologic mechanisms of interaction between micronutrient consumption and periodontal health or disease.

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## Overview

Periodontitis is a chronic inflammatory disease caused by oral microorganisms, characterized by the loss of structures supporting the teeth, i.e., periodontal ligament, cementum, and alveolar bone. In the United States, the prevalence of periodontitis among adults over 30 years of age is approximately 42% and causes significant impact on overall and oral quality of life.<sup>1</sup> While periodontitis is initiated by the microorganisms and their byproducts in dental plaque biofilm, its disease progression is mediated by the individual host immune-inflammatory response.<sup>2</sup> As such, periodontal disease progression may be influenced by many systemic and environmental risk factors, including smoking, diabetes mellitus, alcohol consumption, psychosocial stress, and/or sex hormonal levels. In addition to these systemic influences, recent findings suggest that periodontitis is also influenced by local and/or serum/plasma micronutrient levels. These levels are dependent upon dietary, lifestyle factors, and/or nutrigenetic characteristics.

The old adage, “you are what you eat” relates to periodontal health as well. Eating a balanced diet is critical to overall health and an imbalance, deficit, or overconsumption of dietary components can lead to severe systemic and oral diseases. Both macronutrients (protein, fats, and carbohydrates) and micronutrients can impact cell function, host cell immunity, and biofilm formation and maturation. Findings suggest that dietary consumption may influence

periodontal health and that deficiencies of both macro- and micronutrients may play a role in the onset or exacerbation of periodontal diseases. This course seeks to review the association between micronutrient levels and periodontitis and their underlying physiologic mechanisms.

## Learning Objectives

**Upon completion of this course, the dental professional should be able to:**

- Discuss the role of inflammation in periodontal disease.
- Recognize nutrient requirements for optimal oral health.
- Understand the connection between diet/nutrition and periodontal health.
- Assess the effects of both macro- and micronutrient imbalance on periodontal health status.
- Recognize the potential role of dietary modification in periodontal therapy.
- Identify and utilize tools to counsel and motivate patients to seek dietary modifications.

## Introduction

Dietary consumption and overall nutrition have been associated with the development of many health conditions, including cardiovascular diseases, diabetes mellitus, and cancer.<sup>3</sup> Diet has been defined as the habitual eating patterns of an individual, whereas nutrition refers to the science of food intake and biological processes involved in consumption and utilization of nutrients.<sup>4,5</sup> Nutrients can be categorized into macronutrients (proteins, carbohydrates, lipids), micronutrients (minerals, vitamins), and water.<sup>4</sup> Overall caloric intake as well as macronutrient consumption have been associated with oral and overall health status.<sup>3,5</sup> Further, approximately 10% of the US population is thought to have at least one clinically significant nutritional deficiency.<sup>6</sup> The most common deficiencies identified in US individuals include: Vitamin B, Iron, and Vitamin D.<sup>7</sup> It is also well-established that oral health status can impact the types of foods that individuals consume. Compromised oral health is associated with a marked decrease in whole food consumption and an increase in processed food consumption.<sup>8</sup> Such changes in diet have

the potential to impact both oral and systemic health. Given the impact of diet and on overall health and oral health, oral healthcare providers should strive to understand the interaction between nutritional intake and periodontal health.

### Systemic Inflammation and Periodontal Disease

Periodontal diseases are both infectious and inflammatory diseases of the supporting structures around the teeth: the gingiva, periodontal ligament, alveolar bone, and cementum.<sup>2,9,10</sup> The two most common forms of periodontal disease are gingivitis and periodontitis.<sup>1</sup> Gingivitis is a non-specific inflammatory reaction to the accumulation of dysbiotic bacterial biofilm.<sup>11</sup> All individuals are susceptible to developing gingivitis after oral hygiene procedures are stopped. Gingivitis is also a necessary precursor to periodontitis and, ultimately, a loss of hard and soft tissues around the teeth.<sup>11</sup> Removal of biofilm and local etiologic factors results in the reversal of gingivitis symptoms and resolution of local and systemic levels of inflammatory markers is associated with reestablishment of gingival health.<sup>11-13</sup>

Periodontitis is initiated by dysbiotic biofilm accumulation in a susceptible host and this biofilm dysbiosis triggers an immune-inflammatory response that then leads to the destruction of hard and soft tissues supporting the teeth.<sup>14-15</sup> Periodontal disease progression is generally slow to moderate. Average clinical progression of periodontal disease is approximately 0.1mm of attachment loss and 0.2 teeth lost annually.<sup>15</sup> In longitudinal investigations, groups with the fastest and slowest disease progression differed considerably with regard to demographics and underlying health conditions.<sup>16</sup> In an updated classification system from the American Academy of Periodontology (AAP) and European Federation of Periodontology (EFP), individuals with periodontitis are classified with a Stage and Grade, which are meant to capture both the current state of disease severity and distribution and risk of future disease progression based upon the history of disease progression and patient-level risk factors.<sup>15,17</sup> Periodontitis Stages I-IV are assigned based upon patients' current

clinical presentation of periodontitis, including attachment loss, alveolar bone levels, and tooth loss, and the Stage may be modified by case complexity and need for multidisciplinary care.<sup>15,17</sup> In order to describe the risk of future disease progression, Periodontitis Grades A-C are determined based upon individualized patient risk factors (i.e. smoking status and glycemic control) and direct and/or indirect evidence of disease progression, including the calculation of alveolar bone loss/age.<sup>15,17</sup> The prevalence of periodontitis has been estimated to be over 42% of U.S. adults over 30 years of age.<sup>1</sup> Of those individuals, 7.8% were found to have severe periodontitis.<sup>1</sup> Severe periodontitis was also most prevalent among US adults 65 years or older, Mexican Americans, non-Hispanic blacks, and current heavy (>10 cigarettes daily) smokers.<sup>1</sup> Among US adults, periodontitis prevalence is nearly 4-fold greater than that of diabetes mellitus<sup>18</sup> and over 6-fold greater than that of coronary artery disease,<sup>19</sup> making it extremely common within the population. Periodontal disease progression and destruction of both hard and soft tissues occurs through host-mediated inflammatory pathways,<sup>20</sup> which may vary based upon genetic and environmental risk factors, potentially including nutritional factors.<sup>2,20-23</sup> It should also be noted that a number of systemic diseases that are influenced by diet and nutritional intake, including diabetes mellitus, cardiovascular disease, and obesity have been associated with periodontal disease development or progression.<sup>24-26</sup>

The process of periodontal tissue destruction is mediated by pro-inflammatory cytokines and mediators such as interleukin-1 $\beta$  (IL-1 $\beta$ ), interleukin-6 (IL-6), tumor necrosis factor- $\alpha$  (TNF- $\alpha$ ), prostaglandin E2 (PGE2), receptor activator of nuclear factor kappa B ligand (RANKL), and matrix metalloproteinases (MMPs).<sup>2,9,10</sup> These pro-inflammatory mediators drive the activation of osteoclastic functions and lead to alveolar bone loss. The heterogeneity of the immune-inflammatory response among individuals can influence disease susceptibility and severity.<sup>2</sup> Additionally, periodontal disease severity is correlated to increased levels of pro-inflammatory mediators systemically.<sup>27-30</sup> Because dietary intake can

make a significant impact on systemic levels of inflammation, nutrition has the potential to influence the progression of periodontitis.

### Nutritional Components and their Relation to Periodontal Health and Disease

It is well-established that carbohydrate substrate and cariogenic bacteria are the causative agents for dental caries. The role of diet and nutritional consumption on periodontal health are less well-defined. Nutrients are generally classified as macronutrients or micronutrients and delineated by the amount consumed in a typical diet.<sup>31,32</sup> Macronutrients are consumed in gram quantities and include protein, carbohydrates, and lipids.<sup>31,32</sup> Conversely, micronutrients are required in the diet in milligram or even microgram quantities and include vitamins and minerals.<sup>31,32</sup> Humans also require adequate consumption of water for health and optimal functioning.<sup>31,32</sup> A variety of nutrients have been identified as having a major impact on periodontal health.<sup>33-36</sup> We will discuss the impacts of macronutrients and select micronutrients on periodontal health and disease (Figure 1).

### Macronutrients

Macronutrients required for optimal human processes include protein, carbohydrates, and lipids. Current recommendations allow for more flexibility based upon individualized dietary needs and suggest that adults consume 45% to 65% of their total calories from carbohydrates, 20% to 35% from fat, and 10% to 35% from protein.<sup>37,38</sup> Protein is the most common non-water substance in the body, making up 50% of dry weight of humans.<sup>31,32</sup> In the periodontium, proteins are present as structural proteins, such as collagen, and enzymes.<sup>31,32</sup> When proteins are consumed, they are subsequently broken down into the component amino acids.<sup>31,32</sup> Overall, 22 amino acids are required for protein synthesis and nine are considered essential amino acids (i.e. histidine, isoleucine, leucine, lysine, methionine, phenylalanine, threonine, tryptophan, and valine).<sup>31,32</sup> Major dietary sources for dietary protein are milk, meat, eggs, and legumes.<sup>31,32</sup> Carbohydrates are used primarily as a source of energy and also aid in fat metabolism.<sup>31,32</sup> In periodontal tissues, carbohydrates are found as glycoproteins and glycosaminoglycans, which are required for the synthesis of the ground substances of connective tissues such as chondroitin, keratin,

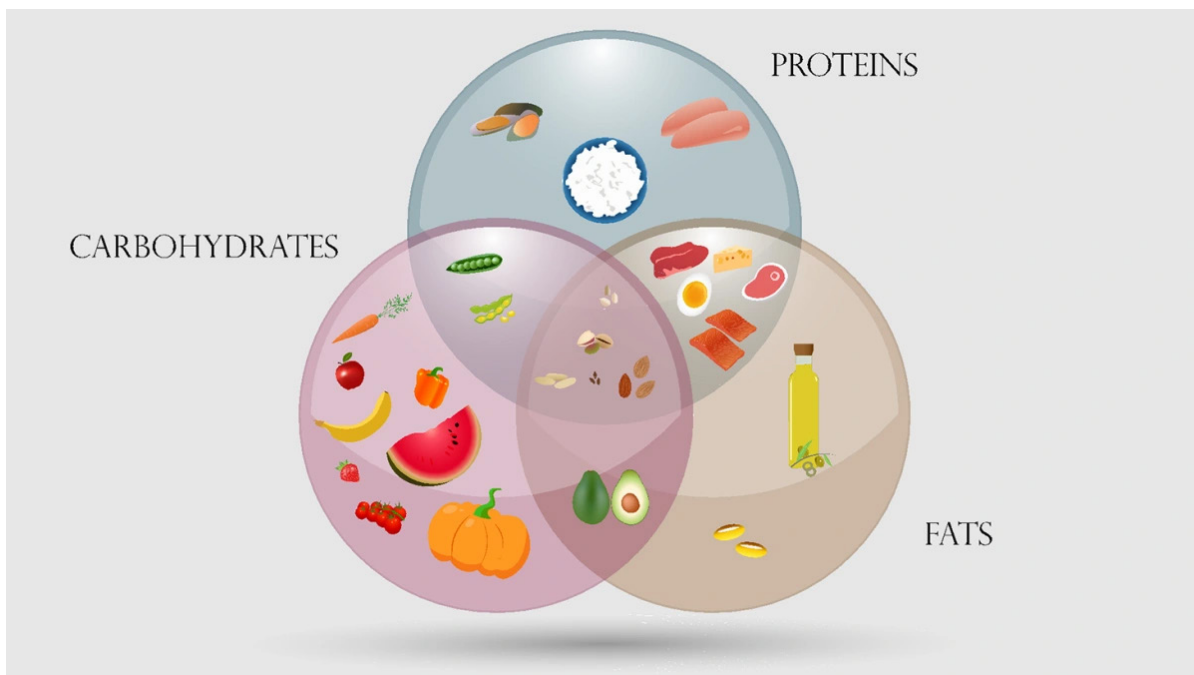


Figure 1. Macronutrients in common foods.

dermatan sulfates.<sup>31,32</sup> Glucose is also required for erythrocyte and neurological functioning.<sup>31,32</sup> Lipids provide energy, energy storage, and thermal insulation.<sup>31,32</sup> Humans require two essential fatty acids: linoleic and linolenic acid.<sup>31,32</sup> Fats are also required for the absorption of fat-soluble vitamins (i.e. Vitamins A, D, E, and K).<sup>31,32</sup> When used for energy, 1 gram of protein or carbohydrates provide 4 kilocalories (kcal) of energy whereas 1 gram of lipid provides 9kcal of energy.<sup>31,32</sup>

An overall excess in calorie consumption can result in several adverse outcomes, including insulin resistance, excess glucose production, and increased adiposity.<sup>39,40</sup> When caloric intake exceeds overall energy needs, metabolic pathways are activated to induce insulin metabolism and lipoprotein synthesis in the liver and, ultimately, increases in free fatty acids and a decrease in lipolysis, which—when sustained—can result in increased adiposity.<sup>40</sup> As adipose tissue deposits increase, a concomitant increase in pro-inflammatory cytokine production and, further, an increase in the production in reactive oxygen species (ROS) and markers of systemic inflammation, like C-reactive protein.<sup>40</sup>

High levels of carbohydrate consumption (defined as > 45% of total caloric intake) have been associated with an increased periodontal disease prevalence and increased gingival inflammation.<sup>41-43</sup> Gingival inflammation and risk of periodontal disease progression was also reduced when carbohydrate consumption was restricted.<sup>43</sup> It is also important to note that refined carbohydrates (e.g. sugars) and other carbohydrates (fiber and sugar alcohols) have divergent effects on periodontal health. Excessive consumption of refined carbohydrates promote increased microbial dysbiosis and, thus, periodontal disease progression.<sup>44,45</sup> Free sugars also act on cells in the periodontal ligament inducing apoptosis and decreasing proliferation.<sup>46</sup> Conversely, fiber and sugar alcohols (e.g. xylitol) may have a protective effect on the periodontium.<sup>42,47-49</sup> Xylitol has been shown to demonstrate an antimicrobial effect on periodontal pathogens like *Porphyromonas gingivalis* (P.g.) and *Aggregatibacter actinomycetemcomitans* (A.a.).<sup>47-49</sup>

Analysis of the National Health and Nutrition Examination Survey (NHANES) data demonstrated a positive association between low fat intake (<23.2% of total caloric intake) and periodontal disease progression.<sup>50</sup> However, the nature of the fat consumed is also a significant consideration for the impact on periodontal health.<sup>51</sup> A diet high in anti-inflammatory omega-3 fatty acids and/or with a favorable omega-3 to omega-6 fatty acid ratio has been demonstrated to promote periodontal health.<sup>43,44,52</sup> Conversely, high dietary consumption of saturated fatty acids or an unfavorable omega-3 to omega-6 fatty acid ratios can promote increased inflammation and a progression of the inflammatory process in periodontal disease.<sup>43,44,52,53</sup>

### **Micronutrients**

Micronutrients consist of vitamins, minerals and trace elements. While micronutrients do not have energy value, they are essential for many biological processes including those that promote periodontal health and/or disease.<sup>54</sup> Figure 2 summarizes micronutrients associated with oral health.

### **Vitamin A Complex**

Beta-carotene, the naturally occurring pigment responsible for red, orange, and yellow colors in many fruits and vegetables, is a precursor to vitamin A, also known as retinol. Beta-carotene can act as a scavenging antioxidant to destroy free radicals and may also promote increased integrity of epithelial cells and cell-to-cell attachments.<sup>54</sup> Considering the antioxidant function of beta-carotene, it has been studied to determine the association between periodontitis and beta-carotene with findings suggesting that increased beta-carotene consumption was associated with decreased periodontitis prevalence.<sup>55</sup> Further, beta carotene has been associated with improved healing in nonsmokers after nonsurgical therapy, including greater reductions in probing depth.<sup>56</sup> These findings suggest that consumption of beta-carotene may improve antioxidant capacity and may significantly improve periodontal health.<sup>56</sup> Beta-carotene deficiency is also associated with increased periodontitis prevalence and gingival bleeding.<sup>56</sup>



Micronutrient	Recommended Adult (19-64) Dietary Allowance (UK NHS)	Common Sources
Vitamin A	0.7mg (Men) 0.6mg (Women)	Meat (liver), dairy, red/orange/yellow vegetables
Vitamin B12 (Cobalamin)	1.5µg	Fortified cereals, meat, fish, carrots
Vitamin B9 folate	200µg	Leafy greens, fortified cereals
Vitamin C	40mg	Fruits and vegetables
Vitamin D	10µg	Fortified cereals, mushrooms, fish
Vitamin E	4mg (Men) 3mg (Women)	Fortified cereals, nuts, seeds
Calcium	700mg	Fortified cereals, dairy products
Magnesium	300mg (Men) 270mg (Women)	Cereals, seeds, nuts
Iron	8.7mg (Men and Women >50yo) 14.8mg (Women <50yo)	Meat
Zinc	9.5mg (Men) 7.0mg (Women)	Nuts, fortified cereals, meat
Potassium	3500mg	Fruits, vegetables, nuts
Copper	1.2mg	Pulses, seeds, nuts

**Figure 2.** Micronutrient sources and recommended daily intake.

### Vitamin C/Ascorbic acid

Vitamin C has long been known to be associated with gingival health. This association between vitamin C and oral health was first described in the scientific literature in what has been called the first randomized controlled trial.<sup>57</sup> This study identified a lack of vitamin C consumption as the causative deficiency for scurvy in British sailors.<sup>57</sup> Scurvy, which is now known to be extreme vitamin C deficiency, was a frequent ailment amongst sailors who lacked access to vitamin C-containing foods during long voyages at sea. Due to scurvy's association with severe gingival bleeding and tooth mobility, vitamin C deficiency has been postulated to play a role in gingivitis. A well-balanced nutrient-rich 7-day diet that omitted vitamin C, resulted in no changes in plaque index or probing depths but increased bleeding on probing was noted.<sup>58</sup> It has been established that vitamin C enhances collagen synthesis and protects against tissue damage by scavenging reactive oxygen species (ROS).<sup>58</sup> Studies have demonstrated an inverse relationship between periodontal disease prevalence and serum vitamin C concentrations and this relationship is more

pronounced in individuals with more severe forms of periodontal disease and in never-smokers.<sup>59</sup> Additionally, consumption of whole food sources of vitamin C (e.g., grapefruit) for two weeks in vitamin C deficient individuals resulted in increased plasma vitamin C levels and improved sulcular bleeding scores.<sup>59</sup> Vitamin C may also blunt the cytotoxic effects of *Porphyromonas gingivalis* on human gingival fibroblasts *in vitro*.<sup>59-61</sup> These findings suggest that dietary vitamin C consumption may play an important role in promoting improved periodontal health and outcomes of periodontal therapy.

### Vitamin E

Vitamin E (tocopherol) is a fat-soluble vitamin that has been identified as a key extracellular antioxidant and has been suggested to improve periodontal treatment outcomes.<sup>62</sup> Dietary sources include: poultry, meat, fish, nuts, seeds, and cereal grains.<sup>63</sup> Serum levels of saturated vitamin E, are negatively associated with clinical signs of periodontal disease including: probing depths and overall assessment of periodontal disease severity.<sup>64</sup> Dietary supplementation with vitamin E results

in a reduction of bleeding upon probing and periodontal inflammation.<sup>65</sup> After nonsurgical periodontal therapy, increased dietary intake of vitamins A, B, C, E combined with with omega-3-fatty acid intake resulted in improved healing in nonsmokers but not in smokers when compared to those who did not take supplements.<sup>56</sup> These preliminary findings may indicate a role for vitamin E supplementation during the periodontal treatment to enhance periodontal outcomes, particularly in some patient populations.

### **Vitamin B Complex**

The vitamin B complex refers to eight water-soluble vitamins, which together perform functions essential to the body including cell metabolism, repair, and proliferation.<sup>32</sup> These vitamins include thiamine (B1), riboflavin (B2), niacin (B3) pantothenic acid (B5), pyridoxine (B6), biotin (B7 or B8), folate (B9), and cobalamin (B12).<sup>32</sup> Vitamin B complex deficiencies demonstrate a range of symptoms from dermatitis to paresthesia and include oral manifestations such as angular cheilitis and glossitis.<sup>32</sup> Vitamins in the B complex may also play a role in periodontal disease progression and severity. B2, B3, B6, B12 deficiencies have been linked to hemorrhagic gingivitis and periodontitis.<sup>66</sup> These vitamins support healthy immune functions by strengthening epithelial barriers and cellular and humoral immune responses.<sup>66</sup> Vitamin B complex supplementation is associated with statistically significantly higher mean clinical attachment gain at shallow and deep periodontal pockets after periodontal therapy.<sup>67</sup> While heterogeneity exists in clinical investigations, the direct effect of these micronutrients may be influenced by other factors like age and smoking status, which can obscure results.<sup>67</sup> Additional research to assess the role of the Vitamin B-complex and periodontal health is needed.

### **Vitamin D and Calcium**

Vitamin D is required for a number of essential functions of the human body, including its role in the enhanced resorption of minerals including calcium, magnesium, iron, phosphate and zinc.<sup>32</sup> While the role of

vitamin D in regulation of plasma calcium and phosphorus levels for bone metabolism has long been established, it is also essential for cell development, neuromuscular functions, and inflammatory system modulation.<sup>68</sup> Vitamin D has also been found to inhibit pro-inflammatory cytokines and T-lymphocyte proliferation.<sup>68</sup> Vitamin D is unique among the vitamins discussed in this course in that rather than naturally occurring in dietary sources, sunlight exposure is the most common source of vitamin D.<sup>68</sup> Vitamin D and Vit-D Receptor complex interact with receptor activator of nuclear factor Kappa-B ligand (RANKL) expression and downregulate osteoprotegerin (OPG), thereby increasing differentiation and activation of osteoclasts and consequently bone resorption.<sup>68</sup> When vitamin D levels become low, parathyroid hormone indirectly stimulates bone resorption in order to increase vitamin D levels, so increased vitamin D intake may reduce bone resorption.<sup>68</sup> Despite its role in mineral absorption and metabolism, equivocal study results have not established a definitive association between dietary vitamin D deficiency and periodontal health and/or post-treatment periodontal healing in the general population.<sup>32</sup> Local administration, however, of vitamin D has demonstrated enhanced bone turnover in clinical scenarios. For example, dental implants coated with vitamin D3 have demonstrated enhanced osseointegration<sup>69</sup> and intraperitoneal injections of vitamin D3 have been shown to accelerate orthodontic tooth movement.<sup>70,71</sup>

It should be noted that co-supplementation of calcium and vitamin D has demonstrated a positive effect on the outcomes of periodontal therapy<sup>72</sup> and in two studies of dietary consumption in a Danish population, higher intake of dairy products high in calcium and vitamin D has been associated with decreased periodontal disease severity.<sup>73,74</sup>

### **Minerals and Trace Elements**

Balanced levels of minerals and trace elements are essential for optimal host immune responses and may prevent progression of chronic conditions such as periodontitis.<sup>32,55</sup> Iron (Fe++) is the most abundant essential trace element with many functions in the human

body and is found primarily in blood.<sup>75</sup> Iron is also required for the synthesis of enzymes and plays a role in the innate and adaptive immune responses.<sup>32,75</sup> The recommended daily allowance for iron varies with age and sex with highest levels recommended for women of reproductive age.<sup>75</sup> While direct evidence linking iron deficiencies to periodontitis development is scant, inflammation from periodontitis may result in increased pro-inflammatory cytokines, which then may suppress erythropoiesis in bone marrow and lead to periodontal disease progression.<sup>32,76</sup>

Other essential trace minerals, including selenium (Se), magnesium (Mg), zinc (Zn), and copper (Cu), have antioxidant enzymes that can aid in neutralizing ROS and prevent tissue damage. They also play important roles in regulating immune function and wound healing.<sup>77</sup> In particularly vulnerable patient populations, adequate levels of these minerals may be critically important. For example, zinc has been identified as a potential factor in preventing diabetes-related periodontal disease progression.<sup>78,79</sup> Therefore, achieving ideal levels of these micronutrient minerals may be a critical component in periodontal care.<sup>77</sup>

### **Nutritional Counseling in the Dental Practice**

Dental healthcare professionals have long established that nutritional counseling for caries prevention is a critical part of our preventative mission. However, emerging data suggest that the role of nutrition in the development, disease progression, and healing potential after periodontal therapy may be significant and should be considered in a dietary analysis to develop a risk assessment for all oral diseases. Tools such as the Rapid

Eating Assessment for Participants (short version) (REAP-S) can be utilized chairside to assess a patients' nutritional intake and may be particularly informative in assessing the overall impact of macronutrients on oral health (Figure 3).<sup>80,81</sup> More targeted assessments and/or laboratory testing may be recommended in patients with other signs of micronutrient deficiencies or in high risk individuals (e.g., Stage IV or Grade C patients). Further, dietary recommendations that include the consumption of more whole food nutritional sources and less processed foods can positively impact appropriate consumption of macronutrients and micronutrients. The current findings also suggest that a diet that includes lower proportions of refined carbohydrates, including more carbohydrates from whole food sources and fiber, higher levels of omega-3 fatty acids, high in casein and whey proteins, and an inclusion of foods with antioxidant properties may promote more optimal periodontal health.

### **Summary**

Healthy dietary habits, including avoiding a caloric surplus, may be beneficial to oral and systemic health. Further, specific macronutrients and micronutrients may enhance periodontal health and healing. Periodontal diseases are microbially-induced diseases that are propagated by host-mediated inflammation that results in disease progression and loss of periodontal tissues. The interaction between nutritional intake and the overall host inflammatory state may allow for improved outcomes with modification of dietary intake. In some instances, dietary changes and/or vitamin or mineral supplementation may improve outcomes for patients with periodontal diseases.



**REAPS (Rapid Eating Assessment for Participants - Shortened Version)**  
**CJSegal-Isaacson, EdD RD, Judy-Wylie-Rosett, EdD RD, Kim Gans, PhD, MPH**

In an average week, how often do you:	Usually/ Often	Sometimes	Rarely/ Never	Does not apply to me	
1. Skip breakfast?	○	○	○		
2. Eat <u>4 or more</u> meals from sit-down or take out restaurants?	○	○	○		
3. Eat <u>less than 2 servings</u> of whole grain products or high fiber starches a day? <b>Serving</b> = 1 slice of 100% whole grain bread; 1 cup whole grain cereal like Shredded Wheat, Wheaties, Grape Nuts, high fiber cereals, oatmeal, 3-4 whole grain crackers, ½ cup brown rice or whole wheat pasta, boiled or baked potatoes, yuca, yams or plantain.	○	○	○		
4. Eat <u>less than 2 servings</u> of fruit a day? <b>Serving</b> = ½ cup or 1 med. fruit or ¾ cup 100% fruit juice.	○	○	○		
5. Eat <u>less than 2 servings</u> of vegetables a day? <b>Serving</b> = ½ cup vegetables, or 1 cup leafy raw vegetables.	○	○	○		
6. Eat or drink <u>less than 2 servings</u> of milk, yogurt, or cheese a day? <b>Serving</b> = 1 cup milk or yogurt; 1½ - 2 ounces cheese.	○	○	○		
7. Eat <u>more than 8 ounces</u> (see sizes below) of meat, chicken, turkey or fish <u>per day</u> ? <b>Note:</b> 3 ounces of meat or chicken is the size of a deck of cards or ONE of the following: 1 regular hamburger, 1 chicken breast or leg (thigh and drumstick), or 1 pork chop.	○	○	○	Rarely eat meat, chicken, turkey or fish  ○	
8. Use <u>regular processed meats</u> (like bologna, salami, corned beef, hotdogs, sausage or bacon) instead of low fat processed meats (like roast beef, turkey, lean ham; low-fat cold cuts/hotdogs)?	○	○	○	Rarely eat processed meats  ○	
9. Eat <u>fried foods</u> such as fried chicken, fried fish, French fries, fried plantains, tostones or fried yuca?	○	○	○		
10. Eat <u>regular potato chips, nacho chips, corn chips, crackers, regular popcorn, nuts</u> instead of pretzels, low-fat chips or low-fat crackers, air-popped popcorn?	○	○	○	Rarely eat these snack foods  ○	
11. <u>Add butter, margarine or oil</u> to bread, potatoes, rice or vegetables at the table?	○	○	○		
12. Eat <u>sweets</u> like cake, cookies, pastries, donuts, muffins, chocolate and candies more than 2 times per day.	○	○	○		
13. <u>Drink 16 ounces or more</u> of non-diet soda, fruit drink/punch or Kool-Aid a day? <b>Note:</b> 1 can of soda = 12 ounces	○	○	○		
	<b>YES</b>			<b>NO</b>	
14. You or a member of your family usually shops and cooks rather than eating sit-down or take-out restaurant food?	○			○	
15. Usually feel well enough to shop or cook.	○			○	
16. How willing are you to make changes in your eating habits in order to be healthier?	1 Very willing	2	3	4	5 Not at all willing

Figure 3. REAPS (Rapid Eating Assessment for Participants – shortened version)<sup>80,81</sup>

## Course Test Preview

To receive Continuing Education credit for this course, you must complete the online test. Please go to: [www.dentalcare.com/en-us/ce-courses/ce664/test](http://www.dentalcare.com/en-us/ce-courses/ce664/test)

- 1. Approximately \_\_\_\_\_ of U.S. adults over 30 years old suffer from periodontal disease.**
  - A. 24%
  - B. 38%
  - C. 42%
  - D. 65%
  
- 2. It is estimated that approximately \_\_\_\_\_ of the US population has at least one clinically significant nutritional deficiency.**
  - A. 1%
  - B. 5%
  - C. 10%
  - D. 50%
  
- 3. In longitudinal studies the average tooth loss due to periodontal disease is approximately \_\_\_\_\_ teeth lost annually.**
  - A. 0.2
  - B. 0.5
  - C. 1.0
  - D. 2.0
  
- 4. All of the following are pro-inflammatory cytokines involved in periodontal tissue destruction EXCEPT one, which is the exception?**
  - A. interleukin-1 $\beta$  (IL-1 $\beta$ )
  - B. interleukin-6 (IL-6)
  - C. tumor necrosis factor- $\alpha$  (TNF- $\alpha$ )
  - D. interleukin-4 (IL-4)
  
- 5. Consider the following two statements:**

**Nutrients are generally classified as macronutrients or micronutrients and delineated by the amount consumed in a typical diet.**

**Micronutrients are required in the diet in gram quantities and include vitamins and minerals.**

  - A. Both statements are true
  - B. The first statement is true, the second statement is false
  - C. The first statement is false, the second statement is true
  - D. Both statements are false
  
- 6. All of the following are macronutrients EXCEPT one, which is the exception?**
  - A. Proteins
  - B. Carbohydrates
  - C. Water
  - D. Lipids

- 7. Protein is the most common non-water substance in the body, making up \_\_\_\_\_ of dry weight of humans.**
- A. 20%
  - B. 35%
  - C. 50%
  - D. 75%
- 8. How many amino acids are considered essential?**
- A. Six
  - B. Nine
  - C. Fifteen
  - D. Twenty-two
- 9. When used for energy, 1 gram of protein or carbohydrates provide 4kcal of energy whereas 1 gram of lipid provides \_\_\_\_\_ kcal of energy.**
- A. 5
  - B. 7
  - C. 9
  - D. 12
- 10. Which sugar alcohol has been shown to demonstrate an antimicrobial effect on periodontal pathogens like Porphyromonas gingivalis (P.g.) and Aggregatibacter actinomycetemcomitans (A.a.)?**
- A. Xylitol
  - B. Sorbitol
  - C. Erythritol
  - D. Mannitol
- 11. Which of the following lipids has been associated with improved periodontal health?**
- A. Omega-3 fatty acids
  - B. Omega-6 fatty acids
  - C. Monounsaturated fatty acids
  - D. Saturated fatty acids
- 12. When vitamin C is omitted from an otherwise well-balanced diet for 7 days, which of the following results?**
- A. Decreased plaque index
  - B. Increased probing depths
  - C. Increased tooth mobility
  - D. Increased bleeding on probing

**13. Consider the following two statements:**

**Serum levels of saturated vitamin E, are associated with increased clinical signs of periodontal disease including: probing depths and overall assessment of periodontal disease severity.**

**Dietary supplementation with vitamin E results in more bleeding upon probing and periodontal inflammation.**

- A. Both statements are true
- B. The first statement is true, the second statement is false
- C. The first statement is false, the second statement is true
- D. Both statements are false

**14. After periodontal therapy, supplementation with vitamin B complex results in \_\_\_\_\_ mean clinical attachment gain at shallow and deep periodontal pockets.**

- A. Greater
- B. No difference in
- C. Less
- D. Inconsistent

**15. Consider the following two statements:**

**Dental implants coated with vitamin D3 have demonstrated enhanced osseointegration.**

**Interperitoneal injections of vitamin D3 have been shown to accelerate orthodontic tooth movement.**

- A. Both statements are true
- B. The first statement is true, the second statement is false
- C. The first statement is false, the second statement is true
- D. Both statements are false

**16. Which of the following trace elements is the most common in the human body?**

- A. Zinc
- B. Iron
- C. Selenium
- D. Copper

**17. Which of the following is a nutritional assessment tool that can be used chairside in the dental office?**

- A. Quick Intake Patient Review (QIPR)
- B. Rapid Eating Assessment for Participants (short version) (REAP-S)
- C. Nutritional Intake Measure for Dentistry (NIMD)
- D. Dietary Intake Evaluation Tool (DIET)

**18. Consider the following two statements:**

**Nutritional assessment allows dental healthcare professionals to make targeted recommendations for patients to promote oral and systemic wellness.**

**For patients at a high risk of periodontitis (e.g., Stage III or IV and/or Grade C Periodontitis) or who present with signs of micronutrient deficiencies, advanced laboratory testing may be warranted.**

- A. Both statements are true
- B. The first statement is true, the second statement is false
- C. The first statement is false, the second statement is true
- D. Both statements are false

**19. Current evidence suggests that all of the following nutritional recommendations could have a positive influence on periodontal health EXCEPT one, which is the exception?**

- A. Limit refined carbohydrates
- B. High casein and whey protein intake
- C. Inclusion of foods with antioxidant properties
- D. Higher omega-6 fatty acid intake



## References

1. Eke PI, Thornton-Evans GO, Wei L, et al. Periodontitis in US Adults: National Health and Nutrition Examination Survey 2009-2014, *J Am Dent Assoc.* 2018;149(7):576-588.e6.
2. Kornman KS, Page RC, Tonetti MS. The host response to the microbial challenge in periodontitis: assembling the players. *Periodontol 2000.* 1997 Jun;14:33-53.
3. Martin-Hadmas RM, Martin SA, Romonti A, Marginean CO. The effect of dietary intake and nutritional status on anthropometric development and systemic inflammation: An observational study. *Int J Environ Res Pub Health* 2021;18(11):5635.
4. Raindi D. Nutrition and Periodontal Disease. *Dent Update* 2016;43:66-68, 71-62.
5. Hujoel PP, Lingström P. Nutrition, dental caries and periodontal disease: a narrative review. *J Clin Periodontol* 2017;44(Suppl 18):S79-s84.
6. US Department of Health and Human Services. US Department of Agriculture. 2015–2020 dietary guidelines for Americans. Skyhorse Publishing Inc., 2017. Accessed June 19, 2023.
7. US Centers for Disease Control and Prevention. Second national report on biochemical indicators of diet and nutrition in the US population 2012. Atlanta (GA), National Center for Environmental Health, 2012. Accessed June 19, 2023.
8. Gondivkar SH, Gadbail AR, Gondivkar RS, Sarode SC, Sarode GS, Patil S, Awan KH Nutrition and Oral Health Disease-a-Month; 2019; 65(6): 147-154.
9. Flemmig TF. Periodontitis. *Ann Periodontol* 1999; 4: 32-37.
10. Page RC and Schroeder HE. Pathogenesis of inflammatory periodontal disease. A summary of current work. *Lab Invest* 1976; 33: 235-249.
11. Chapple ILC, Mealey BL, Van Dyke TE, et al. Periodontal health and gingival disease and conditions on an intact and a reduced periodontium: Consensus report of workgroup 1 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. *J Periodontol* 2018; 89(Suppl 1): S74-S84.
12. Chapple ILC, Van der Weijden F, Doerfer C, et al. Primary prevention of periodontitis: managing gingivitis. *Proceedings of the 11th European Workshop on Periodontology.* *J Clin Periodontol* 2015; 42(Spec Iss 16): S71-S76.
13. Mombelli A. Microbial colonization of the periodontal pocket and its significance for periodontal therapy. *Periodontol 2000.* 2018 Feb;76(1):85-96. doi: 10.1111/prd.12147. *Epub* 2017 Nov 30.
14. AAP Glossary of Terms. Periodontitis. Accessed June 19, 2023.
15. Papapanou PN, Sanz M, Buduneli N, et al. Periodontitis: Consensus report of workgroup 2 of the 2017 Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. *J Periodontol* 2018; 89(Suppl 1): S173-S182.
16. Needleman I, Garcia R, Gkraniias N, et al. Mean annual attachment, bone level, and tooth loss: A systematic review. *J Periodontol* 2018; 89(Suppl): S120-S139.
17. Tonetti MS, Greenwell H, Kornman KS. Staging and grading of periodontitis: Framework and proposal of a new classification and case definition. *J Periodontol* 2018; 89(Suppl 1): S159-S172.
18. Centers for Disease Control and Prevention (CDC). National Diabetes Statistics Report 2020. Estimates of Diabetes and its Burden in the United States. Accessed June 19, 2023.
19. Centers for Disease Control and Prevention (CDC). Heart Disease Facts. Accessed June 19, 2023.
20. Löe H, Anerud A, Boysen H, Smith M. The natural history of periodontal disease in man. The rate of periodontal destruction before 40 years of age. *J Periodontol* 1978; 49: 607-620.
21. Michalowicz BS, Aeppli DP, Kuba RK, et al., A twin study of genetic variation in proportional radiographic alveolar bone height. *J Dent Res* 1991; 70: 1431-1435.
22. Michalowicz BS, Aeppli DP, Virag JG et al. Periodontal findings in adult twins. *J Periodontol* 1991; 62: 293-299.
23. Kornman K, Crane A, Wang H. The interleukine-1 genotype as a severity factor in adult periodontal disease. *J Clin Periodontol* 1997; 24: 72-77.

24. Preshaw PM, Alba AL, Herrera D, et al. Periodontitis and diabetes: A two-way relationship. *Diabetologica* 2012; 10: 257-265.
25. Hujoel PP, Drangsholt M, Spiekerman C, DeRouen TA. Periodontal disease and cardiovascular disease. *Periodontol 2000* 2013; 61:160-176.
26. Beck J, Garcia R, Heiss G, Vokonas PS, Offenbacher S. Periodontal disease and cardiovascular disease. *J Periodontol* 1996; 67:1123-1137.
27. Winning, L., Linden, G. Periodontitis and systemic disease. *BDJ Team* 2, 15163 (2015).
28. Loos BG. Systemic markers of inflammation in periodontitis. *J Periodontol* 2005; 76: 2106-2115.
29. Noack B, Genco RJ, Tevisan M, Grossi S, Zambon JJ, De Nardin E. Periodontal infections contribute to elevated C-reactive protein level. *J Periodontol* 2005; 76: 2075-2084.
30. Loos BG, Craandijk J, Hoek FJ, Weterhin-van Dillen PME, van der Velden W. C-reactive protein and other markers of systemic inflammation in relation to cardiovascular diseases are elevated in periodontitis. *J Periodontol* 2000; 71: 1528-1534.
31. Schifferle RE. Nutrition and periodontal disease. *Dent Clin N Am* 2005; 49:595-610.
32. Najeeb S, Zafar MS, Khurshid Z, Zohaib S, Almas K. The role of nutrition in periodontal health: An update. *Nutrients* 2016; 8:530 doi:10.3390/nu8090530
33. Ritchie CS, Kinane DF. Nutrition, inflammation and periodontal disease. *Nutrition* 2003; 19:475.
34. Dodington DW, Fritz PC, Sullivan PJ, Ward WE. Higher intakes of fruits and vegetables, beta-carotene, Vitamin C,  $\alpha$ -tocopherol, EPA, and DHA are positively associated with periodontal healing after nonsurgical periodontal therapy in nonsmokers and smokers. *J Nutr* 2015; 145:2512-2519.
35. Antonoglou G, Knuuttila M, Niemela O, et al., Low serum level of 1,25(OH)<sub>2</sub>D is associated with chronic periodontitis. *J Periodontal Res* 2015; 50:274-280.
36. Hujoel PP, Lingstrom P. Nutrition, dental caries and periodontal disease: a narrative review. *J Clin Periodontol* 2017;44(Suppl 18):S79-S84.
37. Ryan-Harshman M, Aldoori W. New dietary reference intakes for macronutrients and fibre. *Can Fam Physician*. 2006;52(2):177-179.
38. Food and Nutrition Board, Institute of Medicine. Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids. Washington, DC: National Academy Press; 2002.
39. Raindi D. Nutrition and periodontal disease. *Dent Update* 2016;43:66-68.
40. Chapple IL. Potential mechanisms underpinning the nutritional modulation of periodontal inflammation. *J Am Dent Assoc* 2009;140:178-184.
41. Feinman RD, Pogozelski WK, Astrup A, et al. Dietary carbohydrate restriction as the first approach in diabetes management: Critical review and evidence base. *Nutrition* 2015; 31:1-13.
42. Lula ECO, Ribeiro CCC, Hugo FN, Alves CMC, Silva AAm. Added sugars and periodontal diseases in young adults: An analysis of NHANES III *Am J Clin Nutr* 2014; 11:1182-1187.
43. Wobler JP, Bremer K, Vach K, Konig D, Hellwig E, Ratka-Kruger P, Al-Ahmad A, Tennert C. An oral health optimized diet can reduce gingival and periodontal inflammation in humans A randomized controlled pilot study. *BMC Oral Health* 2017; 17.
44. Bosma-den Boer MM, VanWetten ML, Pruijboom L. Chronic inflammatory diseases are stimulated by current lifestyle: How diet, stress levels, and medication prevent our body from recovering. *Nutr Metab* 2012;9:32.
45. Adler CJ, Dobney K, Weyrich LS, et al. Sequencing ancient calcified dental plaque shows changes in oral microbiota with dietary shifts of the Neolithic and industrial revolutions. *Nat Genet* 2013; 45:450-455.
46. Liu J, Jiang Y, Mao J, Gu B, Liu H, Fang B. High levels of glucose induces a dose-dependent apoptosis in human periodontal ligament fibroblasts by activating caspase-3 signaling pathway. *Appl Biochem Biotechnol* 2013; 170:1458-1471.

47. Park E, Na HS, Kim SM, Wallet S, Cha S, Chung J. Xylitol, an anticaries agent, exhibits potent inhibition of inflammatory responses in THP-1 derived macrophages infected with porphyromonas gingivalis J Periodontol 2014; 85, e212-223.
48. Keukenmeester RS, Slot DE, Rosema NAM, Van Loveren C, Van der Weijden GA. Effects of sugar-free chewing gum sweetened with Xylitol or Malitol on the development of gingivitis and plaque: A randomized clinical trial. Int J Dent Hyg 2014; 12: 238-244.
49. Kim S, Park MH, Song YR, Na HS, Chung J. Aggregatibacter Actinomycetemcomitans-induced AIM2 inflammasome activation is suppressed by Xylitol in Differentiated THP-1 Macrophages. J Periodontol 2016; 87: e116-e126.
50. Forouhi NG, Krauss RM, Taubes G, Willett W. Dietary fat and cardiometabolic health: evidence controversies, and consensus for guidance. BMJ 2018; 361:k2139.
51. Hamasaki T, Kitamura M, Kawashita Y, Ando Y, Saito T. Periodontal disease and percentage of calories from fat using national data. J Periodont Res 2017; 52: 114-121.
52. Varela-Lopez A, Giampieri F, Bullon P, Battino M, Quiles JL. The role of lipids in the onset, progression, and treatment of periodontal disease. A systematic review of studies in humans. Int J Mol Sci 2016; 17, 1202.
53. Milward MR, Chapple I. The role of diet in periodontal disease Clin Dent Health 2013; 52:18-21.
54. Martinon P, Fraticelli L, Giboreau A, Dussart C, Bourgeois D, Carrouel F. Nutrition as a key modifiable factor for periodontitis and main chronic diseases. J Clin Med 2021; 10:197.
55. Gaur S, Agnihotri R. Trace mineral micronutrients and chronic periodontitis—a review. Biol Trace Elem Res. 2017;176(2):225-38.
56. Dodington DW, Fritz PC, Sullivan PJ, Ward WE. Higher intakes of fruits and vegetables, β-carotene, vitamin C, α-tocopherol, EPA, and DHA are positively associated with periodontal healing after nonsurgical periodontal therapy in nonsmokers but not in smokers. J Nutr. 2015;145(11):2512-9.
57. Lind J. A treatise of the scurvy. In three parts. Containing an inquiry into the nature, causes, and cure, of that disease. Together with a critical and chronological view of what has been published on the subject. 1753. Edinburgh. Printed by Sands, Murray, and Cochran for A Kincaid and A Donaldson.
58. Leggott PJ, Robertson PB, Rothman DL, Murray PA, Jacob RA. The effect of controlled ascorbic acid depletion and supplementation on periodontal health. J Periodontol. 1986;57(8):480-5.
59. Haffajee AD, Socransky SS, Patel MR, Song X. Microbial complexes in subgingival plaque. J Clin Periodontol. 1998;25(2):134-44.
60. Staudte H, Sigusch BW, Glockmann E. Grapefruit consumption improves vitamin C status in periodontitis patients. Br Dent J. 2005;199(4):213-7.
61. Staudte H, Güntsch A, Völpel A, Sigusch BW. Vitamin C attenuates the cytotoxic effects of Porphyromonas gingivalis on human gingival fibroblasts. Arch Oral Biol. 2010;55(1):40-5.
62. Varela-López A, Navarro-Hortal MD, Giampieri F, Bullón P, Battino M, Quiles JL. Nutraceuticals in periodontal health: a systematic review on the role of vitamins in periodontal health maintenance. Molecules. 2018;23(5):1226.
63. Garcia-Closas R, Berenguer A, Tormo MH, et al. Dietary sources of vitamin C, vitamin E, and specific carotenoids in Spain. Br J Nutr 2004; 91:1005-1011.
64. Zong, G.; Scott, A.E.; Griffiths, H.R.; Zock, P.L.; Dietrich, T.; Newson, R.S. Serum alpha-tocopherol has a nonlinear inverse association with periodontitis among US adults. J Nutr. 2015;145(5):893–899.
65. Willershausen B, Ross A, Försch M, Willershausen I, Mohaupt P, Callaway A. The influence of micronutrients on oral and general health. Eur J Med Res. 2011;16(11):514.
66. Maggini S, Wintergerst ES, Beveridge S, Hornig DH. Selected vitamins and trace elements support immune function by strengthening epithelial barriers and cellular and humoral immune responses. Br J Nutr. 2007;98(S1):S29-35.
67. Neiva RF, Al-Shammari K, Nociti Jr FH, Soehren S, Wang HL. Effects of vitamin-B complex

- supplementation on periodontal wound healing. *J Periodontol.* 2005;76(7):1084-91.
68. Hennig BJ, Parkhill JM, Chapple IL, Heasman PA, Taylor JJ. Association of a vitamin D receptor gene polymorphism with localized early-onset periodontal diseases. *J Periodontol.* 1999;70(9):1032-8.
  69. Javed F, Malmstrom H, Kellesarian SV, Al-Kheraif AA, Vohra F, Romanos FE. Efficacy of Vitamin D3 supplementation on osseointegration of implants. *Implant Dent* 2016; 25:281-287.
  70. Kawakami M, Takano-Yamamoto T. Local injection of 1,25-dihydroxyvitamin D3 enhanced bone formation for tooth stabilization after experimental tooth movement in rats. *J Bone Miner Metab* 2004; 22:541-546.
  71. Kale S, Kocadereli I, Atilla P, Asan E. Comparison of the effects of 1,25-dihydroxycholecalciferol and prostaglandin E2 on orthodontic tooth movement. *Am J Orthod Dentofac Orthop* 2004; 125:607-614.
  72. Garcia MN, Hildebolt CF, Miley DD, et al. One-year effects of vitamin D and calcium supplementation on chronic periodontitis. *J Periodontol* 2011; 82:25-32.
  73. Adegboye AR, Christensen LB, Holm-Pedersen P, Avlund K, Coucher BJ, Heitmann BL. Intake of dairy products in relation to periodontitis in older Danish adults. *Nutrients.* 2012; 4:1219-1229.
  74. Adegboye AR, Boucher BJ, Kongstad J, Fiehn N, Christensen LB, Heitmann BL. Calcium, vitamin D, casein, and whey protein intakes and periodontitis among Danish adults *Public Health Nutr* 2016; 19: 503-510.
  75. Hou J, Yamada S, Kajikawa T, et al. Iron plays a key role in the cytodifferentiation of human periodontal ligament cells. *J Periodontal Res.* 2014;49(2):260-267.
  76. Gokhale SR, Sumanth S, Padhye AM. Evaluation of blood parameters in patients with chronic periodontitis for signs of anemia. *J Periodontol.* 2010;81(8):1202-6.
  77. Apon A, Kamble P. Role of trace minerals in periodontal health: a review. *Clin Trials Degener Dis.* 2019;4(2):30.
  78. Pushparani DS. Serum zinc and  $\beta$  D glucuronidase enzyme level in Type 2 diabetes mellitus with periodontitis. *Curr Diabetes Rev* 2015; 11.
  79. Pushparani D. Zinc and type 2 diabetes mellitus with periodontitis—a systematic review. *Curr Diabetes Rev* 2014; 20:397-401.
  80. Segal-Isaacson CJ, Wylie-Rosett J, Gans KM. Validation of a short dietary assessment questionnaire: the Rapid Eating and Activity Assessment for Participants short version (REAP-S). *Diabetes Educ* 2004; 30:774,776,778.
  81. US Department of Agriculture (USDA) SNAP-Ed Connection. Accessed June 19, 2023.

### Additional Resources

- No Additional Resources

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