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Role of Dietary Habits in the Prevention of Dental Caries

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Abstract:

Background: Dental caries is a diet-influenced infectious disease caused by the interaction between fermentable carbohydrates and acidogenic oral bacteria, leading to enamel demineralization. Secondary factors such as saliva quality, fluoride exposure, and enamel integrity also influence caries development.

Aim: This article examines the role of dietary habits in caries prevention, focusing on cariogenic and cariostatic foods, eating frequency, and dietary interventions to mitigate caries risk.

Methods: A comprehensive review of existing literature was conducted, analyzing studies on sugar metabolism, plaque pH dynamics, food retention properties, and dietary patterns. Key studies, including the Vipeholm Dental Caries Study and research on sugar substitutes, were evaluated.

Results: Frequent consumption of fermentable carbohydrates, particularly sticky and retentive foods, significantly increases caries risk. Conversely, dairy products (especially cheese), sugar alcohols (xylitol), and fibrous foods stimulate saliva and promote remineralization. Dietary frequency and meal sequencing also impact caries development, with between-meal snacking posing the highest risk.

Conclusion: Effective caries prevention requires reducing fermentable carbohydrate intake, promoting protective foods, and integrating dietary counseling into dental care. Public health strategies should emphasize balanced nutrition, fluoride use, and saliva stimulation.

Keywords: Dental caries, fermentable carbohydrates, cariogenic foods, cariostatic foods, dietary prevention, xylitol, saliva stimulation.

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Introduction:

Dental caries is a diet-influenced infectious disease that results primarily from the interaction between specific oral bacteria and fermentable dietary carbohydrates. These bacteria metabolize sugars and starches into organic acids, which then cause demineralization of the enamel surface. The prevalence and severity of dental caries are also significantly shaped by secondary biological and environmental factors such as the quantity and quality of saliva, the availability of fluoride,

and the structural integrity of the enamel layer of the teeth (1). An effective approach to both the prevention and management of caries requires a comprehensive understanding of the interplay between these variables over time. The microbial basis of dental caries is well established in dental literature. Certain species of oral bacteria, notably *mutans streptococci* and *lactobacilli*, play a central role in the caries process due to their ability to thrive in acidic environments and synthesize extracellular polymers. These polymers contribute to the formation of a dense, sticky plaque matrix that anchors the bacteria onto the enamel surface, enabling persistent colonization. Within this biofilm, the bacterial community rapidly ferments dietary carbohydrates—primarily sucrose, glucose, and fructose—producing lactic acid and other organic acids that lower the pH of the local environment (2). A sustained drop in pH, especially below the critical threshold of 5.5, initiates demineralization of the enamel. Repeated acid attacks over time can lead to irreversible mineral loss and cavity formation (3).

Dietary habits directly influence the rate and extent of this microbial-acid interaction. Frequent consumption of fermentable carbohydrates, especially in the form of snacks and sugar-sweetened beverages, is strongly associated with higher caries risk. The frequency of sugar intake is more critical than the total quantity, as it affects how often the oral pH falls into the demineralization range. Sticky, retentive foods such as candies, dried fruits, and starchy snacks also promote caries by prolonging the exposure time of carbohydrates on the tooth surface. Conversely, dietary choices that stimulate salivary flow, such as fibrous fruits and vegetables, and those rich in calcium and phosphate, can contribute to remineralization and neutralizing acids. The presence and flow rate of saliva are fundamental in modulating caries development. Saliva acts as a natural buffer, diluting and clearing sugars from the oral cavity, neutralizing acids, and providing essential minerals for enamel repair. In patients with compromised salivary function—due to medical conditions, medications, or aging, the buffering and protective effects of saliva are reduced, making them more vulnerable to caries. Fluoride, whether from systemic sources like drinking water or topical sources like toothpaste and mouth rinses, also enhances remineralization and reduces acid solubility of enamel crystals, thereby offering significant protection against caries.

Understanding these factors supports the integration of dietary counseling into dental practice. Evidence suggests that effective caries prevention includes not only mechanical plaque control and fluoride use but also education on appropriate dietary behavior. Dental professionals should assess patients' dietary practices as part of routine care and provide targeted advice to limit fermentable carbohydrate exposure and encourage protective foods. Nutrition education in dental settings should emphasize not just sugar reduction but also meal frequency, snack choices, and the role of saliva-stimulating foods.

Such dietary guidance becomes especially important in managing high-risk patients, including children, older adults, and individuals with chronic illnesses or medications that reduce salivary flow. Preventive dietary interventions tailored to individual risk profiles can help reduce caries incidence and progression. Moreover, incorporating dietary counseling into standard dental care can enhance patient outcomes and improve quality of life by preserving tooth function and

reducing treatment needs over time. Dental caries, while influenced by multiple biological and behavioral variables, is ultimately preventable through informed dietary management and patient-centered care. By addressing the dietary causes and modifiers of this disease, dental professionals can significantly reduce its burden across populations and throughout the life span.

Nutrition and Its Systemic and Local Influence on Dental Caries

Nutrition influences the development and health of teeth during the pre-eruptive stage, encompassing the prenatal, perinatal, and postnatal periods. Deficiencies in protein energy, vitamin D, and vitamin A during these stages are linked to enamel hypoplasia, which increases the vulnerability of teeth to caries lesions (4,5). Even a single episode of mild-to-moderate malnutrition in the first year of life has been associated with a higher incidence of dental caries in both deciduous and permanent dentition later in life (4). These nutritional deficiencies are also associated with salivary gland atrophy and decreased salivary flow, both of which alter dental plaque acid activity and thereby increase caries risk (5). Diet, distinct from overall nutrition, refers to the specific foods and beverages consumed and the pattern of their intake over time. In the context of caries development, the local or topical effects of the diet on the tooth surface are of greatest relevance. Dental caries is fundamentally a disease that arises from the interaction between dietary fermentable carbohydrates and acidogenic bacteria on the tooth surface (6). Thus, while dietary choices are the primary factor associated with caries, nutritional status can modify this risk throughout a person's life.

The interaction of diet and oral bacteria begins with the formation of the salivary pellicle on the tooth surface, which serves as a foundation for dental plaque development. This plaque represents the earliest visible sign of dietary and microbial interaction in the mouth. Over time, the metabolism of fermentable carbohydrates, especially sugars, supports the growth of bacterial colonies on the tooth surface and contributes to shifts in the oral pH environment (7). Among the most significant bacteria involved in this process are *mutans streptococci* and *lactobacilli*. These organisms produce lactic acid as a byproduct of sugar fermentation. This acid then leaches calcium and phosphate ions from the tooth's enamel or dentin, initiating and advancing caries lesions (3). The concentration and ratio of acid-producing versus base-producing bacteria on the tooth surface affect the balance of the demineralization-remineralization process (3). Demineralization occurs when the oral pH drops below the critical threshold of approximately 5.5. If this acidic condition is sustained, mineral loss from the enamel continues. Conversely, when the oral pH rises above 5.5 and remains elevated for a sufficient period, remineralization can occur, allowing the enamel to repair some of the surface damage. However, the rate of remineralization is significantly slower than that of demineralization. Thus, the direction and extent of mineral loss or gain are shaped not only by bacterial activity and pH fluctuations but also by specific dietary components and consumption habits.

Several other variables can influence this demineralization-remineralization balance, including the presence and composition of saliva, the availability of topical fluoride, tooth anatomy, oral hygiene

practices, time, age, and general health status. While these factors were not addressed in detail in this context, they collectively contribute to modifying the environment in which caries either progress or are controlled. Saliva plays a vital role in caries prevention. It helps lubricate oral tissues, facilitates the clearance of food particles and acids, and contains antimicrobial agents and buffering systems that neutralize acid (8,9). Importantly, saliva also supports the natural repair of tooth surfaces by supplying calcium, phosphate, and proline-rich proteins necessary for enamel remineralization (8). However, aging, health changes, and medical interventions can impair salivary function, thereby increasing caries risk (9). One example is xerostomia, or dry mouth, a condition commonly associated with reduced salivary flow. It correlates with higher concentrations of cariogenic bacteria and elevated caries prevalence (9).

Fluoride, when applied topically, enhances remineralization by promoting the absorption of minerals into enamel and reducing the demineralization rate driven by plaque acids (10–12). These mechanisms help explain the widespread use of fluoride in dental caries prevention strategies. Still, the protective effect of fluoride is optimized only when other factors, such as saliva flow and dietary control, are also favorable. The dynamic balance between demineralization and remineralization is multifactorial and influenced by both systemic and local elements (13). On one hand, a deficiency in base formation due to inadequate saliva production can have an effect equal to or greater than excessive acid formation caused by frequent consumption of fermentable carbohydrates (3). On the other hand, an environment consistently exposed to fermentable dietary carbohydrates fosters a shift in oral microbiota from non-cariogenic to cariogenic populations. This ecological shift makes the oral cavity more susceptible to acid attack and thus more prone to dental caries. However, caries only occur when both fermentable carbohydrates and acid-producing bacteria are present together in sufficient quantities and for adequate duration. Dental caries remains one of the leading causes of tooth loss in the United States. Prevalence data show that about 20% of young children, nearly 80% of young adults, and close to 95% of older adults have experienced dental decay at some point in their lives (14,15). These figures highlight that caries is not a problem confined to childhood but continues to affect individuals across the lifespan.

There is a common misconception that simply removing “sugar” from the diet and emphasizing good oral hygiene and fluoride exposure will fully protect against caries (16). While such steps are beneficial, they are insufficient on their own. Effective caries management must also incorporate targeted changes in dietary behaviors. Specifically, strategies must aim to reduce the frequency and amount of fermentable carbohydrate intake and simultaneously support optimal saliva production (1,12,17). This dual focus not only limits the availability of substrate for acidogenic bacteria but also enhances the tooth’s ability to repair early damage through remineralization. Overall, the development and prevention of dental caries involve a complex interplay of systemic nutritional status, local dietary practices, microbial activity, salivary function, and fluoride exposure. Addressing only one of these components will not fully mitigate the risk. A comprehensive approach is necessary to control and reverse the factors that drive caries initiation and progression. Recognizing the influence of both systemic nutrition and local diet reinforces the

need for interdisciplinary care that includes dietitians, dental professionals, and medical practitioners to protect and promote oral health across all stages of life.

Dietary Factors in Dental Caries Promotion

Dieting plays a direct and measurable role in the development and promotion of dental caries. Specific foods, their physical form, and the frequency of consumption all contribute to caries risk. Among the dietary components, sugars and other fermentable carbohydrates are the most significant contributors to the caries process. The presence of these substrates in the oral cavity supports acid production by plaque bacteria, ultimately leading to enamel demineralization. Sugars such as sucrose, glucose, fructose, maltose, and lactose are widely studied for their cariogenic potential. Simple sugars found in soft drinks, confectionery, baked goods, and processed foods are readily fermented by bacteria into acids. Sucrose, in particular, has a high caries-promoting potential due to its role in the synthesis of insoluble extracellular polysaccharides by oral bacteria. These polysaccharides contribute to plaque adhesion and microbial colonization on the enamel surface (18,19). Lactose, the sugar found in milk, has lower cariogenic potential, as galactose has not demonstrated a comparable impact on plaque acidogenicity. Other fermentable carbohydrates, including cooked starches from potatoes, rice, legumes, and grains, also contribute to acid production under specific conditions. Heat and mechanical processing of these foods alter their structure, forming gelatinized starch that becomes susceptible to enzymatic hydrolysis by salivary and bacterial amylases. This enzymatic activity releases maltose and maltotriose, which bacteria can ferment into acids (40,42,43). The cariogenicity of starch-rich foods varies according to genetic composition and food processing methods such as frying or baking. Unprocessed whole grains and raw vegetables, which resist enzymatic breakdown, show a lower risk of promoting caries compared to processed starchy snacks like crackers, chips, and cereals (45). The form and texture of foods also influence caries risk. Sticky and retentive foods that adhere to tooth surfaces are more cariogenic because they prolong the exposure of fermentable substrates to oral bacteria. Foods consumed in liquid form, though rapidly cleared from the oral cavity, can also be problematic if consumed frequently throughout the day. The frequency of sugar intake is more critical than the total quantity. Each exposure to fermentable carbohydrate results in a drop in plaque pH, with recovery to baseline pH taking approximately 40 minutes. The Stephan curve illustrates this dynamic, showing the sharp decrease in plaque pH following the intake of a 10% sucrose solution and the gradual return to neutrality (31).

Empirical studies, including human, animal, and laboratory research, confirm the role of dietary sugars in the etiology of caries (20–30). However, not all research findings align. Population-level data on sugar consumption and caries prevalence show inconsistencies. While some studies report positive correlations, others do not (33,34,35). This inconsistency is attributed to variations in fluoride exposure, study designs, and dietary assessment methods. For example, in the United States, sucrose consumption declined between 1970 and 1996, but total added-sugar intake increased due to higher use of high-fructose corn syrup (36). These changes in sugar types and consumption patterns complicate assessments of sugar’s impact on dental caries. The Vipeholm

Dental Caries Study, a foundational clinical trial conducted from 1945 to 1954, provided strong evidence for the influence of sugar form and frequency on caries development (38). The study demonstrated that consuming retentive sugary foods like toffee candies between meals significantly increased caries incidence. In contrast, sugar consumed during meals had a limited impact on caries when between-meal snacking was restricted. These findings suggest that both the physical consistency of the sugar source and its timing relative to meals are critical in modulating caries risk (39). A more recent systematic review of studies examining sugar consumption and caries found that sugar is a moderate risk factor for caries, especially in individuals with consistent fluoride exposure (22). Many of these studies were cross-sectional in design, with varying quality and assessment tools that limited their ability to capture cumulative and long-term sugar exposure. The review concluded that in populations with limited fluoride access, sugar consumption plays a more dominant role in caries development. Thus, fluoride availability acts as a moderating factor, partially offsetting the impact of sugar on oral health.

It is important to note that the cariogenic potential of fermentable carbohydrates is not solely dependent on their concentration in foods. The amount of acid produced in the mouth does not correspond linearly to the sugar content. Factors such as food form, texture, composition, and the presence of protective components like fiber, calcium, or polyphenols all influence the overall cariogenicity of a given food (32). This complexity limits the accuracy of simple classifications and requires a more nuanced understanding of how different foods interact with the oral environment. Non-cariogenic dietary components, including fiber and polyol sugar alcohols like sorbitol, mannitol, and xylitol, do not promote acid formation by bacteria. These compounds are less fermentable or non-fermentable and can even contribute to oral health by reducing plaque acidity and enhancing saliva production. High-intensity sweeteners such as aspartame, sucralose, and saccharin also do not support acidogenic bacterial growth and are often used as substitutes in sugar-free products designed to minimize caries risk. In summary, the role of diet in promoting dental caries is clearly established, particularly through the action of sugars and processed starches that serve as substrates for acidogenic bacteria. Frequency and form of intake are critical factors that modify this risk. The addition of fluoride and effective saliva function can mitigate some of the harmful effects, but without careful control of dietary exposures, especially in high-risk individuals, the likelihood of caries development remains high. Understanding these dietary mechanisms allows for the development of effective caries prevention strategies that include not only fluoride use and oral hygiene but also comprehensive nutritional counseling and behavior modification.

Table 1: Cariogenic vs Cariostatic Foods and Their Effects on Oral Health

Food Type	Examples	Cariogenicity	Oral Health Effect
Simple Sugars	Sucrose, glucose, fructose, honey, HFCS	High	Promote acid production, lower plaque pH, cause demineralization

Food Type	Examples	Cariogenicity	Oral Health Effect
Cooked Starches	White bread, crackers, chips	High	Retentive, slow salivary clearance, support acid production
Processed Snacks	Cookies, caramel, dry cereals	High	Prolonged contact with teeth, low salivary stimulation
Dairy Products	Milk, cheese, yogurt	Low (Lactose), Cariostatic	Buffer acids, support remineralization via calcium/phosphates
Sugar Alcohols	Xylitol, sorbitol, mannitol	Non-cariogenic	Reduce bacterial growth, stimulate saliva, lower caries risk
Plant-Based Whole Foods	Raw vegetables, nuts, whole grains	Non-cariogenic	Stimulate saliva, improve clearance, low fermentability
Tea	Green, oolong, black	Cariostatic	Contain polyphenols, reduce acidogenic bacteria

Unique Forms of Food and Frequency of Dietary Intake in Relation to Dental Caries

The classification of sugars and carbohydrates is essential for understanding their role in dental caries. Sugars fall into two primary categories: monosaccharides, such as glucose, fructose, galactose, and disaccharides, including sucrose, lactose, and maltose. These sugars, particularly when extrinsically added to foods and beverages, are key substrates for acidogenic oral bacteria. Common nutritive sweeteners—like table sugar, high-fructose corn syrup, honey, dextrose, and molasses—are used widely in processed foods and provide fermentable carbohydrates that support bacterial metabolism. Sugar alcohols, though technically alcohol forms of monosaccharides, are also added to sweeten foods but generally possess lower cariogenic potential due to reduced fermentability by plaque bacteria (18). Among disaccharides, sucrose, glucose, fructose, and maltose share similar acidogenic and cariogenic potentials. Lactose, in contrast, is considered less cariogenic (42). Intrinsic sugars found in whole grains, fruits, and vegetables are encapsulated within a matrix of fiber, protein, and lipids, which impedes their rapid fermentation in the oral cavity. These structural properties of natural foods reduce their cariogenic impact. Current evidence does not support a significant role for intrinsic sugars in the caries process (46). Scientific efforts to determine the cariogenicity of foods often rely on artificial methodologies, such as plaque pH response testing following food exposure. While informative, these studies do not fully replicate the complexity of human dietary behavior. Nonetheless, such research has provided valuable insights. For instance, Edgar et al. (47) found a wide variation in the acidogenicity of 54 snack foods. Bibby and Mundroff (32) examined 180 foods and reported that items with high sugar content did not necessarily cause more enamel demineralization than those containing lower sugar levels but combined with starches, such as breads or cookies. Processed starchy snacks, although

slower to initiate acid production—can generate acid levels equivalent to those of sucrose alone (48,49).

Food texture, solubility, retentiveness, and the rate of oral clearance all influence a food's cariogenic potential. High-sugar foods such as caramels and chocolate bars are often cleared more rapidly from the oral cavity than high-starch foods like potato chips and crackers (32,43). When sucrose is combined with cooked starch, the result is a more adherent food substance that remains in contact with the tooth surface longer, enhancing its cariogenic effect (50). This prolonged exposure is more detrimental than sugar alone due to increased retention and delayed salivary clearance. Thus, cariogenicity is influenced more by the interaction of food properties with oral biology than by sugar content alone (32,51). No single food can be universally labeled as cariogenic or non-cariogenic without considering these interactions. The classification of foods based on acidogenic potential, often derived from animal models, can aid dental professionals in patient counseling. However, this approach must be interpreted with caution. For example, studies that ranking raw vegetables as less acidogenic than dried fruits may incorrectly suggest that corn chips are a better dietary option than nutrient-dense dried fruit (52). Nutritional guidance should consider context, including total dietary patterns, nutrient content, frequency of intake, and overall diet quality, rather than focusing exclusively on acidogenicity measures. Frequency of dietary intake is arguably the most critical factor linking diet to caries development. Although total sugar intake plays a role, frequent consumption, particularly snacking on fermentable carbohydrate-rich foods, poses a much higher risk. Most individuals consume food four to six times per day. When this pattern is exceeded, especially with repeated intake of sugar-laden or starchy foods, the oral environment is continually exposed to acidogenic challenges. These prolonged episodes of plaque acidification promote demineralization over remineralization, especially when plaque removal is inadequate and salivary buffering is compromised (53–55).

Frequent snacking results in extended periods during which oral pH remains below the critical level for enamel demineralization. If this low pH environment is maintained across the day, the cumulative effect significantly increases the likelihood of caries development. Furthermore, cooked starches and processed carbohydrate-sugar combinations tend to adhere to teeth longer and clear more slowly, compounding their cariogenic potential. When these foods are consumed often, particularly between meals, they maintain a steady supply of fermentable substrate for acidogenic bacteria. Epidemiological studies support the correlation between eating frequency and caries prevalence. Children who consume sugary foods four or more times daily or snack three or more times between meals exhibit significantly higher caries scores compared to peers with lower snacking frequency (56–58). These data highlight the combined effect of eating frequency and sugar quantity, showing that both dimensions contribute to caries risk. Importantly, controlling one factor, either reducing frequency or limiting quantity—can positively influence the overall outcome. Taken together, these findings emphasize that food form, processing, retention characteristics, and frequency of intake are central to understanding dietary contributions to dental caries. While sugars remain the primary culprits, their impact is mediated by the physical and

chemical properties of the food and how frequently they are consumed. Effective dietary counseling should incorporate these variables to provide balanced, evidence-informed advice tailored to individual dietary habits and caries risk. Such guidance can support patients in making food choices that align with both oral and overall health goals.

Dietary Factors in Caries Prevention

Just as certain dietary factors contribute to the promotion and progression of dental caries, there are foods and nutrients that serve to protect against caries or reduce its severity. These components are described as "anticariogenic" or "cariostatic." They do not contribute to enamel demineralization and may support protective oral conditions by neutralizing acids, enhancing enamel remineralization, or stimulating saliva production. Understanding these protective dietary elements is critical for the development of effective nutritional guidelines in dental practice. Dairy products play a significant role in caries prevention. Although milk contains lactose, a disaccharide sugar that has some potential to contribute to early childhood caries, especially when infants are exposed to it for prolonged periods during bottle-feeding, it also possesses properties that help prevent demineralization and promote remineralization of tooth enamel. Several human and animal studies have demonstrated milk's effectiveness in reducing the cariogenicity of sugar-laden foods. The protective mechanism of milk is attributed primarily to its content of calcium and phosphate, minerals essential for enamel integrity, which are tightly bound to the milk protein casein (60–62). These minerals are readily available to restore demineralized enamel when the pH in the oral cavity becomes favorable for remineralization. Additionally, casein phosphopeptides in milk and other dairy products stabilize calcium and phosphate ions and support their integration into tooth structure (58,63,64).

Cheese, another dairy product, exhibits even stronger anticariogenic properties than milk. When consumed after a sugary food, cheese stimulates salivary flow significantly, which assists in neutralizing plaque acids and restoring pH balance. The casein phosphopeptides in cheese, along with calcium and phosphate, actively promote enamel remineralization. The effectiveness of cheese in caries prevention has been well-documented; a small cube consumed at the end of a meal or snack has been shown to reverse acid attacks and support oral health (65–67). The role of cheese in stimulating saliva and neutralizing acids is a simple, accessible preventive strategy for individuals at risk of dental caries. Sugar substitutes and alternative sweeteners offer another means of reducing caries risk. Nutritive sugar alcohols such as sorbitol, mannitol, and xylitol are lower in caloric content than sucrose and exhibit significantly reduced cariogenic potential. Among these, xylitol has been studied most extensively. It is derived from natural sources including birch trees, corn cobs, and various fruits. Controlled human studies have shown that regular use of xylitol-sweetened products—particularly chewing gums and mints—can reduce the incidence of dental caries by 30% to 60% compared with control groups (69). The mechanisms underlying this reduction are both chemical and behavioral. Xylitol is not readily metabolized by oral bacteria, thus inhibiting acid production. Furthermore, the act of chewing gum itself increases salivary flow, which contributes to the clearance of food debris and acid neutralization (70).

The benefits of sugar alcohols are reflected in regulatory standards as well. The U.S. Food and Drug Administration has permitted specific health claims for food products containing sugar alcohols, such as "does not promote dental caries" or "useful in not promoting caries," acknowledging their favorable impact on oral health (71). These sweeteners, particularly when used in place of fermentable sugars in candies, gums, and baked goods, serve as valuable tools in dietary-based caries prevention strategies. Nonnutritive sweeteners also demonstrate promise in caries prevention. These include saccharin, aspartame, acesulfame potassium (K), sucralose, and cyclamate. These compounds are significantly sweeter than sucrose and are used in minute quantities in various foods and beverages. Because they are not metabolized by oral bacteria, they do not contribute to acid production and are therefore considered non-cariogenic. As new formulations and food products are developed, especially those aimed at individuals seeking sugar alternatives for metabolic health reasons, their oral health benefits may gain further recognition. Plant-based foods also provide caries-protective benefits. Whole grains, fruits, vegetables, and legumes contain various bioactive components such as organic and inorganic phosphates, polyphenols, phytates, and dietary fiber. These compounds may act independently or synergistically to inhibit bacterial activity or enhance mineral retention in the oral cavity. Although current evidence does not confirm a direct protective role of these compounds when consumed in natural food forms, their presence in unprocessed plant foods contributes to the maintenance of a healthy oral environment. Fibrous foods, in particular, stimulate mechanical cleansing and salivary secretion, which indirectly supports caries prevention (72). As part of a balanced diet, plant foods enhance overall nutrition and oral health, particularly when they replace processed snacks that are high in fermentable sugars and refined starches.

Other natural foods and beverages have also demonstrated potential for reducing caries risk. Teas—including green, black, and oolong varieties—contain fluoride as well as polyphenols and flavonoids, which have antibacterial properties. Laboratory studies have shown that these compounds suppress the growth of *Streptococcus mutans* and other cariogenic bacteria while reducing acid production in response to sugar exposure (44,73,74). Animal models support these findings, suggesting that regular tea consumption may lead to a reduction in the incidence and severity of dental caries (75). If confirmed by future clinical trials, tea consumption could represent a cost-effective public health intervention with the dual benefit of hydration and oral health protection. Research has also explored the potential protective roles of specific food components. Fatty acids found in cocoa bean husk, such as oleic and linoleic acid, have demonstrated bactericidal activity against *S. mutans* in vitro (76,77). While these findings suggest that dark chocolate may contain components with anticariogenic properties, the high sucrose content in most commercial chocolate products largely neutralizes any protective effect (78). Similarly, licorice, which contains glycyrrhizinic acid, has been shown to enhance plaque buffering and inhibit bacterial metabolism. However, its use is limited due to potential adverse effects such as enamel staining and systemic effects associated with excessive consumption (79). Peanuts, which are high in monounsaturated fats, contribute to salivary stimulation during chewing and are characterized by a low caries-promoting potential. Their mechanical properties promote oral cleansing, and their

high fat content helps inhibit bacterial acid production. However, when combined with fermentable carbohydrates in processed snacks, these benefits can be outweighed by the increased cariogenic potential of added sugars and starches (80). The net impact of such foods depends on the overall composition and the context in which they are consumed.

The integration of these dietary insights into preventive oral health strategies is crucial. While much focus is placed on avoiding high-sugar foods, an effective approach must also emphasize the inclusion of protective foods. Dairy products, especially cheese, should be recommended following sugary meals to neutralize acids and promote enamel repair. Chewing gums containing xylitol should be encouraged after meals for individuals at high risk of caries. Plant-based diets rich in fibrous vegetables and whole grains support overall and oral health by reducing reliance on processed, fermentable carbohydrates. Public health messages and patient education should reflect the complexity of dietary effects on oral health. Labeling foods as simply "good" or "bad" oversimplifies the influence of diet on caries. Instead, dietary counseling should consider individual dietary patterns, food combinations, timing of consumption, and access to fluoridated water or oral hygiene resources. A piece of dried fruit consumed as part of a meal, followed by water and good oral hygiene, poses far less risk than frequent snacking on low-quality carbohydrate-rich foods between meals. As dietary habits continue to evolve with modern food processing and global food distribution, continued research into food-based caries prevention is necessary. Greater understanding of the bioavailability of protective food components, the role of food structure, and the effect of food combinations will help refine nutritional recommendations for oral health. For dental practitioners, incorporating dietary advice into routine care—backed by current evidence—is a necessary step in improving patient outcomes and reducing the burden of dental caries.

Dietary Recommendations

Although some foods considered protective against dental caries may carry health-related risks if consumed excessively or inappropriately, the opportunity remains to craft dietary messages that help translate good nutrition into practical, preventive behavior. Terms like "cariogenic" and "caries-promoting" are commonly used to describe food items or dietary patterns linked to dental decay. However, these terms may unintentionally reinforce a restrictive mindset around food. Instead of focusing solely on avoidance, dental professionals should guide patients toward making balanced food choices that support oral health while also promoting general nutritional well-being. The relationship between diet and dental caries is not limited to the nature of single foods but reflects broader dietary patterns, which involve not only food types but also how foods are combined and how frequently they are consumed over time (16). Understanding these patterns is essential for developing individualized dietary strategies that support oral health. A helpful starting point for dental professionals is the assessment of a patient's food choices and eating behaviors. Through this assessment, strategies can be proposed that involve pairing cariogenic and cariostatic foods to reduce the risk of caries development (81). For instance, sugary foods can be less harmful when consumed as part of a balanced meal rather than alone. When sugar is ingested with proteins

and small amounts of fat, its impact on plaque pH may be moderated. Moreover, the sequence in which foods are consumed can play a role. If a food item that stimulates salivary flow is eaten before or after a sugary item, the resulting salivary increase can buffer acid production and help return oral pH to a neutral level (82).

This principle also applies to calcium- and fluoride-rich foods. When these are present in the oral cavity during or after the consumption of fermentable carbohydrates, the potential for enamel remineralization is enhanced. Incorporating milk or nonfat yogurt with a meal or snack can support a cariostatic effect by not only providing beneficial minerals but also increasing saliva production. Clinical observations have confirmed this benefit in specific populations; for example, elderly individuals who consumed cheese regularly were found to have lower rates of root caries (83). Such findings emphasize the importance of including mineral-rich foods, particularly dairy products, in daily dietary habits. The sequence and combination of foods consumed in a single eating occasion significantly influence the extent of acid production in dental plaque. When fermentable carbohydrates are consumed sequentially—such as eating crackers followed by cola, then sticky candies like caramels or raisins, and then cookies—the cumulative acidogenic effect is substantial, especially over prolonged time frames (53,54). In contrast, modifying these sequences can reduce the risk. For instance, consuming peanuts, which require thorough chewing and stimulate saliva, along with apple juice, which has minimal salivary impact, can produce a more favorable oral environment than either item alone. Similarly, eating fermentable carbohydrate-rich crackers along with tuna, a protein-dense food with a basic pH profile, may help mitigate enamel demineralization by promoting remineralizing conditions.

Encouraging patients to adopt these strategies requires more than listing specific foods. It involves providing context and personalization. Food selection should aim not only to reduce exposure to fermentable carbohydrates but also to promote overall diet quality. Nutrient adequacy—the balance and variety of nutrients in the diet—is a strong indicator of caries risk. Research has shown that individuals who snack frequently and obtain a high percentage of their daily energy intake from fermentable carbohydrates, while consuming low amounts of high-protein foods, tend to have higher rates of dental caries (84). On the other hand, dietary patterns rich in vegetables and dairy products are consistently associated with lower caries incidence (85,86). This data underscores the importance of promoting dietary variety and moderation. Rather than eliminating entire food groups or demonizing certain ingredients, the goal should be to build meals and snacks that incorporate nutrient-dense options capable of supporting both oral and systemic health. High-protein foods, such as lean meats, legumes, nuts, and dairy, should be consumed regularly. Fruits and vegetables, especially those with high fiber content, should also be included to aid in oral clearance and stimulate saliva. Meanwhile, foods and beverages high in added sugars or refined starches should be limited in frequency, quantity, and timing—preferably consumed with meals rather than between them.

Another important aspect of dietary guidance is recognizing that behavior change is often incremental and influenced by accessibility, habits, and cultural preferences. Dental professionals

must frame recommendations in a way that respects these individual factors. Tailored advice, such as suggesting that a patient finish a meal with cheese or switch from sugary beverages to water or milk, can be more effective than generalized statements about “avoiding sugar.” In populations with limited access to dental care or fluoridated water, these nutritional strategies become even more critical. Dietary counseling should be integrated into regular dental assessments, particularly for patients identified as having high risk due to existing caries, poor oral hygiene, or systemic conditions like diabetes that increase vulnerability to oral disease. Nutritional assessments can help identify deficiencies or imbalances that may compromise oral health. This approach supports a preventive care model in which diet is addressed alongside hygiene and fluoride use. In summary, dietary recommendations for caries prevention should focus on optimizing meal structure, food combinations, and eating frequency. Emphasis should be placed on pairing cariogenic foods with protective foods, choosing snacks that stimulate saliva, and ensuring that calcium-rich foods are regularly included. Encouraging balanced, nutrient-dense diets rich in vegetables, whole grains, dairy, and protein sources will contribute to both oral and general health outcomes. Effective dietary advice goes beyond restriction; it promotes informed choices, sustainable habits, and practical routines that align with a patient’s individual context.

Dietary Screening and Education in the Dental Practice

Routine dietary screening in dental practice is essential for the prevention and management of dental caries. Diet is a well-established factor influencing caries development, and dietary behavior plays a critical role in both risk assessment and patient education. Although dentists are not trained to conduct comprehensive nutritional assessments, including anthropometric or biochemical analyses, they can perform effective dietary screening and provide basic dietary counseling. More complex cases can be referred to registered dietitians for in-depth evaluation and personalized nutrition plans (81). The primary objective is to assess behaviors and determinants related to dietary intake that influence oral health and caries risk, and to provide clear, actionable messages that promote oral health without disregarding the role of taste and personal food preferences in dietary choices (16). To support dietary screening in a clinical setting, validated tools such as the questionnaire. This questionnaire includes two parts. Part A focuses on dietary behaviors that strongly predict caries risk. These include frequency of snacking, particularly on fermentable carbohydrates; regular consumption of sugar-sweetened beverages; and chewing of regular (non–sugar-free) gum. A "yes" response to any of these items may indicate increased risk, while two or more positive responses suggest a dietary pattern likely exceeding sugar intake guideline. In such cases, Part B should be completed to evaluate nutrient adequacy. This section inquiries about daily intake of dairy products, fruits and vegetables, whole grains, protein sources, and water. This screening tool helps dental professionals contextualize caries risk in terms of both harmful and protective dietary practices. More importantly, it serves as a foundation for developing individualized health-promotion messages. These messages can focus on reducing cariogenic behaviors and encouraging nutrient-dense dietary patterns that support oral health. The integration of diet-based preventive strategies into routine dental care helps patients understand the connection

between oral and general health and empowers them to make informed choices that support long-term well-being.

These guidelines define appropriate serving sizes, food group targets, and the underlying rationale for each recommendation. For example, daily added sugar intake should not exceed 40 grams or about 12 teaspoons. Exceeding this threshold is associated with increased caries incidence, particularly in populations with limited fluoride exposure (34,46,88). Patients who frequently consume sugary snacks or beverages should be encouraged to replace these with sugar-free alternatives or to pair them with cariostatic foods such as cheese, nuts, or milk. Sugar-free chewing gum is a practical intervention. When used after meals or snacks, it increases salivary flow and supports acid neutralization. Products containing sugar alcohols (like xylitol or sorbitol) have been shown to reduce caries by 30% to 60% in various studies (69,70). Chewing gum offers a dual benefit: chemical action through non-cariogenic sweeteners and mechanical stimulation of saliva, which helps cleanse the mouth and promote remineralization. Food combinations are another important consideration. For instance, acidogenic foods such as cookies or raisins can be paired with more neutral items like milk or cheese to reduce their cariogenic effect (82). Examples include yogurt with granola, apple slices with peanuts, or crackers with tuna. These combinations encourage salivary buffering and reduce acid exposure duration, minimizing enamel demineralization. The timing of food intake also plays a role. Spacing eating occasions at least two hours apart gives the oral environment time to return to a neutral pH, reducing cumulative caries risk.

Frequency of meals and snacks should ideally range between four and six times daily, particularly in children. This allows for nutritional adequacy without sustained acidic conditions in the mouth. For adults, three meals and one or two snacks are often sufficient, provided that overall energy and nutrient needs are met. Importantly, consistency in eating patterns and food types of aids in maintaining a stable oral environment. Unpredictable or excessive snacking, especially on high-sugar or refined starch products, contributes to greater caries risk. Fat intake is another dietary component with implications for oral health. Although the focus in dentistry is often on carbohydrates, dietary fats—particularly monounsaturated fats such as oleic and linoleic acid—can also influence oral biofilm and adherence properties. These fats may help reduce bacterial adhesion to tooth surfaces and, in some contexts, act as barriers against plaque formation (77,88). Therefore, the type and quality of fats consumed should be considered in dietary counseling. Whole grains and unprocessed cereals are encouraged due to their low fermentability and high fiber content, which stimulates saliva and supports oral cleansing. A minimum of six servings per day is advised, corresponding to items such as whole-grain bread, brown rice, and unrefined cereal. In contrast, refined grain products, including white bread and sugary breakfast cereals, are more cariogenic and should be consumed with caution.

Fruits and vegetables provide fiber and antioxidants that benefit oral soft tissue health. Five servings per day, consisting of a mix of raw and cooked options, are recommended. Though natural sugars in fruits are present, they are generally less cariogenic due to their encapsulation in fibrous

matrices and their pairing with other nutrients. Juices and processed fruit products, however, should be consumed in moderation and preferably with meals. Proteins from meat, poultry, fish, eggs, nuts, and legumes contribute to soft tissue repair and buffer the oral environment. Consuming two to three servings daily ensures adequate intake of essential nutrients such as iron, zinc, and B vitamins, all of which support gingival and periodontal health. Proteins are also useful in reducing the net acid load of meals, particularly when eaten alongside fermentable carbohydrates. Dairy products, especially milk, yogurt, and cheese, are emphasized for their high calcium and phosphorus content, along with casein protein, which supports remineralization. Two to three servings daily are advised. Cheese, in particular, offers unique benefits when consumed after acidic or sugary foods. It increases salivary flow, raises plaque pH, and introduces protective compounds that reduce enamel erosion (66,88). Water intake also deserves specific attention. The recommendation is to consume eight cups of water daily. Water supports oral clearance, maintains mucosal hydration, and aids in the neutralization of acidic byproducts. In communities without fluoridated water supplies, dental professionals may consider recommending fluoride supplements or topical fluoride applications to augment caries prevention.

Ultimately, the dietary education provided in dental settings must align with the patient's overall health goals and readiness to change. Personalized messages that consider socioeconomic status, cultural food preferences, and existing health conditions are more likely to be adopted and sustained. Collaborative care models involving dietitians, physicians, and dental professionals enhance the scope and effectiveness of such interventions. By adopting structured dietary screening protocols and offering evidence-based education, dental professionals can significantly influence patients' oral health behaviors. This approach bridges the gap between dental care and nutritional health and reinforces the role of diet as a modifiable risk factor in caries prevention and management. When combined with oral hygiene instruction, regular fluoride exposure, and professional cleanings, dietary counseling becomes a cornerstone of comprehensive preventive dental care. In conclusion, integrating dietary screening and nutrition education into dental practice offers a critical opportunity to address the dietary behaviors that contribute to caries development. Through validated tools, practical guidelines, and patient-specific counseling, dental professionals can promote sustainable dietary changes that enhance both oral and systemic health (89).

Table 2: Dietary Screening Questionnaire for Dental Settings

Screening Topic	**Sample Question**	**Risk Indicator**
Snack Frequency	Do you snack between meals or instead of meals?	Yes = Increased risk
Fermentable Carb Snack Frequency	Do you eat candies, pastries, crackers between meals 3 out of 6 days?	Yes = Increased risk
Sugary Beverage Intake	Do you drink soda, lemonade, sugary coffee between or after meals?	Yes = Increased risk

Screening Topic	Sample Question	Risk Indicator
Regular Gum Use	Do you chew regular (not sugar-free) gum most days of the week?	Yes = Increased risk
Dairy Intake	Do you eat/drink dairy at least twice daily?	No = Potential deficiency
Fruit and Vegetable Intake	Do you eat fruits/bright vegetables at least 5 times per day?	No = Potential deficiency
Whole Grain Intake	Do you eat whole grain cereals/bread at least 4 times daily?	No = Potential deficiency
Protein Intake	Do you eat meat, eggs, fish, or legumes at least twice daily?	No = Potential deficiency
Water Intake	Do you drink 6–8 cups of water daily?	No = Potential deficiency

Table 3: Dietary Guidelines for Oral Health Promotion

Food/Behavior	Guideline	Recommended Serving	Oral Health Rationale
Added Sugars	< 40g/day (approx. 10 tsp)	1 tsp = 4g	Intake >55g/day increases caries risk
Sugar-Free Gum	Use after meals	1 slice for 5–10 minutes	Increases saliva, buffers acid
Food Combinations	Mix acidogenic with basic/neutral foods	Cheese with crackers, yogurt + fruit	Alters oral pH, promotes remineralization
Meal/Snack Frequency	4–5/day (adults), 4–6/day (children)	Spaced 2 hours apart	Prevents prolonged acidic pH
Whole Grains	Choose unprocessed sources	6–11 servings/day	Stimulate saliva, low fermentability
Fruits and Vegetables	Include raw and cooked	≥5 servings/day	Provide antioxidants, stimulate saliva
Protein Foods	Meat, legumes, nuts	2–3 servings/day	Provide buffering capacity
Dairy Products	Milk, cheese, yogurt	2–3 servings/day	Contain casein, calcium, phosphates for remineralization
Water	Daily intake	6–8 cups/day	Promotes clearance and neutral oral environment

Conclusion:

Dental caries remains a prevalent, yet preventable disease influenced significantly by dietary habits. The interaction between fermentable carbohydrates and acidogenic bacteria, particularly *Streptococcus mutans* and lactobacilli, drives enamel demineralization. However, caries risk is modifiable through informed dietary choices and preventive strategies. The evidence underscores that frequency of sugar intake is more critical than total quantity, as repeated acid attacks disrupt the demineralization-remineralization balance. Sticky, retentive foods prolong carbohydrate exposure, while liquid sugars, when consumed frequently, also contribute to caries. Conversely, protective foods such as cheese, milk, and xylitol-containing products counteract acid production and enhance remineralization. Cheese, in particular, stimulates salivary flow and buffers plaque pH, making it a valuable post-meal supplement. Dietary interventions must extend beyond sugar reduction to include meal timing and food combinations. Pairing cariogenic foods with cariostatic options (e.g., nuts with dried fruit) can mitigate acidogenic effects. Additionally, sugar-free gum with xylitol reduces bacterial adhesion and boosts saliva production, offering a practical preventive measure. Saliva's role cannot be overstated, neutralizes acids, clearing food debris, and supplies remineralizing ions. Patients with xerostomia or low salivary flow require tailored dietary advice to compensate for reduced natural protection. Fluoride, whether systemic or topical, further enhances enamel resistance but works best alongside dietary modifications. Clinical implications highlight the need for dietary screening in dental practice. Simple questionnaires can identify high-risk behaviors (e.g., frequent snacking), allowing targeted counseling. Public health messages should promote nutrient-dense diets rich in dairy, whole grains, and fibrous foods while discouraging excessive processed sugar intake. Ultimately, a multidisciplinary approach—combining dietary education, fluoride therapy, and oral hygiene—is essential for caries prevention. Dental professionals must collaborate with nutritionists to provide personalized guidance, especially for high-risk groups like children, elderly patients, and those with dry mouth conditions. By addressing diet as a modifiable risk factor, the burden of dental caries can be significantly reduced, improving long-term oral health outcomes.

References:

1. König, K. G. (2000). Diet and oral health. *International Dental Journal, 50*, 162-174. https://doi.org/10.1111/j.1875-595X.2000.tb00556.x

2. Miller, W. D. (1973). *The microorganisms of the human mouth: The local and general diseases which are caused by them*. S. Karger.

3. Kleinberg, I. (2002). A mixed-bacteria ecological approach to understanding the role of the oral bacteria in dental caries causation: An alternative to Streptococcus mutans and the specific-plaque hypothesis. *Critical Reviews in Oral Biology & Medicine, 13*(2), 108-125. https://doi.org/10.1177/154411130201300204

4. Alvarez, J. O. (1995). Nutrition, tooth development, and dental caries. *The American Journal of Clinical Nutrition, 61*(Suppl), 410S-416S. https://doi.org/10.1093/ajcn/61.2.410S

5. Nunn, J. (2001). Nutrition and dietary challenges in oral health. *Nutrition, 17*(5), 426-427. https://doi.org/10.1016/S0899-9007(01)00570-8

6. Bowen, W. H. (2002). Do we need to be concerned about dental caries in the coming millennium? *Critical Reviews in Oral Biology & Medicine, 13*(2), 126-131. https://doi.org/10.1177/154411130201300205

7. Tanzer, J. M., Livingston, J., & Thompson, A. M. (2001). The microbiology of primary dental caries in humans. *Journal of Dental Education, 65*(10), 1028-1037. https://doi.org/10.1002/j.0022-0337.2001.65.10.tb03447.x

8. Kaufman, E., & Lamster, I. B. (2002). The diagnostic applications of saliva - a review. *Critical Reviews in Oral Biology & Medicine, 13*(2), 197-212. https://doi.org/10.1177/154411130201300209

9. Stack, K. M., & Papas, A. S. (2001). Xerostomia: Etiology and clinical management. *Nutrition in Clinical Care, 4*(1), 15-21. https://doi.org/10.1046/j.1523-5408.2001.00004.x

10. American Dietetic Association. (2000). Position of the American Dietetic Association: The impact of fluoride on dental health. *Journal of the American Dietetic Association, 100*(10), 1208-1213. https://doi.org/10.1016/S0002-8223(00)00360-4

11. Petersson, G. H., & Bratthall, D. (1996). The caries decline: A review of reviews. *European Journal of Oral Sciences, 104*(4), 436-443. https://doi.org/10.1111/j.1600-0722.1996.tb00104.x

12. Stookey, G. K. (2000). Current status of caries prevention. *Compendium of Continuing Education in Dentistry, 21*(10A), 862-867.

13. Featherstone, J. D. B. (2000). The science and practice of caries prevention. *The Journal of the American Dental Association, 131*(7), 887-899. https://doi.org/10.14219/jada.archive.2000.0307

14. National Institutes of Health. (2001). Diagnosis and management of dental caries throughout life. *NIH Consensus Statement, 18*(1), 1-30.

15. U.S. Department of Health and Human Services. (2000). *Oral health in America: A report of the Surgeon General*. National Institute of Dental and Craniofacial Research.

16. Mobley, C. (2002). Dietary analysis in a restorative practice. In E. S. Duke (Ed.), *Proceedings of the 5th Annual Indiana Conference on the Changing Practice of Restorative Dentistry* (pp. 139-156). Indiana University School of Dentistry.

17. Jensen, M. E. (1999). Diet and dental caries. *Dental Clinics of North America, 43*(4), 615-633.

18. American Dietetic Association. (1998). Position of the American Dietetic Association: The use of nutritive and nonnutritive sweeteners. *Journal of the American Dietetic Association, 98*(5), 580-587. https://doi.org/10.1016/S0002-8223(98)00131-3

19. Coulston, A. M., & Johnson, R. K. (2002). Sugar and sugars: Myths and realities. *Journal of the American Dietetic Association, 102*(2), 351-353. https://doi.org/10.1016/S0002-8223(02)90069-1

20. Al-Malik, M. I., Holt, R. D., & Bedi, R. (2001). The relationship between erosion, caries and rampant caries and dietary habits in preschool children in Saudi Arabia. *International Journal of Paediatric Dentistry, 11*(6), 430-439. https://doi.org/10.1046/j.0960-7439.2001.00305.x

21. Beighton, D., Adamson, A., & Rugg-Gunn, A. (1996). Associations between dietary intake, dental caries experience and salivary bacterial levels in 12-year-old English schoolchildren. *Archives of Oral Biology, 41*(3), 271-280. https://doi.org/10.1016/0003-9969(95)00135-2

22. Burt, B. A., & Satishchandra, P. (2001). Sugar consumption and caries risk: A systematic review. *Journal of Dental Education, 65*(10), 1017-1023. https://doi.org/10.1002/j.0022-0337.2001.65.10.tb03447.x

23. Garcia-Closas, R., Garcia-Closas, M., & Serra-Majem, L. (1997). A cross-sectional study of dental caries, intake of confectionery and foods rich in starch and sugars, and salivary counts of Streptococcus mutans in children in Spain. *The American Journal of Clinical Nutrition, 66*(5), 1257-1263. https://doi.org/10.1093/ajcn/66.5.1257

24. Karjalainen, S., Soderling, E., Sewon, L., Lapinleimu, H., & Simell, O. (2001). A prospective study on sucrose consumption, visible plaque and caries in children from 3 to 6 years of age. *Community Dentistry and Oral Epidemiology, 29*(2), 136-142. https://doi.org/10.1034/j.1600-0528.2001.290208.x

25. Milgrom, P., Riedy, C. A., Weinstein, P., Tanner, A. C., Manibusan, L., & Bruss, J. (2000). Dental caries and its relationship to bacterial infection, hypoplasia, diet, and oral hygiene in 6- to 36-month-old children. *Community Dentistry and Oral Epidemiology, 28*(4), 295-306. https://doi.org/10.1034/j.1600-0528.2000.280408.x

26. Rodrigues, C. S., & Sheiham, A. (2000). The relationships between dietary guidelines, sugar intake and caries in primary teeth in low income Brazilian 3-year-olds: A longitudinal study. *International Journal of Paediatric Dentistry, 10*(1), 47-55. https://doi.org/10.1046/j.1365-263x.2000.00169.x

27. Rugg-Gunn, A. J., Hackett, A. F., Appleton, D. R., Jenkins, G. N., & Eastoe, J. E. (1984). Relationship between dietary habits and caries assessed over two years in 405 English adolescent school children. *Archives of Oral Biology, 29*(12), 983-992. https://doi.org/10.1016/0003-9969(84)90099-8

28. Shaw, J. H. (1986). Animal caries models - resource paper. *Journal of Dental Research, 65*(Spec Iss), 1485-1490. https://doi.org/10.1177/00220345860650090501

29. Clarkson, B. H. (1986). In vitro methods for testing the cariogenic potential of foods. *Journal of Dental Research, 65*(Spec Iss), 1516-1519. https://doi.org/10.1177/00220345860650091001

30. Ten Cate, J. M. (1986). Demineralization models: Mechanistic aspects of the caries process with special emphasis on the possible role of foods. *Journal of Dental Research, 65*(Spec Iss), 1511-1515. https://doi.org/10.1177/00220345860650090901

31. Stephan, R. M. (1940). Changes in hydrogen-ion concentration on tooth surfaces and in carious lesions. *The Journal of the American Dental Association, 27*(5), 718-723. https://doi.org/10.14219/jada.archive.1940.0218

32. Bibby, B. G., & Mundorff, S. A. (1975). Enamel demineralization by snack foods. *Journal of Dental Research, 54*(3), 461-470. https://doi.org/10.1177/00220345750540030501

33. Blinkhorn, A. S., & Davies, R. M. (1996). Caries prevention. A continued need worldwide. *International Dental Journal, 46*(2), 119-125.

34. Sreebny, L. M. (1982). Sugar and human dental caries. *World Review of Nutrition and Dietetics, 40*, 19-65. https://doi.org/10.1159/000406989

35. Woodward, M., & Walker, A. R. P. (1994). Sugar consumption and dental caries: Evidence from 90 countries. *British Dental Journal, 176*(8), 297-302. https://doi.org/10.1038/sj.bdj.4808439

36. Kantor, L. S. (1998). *A dietary assessment of the U.S. food supply: Comparing per capita food consumption with Food Guide Pyramid serving recommendations* (Agricultural Economic Report No. 772). U.S. Department of Agriculture, Economic Research Service.

37. Guthrie, J. F., & Morton, J. F. (2000). Food sources of added sweeteners in the diets of Americans. *Journal of the American Dietetic Association, 100*(1), 43-51. https://doi.org/10.1016/S0002-8223(00)00018-3

38. Gustafsson, B. E., Quensel, C. E., Lanke, L. S., Lundqvist, C., Grahnen, H., Bonow, B. E., & Krasse, B. (1954). The Vipeholm dental caries study: The effects of different levels of carbohydrate intake in 436 individuals observed for five years. *Acta Odontologica Scandinavica, 11*(3-4), 195-206. https://doi.org/10.3109/00016355308993925

39. Krasse, B. (2001). The Vipeholm dental caries study: Recollections and reflections 50 years later. *Journal of Dental Research, 80*(9), 1785-1788. https://doi.org/10.1177/00220345010800090201

40. Kashket, S., Yaskell, T., & Murphy, J. E. (1994). Delayed effect of wheat starch in foods on the intraoral demineralization of enamel. *Caries Research, 28*(4), 291-296. https://doi.org/10.1159/000261989

41. Lineback, D. R. (1984). The starch granule organization and properties. *Baker's Digest, 58*(3), 16-21.

42. Imfeld, T. N. (1983). *Identification of low caries risk dietary components* (Monographs in Oral Science, Vol. 11). Karger.

43. Kashket, S., Zhang, J., & van Houte, J. (1996). Accumulation of fermentable sugars and metabolic acids in food particles that become entrapped on the dentition. *Journal of Dental Research, 75*(11), 1885-1891. https://doi.org/10.1177/00220345960750110801

44. Lingström, P., van Houte, J., & Kashket, S. (2000). Food starches and dental caries. *Critical Reviews in Oral Biology & Medicine, 11*(3), 366-380. https://doi.org/10.1177/10454411000110030601

45. Lingström, P., Holm, J., Birkhed, D., & Björck, I. (1989). Effects of variously processed starch on pH of human dental plaque. *Scandinavian Journal of Dental Research, 97*(5), 392-400. https://doi.org/10.1111/j.1600-0722.1989.tb01452.x

46. Sheiham, A. (2001). Dietary effects on dental diseases. *Public Health Nutrition, 4*(2B), 569-591. https://doi.org/10.1079/PHN2001142

47. Edgar, W. M., Bibby, B. G., Mundorff, S., & Rowley, J. (1975). Acid production in plaques after eating snacks: Modifying factors in foods. *The Journal of the American Dental Association, 90*(2), 418-425. https://doi.org/10.14219/jada.archive.1975.0122

48. Grenby, T. H. (1990). Snack foods and dental caries. Investigations using laboratory animals. *British Dental Journal, 168*(9), 353-361. https://doi.org/10.1038/sj.bdj.4807192

49. Mormann, J. E., & Mühlemann, H. R. (1981). Oral starch degradation and its influence on acid production in human dental plaque. *Caries Research, 15*(3), 166-175. https://doi.org/10.1159/000260515

50. Sgan-Cohen, H. D., Newbrun, E., Huber, R., & Tenenbaum, G. (1988). The effect of previous diet on plaque pH response to different foods. *Journal of Dental Research, 67*(11), 1434-1437. https://doi.org/10.1177/00220345880670110801

51. Morrissey, R. B., Burkholder, B. D., & Tarka, S. M., Jr. (1984). The cariogenic potential of several snack foods. *The Journal of the American Dental Association, 109*(4), 589-591. https://doi.org/10.14219/jada.archive.1984.0039

52. Palmer, C. A. (2001). Important relationships between diet, nutrition, and oral health. *Nutrition in Clinical Care, 4*(1), 4-14. https://doi.org/10.1046/j.1523-5408.2001.00001.x

53. Firestone, A. R., Schmid, R., & Mühlemann, H. R. (1984). Effect of the length and number of intervals between meals on caries in rats. *Caries Research, 18*(2), 128-133. https://doi.org/10.1159/000260761

54. Gatenby, S. J. (1997). Eating frequency: Methodological and dietary aspects. *British Journal of Nutrition, 77*(Suppl 1), S7-S20. https://doi.org/10.1079/BJN19970102

55. Kandelman, D. (1997). Sugar, alternative sweeteners and meal frequency in relation to caries prevention: New perspectives. *British Journal of Nutrition, 77*(Suppl 1), S121-S128. https://doi.org/10.1079/BJN19970107

56. Holbrook, W. P., Arnadóttir, I. B., Takazoe, I., Birkhed, D., & Frostell, G. (1995). Longitudinal study of caries, cariogenic bacteria and diet in children just before and after starting school. *European Journal of Oral Sciences, 103*(1), 42-45. https://doi.org/10.1111/j.1600-0722.1995.tb00009.x

57. Holt, R. D. (1991). Foods and drinks at four daily time intervals in a group of young children. *British Dental Journal, 170*(4), 137-143. https://doi.org/10.1038/sj.bdj.4807449

58. Jenkins, G. N., & Ferguson, D. B. (1966). Milk and dental caries. *British Dental Journal, 120*(11), 472-477.

59. Tinanoff, N., & Palmer, C. A. (2000). Dietary determinants of dental caries and dietary recommendations for preschool children. *Journal of Public Health Dentistry, 60*(3), 197-206. https://doi.org/10.1111/j.1752-7325.2000.tb03328.x

60. Bowen, W. H., Pearson, S. K., Wuyckhuyse, B. C., & Tabak, L. A. (1991). Influence of milk, lactose-reduced milk, and lactose on caries in desalivated rats. *Caries Research, 25*(4), 283-286. https://doi.org/10.1159/000261379

61. Reynolds, E. C., & Johnson, I. H. (1981). Effect of milk on caries incidence and bacterial composition of dental plaque in the rat. *Archives of Oral Biology, 26*(5), 445-451. https://doi.org/10.1016/0003-9969(81)90046-5

62. Thompson, M. E., Dever, J. G., & Pearce, E. I. (1984). Intra-oral testing of flavoured sweetened milk. *New Zealand Dental Journal, 80*(361), 44-46.

63. Bibby, B. G., Huang, C. T., Zero, D., & Mundorff, S. A. (1980). Protective effect of milk against in vitro caries. *Journal of Dental Research, 59*(10), 1565-1570. https://doi.org/10.1177/00220345800590100701

64. Weiss, M. E., & Bibby, B. G. (1966). Effects of milk on enamel solubility. *Archives of Oral Biology, 11*(1), 49-57. https://doi.org/10.1016/0003-9969(66)90103-6

65. Edgar, W. M., Bowen, W. H., Amsbaugh, S., Monell-Torrens, E., & Brunelle, J. (1982). Effects of different eating patterns on dental caries in the rat. *Caries Research, 16*(5), 384-389. https://doi.org/10.1159/000260621

66. Kashket, S., & DePaola, D. P. (2002). Cheese consumption and the development and progression of dental caries. *Nutrition Reviews, 60*(4), 97-103. https://doi.org/10.1301/00296640260085822

67. Moynihan, P. J., Jenkins, G. N., & Fletcher, E. (2000). Cheese-containing meals increase plaque calcium following acidic drink consumption [Abstract 3697]. *Journal of Dental Research, 79*(Spec Iss), 606.

68. Mäkinen, K. K., Mäkinen, P. L., Pape, H. R., Jr., Allen, P., Bennett, C. A., Isokangas, P. J., & Isotupa, K. P. (1996). Conclusion and review of the Michigan Xylitol Programme (1986-1995) for the prevention of dental caries. *International Dental Journal, 46*(1), 22-34.

69. Hayes, C. (2001). The effect of non-cariogenic sweeteners on the prevention of dental caries: A review of the evidence. *Journal of Dental Education, 65*(10), 1106-1109. https://doi.org/10.1002/j.0022-0337.2001.65.10.tb03447.x

70. Machiulskiene, V., Nyvad, B., & Baelum, V. (2001). Caries preventive effect of sugar-substituted chewing gum. *Community Dentistry and Oral Epidemiology, 29*(4), 278-288. https://doi.org/10.1034/j.1600-0528.2001.290406.x

71. Food and Drug Administration. (1996). Health claims: Dietary sugar alcohols and dental caries. *Federal Register, 61*(165), 43433-43447.

72. Moynihan, P. (2000). Foods and factors that protect against dental caries. *Nutrition Bulletin, 25*(4), 281-286. https://doi.org/10.1046/j.1467-3010.2000.00085.x

73. Bennick, A. (2002). Interactions of plant polyphenols with salivary proteins. *Critical Reviews in Oral Biology & Medicine, 13*(2), 184-196. https://doi.org/10.1177/154411130201300208

74. Linke, H. A., Salgado, T., Retino, M., & Legeros, R. Z. (2000). Effect of black tea on caries formation in hamsters [Abstract 3601]. *Journal of Dental Research, 79*(Spec Iss), 594.

75. Hamilton-Miller, J. M. (2001). Anti-cariogenic properties of tea (Camellia sinensis). *Journal of Medical Microbiology, 50*(4), 299-302. https://doi.org/10.1099/0022-1317-50-4-299

76. Bowen, W. H. (1994). Food components and caries. *Advances in Dental Research, 8*(2), 215-220. https://doi.org/10.1177/08959374940080021101

77. Ooshima, T., Osaka, Y., Sasaki, H., Osawa, K., Yasuda, H., & Matsumura, M. (2000). Cariostatic activity of cacao mass extract. *Archives of Oral Biology, 45*(9), 805-808. https://doi.org/10.1016/S0003-9969(00)00035-6

78. Osawa, K., Miyazaki, K., Shimura, S., Okuda, J., Matsumoto, M., & Ooshima, T. (2001). Identification of cariostatic substances in the cacao bean husk: Their anti-glucosyltransferase and antibacterial activities. *Journal of Dental Research, 80*(11), 2000-2004. https://doi.org/10.1177/00220345010800111001

79. Edgar, W. M. (1978). Reduction in enamel dissolution by liquorice and glycyrrhizinic acid. *Journal of Dental Research, 57*(1), 59-64. https://doi.org/10.1177/00220345780570011101

80. Mundorff, S. A., Featherstone, J. D., Bibby, B. G., Curzon, M. E., Eisenberg, A. D., & Espeland, M. A. (1990). Cariogenic potential of foods. I. Caries in the rat model. *Caries Research, 24*(5), 344-355. https://doi.org/10.1159/000261296

81. American Dietetic Association. (1996). Position of the American Dietetic Association: Oral health and nutrition. *Journal of the American Dietetic Association, 96*(2), 184-189. https://doi.org/10.1016/S0002-8223(96)00051-6

82. Geddes, D. A. (1994). Diet patterns and caries. *Advances in Dental Research, 8*(2), 221-224. https://doi.org/10.1177/08959374940080021102

83. Papas, A. S., Joshi, A., Palmer, C. A., Giunta, J. L., & Dwyer, J. T. (1995). Relationships of diet to root caries. *The American Journal of Clinical Nutrition, 61*(Suppl), 423S-429S. https://doi.org/10.1093/ajcn/61.2.423S

84. Burt, B. A., Eklund, S. A., Morgan, K. J., Larkin, F. E., Guire, K. E., Brown, L. O., & Weintraub, J. A. (1988). The effect of sugars intake and frequency of ingestion on dental caries in a three-year longitudinal study. *Journal of Dental Research, 67*(11), 1422-1429. https://doi.org/10.1177/00220345880670110601

85. Petridou, E., Athanassouli, T., Panagopoulos, H., & Revinthi, K. (1996). Sociodemographic and dietary factors in relation to dental health among Greek adolescents. *Community Dentistry and Oral Epidemiology, 24*(5), 307-311. https://doi.org/10.1111/j.1600-0528.1996.tb00869.x

86. Rugg-Gunn, A. J. (1994). *Nutrition and dental health*. Oxford University Press.

87. DePaola, D. P., Mobley, C., & Touger-Decker, R. (2002). Nutrition and oral medicine. In C. D. Berdanier (Ed.), *Handbook of nutrition and food* (pp. 1113-1114). CRC Press.

88. U.S. Department of Health and Human Services & U.S. Department of Agriculture. (2000). *Nutrition and your health: Dietary guidelines for Americans* (5th ed.). https://www.health.gov/dietaryguidelines/

89. Mobley, C., & Dodds, M. W. (1998). Diet and dental health. *Topics in Nutrition, 7*(1), 1-19.

دور العادات الغذائية في الوقاية من تسوس الأسنان

الملخص:

الخلفية: تسوس الأسنان مرض معدٍ يتأثر بالنظام الغذائي، وينتج عن التفاعل بين الكربوهيدرات القابلة للتخمر والبكتيريا الفموية المنتجة للأحماض، مما يؤدي إلى إزالة المعادن من مينا الأسنان. كما تؤثر عوامل ثانوية أخرى في تطور التسوس مثل نوعية اللعاب، والتعرض للفلورايد، وسلامة مينا الأسنان.

الهدف: تتناول هذه المقالة دور العادات الغذائية في الوقاية من تسوس الأسنان، مع التركيز على الأطعمة المسببة للتسوس والمضادة له، وتكرار تناول الطعام، والتدخلات الغذائية لتقليل خطر التسوس.

المنهجية: تم إجراء مراجعة شاملة للأدبيات العلمية المتوفرة، وتحليل الدراسات المتعلقة بتمثيل السكر، وديناميكية الأس الهيدروجيني في اللويحة السنية، وخصائص بقاء الطعام في الفم، وأنماط النظام الغذائي. وتم تقييم دراسات رئيسية مثل دراسة فيبيهولم حول تسوس الأسنان وأبحاث بدائل السكر.

النتائج: يرتبط الاستهلاك المتكرر للكربوهيدرات القابلة للتخمر، وخاصة الأطعمة اللزجة والمحتجزة في الفم، بزيادة خطر التسوس بشكل كبير. في المقابل، تساهم منتجات الألبان (خصوصاً الجبن)، وكحوليات السكر (مثل زيليتول)، والأطعمة الغنية بالألياف في تحفيز إفراز اللعاب وتعزيز إعادة تمعدن الأسنان. كما يؤثر تكرار تناول الطعام وتسلسل الوجبات على تطور التسوس، وتُعدّ الوجبات الخفيفة بين الوجبات الرئيسية العامل الأكثر خطورة.

الاستنتاج: تتطلب الوقاية الفعالة من تسوس الأسنان تقليل تناول الكربوهيدرات القابلة للتخمر، وتعزيز الأطعمة الوقائية، ودمج الإرشادات الغذائية في الرعاية السنية. كما ينبغي أن تركز الاستراتيجيات الصحية العامة على التغذية المتوازنة، واستخدام الفلورايد، وتحفيز اللعاب.

الكلمات المفتاحية: تسوس الأسنان، الكربوهيدرات القابلة للتخمر، الأطعمة المسببة للتسوس، الأطعمة المضادة للتسوس، الوقاية الغذائية، زيليتول، تحفيز اللعاب.