We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists



186,000

200M



Our authors are among the

TOP 1% most cited scientists





WEB OF SCIENCE

Selection of our books indexed in the Book Citation Index in Web of Science™ Core Collection (BKCI)

Interested in publishing with us? Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected. For more information visit www.intechopen.com



Chapter

Dietary Factors, Salivary Parameters, and Dental Caries

Efka Zabokova Bilbilova

Abstract

Diet and oral microflora are connected to caries along with host factors such as salivary composition and flow. The only component of the food with potentially cariogenic effect is fermentable carbohydrate sucrose. Sucrose is generally accepted as the most cariogenic dietary factor, and consumption of sucrose is associated with the frequency of dental caries in humans. Saliva is a biological environment, important for the physiology of the mouth. It achieves its mechanical functions of cleaning and protection through various physical and biochemical mechanisms. Bicarbonates, phosphates, and proteins have a buffer role in the saliva environment. Other compounds or enzymes in this group acting as prophylactic buffers are urea, salivary amylases, and fluorides.

Keywords: dental caries, diet, sucrose, saliva, salivary buffer

1. Introduction

From the ancient time, dental caries has existed, even from the time when the only way to eat and drink was hunting and gathering. According to the World Health Organization, 60–90% of schoolchildren worldwide have experienced caries, with the disease being most prevalent in Asian and Latin American countries (WHO, 2008). Dental caries is a multifactor disease which appears when demineralization of the hard tissues of the teeth occurs by organic acids formed by bacteria in dental plaque through the anaerobic metabolism of sugars derived from the diet.

Calcium is lost from the tooth surface, and demineralization occurs only when sugars or other fermentable carbohydrates are ingested in which results fall in dental plaque pH caused by organic acids that increase the solubility of calcium hydroxyapatite in the dental hard tissues.

Lifestyle or dental health habits are the factors that should be connected to dental diseases. Dietary and daily habits, familial and physiological well-being, socioeconomic status and lifestyle, awareness and education, and area where they live are the factors that should be taken into consideration when discussing oral health. The higher the socioeconomic status is, the more the people are exposed to the availability of junk foods and susceptible to its frequent consumption. Those from lower economic group and rural area are not as much exposed to such food habits, and they do not buy them because they are expensive for their pocket. Many adolescents fail to brush their teeth effectively and tend to consume cariogenic foods even though they have basic knowledge of dental health. Children who have caries eat snacks between meals, more than those children without dental caries do. The basic means of avoiding these primary public health measures are compiled with the use of topical fluorides and fluoridated water. When it comes to nutrition perspective, one of the main things is to have balanced diet and adherence to the dietary guidelines and the dietary reference intakes.

1.1 The dental caries process

Dental caries occurs due to the demineralization of enamel and dentin (the hard tissues of the teeth) by organic acids formed by bacteria in dental plaque through the anaerobic metabolism of sugars and other fermentable carbohydrates derived from the diet [1]. Organic acids increase the solubility of calcium hydroxyapatite in dental hard tissues, and demineralization process of the tooth surface occurs due to calcium loss.

Teeth are most susceptible to dental caries soon after they erupt, and therefore the peak ages for dental caries are 2–5 years for the deciduous dentition and early adolescence for the permanent dentition [2]. The age of adolescences is when permanent teeth begin to grow and get their full position in the dental arch. This is a crucial age for the development of several oral diseases. Dental caries, periodontal disease, and orthodontic problems such as overcrowding of the teeth or malocclusions are bringing changes and altering the facial profile and esthetic appearance.

Certain psychological factors like self-confidence and social outlook of the individuals can also be affected, and they can leave permanent effect on the psychology of the child if not appropriately treated.

Neglecting the general problems, the lack of awareness and expertise is one of the reasons that most of the children at this age face these problems. Since the treatment of dental disease is very expensive especially in low-income countries, it would exceed the available resources for health care. The large financial benefits of preventing dental diseases should be emphasized to countries where current disease levels are high [3].

1.2 Effects of dental caries

It is undisputable that the development of dental caries is a result of poor diet, and it has been observed in humans and animals that frequent and prolonged exposure to carbohydrates and sugars results in an appearance of dental caries. Important bacteria in the development of dental caries are *Streptococcus mutans* and *Streptococcus sobrinus*. These bacteria produce organic acids from food sugars and help bacterial colonization of the tooth surface. The bacteria attached to teeth in dental plaque, found as a thin film on the surface of the enamel, utilize mono- and disaccharides (e.g., glucose, fructose, and sucrose) to produce energy, and acid is the by-product of this metabolism.

Consequently, the acidity of dental plaque may decrease to a point where the demineralization of the tooth begins. Demineralization occurs at a low pH when the oral environment is undersaturated with mineral ions, relative to a tooth's mineral content. The enamel crystal, which consists of carbonated apatite, is dissolved by organic acids (lactic and acetic) that are produced by the cellular action of plaque bacteria in the presence of dietary carbohydrates. The "white spot lesion" is the initial stage that occurs just below the enamel surface and produces a visual whitening of the tooth. At this stage of mineral loss, the lesion may not progress any further or could even regain minerals (i.e., remineralize) if the cariogenic environment diminishes. The prevention measures that can remineralize the initial carious lesion are as follows: decreasing the carbohydrate source to the bacteria, treating the tooth with fluoride, reducing the levels of cariogenic bacteria, or reducing the bacterial ability to produce acid.

Dietary Factors, Salivary Parameters, and Dental Caries DOI: http://dx.doi.org/10.5772/intechopen.92392

The initial lesion will continue to lose mineral if the procedure of disease suppression is not initiated and the acidic challenge is unabated. The progressive dissolution of enamel and loss of enamel surface structure eventually give rise to a frank carious lesion [4]. Sugary food products and their everyday consumptions exert our teeth. The reasons behind dental caries are the exposure to junk foods, colas, sweets, and other dietary products which are easy to access and abundantly available for children to consume. That is why dental caries is like a sort of nontransmittable and nonfatal sickness [5].

1.3 Dietary factors in the initiation and progression of dental caries

Some authors emphasize the importance of the dental biofilm and dietary sugars as essential primary etiological factors causing the appearance of the caries; moreover, one of them cannot cause caries in the absence of the other.

The main direct impact of the diet is mediated through its effect on the pH of the dental biofilm. Foods high in fermentable carbohydrates (mainly sugars) cause a low biofilm pH, while foods high in proteins and fats favor a more neutral biofilm pH. High-protein foods increase the urea concentration of saliva, which can be converted by ureolytic bacteria to ammonia; this raises the biofilm pH and is associated with decreased caries risk. Dietary factors can have an indirect effect by modifying the composition and metabolic activity of dental biofilm.

The major dietary factor affecting dental caries prevalence and progression is sucrose [6]. A low consumption example is from a study of the Hopewood House in Australia, conducted between 1947 and 1952. As a matter of fact, children living in this closely supervised environment consumed food that was virtually free of sugar and white flour products. Data collected from these children revealed an extremely low dental caries prevalence, compared to children attending other Australian schools [7].

High sugar consumption's effect is best revealed from the report of the classic Vipeholm study [8]. This study examined three factors leading to these stages as follows: the timing of sugar ingestion, the effects of the frequency of sugar consumption, and finally the consistency of the sugar on dental caries rates. According to the results, the degree of the sugar's consistency was more important than the addition of sugar to the diet and especially if it was consumed between meals, or products, which are sticky, in a form that stayed longer in the mouth such as toffees. These products have a bigger cariogenicity impact than foods that are eliminated quickly from the oral cavity. Therefore, frequent ingestion of foods such as hard candies and throat lozenges that contain fermentable carbohydrates can be extremely harmful to the teeth. The conclusions from this study, conducted a half century ago, are still well regarded today:

- 1. If sugar is taken with meals, then only a small caries increase is noted.
- 2. A marked increase in caries increment is shown if sugar is consumed as snacks between meals.
- 3. If you consume sticky candies containing sugar, then the caries activity will be at the highest form.
- 4. Caries activity may vary greatly among individuals.
- 5. By eliminating sugar-rich foods, caries activity will be declined.

Dental Caries

The detrimental effects of sugar in causing tooth decay are shown in the two major studies of public health importance, and those are the classic Vipeholm study in Sweden and Hopewood House study in Australia. Children generally consume diets which are rich in sugar like sweets, candies, cakes, colas, etc. That is why a lot of awareness has been raised since this food has a negative effect on oral health, and that is the appearance of dental caries. Nowadays, the household food that we generally eat contains certain amounts of sugar. That is why these two studies are of huge public health importance when conducting preventive dental health programs especially in schools where the drawbacks of consuming such diet containing sugars can be addressed.

1.4 Food products that play a main role in the development of dental caries

A direct relationship between dental caries incidence and sugar (carbohydrates) intake is indisputed. The caries will not be developed if there are no fermentable carbohydrates in the food [9].

Free sugars as defined by the World Health Organization present as monosaccharides and disaccharides added to food, and sugars are naturally present in honey, syrups, and fruit juices. Fermentable carbohydrates are free sugars, glucose polymers (syrups and maltodextrins), fermentable oligosaccharides, and highly refined starches. They are added to food in industrialized countries and are as acidogenic as sucrose. However, sucrose and starches today present as the main carbohydrates in modern society diet. Sucrose is the most cariogenic sugar which is a highly soluble substrate transformed into intracellular (IPS) and extracellular polysaccharides (EPS). It diffuses easily into the dental plaque accumulation and induces a lower pH [10]. **Starch** is a carbohydrate that can cause very small amounts of caries, unlike real sugar. It is found in fruits and vegetables and can be consumed raw or cooked. Starchy foods such as rice, potatoes, pasta, and bread have very low cariogenicity, and this is why they can cause less caries than sucrose. Starch can be sorted out to mono- and disaccharides and metabolized by bacteria, so it is retained on the teeth long enough to be hydrolyzed by salivary amylase.

Since the original Miller's study, Stephan in both of his researches (1940, 1944) about the relationship between caries and sugar showed that fermentable carbohydrates can transform into acid in dental plaque. A direct relationship between caries incidence and the frequency of consumption of sweets was also presented [11], and these findings supported those of the Vipeholm study [12].

Sucrose is freely diffusible in dental biofilm and metabolized by oral bacteria *Streptococcus mutans* [13]. Bacteria metabolize sucrose to soluble and insoluble extracellular polysaccharide glucan by enzyme glucosyltransferases (GTFs). Few mechanisms are involved in the role of extracellular glucans as the major caries associated factor. Glucan enables the bacteria to adhere firmly to the teeth [14], and in dental plaque, they contribute to the structural integrity of dental biofilms [15].

Several studies showed that the presence of insoluble glucan enhanced the demineralization potential of *S. mutans*. Glucan altered the diffusion properties of plaque and allowed deeper penetration of dietary carbohydrates [16, 17].

There are several important and critical cariogenic factors to be considered when evaluating starch and caries relationship. They are the size and frequency of tooth exposure, the bioavailability of the starches, the microbial flora of dental plaque, the pH-lowering capacity of dental plaque, and the flow rate of saliva. Starchy foods with higher amounts of sucrose are as cariogenic as a sucrose. Some cooked and processed starches are dissolved by salivary amylase, and they release glucose and maltose metabolized by oral bacteria to acids. In Rugg-Gunn [18] study, the

Dietary Factors, Salivary Parameters, and Dental Caries DOI: http://dx.doi.org/10.5772/intechopen.92392

relationship between starches and dental caries was proved, and several conclusions were made. Rice, potatoes, bread, and cooked staple starchy foods have low cariogenicity in humans. Uncooked starch has low cariogenicity, while heat-treated starch induces lesser caries than sugars. Foods with cooked starch and higher amounts of sucrose are as cariogenic as similar quantities of sucrose.

Fresh fruits contain various sugars and may be capable of causing caries under some conditions. They have low cariogenicity, while citrus fruits have not been associated with dental caries. Increased consumption of fresh fruit in the diet is decreasing the level of dental caries in a population [19]. Although excessive exposure to fructose may produce dental caries, fresh fruits are likely to be much less cariogenic than most sucrose-rich snack foods consumed by children. One hundred percent fruit juice has also been associated with caries, but the relationship is less clear. Children consuming more than 17 oz. 100% juice are more likely to have caries, than children consuming water or milk [20]. Conversely, in a cohort of low-income African-American children, 100% fruit juice was found to be protective of caries. The fact that 100% fruit juice contains about the same amount of sugar as the average sugar-sweetened beverages made it important to understand its role in caries [21]. Animal studies revealed that all fruits cause less caries than sucrose but dried fruits may potentially be more cariogenic since the drying process breaks down the cellular structure, releasing free sugars that tend to have a longer oral clearance.

Flavored drinks, especially aerated beverages like cola, have a much greater cariogenic potential due to high sugar content and regular consumption. Children are frequently offered with these drinks because of their high acceptance, low cost, and parent's belief of being very nutritious [22]. Different campaigns and various forms of advertising by the media changed public health knowledge, and people started to become aware and understand about the bad effect of this kind of food.

Milk is most frequently consumed by schoolchildren. In milk a sugar named lactose is not fermented as the other sugars, so it is less cariogenic because the phosphor proteins inhibit enamel dissolution and the milk antibacterial factors may interfere with the oral microbial flora.

Cheese can lead to protection against creating caries as it stimulates salivary flow and raises the calcium, phosphorus, and protein content of plaque.

The sugar alcohols like sorbitol, mannitol, and xylitol are kind of sweeteners that are metabolized by bacteria at much slower rate than glucose or sucrose, which is not metabolized at all. According to certain clinical studies, xylitol chewing gum has the ability to reverse initial white spot lesions on teeth.

When dental decay happens there is high probability of losing a tooth. That leads to a reduced ability to eat a varied diet. It is in particular associated with a low consumption of fruits, vegetables and non-starch polysaccharides (NSP) in the persons diet [23]. NSP intakes of less than 10 g/day and fruits and vegetable intakes of less than 160 g/day have been reported in edentulous subjects. Therefore, tooth loss may impede the achievement of dietary goals related to the consumption of fruits, vegetables, and NSP. Tooth loss has also been associated with loss of enjoyment of food and confidence to socialize. So, basically, it is clear that dental diseases have a detrimental effect on the quality of life both in childhood and older age [24].

1.5 Eating between meals

An important issue for the appearance of dental caries in older children as well as infants is not only the total quantity but also the form of the carbohydrate as well as the frequency of consumption since the refined carbohydrates exert their effect in the appearance of dental caries by serving as a substrate for caries-producing streptococci, which as a small piece of it adheres to the teeth for almost an hour. In the case of sugars that are not in sticky form, a specified amount consumed at one time is likely to be less conducive to the formation of dental caries than the same amount consumed in small portions throughout the day. There is considerable evidence that between-meal snacks cause the development of dental caries. Foods that must be avoided between meals are the following: sugar, honey, corn syrup, candies, jellies, jams, sugared breakfast cereals, cookies, cakes, chewing gum, and sweetened beverages, including flavored kind of milks, carbonated drinks, sweetened fruit juices, and fruit or fruit-flavored drinks. Finally, eating frequency, particularly constant grazing or sipping of foods and beverages, is also caries promoting. In a recent study in a diverse sample of children aged 2 to 6 years, eating frequency was associated with severe early childhood caries [25].

1.6 Dietary fluoride and water fluoridation

Reduction of dental caries can be achieved with the help of fluoride or in other words dietary fluoride drinking water, which also has rich sources. The ingested fluoride becomes incorporated into enamel during tooth formation and increases the resistance of the tooth to decay. However, the main protection from dietary fluoride is the localized intraoral effect. Fluoride promotes the remineralization of damaged enamel with resistant fluorapatite and also inhibits bacterial metabolism of sugars. As we can see, the benefits to the exposure of teeth to fluoride are therefore beneficial lifelong. It may be added to an optimum concentration of 1 mg/L as a caries preventive measure if natural water supplies are low in fluoride; Murray et al. [26] have reviewed the published data on the effect of water fluoridation on caries and have concluded that on average water fluoridation reduces dental caries by 50%. In a study of 5-year-old children, Carmichael et al. have demonstrated that water fluoridation is effective in reducing dental caries across social classes and, in terms of the number of teeth saved per child, the benefits are greatest in the lower social classes [27].

According to UK national surveys, it has been indicated that those from lower social classes have higher levels of dental diseases and poorer oral hygiene practice and are less likely to visit the dentist [28]. In these cases, dental caries is not eliminated even though the benefit of fluoride is reducing caries. Fluoride repairs the damage caused by acids produced by plaque bacteria but does not remove the cause of caries, i.e., dietary sugars. The process of prevention requires both a reduction in sugar intake as well as optimum exposure to fluoride. Very extensive and comprehensive research by the National Health Survey concluded that a preventive dentistry program is water fluoridation.

1.7 Dietary advice

Dietary advice by dental health professionals should be consistent and not conflict with the advices from other health professionals, based on the evidence in the various professional fields and based on the national dietary guidelines. The advices may be more readily accepted from the people when the oral healthcare professionals can make unequivocally clear that the advice benefits caries prevention. If not, the person may not understand why the dental professional interferes with his diet and not accept the advices. However, this does not dismiss the dental professional from also explaining the benefits for general health on limiting or reducing the intake of sugars. Under the premise that it benefits oral health, the dental health professional can make stronger restrictions than the general guidelines as long as they do not harm general health. Generally speaking a diet that is beneficial to both general and dental health is one that is low in free sugars, saturated fat, and salts, as well as high in fresh fruits, vegetables, nuts and seeds, and wholegrain carbohydrates with modest amounts of legumes, fish, poultry, and lean meat and plenty of fluids preferably water and milk and, thus, modest with sugar sweetened beverages [29].

2. Saliva and oral health

The teeth and oral mucosa are cleaned with the help of saliva, which is a mixed glandular secretion. Saliva by itself is consisted of three glands, and they are as follows: submandibular, sublingual, and finally the parotid. It also has hundreds of small glands inside the oral mucosa and submucosa as well as gingival cervical fluid.

The maintenance of healthy teeth and oral tissues could be achieved only with the help of saliva's presence. If there were a severe reduction of the saliva's production, then there would be a very fast deterioration of oral health as well as the patient's life. The results from such a condition could lead to eating difficulties like: swallowing difficulties, bad oral hygiene, dental caries that progresses very fast, mucosa's burning sensation, difficulty in talking, wearing denture, oral infections like *Candida*, and ulceration of oral mucosa.

Dry mouth is a problem, which appears in huge proportions. Xerostomia or in other words dry mouth is very common for people with Sjogren's syndrome, as a result of radiotherapy in the head and neck in cancer treating and especially in the case of older generations when they are prescribed with drugs. The saliva's role in oral health is huge especially taking into consideration the sicknesses that appear because of decreased quantity or quality of saliva. That is why it is very important to early diagnose and prevent this condition.

Saliva is considered as the most easily available diagnostic fluid for noninvasive collection and analysis because through it we can diagnose caries susceptibility, systemic, physiological, and pathological, and we can monitor the level of hormones, drugs, antibodies, microorganisms, and ions.

In this research, we will try to present the main functions of saliva, the anatomy and histology of salivary glands, the physiology of saliva formation, the constituents of saliva, and the use of saliva as a diagnostic fluid, including its role in caries risk assessment.

2.1 Saliva's functions

Saliva has several functions which are very protective, but it has also other functions presented in **Figure 1**. Salivary function can be organized into five major categories that serve to maintain oral health and create an appropriate ecologic balance: (1) lubrication and protection, (2) buffering action and clearance, (3) maintenance of tooth integrity, (4) antibacterial activity, and (5) taste and digestion [30].

Figure 2 presents the changes in plaque pH following as a result of sucrose rinse. The graphs are named as Stephan's curve according to the name of the scientist who was the first one who described it in 1944. By using antimony probe microelectrodes in a series of experiments, he also measured changes in plaque pH.

The unstimulated plaque pH in **Figure 2** is approximately 6.7. After the process of sucrose rinse, the plaque pH within a few minutes is reduced to less than 5.0. When the enamel is below the critical pH 5.5, then there is demineralization of the enamel. For about 15–20 min, plaque pH stays below the critical pH and does return to normal for about 40 min. In the presence of saliva and other fluids that are super-saturated with the help of hydroxyapatite and fluorapatite, the enamel itself could be remineralized only when the plaque pH recovers to a level above the critical pH.



Figure 1. *Functions of saliva.*





The buffering capacity, the degree of access to saliva, the velocity of the salivary film, and the saliva's urea content are the ones that determine the variation of the shape of Stephan's curve among individuals and the rate of recovery of the pH plaque.

The major buffer in stimulated saliva is the carbonic acid/bicarbonate system. As the bicarbonate ion concentration gets higher, also the buffering capacity of saliva increases.

2.2 Saliva as a diagnostic fluid

2.2.1 General diagnostics

Nowadays for the study of bacteria, proteins, and genes, there are very highlevel techniques where they apply saliva in order to spread out the field of oral diagnostics in the process of learning and understanding the oral diseases, systemic diseases, as well as metabolism. Saliva by itself presents an opportunity for the identification of biomarkers for the diseases like dental caries, periodontal diseases, and oral diseases, but all this should be easily done with careful collection and handling.

2.3 Caries risk assessment

There have been developed a series of caries risk assessment tests based on saliva's measurements. These tests measure the capacity of salivary buffering and salivary mutans streptococci and lactobacilli. The increased risk of developing caries comes because of high levels of mutans streptococci, i.e., >105 colony-forming units (CFUs) per mL of saliva. Individuals with high levels of lactobacilli (>105 CFUs per mL saliva) are the ones who consume frequently carbohydrates, and because of that they have an increased risk of caries.

As an answer to the question what is buffering capacity, one could answer that it is the host's capability to neutralize reduction pH's plaque constructed by acidogenic organisms. Useful caries indicators for monitoring, preventive measures, and profiling patient's disease are the salivary tests.

Table 1 lists some salivary variables measured for caries risk assessment in dentistry, which are more used for measurement than the other types.

While either measuring unstimulated or stimulated saliva's flow rates, we should bear in mind the conditions of saliva's collection process. When measuring unstimulated flow, which is usually at rest, repeated measurements should be assessed during the same day as a result of circadian rhythm and also because chewing (mechanical) and citric acid (gustatory) produce different results.

The best way of measuring unstimulated or stimulated saliva is using commercial kit. When it comes to buffering capacity of unstimulated saliva which is lower or stimulated saliva, they are very easily measured at the chairside. In order to do bacteriological tests as chewing dislodges the flora into the saliva, then the best way is to use paraffin wax-stimulated saliva samples. From stimulated saliva samples, you can culture mutans streptococci and lactobacilli. Their measurements could also be facilitated with the help of commercially available chairside tests. However, when it comes to fluoride, calcium, and phosphate biochemical measurement, then these must be done with the help of special laboratory facilities that are not available to practitioners.

2.4 Unstimulated saliva

As an answer to the question what is unstimulated whole saliva, one could answer that it is the mouth's secretion mixture with tastants or chewing in the absence of exogenous stimuli. It is composed of parotid, submandibular, and sublingual secretions as well as the minor mucous glands, but it also contains

Fluid/lubricant	It coats hard and soft tissue. Helps to protect against mechanical, thermal, and chemical irritation and tooth wear. Assists smooth air flow, speech, and swallowing.
Ion reservoir	Solution supersaturated with respect to tooth mineral facilitates remineralization of the teeth. Acidic proline-rich proteins and statherin in saliva inhibit spontaneous precipitation of calcium phosphate salts.
Buffering action and clearance	Helps to neutralize plaque pH after eating, thus reducing time for demineralization.
Mechanical function of cleaning the tooth surface	Clears food and aids swallowing.
Antimicrobial activity	Specific (e.g., sIgA) and non-specific (e.g. lysozyme, lactoferrin, and myeloperoxidase) anti-microbial mechanisms help to control the oral microflora.
Digestion	The enzyme α -amylase is the most abundant salivary enzyme; it splits starchy foods into maltose, maltotriose, and dextrins.
Protective remineralization (promoted by fluoride)	Saliva also inhibits caries by protective remineralization. This is promoted by fluoride ions in saliva.

Table 1.

Salivary variables measured for caries risk assessment.

desquamated epithelial cells, gingival crevicular fluid, leucocytes (mainly from the gingival crevice), bacteria, and possibly food residues, blood, and viruses.

The collection of saliva from the patient is done in that way that the patient spits out saliva in regular intervals of time without swallowing it, and there is another way when the patient keeps his or her head down and mouth just a bit open so that saliva can drip down from the mouth into a beaker during a time interval. However, one should bear in mind that when saliva is spit down, the number of desquamated epithelial cells as well as bacteria are increased. The difference between the secreted amount by the different salivary glands and the evaporated volumes is the measured flow rate. The unstimulated salivary flow rates in healthy individuals and the average value for whole saliva is about 0.3–0.4 mL/min. Patients say that they have dry mouth (xerostomia) only when saliva is almost completely absent. Objective evidence of hyposalivation is considered a flow rate of <0.1 mL/min.

Dentists should also measure salivary flow as part of their regular examination so that when patients complain of dry mouth, they will have the tests. The usual problems are related to swallowing difficulty that often leads to individuals with very little saliva but without discomfort and others with saliva flow rates within the normal range who feel that their mouth is drowning in saliva.

2.5 Stimulated saliva

Stimulated saliva is produced in response to a mechanical, gustatory, olfactory, or pharmacological stimulus, contributing to around 40–50% of daily salivary production. Several studies of stimulated salivary flow rates have been done in healthy populations and show a wide variation among individuals. The salivary flow (SF) index is a parameter allowing stimulated and unstimulated saliva flow to be classified as normal, low, or very low (hyposalivation). In adults, normal total stimulated SF ranges 1–3 mL/min, and low ranges 0.7–1.0 mL/min, while hyposalivation is characterized by a stimulated SF <0.7 mL/min. Many factors influence the stimulated salivary flow rate which, for whole saliva, has an average maximum value of about 7 mL/min.

2.5.1 Mechanical stimuli

Eating is a strong stimulus for the secretion of saliva by the major salivary glands. Large volumes of saliva are secreted before, during, and after eating via the gustatory-salivary reflex, masticatory-salivary reflex, olfactory-salivary reflex, and esophageal-salivary reflex. The action of chewing, in the absence of any taste, will stimulate salivation to a smaller degree than maximum gustatory stimulation with citric acid. Mastication also serves to mix the contents of the mouth, thus increasing slightly the distribution of the different types of saliva around the mouth. Mechanical stimulation of the fauces (the gag reflex) leads to increased salivation.

2.5.2 Gustatory and olfactory stimuli

Acid is the most potent of the five basic taste stimuli, the other four being salty, bitter, sweet, and umami. A study performed with different concentrations of citric acid revealed that 5% citric acid stimulated an average maximum salivary flow rate of about 7 mL/min. The citric acid was continuously infused into the mouth, and the teeth were covered with a paraffin film to protect them against the acid. For a clinical evaluation of the residual secretory capacity in patients with hyposalivation, a 3% citric acid solution can be applied to the patient's tongue at regular intervals so that the degree of stimulation is relatively standardized. If a gustatory stimulus is held in the mouth without movement, salivary flow decreases to nature of stimulus gland size, mechanical unilateral stimulation, gustatory vomiting, pharmacological olfaction, food intake smoking, and gag reflex.

Dawes [31] has stimulated the flow of saliva alters its composition and noted that the rate of salivary flow increases the concentration of protein, sodium, chloride, and bicarbonate and decreases the concentration of magnesium and phosphorus. Perhaps of greatest importance is the increase in the concentration of bicarbonate, which increases progressively with the duration of stimulation. The increased concentration of bicarbonate diffuses into the plaque, neutralizes plaque acids, increases the pH of the plaque, and favors the remineralization of damaged enamel and dentin.

2.6 Saliva's buffering ability

Buffer solutions are solutions that maintain an approximately constant pH when small amounts of either acid or base are added or when the solution is diluted. These solutions own the capacity of resisting changes of pH when either acids or alkalis are added to them. There are three possible buffer systems in saliva—the carbonic acid/bicarbonate system, the phosphate system, and the proteins.

2.6.1 Bicarbonate

Bicarbonate is one of the most important systems in saliva, which is produced by dental plaque, and its concentration could be from less than 1 mmol/L in unstimulated parotid saliva to a very high flow rate of 60 mmol/L which is elicited by chewing gum thus having a bicarbonate concentration of about 15 mmol/L. The level of bicarbonate ions in unstimulated saliva is too low to be an effective buffer. For those who suffer from the gastroesophageal reflux disease, the bicarbonate in saliva will help them in the clearance process of acid from the esophagus.

The carbonic acid/bicarbonate system is one of the components of the saliva that modifies the creation of caries. It does this by changing the environmental pH and possibly the virulence of bacteria that cause decay. Tanzer et al. [32] tasted the efficacy of a sodium bicarbonate-based dental power and paste with the addition of fluoride on dental caries and on *Streptococcus sobrinus* or *Streptococcus mutans* recoveries in rats. These authors observed that the caries reductions in these studies ranged from 42 to 50% in the rats treated with bicarbonate dentifrices when compared with rats treated with water [33, 34].

2.6.2 Phosphate

The concentration of phosphate in non-stimulated saliva is about 5–6 mmol/L, compared to a level of about 1 mmol/L in plasma; there is still too little phosphate in saliva to act as a significant buffer. The pH of unstimulated saliva is less than the pK₂ value of 7.2 for phosphate so that most of the phosphate is present as $H_2PO_4^-$ and cannot accept another hydrogen ion until the pH is close to 2.1, the pK₁ for phosphate.

2.6.3 Proteins

In saliva's plasma there is about one-thirtieth protein concentration as well as few amino acids with acidic or basic side chains which present an important buffering effect at the usual pH of the oral cavity.

2.6.4 pH

When the bicarbonate concentration increases, the salivary pH increases too. Henderson and Hasselbalch give the equation of the relationship between the pH and the bicarbonate concentration, which is $pH = pK + log[HCO_3^-]/[H_2CO_3]$, in which the pK (about 6.1) and $[H_2CO_3]$ (about 1.2 mmol/L) are virtually independent of the flow rate. The latter is in equilibrium with the pCO₂ which, in saliva, is about the same as that in the venous blood. If we try to measure the pH of saliva, then it is very obligative to avoid exposure of the saliva to the atmosphere because the pH will be artificially elevated and CO₂ will be released. At very low flow rates, the pH of parotid saliva can be as low as 5.3, rising to 7.8 at very high flow rates. Because of the low bicarbonate concentration, patients with hyposalivation will have a low salivary buffering capacity and a low salivary pH (**Figure 3**).

2.6.5 Urea

The importance of salivary urea was acknowledged early in dental literature [35, 36]. The pH-raising effect of intraoral urea application was first described by Stefan [37]. This author found that in both in vivo and in vitro, urea could raise plaque pH up to pH 9 and that the addition of 40–50% urea to carbohydrates largely overcame the pH-lowering effect for up to 24 h. The value of salivary urea ranges from 2 to 6 mmol/L.

Urea possesses the capability to inhibit the metabolism and multiplication of bacteria in the saliva, which indirectly neutralize the acids in the oral environment and maintain the salivary acidobasic balance due to its buffer capacity [37, 38].

Less aciduric oral bacteria (*Streptococcus sanguinis* and *Streptococcus gordonii*) associated with dental health have the ability for alkali generation by hydrolyzing urea or arginine to **ammonia**. Production of ammonia is a mechanism that influences the balance remineralization-demineralization of the tooth, maintains neutral pH in oral cavity, and prevents the appearance of a cariogenic microflora [39, 40].



Figure 3. *The effects of flow rate on the concentrations of some components of saliva.*

Urea can be used as a constituent of chewing gums for neutralized acids. Imfeld [41] explored the effect of sugar-free chewing gums containing various amounts of urea on the pH recovery in dental plaque.

After rinsing the mouth with 10 or 50% (w/v) sucrose solution, the respondents chewed the gum with different content of urea (10, 20, 30 mg) for 10 min. Increased value of salivary or plaque pH was found in the first minutes of chewing, and the effect of urea continued and lasted over 10 min. The higher concentrations of urea in chewing gum resulted in a faster leveling of the pH. As a result, the highest values of pH in the examined groups were observed in cases where they were treated with chewing gum containing 30 mg urea. With the use of such chewing gum, the salivary pH value does not fall below the level which is risky for the occurrence of dental caries, and there is a positive effect of chewing on the salivary flow that also affects neutralizing the acids in saliva or plaque [42, 43]. For the purpose of demonstrating the effect it can have on unstimulated saliva, a mathematical model of the influence of salivary urea on dental plaque was constructed. Data from study indicated that urea present in unstimulated saliva has a significant effect on plaque pH by elevating and counteracting the fall of plaque pH in the fasting state. The correlation of higher salivary urea concentrations and low salivary caries activity was registered in patients with chronic renal disease. These patients, who have elevated salivary urea concentration, have a reduced incidence of dental caries [44].

2.6.6 Calcium and phosphate concentrations

Saliva contains a supersaturated solution of calcium and phosphate, which neutralizes acids. Some epidemiological studies have revealed that humans with relatively high Ca and P in their plaque experience correspondingly lower caries. Higher Ca concentration of plaque is associated with low caries incidence. The process of undersaturation of the saliva with respect to tooth mineral content is a result of decreasing total phosphate concentration at high flow rates which would be bad for the teeth.

However, if the flow rate increases, then the saliva's pH increases together with the bicarbonate concentration, and therefore high pH is altered. In the proportions of four different phosphate species (H_3PO_4 , $H_2PO_4^-$, HPO_4^{2-} , and PO_4^{3}) together with the fall in total phosphate concentration, there is a fall in $H2PO_4^-$ and a slight increase in HPO_4^{2-} but a dramatic increase in PO_4^{3-} , all as a result high pH. It is the PO_4^{3-} that is an important ionic species with respect to the solubility of tooth mineral. So, although the total level of phosphate falls with increasing flow rate, the concentration of PO_4^{3-} actually increases as much as 40-fold when flow rate increases from the unstimulated level to high flow rates. The three components (Ca^{2+} , PO_4^{3-} , and OH^-) increase with salivary flow if taking into consideration the components of the ion product determining the solubility of tooth mineral in saliva. The saliva is more effective in reducing demineralization and promoting remineralization of the teeth if the flow rate is higher as well as the potential for calculus formation.

3. Conclusion

It can be concluded that tooth decay is a disease of great importance for general health. As a result, strategies to reduce the risk for dental caries are extremely important. The strategies may involve decreasing the growth or activity of bacteria especially *S. mutans*. To do so, people need to change their daily diet. Parents should advise children to avoid eating between meals, especially food containing carbohydrate.

Diet and oral microflora are connected to caries along with host factors such as salivary composition and flow.

Diet rich in fermentable carbohydrates is responsible for causing caries. Sucrose is one of the most cariogenic sugars, and glucose and fructose have also been shown to be less cariogenic. The cariogenic potential of carbohydrate-containing foods depends on their stickiness characteristics, frequency, and amount.

The saliva with its components plays an important role in maintaining oral, especially dental, health. Saliva is a natural factor that protects against demineralization. Apart from the activity of human saliva in diluting, clearing, neutralizing, and buffering acids, it also reduces demineralization and enhances the remineralization process.

Saliva performs its mechanical cleaning and protective functions though several physical and biochemical mechanisms. Saliva has buffer capacity which neutralizes acids in the mouth. The carbonic acid/bicarbonate system is the most important buffer in stimulated saliva.

The urea contributes to maintaining the acidobasic balance of saliva and thus affects the incidence of caries.

Intechopen

Author details

Efka Zabokova Bilbilova

Faculty of Dentistry, Department of Pediatric and Preventive Dentistry, "Ss. Cyril and Methodius" University, University Dental Clinic Center "St. Panteleimon", Skopje, Republic of North Macedonia

*Address all correspondence to: efka_zabokova@hotmail.com

IntechOpen

© 2020 The Author(s). Licensee IntechOpen. This chapter is distributed under the terms of the Creative Commons Attribution License (http://creativecommons.org/licenses/by/3.0), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

References

[1] Arens U. Oral Health, Diet and Other Factors: Report of the British Nutrition foundation Task Force. Amsterdam, New York: Elsevier; 1998

[2] Moynihan P. The interrelationbetween diet and oral health.Proceedings of the Nutrition Society.2005;64:571-580

[3] Jurgensen N, Petersen PE. Oral health and the impact of sociobehavioural factors in a cross sectional survey of 12-year old school children in Laos. BMC Oral Health. 2009;**9**:29. DOI: 10.1186/1472-6831-9-29

[4] Tinanoff N, Palmer CA. Dietary determinants of dental caries and dietary recommendations for pre school children. Journal of Public Health Dentistry. 2000;**60**(3):197-206

[5] Moynihan P, Petersen PE. Diet, nutrition and the prevention of dental diseases. Public Health Nutrition. 2004;7:201-226. DOI: 10.1079/ PHN2003589

[6] Harris R. Biology of the children of Hopewood house, Bowral, Australia.
4. Observations on dental caries experience extending over five years (1957-61). Journal of Dental Research.
1963;42:1387-1399

[7] Sheiham A. Dental caries affects body weight, growth and quality of life in pre-school children. British Dental Journal. 2006;**201**:625-626. DOI: 10.1038/sj.bdj.4814259

[8] Gustafsson B, Quensel C, Lanke L. The Vipeholm dental caries study: The effect of different levels of carbohydrate intake on caries activity in 436 individuals observed for five years. Acta Odontologica Scandinavica. 1954;**11**(3-4):232-264

[9] Linke HA, Birchmeier RI. Effect of increasing sucrose concentrations

on oral lactic acid production. Annals of Nutrition & Metabolism. 2000;**44**:121-124

[10] Bernabé E, Vehkalahti MM, Sheiham A, Lundqvist A, Suominen AL. The shape of the doseresponse relationship between sugars and caries in adults. Journal of Dental Research. 2016;**95**:167-172

[11] Marshall T, Levy S, Broffitt B, et al. Dental caries and beverage consumption in young children. Pediatric Dental Journal. 2003;**112**(3):184-191

[12] Mobley C, Marshall TA, Milgrom P, Coldwell SE. The contribution of dietary factors to dental caries and disparities in caries. Academic Pediatrics. 2009;**9**(6):410-414. DOI: 10.1016/j. acap.2009.09.008

[13] Decker RT, Loveren CV. Sugars and dental caries. The American Journal of Clinical Nutrition.2003;78(Suppl):881S-892S

[14] Burt B, Eklund S, Morgan K. The effects of sugars intake and frequency of ingestion on dental caries increment in a three-year longitudinal study. Journal of Dental Research. 1988;**67**(11):1422-1429

[15] Lingström P, van Houte J, Kashket S. Food starches and dental caries. Critical Reviews in Oral Biology and Medicine. 2000;**11**(3):366-380

[16] Shils ME, Shike M, Ross AC, et al. Modern Nutrition in Health and Disease. 10th ed. Philadelphia, PA: Lippincott, Williams & Wilkins; 2006

[17] Dodds MWJ, Edgar WM. Effects of dietary sucrose levels on pH fall and acid-ion profile in human dental plaque after a starch mouth-rinse. Archives of Oral Biology. 1986;**31**:509-512

[18] Rugg-Gunn AJ. Diet and dental caries. In: Murray JJ, editor. Prevention

Dietary Factors, Salivary Parameters, and Dental Caries DOI: http://dx.doi.org/10.5772/intechopen.92392

of Oral Disease. Oxford: Oxford University Press; 1996. pp. 3-3146

[19] Dennison BA. Fruit juice consumption by infants and children: A review. Journal of the American College of Nutrition. 1996;**15**(5):26-29

[20] Sohn W, Burt B, Sowers M. Carbonated soft drinks and dental caries in the primary dentition. Journal of Dental Research. 2006;**85**(3):262-266

[21] Kolker J, Yuan Y, Burt B, et al. Dental caries and dietary patterns in low-income African American children. Pediatric Dental Journal. 2007;**29**(6):457-464

[22] Marshall TA, Eichenberger-Gilmore JM, Broffitt B, Warren JJ, Levy SM. Dental caries and childhood obesity: Roles of diet and socioeconomic status. Community Dentistry and Oral Epidemiology. 2007;**35**:449-458

[23] Kleinberg I. Oral effects of sugars and sweeteners. International Dental Journal. 1985;**35**:180-189

[24] Kashket S, Zhang J, van Houte J. Accumulation of fermentable sugars and metabolic acids in food particles that become entrapped on the dentition. Journal of Dental Research. 1996;**75**:1885-1891

[25] Palmer C, Kent R, Loo C, et al. Diet and caries-associated bacteria in severe early childhood caries. Journal of Dental Research. 2010;**89**(11):1224-1229

[26] Murray J, Rugg-Gunn A, Jenkins G. Fluorides in Caries Prevention. 3rd ed. Oxford, UK: Butterworth-Heinemann; 1991

[27] Carmichael CL, French AD, Rugg-Gunn A, Furness JA. The relationship between social class and caries experience in five-yearold children in Newcastle and Northumberland after twelve years of fluoridation. Community Dental Health. 1984;**1**:47-54

[28] van Loveren C. Sugar restriction for caries prevention: Amount and frequency. Which is more important? Caries Research. 2019;**53**:168-175. DOI: 10.1159/000489571

[29] Moynihan P, Makino Y, Petersen PE, Ogawa H. Implications of WHO guideline on sugars for dental health professionals. Community Dentistry and Oral Epidemiology. 2018;**46**:1-7

[30] Leone WC, Oppenheim GF. Physical and chemical aspects of saliva as indicators of risk for dental caries in humans. Journal of Dental Education. 2001;**65**(10):1054-1062

[31] Dawes C, Dong C. The flow rate and electrolyte composition of whole saliva elicited by the use of sucrose-containing and sugar-free chewing-gums. Archives of Oral Biology. 1995;**40**:699-705

[32] Tanzer J, Grant L, McMahon T. Bicarbonate-based dental powder, fluoride and saccharin inhibition of dental caries associated with S. mutans infection of rats. Journal of Dental Research. 1988;**67**:969-972

[33] Bardow A, Moe D, Nyvad B, Nauntofte B. The buffer capacity and buffer systems of human whole saliva measured without loss of CO2. Archives of Oral Biology. 2000;**45**:1-12

[34] Bardow A, Hofer E, Nyvad B, Cate JM, Kirkeby S, Moe D, et al. Effect of saliva composition on experimental root caries. Caries Research. 2005;**39**:71-77

[35] Nauntofte B, Tenovuo JO, Lagerlöf F. Secretion and composition of saliva. In: Fejerskov O, EAM K, editors. Dental Caries The Disease and its Clinical Management. Oxford: Blackwell, Munksgaard; 2003. pp. 7-27 [36] Arends J, Jongebloed WL, Goldberg M, Schuthof J. Interaction of urea and human enamel. Caries Research. 1984;**18**:17-24

[37] Macpherson LMD, Dawes C. Urea concentration in minor mucous gland secretions and the effect of salivary film velocity on urea metabolism by streptococcus vestibularis in an artificial plaque. Journal of Periodontal Research. 1991;**26**:395-401

[38] Sjögren K, Ruben J, Lingstrom P, Lundberg AB, Birkhed D. Fluoride and urea chewing gums in an intra-oral experimental caries model. Caries Research. 2002;**36**:64-69

[39] Dawes C, Dibdin GH. Salivary concentrations of urea released from a chewing gum containing urea and how these affect the urea content of gel-stabilized plaques and their pH after exposure to sucrose. Caries Research. 2001;**35**:344-353

[40] Leone CW, Oppenheim FG. Physical and chemical aspects of saliva as indicators of risk for dental caries in human. Journal of Dental Education. 2001;**65**:1054-1064

[41] Imfeld T, Birkhed D, Lingstrom P. Effect of urea in sugar – Free chewing gums on pH recovery in human dental plaque evaluated with three different methods. Caries Research. 1995;**29**:172-180

[42] Raymond G, Schipper A, Erika Silletti A, Vingerhoeds MH. Saliva as research material: Biochemical, physiochemical, and practical aspects. Archives of Oral Biology. 2007;**52**:1114-1135

[43] Zabokova-Bibilova E, Sotirovska-Ivkovska A, Ambarkova V. Correlation between salivary urea level and dental caries. Contributions. 2012;**33**(1):289-302 [44] Dibdin GH, Dawes C. A mathematical model of the influence of salivary urea on the pH of fasted dental plaque and on the changes occurring during a cariogenic challenge. Caries Research. 1998;**32**:70-74

