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# Dental Caries and Sugar Substitutes: A Review

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***Abstract:*** *A dynamic relation exists between sugars and oral health. Dental caries is one of the most common contagious diseases found among human beings. Diet plays an important role in the advancement of dental caries. About 50 years back dietary sugars were concerned as the major cause for dental caries. Today's diet contains more number of fermentable carbohydrates, these includes highly processed starch-containing foods, and food products that contain synthetic carbohydrates . .Fermentable dietary carbohydrates undergo bacterial action that leads to the production of acids which demineralizes the tooth structure and ultimately leads to the formation of dental caries. Diet affects the integrity of the teeth; quantity, pH, and composition of the saliva; and plaque pH. Sugars and other fermentable carbohydrates, after being hydrolyzed by salivary amylase, provide substrate for the actions of oral bacteria, which in turn lower plaque and salivary pH. The resultant action is the beginning of tooth demineralization. Consumed sugars are naturally occurring or added. Many factors in addition to sugars affect the caries process, including the form of food or fluid, the duration of exposure, nutrient composition, sequence of eating, salivary flow, presence of buffers, and oral hygiene. No doubt, it is difficult to avoid sugar in diet but reducing the amount and exposure to sugar in diet of humans especially children is an important consideration in preventing caries thus non-cariogenic sweeteners offer good alternative to sugar if used in moderation. These sugar substitutes do not promote caries. As dental professionals we have the responsibility to reduce the incidence of dental caries by emphasizing the role of sugars in caries formation. Individual dietary counselling is highly recommended for patients at high caries risk It is important that the dentist must be familiarised with alternatives to sugar and type of food products that are available with substitute sweetening agents. Currently, a large number of investigations are on their way, focused on identifying various foods and factors that defend and control dental caries*

***Keywords****: Dental caries, Microorganism, Sugar substitutes*

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## I. Introduction

Dental caries is one of the most established and worldwide disease of prehistoric and current times. Dietary control makes a vital contribution to the multifacet approach for caries control. Sugars are surely significant dietary components in the etiology of dental caries. Now a days diet contains wide range of fermentable carbohydrates, such as exceptionally prepared starch containing meals and ingredients that contain novel manufactured carbohydrates like sucralose, glucose and oligofructose polymers.

Along with this there present an extensive varieties of non-cariogenic sweeteners which have a crucial position to play in caries management.(1)

In 1960,Keyes,introduced a circle which included four factors namely host, agent, dental plaque and microflora.The prolonged interplay of these factors results in caries development and loss of tooth structure.(2)

The two essential microbes involved in dental caries are Streptococcus Mutans and Lactobacillus. S.mutans produce extracellular polysaccharides like dextran and levans from dietary carbohydrates, mainly sucrose; which can contribute to bacterial adhesion to tooth surface by increasing the bulk of dental plaque. Intracellular polysaccharides act as a resource medium during starvation periods and prevent neutralizing substances diffusing through plaque. Though microorganisms are essential in pathogenesis of dental caries, there mere presence will not lead to dental decay.

Indigenous natural sweeteners like Honey has been utilized as a supply of nutrients in addition to a medicinal drug since ancient times. Honey, was found to cause less demineralization when compared to glucose and fructose.It is used as a sweetener in toothpaste, gums, candies, chocolates and so on. Recent studies reported the anticariogenic effect of honey and palmsugar.(3)

Stevia rebaudiana Bertoni is a perennial shrub of family Asteraceae, native of Paraguay and Brazil that is accredited as a food supplement in several countries such as Brazil, Japan, the United States and recently the European Union. Other natural sweeteners are glycirrhicine, monellin, thaumatin, mirakulin etc.[4]

Intense sweeteners (non caloric) like aspartame, saccharine, neotame (which are chemically synthesized) are used in a variety of food products including soft drinks, icecreams, desserts, confectioneries etc.

Among the bulk sugars, Sorbitol and xylitol are the most commonly used sugar substitute. Many studies are available that have shown the anticariogenicity of xylitol. Apart from preventing adherence to microorganisms, it also promotes salivation and remineralization, when used as a chewing gum. Bulk sugars are currently used in confectioneries, chocolates, jellies and other sweets.

The usefulness of a sugar substitute must be looked upon not just from a cariological, but additionally from a nutritional, toxicological, economical and technical point of view.[5]

This review article will give a discriptive idea about dietary relation to development of dental caries and the part of sugar substitutes in the counteractive action of caries.

## II. Sources

The three principal monosaccharides are glucose, fructose and galactose, which are the building blocks of naturally occurring di, oligo and polysaccharides. Free glucose and fructose occur in honey and cooked or dried fruit (invert sugar), in small amounts, and in larger amounts in fruit and berries where they are the main energy source. Corn syrup, a glucose syrup produced by the hydrolysis of cornstarch, and high fructose corn syrup, containing glucose and fructose, are increasingly used by the food industry in many countries. Fructose is the sweetest of all the food carbohydrates. The polyols, such as sorbitol are alcohols of glucose and other sugars. They are found naturally in some fruits and are made commercially by using aldose reductase to convert the aldehyde group of the glucose molecule to the alcohol. Sorbitol is used as a replacement for sucrose in the diet of people with diabetes.

The principal disaccharides are sucrose and lactose. Sucrose is found very widely in fruits, berries and vegetables, and can be extracted from sugar cane or beet. Lactose is the main sugar in milk. Of the less abundant disaccharides, maltose, derived from starch, occurs in sprouted wheat and barley. Trehalose (a-Glc(1-4)a-Glc) is found in yeast, fungi (mushrooms) and in small amounts in bread and honey. It is used by the food industry as a replacement for sucrose where less sweet taste is desired but with similar technological properties.[6]

Sucrose represents the main source of sugar in the diet. In studies in rats, using an experimental caries model, sucrose was shown to be more cariogenic than other dietary sugars such as glucose, fructose, and lactose. This cariogenic effect appears to be strain specific, and is also influenced by the type of animal model and class of caries.[7]

## III. Biochemical and Microbiologic Evidence

Key role of sucrose as a dietary substrate in the caries process, particularly on smooth surfaces, can be explained on biochemical grounds. Smooth surface caries depends on the growth of dental plaque. Extracellular polysaccharides, both glucans and levans, have been demonstrated in plaque. The glucans, particularly the water insoluble fraction, can serve as structural components of the plaque matrix, in effect gluing certain bacteria to the teeth. Soluble levans and some of the soluble glucans are degradable by the plaque flora and may function as transient reserves of fermentable carbohydrates, thereby prolonging the production of acid. In the bio synthesis of these polysaccharides by bacterial enzymes, glucose units are transferred from sucrose to the active site and inserted between the enzyme and the reducing end of the polymer. The enzyme conserves the relatively high energy of the link between the two anomeric carbons, Cl of glucose and C2 of fructose, found in sucrose. Disaccharides other than sucrose, such as maltose and lactose, are hemiacetals, not dihemiacetals. Therefore their free energy of hydrolysis is significantly less and they cannot serve directly as glycosyl donors in the system. Sucrose, on the other hand, has a free energy of hydrolysis of six thousand and six hundred calories per mole, which is in the same range as the nucleoside diphosphate sugars, seven thousand and six hundred calories per mole, and higher than glucose-I-phosphate. Because sucrose is unique in serving as a substrate not only for fermentation but also for synthesis of extracellular polysaccharides, it influences the ecology of the oral microflora. It does this by favoring the colonization of the plaque by caries-inducing organisms such as Streptococcus mutans.[8]. This specific property of sucrose has been shown in past investigations utilizing an intraoral caries model, where particularly increased demineralization was related with S mutans test plaques prepared from sucrose-containing cultures contrasted with glucose-developed plaques. This was credited to a change in the diffusion properties of plaque due to formation of extracellular polysaccharide (glucan) produced from sucrose. The extracellular matrix permits greater penetration of dietary carbohydrates into the deeper layer of dental biofilm adjacent to the tooth surface, which from a caries lesion.

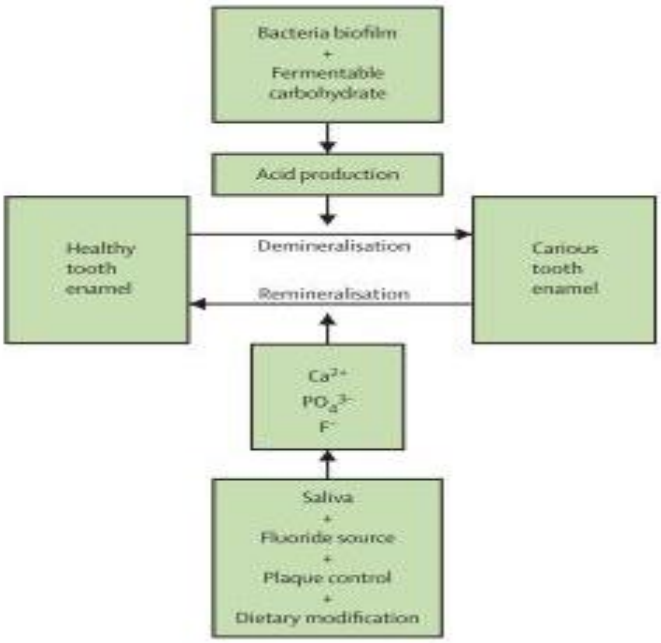


**Fig.** The Demineralization –Remineralization cycle

It is important to keep in mind that the relationship of diet with caries disease has changed. All historical and epidemiologic evidence before 1970 clearly associated the availability of sugar and refined carbohydrates with increased caries disease prevalence. Caries disease increased in severity as the standard of living and nutrition improved. From the early1970s until the most recent, US National Health and Nutrition Examination Survey, a change has been observed in that there has been a reduction in the caries prevalence in adolescents (age twelve to nineteen), adults (age twenty to sixty four), and seniors (age sixty five and older). There was, however, a small but significant increase in caries in the primary teeth of children (age two to eleven). Over the past thirty years there is a decrease in caries prevalence which has been generally attributed to topical effects of fluoride. The reduction in sucrose intake and replacement with alternative sugars may also be a contributing factor. Individual guidance on dietary changes should be provided by dental care practitioners. This includes the assessment of the patient's dietary habits with analyses of the sugar exposure patterns and type of foods. This information can be obtained through the use of dietary diaries and also by recall interviews. Diet counseling should focus not only on reducing the exposures to sugar but also on providing recommendations of healthier alternatives.[(9)]

In summary, then, the specific biochemical characteristics of sucrose affects the oral ecology and promotes dental plaque colonization of caries inducing microbes. In both human beings and animals, ingestion of sugar has been clearly demonstrated as the major dietary factor contributing to caries. The sugar content of food is obviously not the only factor affecting cariogenicity; the physical texture and especially the frequency of ingestion are also of importance. Nevertheless, labelling that discloses the percentage of sucrose in a food would be a useful guide to consumers and would help professionals giving dietary advice[(8)]

Key issues to consider in deciding dietary cariogenic, cariostatic, and anticariogenic properties are food form, recurrence of sugar intake and other fermentable carbohydrates, retention in oral cavity, nutrient composition, the capacity of the food to stimulate salivary secretion, and blends of foods. Longer retention of cariogenic food results in increased periods of acid production and demineralization and to shortens remineralization. Frequency and amount of fermentable carbohydrate ingested also affects duration. Liquid sugars quickly wash out from oral cavity with limited contact time or adherence to tooth surfaces. Prolonged retention of sugars in mouth also increases caries risk.

## IV. Sugar Substitutes

As mentioned previously, there is a definite relationship between the dietary consumption of sucrose and the incidence of dental caries. Therefore, intensive analysis for a low calorie, non cariogenic sweetener has been performed to supply alternative compound to sugar to be used in food and drugs, and several other artificial sweeteners have been introduced by the food industry to sweeten food and beverages. This research lead to the development of synthetic sweeteners, many of which are considered safe for teeth, such as aspartame, saccharin, cyclamate, xylitol, and mannitol. These sweeteners have also been used as sugar substitutes for caries active patients.

However, in addition to their benefits, animal studies have proved that artificial sweeteners cause health hazards like weight gain, brain tumours, bladder cancer etc[10]. Thus, research should still determine natural foods and components that shield against dental caries, notably those that have practical dietary application and might facilitate dietary advice effective.

## V. Ideal Sweetener

The ideal product would be calorie free, non carcinogenic, non mutagenic, not heat degradable, economical to produce, and it would provide sweetness with no unpleasant aftertaste. However, obtaining these properties in a single product has been extremely challenging. Since past, active compounds from plant origins are used as medicines for numerous diseases and microbial infections. Recent research has also been oriented towards the invention and analysis of novel, potentially non cariogenic, sweeteners from nature.[11]

## VI. Classification of Sugar Substitutes

**TABLE1:**Classification of Sugar substitutes

A) Based on sugar substitute being caloric or non- caloric

| **A. Caloric/nutritive sweetener** | **B. Non-Caloric/Non-nutritive sweetener** |
|---|---|
| 1.Poly alcohols/sugar alcohols<br>● Xylitol<br>● Sorbitol<br>2.Hydrogenated starch hydrosylates<br>● Lycasin<br>● Palatinit<br>3.Coupling sugars<br>● Sorbose<br>● Palatinose | 1. Cyclamate<br>2. Saccharin<br>3. Aspartame<br>4. Sucralose<br>5. Neotame |

**B)** Based on their origin being natural and artificial.

| Natural sugar substitute | Artificial sugar substitute |
|---|---|
| Xylitol<br>Sorbitol<br>Mannitol<br>Trehalose<br>Stevia<br>Tagatose<br>Starch Hydrolysates<br>Honey<br>Jaggery | Acesulfame K<br>Alitame<br>Neotame<br>Aspartame<br>Cyclamate<br>Saccharin<br>Sucralose<br>Neosporidine Dihydrochalone |

## VII.Artificial Sugar Substitutes

### 1. ACESULFAME-K

It has been developed as sweetener by Hoechst (Clauss and Jensen 1970). This high intensity sweetener is K salt of 6-methyl-123-axathiazine-4 (3H)-one 2,2-dioxide with molecular formula C4H4KNO4S and relative molecular mass of 201.24. It is a white crystalline powder, about one twenty times sweeter than sucrose and has high water solubility[12]

### 1.1. Properties

Acesulfame K is about two hundred times more sweeter than of a three percent sucrose solution. Its sweetness intensity decreases with increasing sucrose concentrations to values from hundred and thirty to hundred times the sucrose value. It has identical sweetness as aspartame, about half as sweet as sodium saccharin, and four to five times sweeter than sodium cyclamate [13].

### 1.2 Toxicology

It is practically non toxic. A recommended daily intake (ADI) of zero to nine mg/kg of body weight was allocated by the Joint Expert Committee on Food Additives of the FAO and WHO,, which was later enhanced to zero to fifteen mg/kg[14]

### 1.3 Metabolism and Physiological Characteristics

Acesulfame K isn't metabolized by the human body and doesn't have any caloric value. Acesulfame K is not metabolized by bacteria.[15]

## 2.ASPARTAME

### 2.1 Introduction

Aspartame was discovered accidentally in 1965 by G. D. Searle and Co. chemist James Schlatter. Aspartame is an artificial, dipeptide, intense sweetener, that is nearly 180-200 times sweeter than sucrose. It has a low calorific value. It metabolizes slowly than sucrose and so maintains a stable blood glucose level. Unlike sucrose, the micro flora present in the dental plaque does not utilize aspartame. Due to this property use of aspartame is recommended in the form of prescribe sugar free medicines[16].

### 2.2Properties

It is a white crystalline, unscented intensively sweet powder has the molecular formula $C_{14}H_{18}N_2O_5$ along with the molar mass 294.31 g $mol^{-1}$ [17]

### 2.3Mechanism of action

Aspartame is hydrolyzed in the intestinal lumen into aspartic acid, phenylalanine and methanol. These components are absorbed into the blood and metabolized. [18]

It has a flavour enhancing property which is evident in chewing gum, which is an important characteristic in many food applications. It is one sixty to two twenty time sweeter than sucrose.It has a synergistic effect when mixed with other sweeteners including sugar.[16]

### 2.4 Role of Aspartame on Dental Caries[19]

In a study to test the effect of aspartame alone and alongside sucrose, on dental caries in rats, they found that colonisation by S.sobrinus was insignificant in those fed aspartame and there was no caries development and concluded that aspartame is noncariogenic, or anticariogenic.

### 2.5 Side effects of aspartame:

Negative effects like headaches, brain tumors, brain lesions, and lymphoma. High level of the naturally occurring essential amino acid phenylalanine is a health hazard to those born with phenylketonuria (PKU) a rare inherited disease. must be labelled with the statement phenylketonurics: contains phenylalanine. Aspartame causes behavioural problems.[44]Conceiving aspartame during gestation could be prejudicial to the fetal embryo. [20]

## 3. CYCLAMATES

### 3.1 Introduction

Sodium cyclamate was synthesized in 1937, but it was first produced commercially in the United States in 1950. It is produced in many countries including Japan, Germany, Spain, Taiwan, and Brazil.[21] Because of regulatory decisions the utilization of cyclamates was diminished in the 1960s. Recently, after the allocation of a new acceptable daily intake value by JECFA, the utilization has begun to increase again[21].Cyclamate was first promoted as tablets that were recommended for use as a tabletop sweetener for diabetics and other people who had to confine their utilization of sugar.[22]

### 3.2 Chemistry

Cyclamates is a group name used for the following compounds: cyclamic acid, sodium cyclamate, and calcium cyclamate. The atomic formula for calcium cyclamate is $C_{12}H_{24}CaN_2O_6S_2.2H_2O$. They give a sweet taste that is thirty times sweeter than sugar. Sodium and calcium cyclamates have been utilized mostly in the form of the 10:1 cyclamate/saccharin mixture.[21]

### 3.3 Role of Cyclamate on Plaque and Dental Caries

The effect of mouth washes containing cyclamate, xylitol and sucrose sweeteners on the oral well being were evaluated by Paunia K et al. (1984). A concurrent neglect of oral hygiene was maintained and subjects were made to rinse their mouth six times a day with 15ml of the respective sweetener .Less dental plaque was observed in cyclamate and xylitol groups in contrast to sucrose group.[23]

### 3.4 Metabolism

Cyclamate is absorbed partially from the intestine, and a variable amount is converted to cyclohexylamine by microorganisms in the large bowel [24]

**3.5 Physical and chemical properties**

It is commercially available in the sodium and calcium salt forms.Sodium cyclamate, C6H12NNaO3S, Mr 201.2[139-05-9], is the most important cyclamate sweetener. Compared with 0.1 mol/L sucrose solutions, the sweetness intensity of sodium cyclamate is ca. 35. Aftertastes become perceptible at elevated concentrations only, rendering cyclamate generally superior to saccharin.It exhibit excellent solubility and are quite stable.[25]

**3.6 Side effects of cyclamate:**

Bladder cancer and testicular atrophy had been reported.[23]

**4.SACCHARIN**

**4.1 Introduction**

Saccharin is commercially available in sodium and calcium salt forms, both of which are readily soluble in water. Saccharin is model of the N-sulfonyl amide structural class of sweeteners, which is recognized by the regular -CONHSO2- substructure.[25]

**4.2 Chemistry**

**S**accharin is a general name used for saccharin, sodium saccharin, and calcium saccharin. The molecular formula of saccharin is C7H5 NO3 S. Chemically saccharin is 1,2-benzisothiazol-3(2H)-one-1,1-dioxide and its sodium or calcium salt. Saccharin can be produced by two methods, either from toluene and chlorosulfonic acid or from methyl anthranilate.

**4.3 Properties**

In dilute aqueous solutions they are about three hundred times sweeter than a solution containing an same concentration by weight of sucrose [21]

**4.4 Metabolism:**

Saccharin is not digested in humans or rats. Saccharin is largely absorbed from the small intestine. Excretion: In humans, oral doses are excreted almost totally by the kidneys with the balance recovered in the faeces.[25]

Saccharin is not metabolized in the gastrointestinal (GI) tract and therefore does not influence blood glucose levels[26], which makes saccharin a viable sugar substitute for patients with diabetes. The Adequate Dietary Intake (ADI) for saccharin is set at 5 mg/kg body weight per day for adults and children. [20]

**4.5 Toxicology**

In some long term rat studies an induction of a higher occurence of bladder cancer was found among rats devouring saccharin.[27]

**4.6 Role of Saccharin on Dental Caries:**

J. M Tanzer (1988), in an experiment studied the effect of bicarbonate based dental powder containing fluoride and saccharin on inhibition of dental caries associated with S. mutans in rats in contrast with use of topical fluoride and fluoride with de ionized water and the outcome of study showed dental powder containing fluoride and saccharin showed maximum decrease in dental caries when contrasted to topical fluoride alone or when utilized with de ionized water.[23]

**5. SUCRALOSE**

**5.1 Introduction**

Sucralose was discovered in 1976 by researchers Tate & Lyle, working with scientists Leslie Hough and Shashikant Phadnis at Queen Elizabeth College. It was first approved for use in Canada (marketed as Splenda) in 1991. It had been endorsed in more than eighty countries, including Australia, NewZealand, Europe, Mexico, Brazil, China, India and Japan.[23]

**5.2 Production**

Sucralose is manufactured by the particular substitution of three hydroxyl groups on the sucrose particles by three chlorine atoms to produce 1,6-dichloro-1,6-dideoxy-beta-d-fructofuranosyl-4-chloro-4-deoxy-alpha-d-galactopyranoside. [25]

**5.3 Metabolism and Health Aspect**

The human body does not recognize it as a sugar and does not metabolize it therefore it provides no calories [54]. The amount that is ingested from the gastro intestinal tract is largely expelled from the blood

stream by the kidneys and eliminated in the urine. ([28] The acceptable daily intake (ADI) for sucralose in US is 5mg/kg body weight/day. The estimated daily intake for percentile consumers as calculated by USFDA is 1.6mg/kg body weight/day.

**5.4 Cariogenicity**

Sprague Dawley rats infected with Streptococcus mutans were given sucrose or sucralose along with a diet that contained no extra sucrose. Following thirty five days, the level of infection of S. mutans was determined and the sulcal caries scored. Rats fed sucralose had same number of total viable flora; however, the level of S. mutans of the total viable flora was reduced by up to twenty fold in contrast with the sucrose control. Sucralose fed rats had less than fifty percent of the sulcal caries evident in the sucrose control. Rats were desalivated and inoculated with S. mutans and Actinomyces viscosus and fed diets that contained either fifty six percent sucrose or sucralose at 93 mg/100 g. The number of coronal lesions did not vary significantly among groups, but the severity of the lesions was comparatively lower following thirty five days of feeding the sucralose diet compared with the sucrose control. Substitution of sucralose for sucrose brought about substantially fewer lingual and proximal lesions. Although comparable levels of root surface exposures were accomplished in the test and control diet, only rats fed sucralose stayed free of root surface caries. These results shows that sucralose is non cariogenic[29]

**5.5 Side Effects**

Sucralose is said to act as a triggering agent for migraine, causes weight gain and some hypersensitive reactions. [23]

## VIII. Polyols

**1..XYLITOL**

**1.1Introduction**

Xylitol is a five-carbon polyol with a sweetness similar to sucrose[30] It is found in small amounts in a variety of fruits and vegetables and is formed as a normal intermediate in the human body during glucose metabolism. Xylitol has been appeared to be of value in the prevention of dental caries because it is not a compelling substrate for plaque bacteria[31].

**1.2Production**

Fundamental substrate for its production is xylan, which is usually acquired from birch trees and other hardwood[65]. Likewise to other sugar alcohols, xylitol is produced by metal catalyzed hydrogenation of a corresponding sugar, i.e., D-xylose[32]

**1.3Metabolism**

Xylitol is approximately absorbed in fifty percent in small intestine and fifty to seventy five percent of its fermentation, which takes place in large bowel[33]. Direct metabolization happens in the liver or indirect metabolization by fermentative degradation by intestinal flora[34]. ADI intake by humans is about 100 g per day.

**1.4Properties**

Xylitol is the sweetest among all sugar alcohols. It characterizes with the same sweetness and bulk as sucrose with one-third fewer calories and no unpleasant aftertaste. Insulin is not required for its metabolism [59). It readily dissolves and produces a cooling sensation in the mouth[35]

**1.5 Mechanism of action**

Streptococcus mutans transports the sugar into the cell in an energy consuming cycle that is responsible for growth inhibition. Xylitol is then changed over to xylitol-5-phosphate by means of phosphoenolpyruvate: fructose phosphotransferase system by S. mutans resulting in development of intracellular vacuoles and cell membrane degradation. Unwittingly contributing to its own death, S. mutans then dephosphorylates xylitol-5-phosphate. The dephosphorylated molecule is then expelled from the cell. This expulsion occurs at an energy cost with no energy gained from xylitol metabolism. Thus, xylitol inhibits S. mutans growth essentially by starving the bacteria. Xylitol can inhibit the growth of harmful oral bacteria such as S. mutans, but its benefits do not stop in the oral cavity.Xylitol alcohol has been shown to impact growth of nasopharyngeal bacteria such as S. pneumonia and S. mitis, and hence has a role to play in nasopharyngeal pneumonia[5]

**1.6 Dental benefits**

**A. Cariogenicity**

It has non cariogenic and cariostatic properties, and thus considered as a suitable sugar substitute. [22]

**B. Acidogenicity**

Considerable evidence records the fact that xylitol is non fermentable by most oral microorganisms and that exposure of dental plaque to xylitol doesn't bring about a decrease in plaque pH[36].

**C. Efficacy of Xylitol in Human Caries Studies**

The clinical benefit of xylitol for caries prevention has been recorded in several studies with children and adult human volunteers in which consumption of xylitol was consistently associated with a significant reduction of the caries increment. Daily fluoride brushing plus xylitol consumption was found more beneficial than daily fluoride brushing alone

**D. Discrimination against Streptococcus mutans**

The use of xylitol as a sucrose substitute becomes an extremely attractive means to control and prevent dental caries for two different reasons. First, no acid is formed from xylitol by the dental plaque. In fact, during and after chewing of a xylitol sweetened gum, the plaque pH increases and under such conditions, however, the metabolism of S. mutans is not optimally active, and other bacteria may successfully compete with S. mutans. Second, in addition to this indirect, pH-mediated effect, xylitol appears to inhibit the growth and metabolism of S. mutans in a more direct way. [37]. This inhibition by xylitol appears to be related to the accumulation of xylitol-5-phosphate and xylulose-5-phosphate within the cells and subsequently the ability of S. mutans to adhere to surfaces is decreased, and disintegration of the ultrastructure of the cells may occur.

**E. General Effects on Dental Plaque**

Records suggest that xylitol inhibits the formation of insoluble glucans and lipoteichoic acid, two products of bacterial metabolism that plays a crucial role in the adhesion and cohesion of dental plaque. Xylitol/Sorbitol Chewing gums have been demonstrated regarding their effect on the formation of plaque. The results of these studies suggest that chewing gums sweetened with xylitol have a higher impact on plaque reduction[5]

**1.7 Side effects of xylitol:**

Xylitol intake can cause stomach discomfort and above 40 grams per day, might cause diarrhoea. [23]

**2. SORBITOL**

**2.1 Introduction**

Sorbitol is a six-carbon polyol that was originally present in the berries of mountain ash. It is available in many fruits and vegetables.

It's sweetness is equivalent to sixty percent of the sweetness of sucrose [34]

**2.2 Production of Sorbitol**

Sorbitol is derived from the catalytic hydrogenation of glucose, sucrose and starch.

**2.3 Metabolism**

Sorbitol is absorbed and metabolized in the liver [34]

**2.4 Anticariogenicity**

Sorbitol is impervious to digestion by oral microbes which break down sugars and starches to release acids that may lead to cavities or disintegrate tooth enamel. Because of its non cariogenic properties, it is utilized in products for special nutritional purposes designated for people with diabetes .

**2.5Side effects**

Intake of higher amounts of sorbitol might cause abdominal discomfort pain, and mild to severe diarrhoea. Sorbitol can cause irritable bowel syndrome and fructose malabsorption. [34]

## IX. Natural Sugar Substitutes

**1. STEVIA**

**1.1 Introduction**

Stevia rebaudiana Bertoni is the biological name of stevia. It is a perennial bush belongs to the family Compositae(Asteraceae). Stevia is native to Paraguay and Brazil and it is regularly referred to as the sweet herb of Paraguay.[38]

Stevia has two primary glycosides that are stevioside (110-270 times sweeter than sugar) and rebaudioside A (180-400 times sweeter than sugar), the latter with higher commercial value because it shows a

nice flavor profile unlike artificial sweeteners that have metallic taste or the same stevioside that has a subtle bitter flavor.[39]

**1.1 Chemical composition**

The fresh leaves contain a high water content ( eighty to eighty five percent), ascorbic acid, ß-carotene, chromium, cobalt, magnesium, iron, potassium, phosphorus, riboflavin, thiamin, tin, zinc, and chemicals products found are apigenin, austroinilina, avicularin, ß-sitosterol, caffeic acid, campesterol, caryophyllene, centaureidin, chloro-genic acid, chlorophyll, kaempferol,luteolin, quercetin, stigmasterol,

About a quarter of a teaspoon of leaves is equivalent to one sugar tea spoon.

**1.2Production**

Extraction in water; soaked in warm water at 15:1 v/ w for three hours, then it is filtered and purified using calcium hydroxide, the filtrate passes through the ion exchange column to remove unwanted pigments and then concentrate by rotary evaporator at a temperature of 45°C.

**1.3 Mechanism of action**

In the digestive tract, rebaudiosides are metabolised into stevioside and is broken down into glucose and steviol. The glucose released by this process is used by bacteria in the colon but is not absorbed into the bloodstream. Steviol is not further digested and is excreted. [40]

**1.4 Anticariogenic properties**

Das et al. did an in vivo study about rats to examine the cariogenic potential of stevioside and rebaudioside A with long term utilization. The rats were fed a diet containing 0.5% of stevioside or 0.5% rebaudioside A for five weeks. Neither of these compounds showed the potential to increase the risk of the development of dental caries[41]

**1.5 Low acidogen potential:**

It has seen that Streptococcus mutans generates a lower production of acid when are grown on medium that contain Stevioside in comparison with the ones that are grown in sucrose, glucose or fructose

**1.6 Anti-plaque effect**

The phytochemicals present in Stevia are austroinullin, ß-carotene, dulcoside, niacin, oxides rebaudi, riboflavin, steviol, stevioside and thiamine.These nutritious substances affect the microbial flora of the mouth and also, the content of tannin, xanthines (theobromine and caffeine) and flavonoids have anti-plaque activity. In addition, the leaf extracts of Stevia and its major polyphenolic constituents, stevioside and rebaudioside A, are not cariogenics.

**1.7 Safety of stevia**

Generally safe and an acceptable intake of stevioside in humans 7.94 mg/ kg/day [34]

Trade names: Rebiana, truvia

**2. HONEY**

**2.1 Introduction**

Honey is a super-saturated, delicious, and naturally sweet nectar popular worldwide and is collected by bees from a wide variety of plants. Thus, using a sweetener with a lower chance to do harm in the diet such as honey is important.

**2.2 Composition**

Honey contains sugars as fructose (18.4%), glucose (30.3%), sucrose (1.3%), other carbohydrates (12%), minerals (0.169%), proteins (169 mg/100 g) with a water content of about 17.2% [1].[42]

**2.3 Anticariogenicity**

It was found that mouth washes containing propolis (present in bee products) possessed antimicrobial activity against Streptococcus mutans and can be used as an alternative treatment in dental caries prevention and in the reduction of plaque accumulation and polysaccharide formation[43,85].

**3.JAGGERY**

**3.1 Introduction**

Jaggery is a natural ancient sweetener prepared concentrating the extracted sugarcane juice. It is a standard unrefined non-centrifugal sugar consumed in Asia, Africa, Latin America and also the Caribbean. [44]

**3.2 Composition**[45]

Jaggery, a product of sugarcane, is such a product which is rich in vital minerals (viz Calcium-40-100 mg, Magnesium-70-90 mg, Potassium-1056 mg, Phosphorus-20-90 mg, Sodium-19-30 mg, Iron-10-13 mg, Manganese-0.2-0.5 mg, Zinc-0.2- 0.4 mg, Copper-0.1-0.9 mg, and Chloride-5.3 mg per 100g of jaggery), vitamins (viz Vitamin A-3.8 mg, Vitamin B1-0.01 mg, Vitamin B2-0.06 mg, Vitamin B5-0.01 mg, Vitamin B6-0.01 mg, Vitamin C-7.00 mg, Vitamin D2-6.50 mg, Vitamin E-111.30 mg, Vitamin PP-7.00 mg), and protein.

**3.3 Nutritional Aspects**

Jaggery is far complex than sugar, because it is formed of longer chains of sucrose. Hence, it is digestable slower than sugar and releases energy slowly and not spontaneously. This provides energy for a extended time and isn't harmful for the body. [46]

**3.4 Benefits**

1. Easily digestable
2. Rich in minerals
3. Enhances distinctive taste as sweetener
4. Sulphur less organic composition, a best to suite as preferred health alternative used in confectionary items

## X. Conclusion

There is substantial amount of knowledge on the effect of non-sugar sweeteners on dental health. This comes from numerous types of studies like incubation experiments, plaque pH studies, enamel slab experiments, animal studies, and human clinical trials**.**[1]Each of the sucrose substitutes has explicit characteristics that ought to be utilized in order that the requirements of specific individuals are met. The prevalence of dental caries in population is declining, however people at high risk of developing dental caries are still an vital public health concern. To ensure success, a greater variety of sweets is needed and new sucrose substitutes of nutritionary worth ought to even be developed .

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