

Review Article

Prevention of Enamel and Dentin Carious Lesions

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Abstract

Four main preventive strategies have been developed: fluorides administration, fissure sealing, dietary choice, and plaque control. Caries is a dynamic and reversible process. The initial demineralization step occurs when plaque acids on the tooth surface dissolve calcium and phosphate ions from the hydroxyapatite. The remineralization step occurs when the salivary free calcium and phosphate ions are driven back into the demineralized zone. *S.mutans* (SM) and *S.sobrinus* (SS) are the most important colonizers in terms of human caries. SM ferment sucrose and produce organic acids including lactic, acetic, formic and propionic acids. Cariogenic bacteria produce insoluble extracellular polysaccharides, that enhance their adherence to the tooth surface and contribute to biofilm formation. Strategies consistent with the prevention of disease via the principles of the ecological plaque hypothesis include: inhibition of plaque acid production, avoidance between main meals of foods and drinks containing fermentable sugars, consumption of foods/drinks that contain non-fermentable sugar substitutes, stimulation of saliva flow after main meals. Prevention program is based on plaque control and topical application of fluorides, after scaling, root planning and conventional caries therapy.

INTRODUCTION

Four main factors are involved in the carious process: the tooth structure, plaque flora (bacteria), fermentable carbohydrates (dietary sugars) and time. Consequently, four main preventive strategies have been developed: fluorides administration, fissure sealing, dietary choice, and plaque control. These strategies encompass:

- Inhibition of plaque acid production
- Avoidance between main meals of foods and drinks containing fermentable sugars
- The consumption of foods/drinks that contain non-fermentable sugar substitutes
- The stimulation of saliva flow after main meals

Prevention program is based on plaque control and topical application of fluorides, after scaling, root planning and conventional caries therapy. These strategies include [1]:

There is a shift towards community dominance by acidogenic and acid tolerant Gram-positive bacteria (e.g. *mutans streptococci* and *lactobacilli*) at the expense of the acid-sensitive species associated with sound enamel [2].

ETIOLOGY, PREVALENCE, AND RISK FACTORS

Dental plaque biofilms is a spatially organized structure containing a metabolically integrated community of bacteria

which interact and intercommunicate. Significant transformation lead to overgrowth of previously minor species that trigger changes from a “healthy,” to a more “pathogenic” plaque. Dental plaque bacteria create their own hydrated exopolysaccharide matrix. They include glucans, which acts as a “glue”. Sugar, especially sucrose, promotes this matrix formation. A group of glucosyltransferases (GTFs), are produced on the tooth surface by specific species of oral bacteria, especially the cariogenic *mutans streptococci* (SM). The acid-producing bacteria rapidly metabolize the sugar-producing acids at the tooth surface. Therefore, the cariogenic bacteria and sugar play an important role in plaque pathogenicity. When acid is formed in sufficient amounts that favor dissolution of calcium and phosphate from the tooth enamel, demineralization occurs and the tooth mineral is partially lost. The pH values of 4.0 are observed at the tooth surface within a few minutes following exposure to sugar. Lactic, acetic, propionic and formic are the most commonly detected acids.

BACTERIOLOGY OF DENTAL CARIES

Dental caries is a chronic site-specific disease caused by protective factors favoring tooth remineralization and destructive factors leading to demineralization. *Mutans streptococci* are the most cariogenic highly acidogenic pathogens, producing short-chain carboxylic acids resulting from many cycles of de- and re-mineralization. The group of *streptococci* comprises seven species. *S.mutans* (SM) and *S.sobrinus* (SS) are most important in terms of human caries. SM ferment sucrose and produce organic acids. Cariogenic bacteria produce insoluble extracellular polysaccharides, which enhance their adherence to the tooth

surface and contribute to biofilm formation. The attachment of bacteria is the preliminary step for colonization. Initial adherence of *S. mutans* to the tooth is mediated by cell-surface adhesin-like proteins. Not only it mediates the adhesion of the bacteria to the tooth, but it also provides the sites for further attachment. SM metabolizes sucrose and synthesizes water-soluble and insoluble glucans. The reactions are catalyzed by three isozymes of glucosyl-transferases (GTFs): GTF-B, GTF-C, and GTF-D. These enzymes catalyze the transfer and addition of a glucosyl moiety to the terminal site of a primer or elongating glucan.

The sticky nature of glucan facilitates the adherence of bacteria to the tooth and resists its detachment by normal mechanical forces. SM produces also three glucan-binding proteins (Gbps). The role of these proteins in mediating sucrose-dependent adherence has been established. They produce lactic acid, and have the ability to survive at a low pH. They are implicated in intracellular polysaccharide synthesis and in the production of a dextran-hydrolyzing enzyme (endodextranase).

In addition to glucan synthesis, bacteria use fructosyltransferases. Fructans are believed to function exclusively as extracellular storage reservoirs. Initial attachment of SM is followed by its accumulation and proliferation, leading to the formation of a sessile, exopolymer community known as a biofilm. Dental biofilm formation occurs through a series of stages.

- The first stage involves deposition of an acquired dental-enamel pellicle. This acellular coating includes salivary components and bacterial constituents.
- The second stage involves the adherence and co-adherence of bacteria from the oral cavity with the help of polysaccharides.
- Proliferation occurs in the third stage, whereas the biofilm reaches a steady state in relation to the surrounding environment.

A considerable pH decrease is observed in the oral cavity within a few minutes after a glucose rinse. This implies that the bacterium is rapidly acidogenic (Stephan's curve). This is mediated by a highly efficient phosphotransferase system for glucose and sucrose. This is achieved by upregulation of a proton-translocating ATPase that extrudes H⁺. SM synthesizes intracellular glycogen-like polysaccharides (IPSS). These IPSSs are similar to those of other oral streptococci and are glucose homopolymers with α -(1-4) and α -(1-6) linkages. The synthesis of IPSS is proportional to the extracellular carbohydrate concentration [3,4].

It is known that caries led to improvements in the prevention, diagnosis and treatment of dental caries, although there is still a need for new diagnostic tools and treatment methods [5].

As a possible *etiologic organism* associated with dental caries, the SM group has shown a great interest. Researchers initially isolated *mutans* from human carious lesions. The key caries-associated with microbial virulence traits include acidogenesis and acid tolerance, intracellular polysaccharide storage and extracellular glucan formation, which promotes SM attachment and increases plaque's pH-lowering ability. Recent

evidence also has supported the role of yeast (*Candida albicans*) as a member of the mixed oral microbiota acting as causing a relationship with the carious decay. SM is only one of many endogenous microorganisms involved in the pathogenesis of caries.

In order to identify the key determinants that influence patterns of colonization, it is necessary to understand the properties that influence microbial colonization. The mouth is continuously bathed with saliva, which keeps warm conditions (35-36°C) and moist at a pH between 6.75 and 7.25, that is optimal for the growth of many micro-organisms. The saliva ionic composition promotes its buffering properties and its ability to remineralize enamel. In addition, the organic components (glycoproteins and proteins)

- Influence the establishment and selection of the oral microflora by either coating oral surfaces, promoting the adhesion of certain organisms, or by aggregating other species and facilitating their clearance by swallowing,
- Act as endogenous nutrients. Saliva also contains components of innate (e.g. lysozyme, lactoferrin, sialoperoxidase, antimicrobial peptides) and adaptive immunity (IgA) and so can directly inhibit some exogenous micro-organisms.
- Teeth do not provide a uniform habitat for microbial growth, but possess several distinct surfaces (pits and fissures, lining points, and smooth surfaces bearing structural defects).

DIETARY FACTORS

Caries cannot occur in the absence of dietary fermentable carbohydrates. It has been characterized as a "dietobacterial" disease. Sucrose has a unique role as the substrate for glucosyltransferases involved in the synthesis of extracellular glucan.

The presence in pellicle of glucosyltransferases and fructosyltransferases in an active form, and other unidentified bacterial products is known for more than a decade. SM and other oral micro-organisms adhere effectively to glucan surfaces formed when pellicle is exposed to sucrose. Furthermore, there is a considerable degree of selectivity in the surfaces to which the various glucosyltransferases bind. Glucosyltransferase B binds preferentially to the surface of bacteria. Glucosyltransferases binding to oral bacteria has been already well-described. It involves opening the proline ring and the acceptance of two protons from lactic acid, giving rise to delta amino valeric acid.

Sugars and other fermentable carbohydrates provide substrates for the actions of oral bacteria. Sugars, both naturally occurring or added, and fermentable carbohydrates stimulate bacteria to produce acids and decrease the pH. It has been consistently shown that when the intake of free sugars is < 15 kg/person/year, the level of dental caries is low.

Fermentable carbohydrates are the class of nutrients that most affect the microbial ecology of the mouth. They are catabolized to acids which acidify plaque biofilms, before saliva returns the pH to normal values. Frequent exposure to such conditions of low

pH can lead to the enrichment of such acidogenic and aciduric species.

As soon as the teeth erupt, the enamel surfaces are coated with a conditioning film containing molecules derived from both the host (primarily saliva) and bacteria. The early colonizers then grow and modify local environmental conditions, making the site suitable for colonization by more species (e.g. obligate anaerobes). The later colonizers bind to the already attached species via similar adhesin-receptor mechanisms (a process termed co-aggregation or co-adhesion).

Caries are associated with increases in the proportions of acidogenic and aciduric (acid-tolerating) bacteria. The 'specific plaque hypothesis' proposed that out of the diverse collection of species present in plaque, only a relatively small number were directly involved in causing disease.

An alternative view was expressed in the 'non-specific plaque' hypothesis. This hypothesis proposed that disease is the result of the overall interaction of all the groups of bacteria within plaque, and recognized the concept that plaque is a microbial community.

The detection of low levels of several pathogens at a wide range of sites strongly suggests that plaque-mediated diseases result from imbalances in the resident microflora resulting from an enrichment. Caries is associated with more frequent exposure to fermentable carbohydrates, and with a lower pH in plaque. Early studies showed

- That SM was able to grow over a wider pH range,
- That its growth was optimal at acidic pH (~ pH 5.5)
- That rates of sugar uptake and glycolysis were greater
- And the terminal pH reached from sugar metabolism was lower than for *S. sanguinis*.

The transition seen in the composition of plaque between health and disease is driven by a response of the members of the microbial community to environmental change, resulting in the selection of previously minor components of the microflora [2].

- The antibacterial effects of fluoride have been largely ignored, despite evidence that fluoride can affect bacterial metabolism. Fluoride in combination with aluminum is a potent inhibitor of ATPase.
- Fluoride also inhibits enolase at low pH values and impedes the uptake of sugars.
- Finally, it has been shown that fluoride affects the production of glucosyltransferase, which plays an essential role in the etiology and pathogenesis of dental caries.
- Fluoride behaves as a weak acid at low pH values. In the protonated form, it can diffuse into cells, where it can dissociate and affect the pH [4,5].

Host salivary and genetic factors

Host salivary and genetic factors seem also to be involved in caries development [6]. The effectiveness of dietary measures to control caries is limited because modern diets are complex

and contain many natural sugars, refined sugars and sugar substitutes.

The cusps morphology, pitting and fissure on the occlusal surface of teeth, the anatomy of enamel, dentin, cementum junction, at some distance from each other or overlapping, the relationship between the proximal surfaces of teeth (leaning point or surfaces between teeth accessible or not to toothbrushing) are genetically determined and therefore they were found to be associated with the carie-free group of patients or with the carious susceptible group. The composition and flow of saliva is also gene-determined.

Chewing sugar free gum after meals (sorbitol-containing so-called sugar-free gum) stimulate salivary flow to restore the pH of the dental plaque to its resting levels. It results in a significant decrease in the incidence of dental caries and the benefit is due to stimulating salivary flow rather than any chewing gum ingredient.

Partially demineralized enamel and dentin apatite crystals can be remineralized to almost their original size under optimal laboratory conditions. The process is diffusion-controlled. This leaves a sealed surface that is more resistant to subsequent demineralization than sound enamel.

CARIOGENICITY OF FOODS

Sugars are implicated as the main cause of dental caries. Removal of the dentin matrix could be due to salivary or leukocytic proteases. Bacterial proteolytic enzymes are necessary and not only contributory for destruction of the dentin matrix. Early childhood caries cause extensive destruction of the deciduous teeth, leading to the terms of « baby bottle tooth decay » or « nursing caries ». The frequency of intake of sucrose seems to be more important than the total amount consumed. Teeth erupt into the mouth with immature enamel. The process of enamel maturation continues following tooth eruption, so that teeth become less susceptible to decay over time. The enamel matures incorporating orally available ions including fluoride. Therefore, a tooth is more susceptible to caries immediately after eruption than at a final stage of maturation.

As they were acidogenic and aciduric organisms, bacteria were of special significance in the caries process. Other organisms such as *staphylococci*, *lactobacilli*, and yeasts are frequently present but in much lower numbers. The low pH present in various plaques may be responsible for the increase in the former organisms, since they are somewhat acid tolerant. The increased lactate concentration present in carious plaque is probably responsible for their increase. Besides these changes, plaque material over various areas appears to contain larger numbers of organisms per unit wet weight than caries-free plaques.

Until recently, few data were suggesting that the composition of the plaque microbiota was important in determining cariogenicity. Organisms present in plaque synthesize intracellular polysaccharides of the glycogen-amylopectin type from glucose and other carbohydrates. Acids are produced from metabolism of polysaccharide to lower the pH of packed cells of *Streptococcus Mitis* to below pH 6 and maintain this acidity for several hours in the presence of continuously flowing buffer of pH 7. These findings suggest that polysaccharide-storing

organisms might be responsible for maintaining various plaque at a low resting pH. Some of the characteristics of cariogenic micro-organisms appear to be the ability to form acid from carbohydrates, to persist in large numbers in dental plaque, and to form intracellular polysaccharide [6].

Streptococcus oralis and *Actinomyces naeslundii* provided sialidase activity, and could remove terminal sialic acid residues, exposing new substrates. The ability to replace simple sugars with a glycoprotein has enabled more realistic simulations to be made of the influence of dietary carbohydrates. Individuals who frequently consume sugar in their diet generally have elevated levels of cariogenic bacteria such as SM and lactobacilli in their plaque, and are at greater risk of dental caries.

The sugars consumed more commonly are the monosaccharides glucose and fructose. Fructose is less cariogenic than sucrose. In patients with excellent oral hygiene there is virtually no 'plaque' on the tooth surfaces and therefore SM may be below the detection level. The relationship between SM and dental caries is not absolute. Relatively high proportions of SM may persist on tooth surfaces without caries progression while caries may develop also in the absence of these species. This should be combined with the fact that SM are not primary colonisers of tooth surfaces.

The paradigm asserting that the biofilm is the cause of dental caries has several consequences. Lesions develop where biofilms are allowed to mature. Mechanical methods of plaque control including brushing, flossing and professional scaling are only temporarily effective in eliminating SM. The control of plaque growth by chemical has attracted considerable attention and chlorhexidine has been shown to be effective. Classical antibiotics interfere with plaque development, but they are not appropriate for long-term application because they can lead to resistance development. The relatively narrow spectrum natural bacterial antibiotics known as bacteriocins appear to offer considerable potential benefits as anti-SM agents. These small peptide molecules could be incorporated into mouthwashes or toothpastes or, alternatively, antimutans-producing bacteria could be implanted within the oral microbiota.

Modern diets of industrialised countries contain a mix of sugars and other carbohydrates that are fermentable in the mouth. Oral bacteria metabolise all mono- and di-saccharides to produce acids. However, early plaque pH studies have shown plaque bacteria produce less acid from lactose compared with other sugars.

The level of dental caries is low in countries where the consumption of free sugars is below 15-20 kg/person/yr. This is equivalent to a daily intake of 40-55 g. It is of particular importance that countries which currently have low consumption of free sugars (<15-20 kg/person/yr) do not increase consumption levels. Infection with SM occurs vertically from the mother to early childhood. Mothers with high levels of MS have a greater chance of transmitting the bacteria, particularly if they engage in practices such as tasting the infant's food and sharing eating utensils [7,8].

In a three-year investigation, groups of children aged 9 to 14 years were assigned to one group: sorbitol-containing chewing

gum, xylitol-containing chewing gum, control chewing gum and a no-gum group. The results for the sorbitol-/carbamide-containing chewing gum group were not different from those for the no-gum group. It was concluded that the appreciable benefits from the chewing gums were attributable to the chewing process rather than to the presence of the sorbitol or xylitol. Clinical caries studies have indicated that chewing sugar-free gum after meals results in a significant reduction in the formation of dental caries. This effect is caused by the increased salivary flow attributed to the chewing process rather than to the sorbitol in these chewing gums [8-10].

Free sugars, at even very low intake, are cariogenic [11]. They include the previous mono and disaccharides added to food plus those naturally present in honey, syrups fruit juices and concentrates. Sometime the term of fermentable carbohydrates is also used. The group of patients receiving xylitol developed very little caries compared with the sucrose group.

A sugar substitute is a food additive that duplicates the effect of sugar in taste, but usually has less food energy. Sugar substitute's are natural, as well as artificial and very much popular because of their beneficial effect on overall dental, as well as general health (Table 1,2).

Sugars replacers or bulk sweeteners include the sugar alcohols, (also called polyols), sorbitol, mannitol, xylitol, isomalt, erythriol, lactitol, mantitol, hydrogenated starch hydrolysates, and hydrogenated glucose syrups. Polyols and other bulk

Table 1: Sugars substitutes.

S.no	Caloric/Nutritive sweetner	Non-Caloric non-nutritive Sweetner
1	Poly alcohols	Cyclamate
	Xylitol	
	Sorbitol	
2	Hydrogenated starch hydrolysates	Saccharin
	Lycasin	
	Palatinit	
3	Coupling sugars	Aspartame
	Sorbose	
	Palatinose	
4		Sucralose
5		Neotame

Table 2: Comparison of sweetness of various sweeteners (sugar substitutes) to that of sucrose (sweetness of sucrose).

1	Xylitol 1.0x
2	Sorbitol 0.6x
3	Malitol 0.9x
4	Mannitol 0.5x
5	Glycerol 0.6x
6	Monellin 3000x
7	Stevia 250x
8	Thaumatococin 2000x

sweeteners have three potential advantages over sugar as food ingredients.

- First, unlike sugars, they do not promote the development of dental caries.
- A 2nd potential advantage of polyols and functionally similar sweeteners such as tagatose and trehalose is that they produce a lower glycemic response than most sugars and starches do.
- A 3rd potential advantage is that most polyols are lower in calories than sugar. Sugar provides 4 Cal/g. However, most of the commonly used polyols provide fewer calories.
- Fourth- Xylitol and sorbitol are majorly used sugar substitutes in chewing gum. Xylitol in chewing gum is reported to reduce not only the proportions of SM in plaque or saliva, but also the amount of plaque present.

There is a considerable amount of information on the effect of non-sugar sweeteners on dental health. Xylitol has been subjected to many studies of different types and can be classified as non-cariogenic.

From a dietary point of view, the best advice for reducing caries risk is to diminish the frequency of consumption of sugars containing food and drinks and to limit their consumption to mealtimes only. It is also advisable to avoid sugars-containing food and drinks close to bedtime (within one hour), as salivary flow is low and its buffering capacity is reduced at night.

Giving dietary advice to patients and selecting the two groups of patients carious-resistant and carious-susceptible the main points and advice to give patients are:

- Limit the frequency of consumption of sugary food and drinks.
 - Eat more vegetables and fruit and starchy staple foods such as bread, potatoes,
 - Un sweetened breakfast: cereals and grains.
 - Drink milk and water rather than sugary acidic soft drinks [11] (Figure 1,2).
1. Strategies that are consistent with the prevention of disease include: inhibition of plaque acid production,
 2. avoidance between main meals of foods and drinks containing fermentable sugars,
 3. the consumption of foods/drinks that contain non-fermentable sugar substitutes,
 4. the stimulation of saliva flow after main meals (Figure 3).

CONCLUSIONS

Prevention program is based on plaque control and topical application of fluorides, after scaling, root planning and conventional caries therapy. After a 6 years follow-up and during the next 9 years period patients were recalled to see the effects of preventive sessions [12].

Agents at sub-MIC levels reduce metabolism by inhibiting

glycolysis, sugar transport and proteases, which will stabilize microbial communities and maintain homeostasis.

Bulk agents, such as sugar alcohols (sorbitol, xylitol), and intense sweeteners will stimulate saliva in the absence of significant acid production. This can even lead to the remineralization of early lesions.

Identification of the causal factor(s) driving this local 'ecological catastrophe' in plaque, and deal with both the cause and the effect of the disease. Examples of potential causal factors include poor oral hygiene, inappropriate diet, smoking and

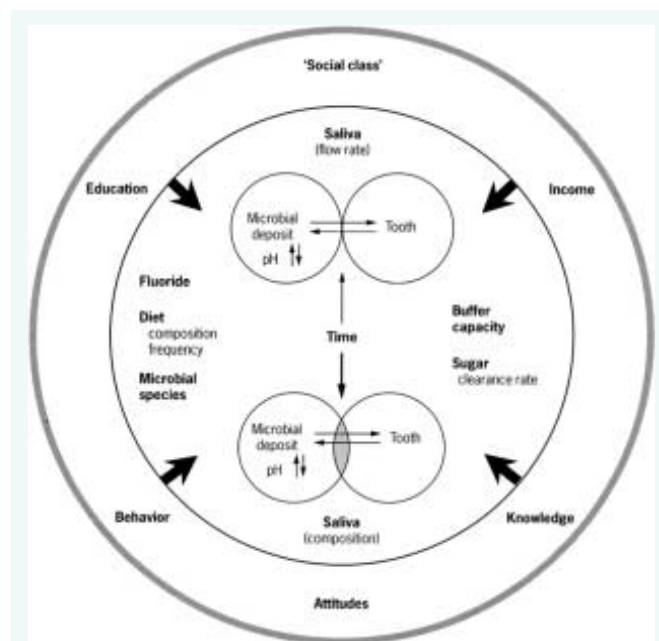


Figure 1 Schematic illustration of the relationship between the aetiological factor – the microbial deposit on the tooth and biological determinants (inner circle) which influence lesion developments at the single tooth surface. In the outer circle are listed various behavioural and socio-economic factors (or confounders) which influence the likelihood for lesion development at an individual and population level [11].

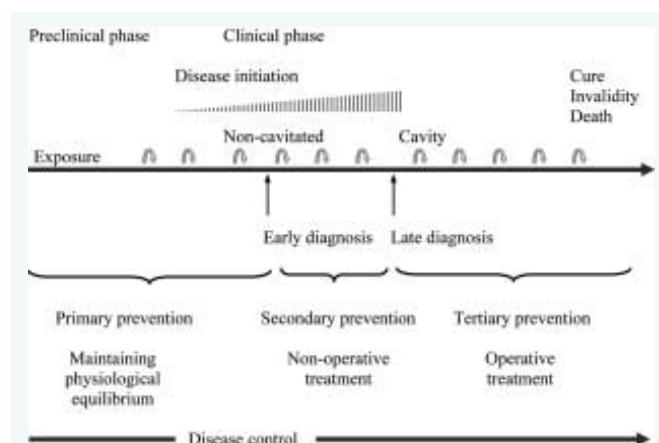


Figure 2 The concept of dental caries- exposure to metabolically active biofilm disease control must be maintained life long [11].

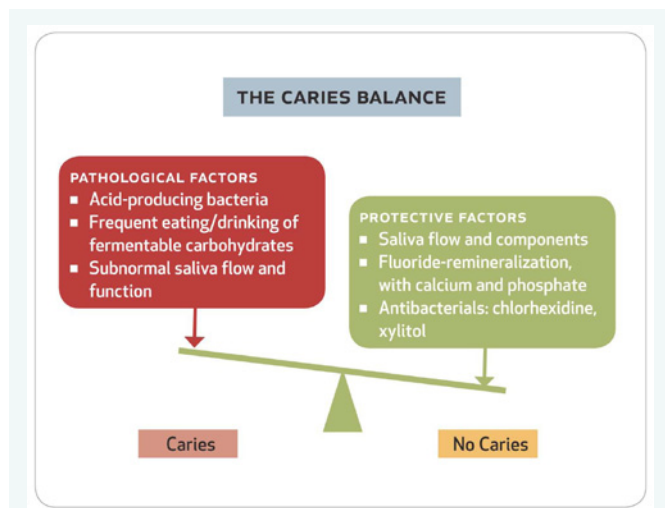


Figure 3 The caries balance concept [13,14]. The key pathological and protective factors determine which side the balance swings and whether the caries process progresses, reverses, or is in balance.

the long term use of medications that reduce the flow of saliva or suppress the activity of components of the adaptive host defences. Fluoride as a gel or varnish, is acting as a preventing agent. Chlorhexidine, Pitts and fissures sealants, and patient counseling prevent the development of carious lesions.

- Chlorhexidine has substantial antimicrobial properties, namely against caries-causing bacteria. Mouthrinses containing 0.12% CHX are in the market and act as preventing agent is effective and leads to a caries reduction about 46%. As a gel, the prevention rate was about 47%. The difference is non-significant.
- Pitt-and-fissure sealants provide 71% effectiveness. The effects of prevention on patients at reduced risk for dental caries are low. In contrast, caries reduction is effective with patients at high-risk [13].

Caries prevention has lead during the past decades to a serious decline. However, we are now facing a plateau, and no further decrease is expected shortly. Therefore, new tools are needed to eradicate the carious pathology [14-16].

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