

ILSI EUROPE CONCISE MONOGRAPH SERIES

# ORAL AND DENTAL HEALTH

*PREVENTION OF DENTAL CARIES, EROSION,  
GINGIVITIS AND PERIODONTITIS*



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# Oral and Dental Health

*Prevention of Dental Caries, Erosion, Gingivitis and Periodontitis*

by Cor van Loveren



**ILSI Europe**

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For more information about ILSI Europe, please contact

ILSI Europe a.i.s.b.l.  
Avenue E. Mounier 83, Box 6  
B-1200 Brussels  
Belgium  
Tel.: (+32) 2 771 00 14  
Fax: (+32) 2 762 00 44  
E-mail: [info@ilsieurope.be](mailto:info@ilsieurope.be)  
Website: <http://www.ilsieurope.eu>

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*Author:* Cor van Loveren, ACTA – Academic Centre for Dentistry Amsterdam (NL)

*Scientific Editor:* Monty Duggal, Leeds Dental Institute (UK)

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## FOREWORD

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During recent decades the dental health of children and adolescents has improved steadily in the Western world due to the availability of fluoride. Research has shown the capacity of fluoride to prevent caries, to arrest caries lesions when they occur and to heal caries lesions by enforcing remineralisation. This Concise Monograph reviews the latest scientific knowledge on, and understanding of, the important role of fluoride in the prevention of caries. Additionally, it covers the importance of regular oral hygiene measures for the prevention of periodontal diseases, and addresses the issue of dental erosion and ways to minimise it.

This Concise Monograph was developed under the auspices of the ILSI Europe Oral Health Task Force. We hope that it will contribute to further improvements in the oral health of all generations.

Susanne Ziesenitz  
Südzucker  
Germany

## DENTAL AND ORAL HEALTH OVER THE LAST DECADES

Dental caries is a disease affecting the hard tissues of the teeth, which might result in progressive decay. Bacteria that accumulate in a dense mass, as plaque on the surface of the teeth, ferment carbohydrates from foods and drinks and form acids that demineralise the hard tissue underneath. Over the last 30 years, prevalence of dental caries has decreased tremendously, especially in the more affluent countries. Figures 1 and 2 compare the numbers of carious and filled teeth in 12-year-olds in various European countries in the 1970s and 1980s with those observed about thirty-five years later, at the beginning of the 21<sup>st</sup> century. In the 1970s and early 1980s,

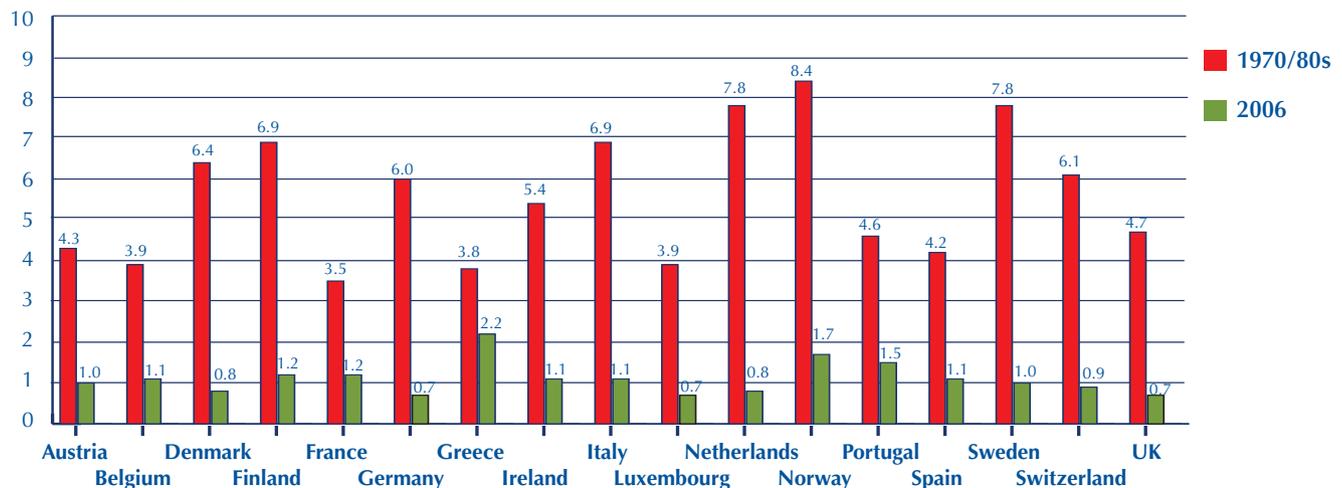
caries prevalence was high with up to eight teeth of the dentitions of the 12-year-olds affected. At the turn of the 21<sup>st</sup> century in Western European countries, only one tooth on average was affected by caries in this age group, with a high proportion of children having a dentition free from cavities (Figure 3). The prevalence of caries in older age groups is also declining. One of the most important factors for the lower prevalence of caries in children and adolescents is the increased awareness of dental health and of regular oral hygiene measures with the daily use of fluoride toothpaste. Surprisingly, the sugar supply in the populations remained stable, at about 34 kg per year in Europe, during this period of caries decline (Figure 4).

This improvement in dental health is unfortunately not yet a reality for everyone. In those subpopulations where fluoride toothpaste is not available or affordable

**FIGURE 1**

### Caries in 12-year-old children in various EU member states in 1970/80s and in 2006

Decayed, missing and filled permanent teeth (DMFT)

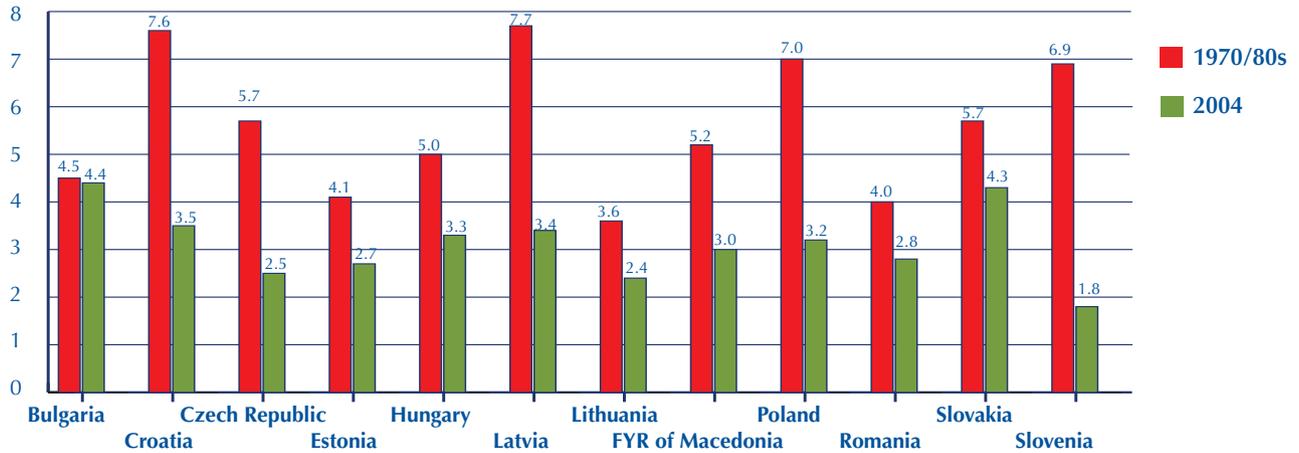


Sources: WHO Europe. Health for all Database & WHO Oral Health Country/Area Programmes, [www.collab.od.mah.se](http://www.collab.od.mah.se)

**FIGURE 2**

**Caries in 12-year-old children in the new EU member states in the 1970/80s and in 2004**

Decayed, missing and filled permanent teeth (DMFT)



Sources: WHO Europe. Health for all Database & WHO Oral Health Country/Area Programmes, [www.collab.od.mah.se](http://www.collab.od.mah.se)

**FIGURE 3**

**Prevalence of caries-free children (12-year-olds) in Germany from 1984 to 2005.**

Percentage of caries-free children plotted against year of survey (Micheelis and Schiffner, 2006)

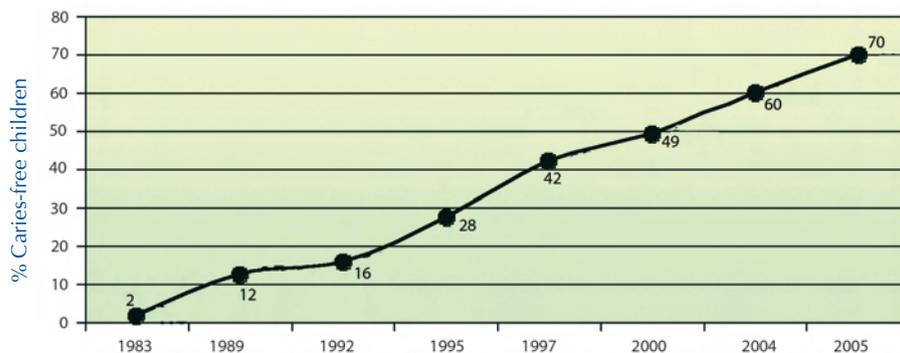
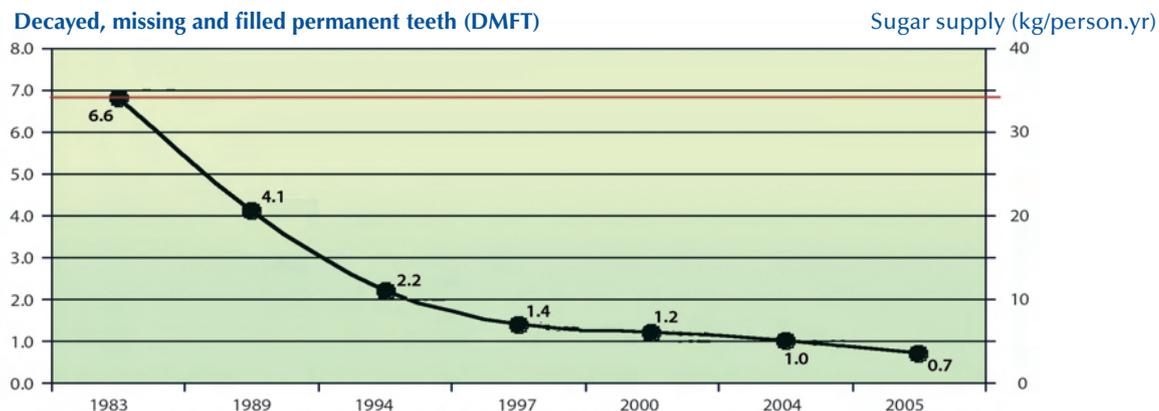


FIGURE 4

Caries decline in 12-year-old children against the background of a stable sugar supply (34 kg per person per year) in Germany (adapted from Micheelis and Schiffner, 2006)



and where the populations do not yet use it routinely or adequately, dental caries is still common. The importance of making fluoride toothpaste available to all, irrespective of the socio-economic status, and motivating people to use it daily are key factors in further reducing the prevalence of caries globally.

Another danger to the integrity of teeth is dental erosion, which is the irreversible loss of tissue resulting from contact with acids without the involvement of the oral bacteria in the dental plaque. The prevalence of dental erosion seems to be increasing. The exact prevalence is not easy to determine because of a lack of homogeneity in the scoring criteria used and because of the different age groups studied. Reports from several European countries show prevalence data varying from 16 to 40% in teenagers.

Periodontal diseases are prevalent and can be present in various forms. Gingivitis is the mildest, reversible, form characterised by inflammation of the gingiva without

involvement of the underlying supporting structures. It is very common where bacterial plaque accumulates and is not removed regularly. Habitual sites of plaque retention are the spaces between the teeth and the gingival margins of the dentition. Gingivitis can be cured, provided oral hygiene measures improve. More advanced stages are called periodontitis and involve loss of connective tissue and bone support, which can result in loosening of teeth, pain and discomfort, impaired capacity to chew and eventually tooth loss. Normally periodontitis will not develop other than on the basis of a long lasting gingivitis; so it is therefore important to fight gingivitis by meticulous oral hygiene. Moderate periodontitis is seen in 20–30% of adults aged 35–44. Periodontitis with advanced loss of connective tissue and bone support in one or more teeth is probably seen in 10% of this age group. The prevalence figures increase with age to 30–60% for the moderate and 20–30% for the advanced stages in seniors of 65–74 years of age.

## DENTAL CARIES

Dental caries is a disease affecting the hard tissues of the teeth resulting in progressive decay. Tooth, plaque and substrate are the three prerequisites for a caries lesion to develop as first postulated by Keyes (1960; Box 1; see the inner three circles in Figure 5). After tooth brushing, plaque formation starts when teeth become colonised with oral bacteria from the saliva. After some time, the number of bacteria is so large that a continuous layer is formed. This so-called dental plaque develops further as a layer consisting of bacteria, bacterial products, salivary proteins and food substances from the diet. Normally dental plaque is not visible to the naked eye but if it is not regularly removed it may become thick enough to be seen. More than one hundred million bacteria per mg (an amount the size of a pinhead) can develop in the dental plaque. Over 500 bacterial species have been identified. Modern molecular methods reveal an even more diverse view of the bacterial flora and suggest that a large proportion of this microbial environment remains uncharacterised. Not all species are able to colonise in high numbers because of local biological and physical properties of the mouth. Nor can all species produce acids, but some we know to be excellent “acid producers” and may produce sufficient acid to dissolve tooth mineral. The most important ones that we know are capable of doing so are the *Lactobacilli* and the mutans streptococci – the collective name for *Streptococcus mutans* and *S. sobrinus*.

The demineralising acids are produced by the fermentation of carbohydrates. It takes time for the carbohydrates to be released from the food, to be taken up by the dental plaque and to be utilised by the acidogenic bacteria to produce acids. Complex carbohydrates are hydrolysed by salivary amylase, breaking them down into a form that can be fermented by the oral micro-flora.

### BOX 1

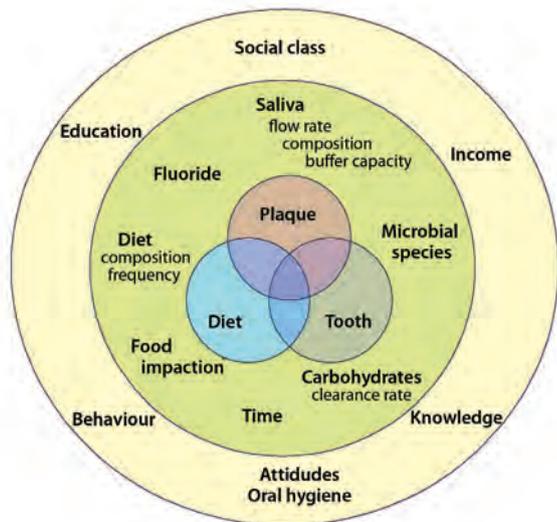
#### Evolution of our understanding of caries

Over the ages there have been many theories on the caries process. Until the 18<sup>th</sup> century, the worm theory - little animals inside your teeth – was very popular. In the 19<sup>th</sup> century it was recognised that caries attacked the teeth from the outside. In 1867, Leber and Rottenstein hypothesised a model in which bacteria fermented carbohydrates to acids in which the tooth would dissolve. Thereafter, the bacteria were supposed to penetrate the enamel and dentine to break down the organic components, a parasitic process. In 1889 Miller postulated a more precise theory supported by good scientific experiments. Experiments *in vitro* with bacteria isolated from the oral cavity demonstrated that these organisms produced organic acids from sugars and from bread, and were also capable of proteolytic activity. By incubating slices of a tooth with saliva, bread or sugar, it was demonstrated that enough acid was produced to decalcify the tooth. On the basis of these and other experiments, Miller formulated the concept we now refer to as the “chemico-parasitic theory” of dental caries. In the 1960s Keyes showed that certain bacteria cause dental caries. He formulated the three prerequisites – plaque, tooth and diet (as substrate for bacteria) – for a caries lesion to develop. Between 1960 and 1970 many studies were performed indicating the important role of *Streptococcus mutans* in the caries process; these studies also unravelled the factors that determine the virulence of this species.

The acid production is limited as the dental plaque acidifies and when the supply of carbohydrates is depleted by the continuous flow and swallowing of saliva. The saliva also contains buffers, which counteract the acidification of the dental plaque by neutralising it – returning the pH to neutrality after acid production stops. Saliva is therefore of paramount importance in the defence against tooth decay.

FIGURE 5

## Modern caries model



In 1960 Keyes designed the central triad in this figure. Plaque, tooth and diet are the three prerequisites for a caries lesion to develop. Subsequent researchers extended the model with factors affecting the interplay between the three prerequisites. The first ring shows modifying factors that play a role in the oral cavity. The second, outer, ring shows behavioural aspects associated with caries risk (modified from Fejerskov and Thylstrup, 1994).

When sufficient acid has been produced and the pH drops below the critical value of 5.5, tooth mineral will dissolve: demineralisation. Calcium and phosphate leave the tooth. This process is reversible and when pH restores towards normal, calcium and phosphate can be rebuilt into the tooth: remineralisation. Unfortunately remineralisation is a much slower process than demineralisation. Therefore, remineralisation only compensates for limited periods of demineralisation. If the demineralising influences on the hard dental tissues exceed the remineralising ones, eventually cavitation will occur.

## Cariogenic dental plaque

Much oral microbial research has focussed on the identification of the oral bacteria assuming that specific bacteria were responsible for the caries process. Traditionally mutans streptococci were associated with the onset of the caries process, while in more advanced caries lesions high numbers of *Lactobacilli* could also be isolated. This specific plaque theory was attractive since it would allow specifically targeted antimicrobial interventions. However, mutans streptococci were not always isolated from caries sites, whereas they could be found in high numbers in dental plaque over apparently sound tissue. The first observation suggests that acidogenic bacteria other than mutans streptococci significantly contribute to the caries process, while the second observation suggests that the harm expected of mutans streptococci is counteracted. This may be due either to the localisation of the mutans streptococci in the dental plaque away from the enamel surface, to the presence of acid consuming bacteria (e.g. *Veillonella*) or to the production of ammonia (e.g. by *Streptococcus salivarius* or *Streptococcus sanguis*) raising the local pH.

In 1994 Marsh formulated the ecological plaque hypothesis. This theory emphasised the relationship between plaque composition and local environmental conditions. Caries could only develop when the natural balance of bacteria in dental plaque is disturbed, allowing an increase in the number of acid-producing bacteria. The disturbance may result from key host factors, such as an increased frequency of fermentable carbohydrate intake, allowing plaque to develop in the absence of mechanical disturbance by tooth brushing or during a reduction of the salivary flow. Implicit in this theory is the concept that interfering with those factors that are driving the detrimental shifts in the microbial balance in dental plaque can also prevent caries.

Today, emphasis is given to the fact that dental plaque is a biofilm and functions as a microbial community, in which bacteria are communicating. The properties of such a community are more than the sum of those of the constituent species. Interactions between bacteria can be beneficial to one or more of the interacting species, while others can be antagonistic. Biofilms and microbial communities are protected against environmental stresses. This implies that higher concentrations of antimicrobials are needed to affect the growth of bacteria when they are living in a biofilm. Also, the penetration of antimicrobials may be hampered and they may not reach the tooth surface. Current research is very much focussed on unravelling the genetic nature of the interactions between the bacteria and their responses to environmental stresses in biofilms.

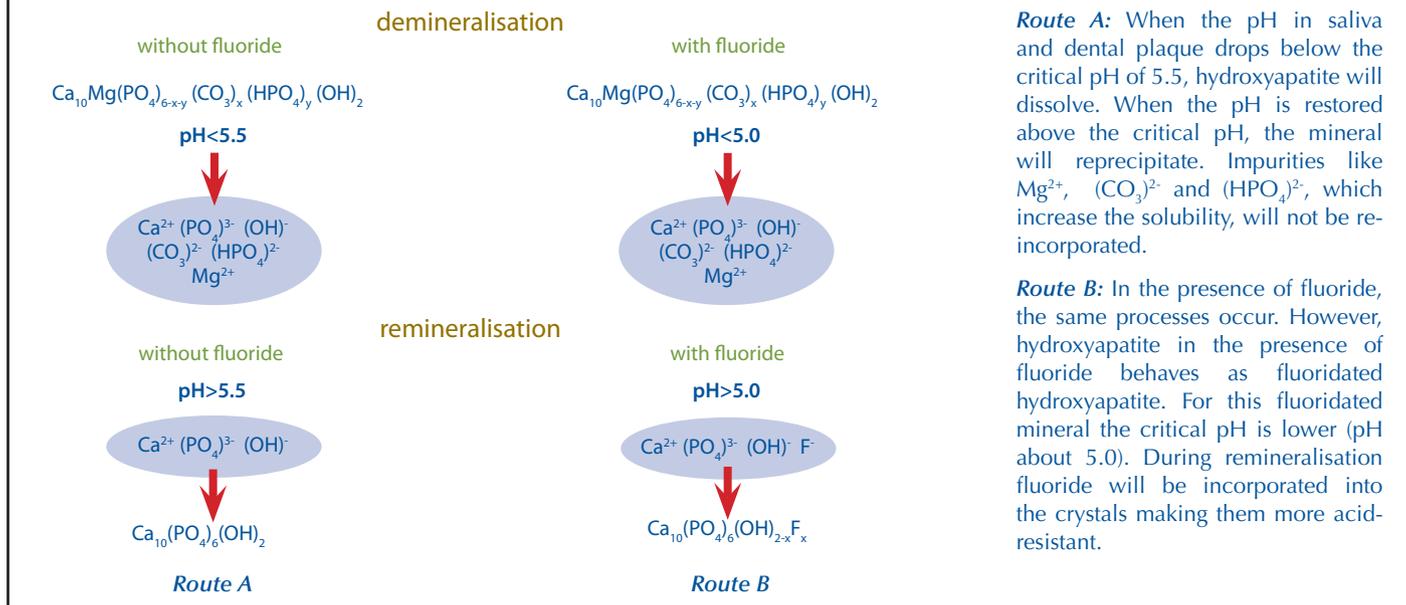
Clinically, the focus is still on mutans streptococci. Their number in saliva predicts the risk of caries, and individuals with  $>10^6$  mutans streptococci per ml of saliva are regarded as being at risk. Mutans streptococci are the target organisms for antimicrobial caries therapy. Antimicrobial caries research comprises validating the efficacy of antimicrobials, assessing the feasibility of passive and active immunisation and assessing the feasibility of the replacement of mutans streptococci with less harmful strains. Research also focuses on preventing the transmission of mutans streptococci from mother to child, which occurs mainly when the child is between 1½ and 2½ years of age (“window of infectivity”). Transmission to the child can be prevented when the number of mutans streptococci in the mouth of the mother is suppressed, for instance by an intensive treatment with chlorhexidin or when mothers frequently chew xylitol gum.

## *The caries lesion in more detail*

Enamel, dentine and root cement consist of an inorganic component (approximately 86, 55 and 45 vol%, respectively), an organic component (approximately 4, 25 and 30 vol%, respectively) and water. The inorganic component is hydroxyapatite,  $\text{Ca}_{10}(\text{PO}_4)_6(\text{OH})_2$ . Since the oral fluid and dental plaque contain calcium and phosphate ions, it depends on the pH whether the environment of the tooth is saturated, under- or super-saturated with respect to the mineral. When the environment is undersaturated, demineralisation will occur and when the environment is supersaturated, remineralisation will take place. During tooth formation impurities may be incorporated in the tooth mineral making the mineral either less or more soluble. Impurities such as carbonate ( $\text{CO}_3$ ), magnesium (Mg) and acid phosphate ( $\text{HPO}_4$ ) will make that part of the mineral more soluble. These impurities will soon be lost during carious attack. When the pH in overlaying dental plaque drops below 5.5, which is called the critical pH, dissolution of enamel starts. When the pH rises again, over 5.5, remineralisation will occur and the impurities will not be built in (Figure 6, route A). As long as remineralisation can keep up with the demineralisation, cycles of de- and re-mineralisation will result in a mineral of better quality. This is part of the post-eruptive maturation of the mineral. When remineralisation cannot keep up with demineralisation, i.e. when remineralisation is not given sufficient time, caries lesions will develop. In the presence of fluoride, hydroxyapatite will behave as fluoridated hydroxyapatite, which dissolves only as the pH drops below approximately 5.0 (Figure 6, route B). This means that the critical pH for demineralisation shifts by approximately 0.5 to a more acidic critical pH value of 5.0. When the pH returns to less acidic values above this “new” critical pH of 5.0, fluoride will be built into the lattice of the mineral making it less soluble.

FIGURE 6

## Demineralisation and remineralisation



Caries lesions do not develop as surface lesions but as subsurface lesions that progress below the surface while the outer surface remains intact (right side of Figure 7). At least three mechanisms contribute to the survival of the outer tooth surface. First, the outer surface is simply less soluble than the mineral just below. Secondly, undissociated acid will transport  $\text{H}^+$  ions over the outer surface deeper into the mineral, where the acids will dissociate. Thirdly, calcium and phosphate ions dissolved in deeper layers diffuse out of the mineral and meet conditions in the outer surface layer, which might cause them to reprecipitate. As long as the outer surface of a lesion is intact, preventive intervention might be sufficient to prevent progression of the lesion into a cavity, even when the caries process has actually reached the dentine (right side of Figure 7). When plaque

is adequately removed and fluoride is presented, the outer surface may reharden to such an extent that it is of even better quality than the original surface. When the caries process reaches the dentine, extensions of the odontoblasts (cells that form dentine and remain alive in the pulp of the teeth) will be triggered to form additional dentine, both within the dental tubules (sclerotic dentine) and within the pulp (tertiary or reactive dentine). Both types of deposition can be seen as a defence mechanism against the progression of the caries lesion.

Dentine is more vulnerable to acid dissolution than enamel, due to its composition and open structure. The mineral crystals are smaller than those in enamel, which means that the crystal surface area is increased and therefore that the crystals are more easily attacked.

FIGURE 7

**Caries lesions, with different degrees of progression**

**Right side:** Subsurface lesion (A). The outer surface is still intact, but the caries lesion already reaches beyond the enamel dentine junction. Adequate preventive treatment can stop lesion progression and rehardens the outer surface. If not, the lesion may progress to a cavitated lesion.

**Left side:** The lesion is cavitated (B). This lesion is beyond the stage where adequate preventive treatment can stop its progression. The plaque in the cavity cannot be removed, neither can fluoride adequately be applied. This lesion needs to be restored by a filling.

Source: Dr. Ch. Penning, Department of Cariology Endodontology Pedodontology ACTA, The Netherlands.

## ***Importance of saliva for the protection of teeth***

Saliva is produced by six major glands, being two parotid, two submandibular and two sublingual glands, and by 200–400 minor glands that open in various places in the mucous membrane bringing saliva into the oral cavity. Approximately 1 ml of saliva is continuously present in the oral cavity spread out as a thin film over all surfaces and in a pool in the floor of the mouth. Many functions have been attributed to saliva, which are all relevant to oral health. Human saliva contains sufficient amylase to break down complex carbohydrates, retained from food remnants in the oral cavity, into maltose, isomaltose and glucose, which are substrates for bacterial fermentation. Saliva also serves as a lubricant for creating a bolus of the food and for facilitating speech and movement. As a solvent it is important for taste perception, but also as a carrier of food-derived substances to the dental plaque. Saliva is important in clearing food remnants and bacterial metabolites from the oral cavity. It contributes to the defence against microorganisms, viruses and fungi colonising the oral cavity. Saliva protects the oral cavity against damaging pH changes because of its buffering capacity, mediated by bicarbonate, phosphate and proteins. Last, but not least, at neutral pH saliva is a powerful remineralisation solution.

Salivary flow can be stimulated by gustatory, mechanical and psychological stimuli and the secretion rate can increase to over 3 ml/min (Table 1). Hyposalivation is, in the absence of an intensive preventive programme, a serious risk factor for dental diseases. Hyposalivation can be the result of malfunction of the salivary glands due to age, an infection, or disease of the glands, systemic diseases, obstruction of the salivary duct radiotherapy, surgical damage, stress, fear or depression. Rarely, glands may be hypoplastic or not present at all.

Dentine also has a much larger organic component (25%) embedded in the mineral compared with enamel (4%). Once the mineral is gone, the organic material is exposed to the oral environment and will be broken down by salivary and bacterial proteolytic enzymes. All these factors together make dentine more vulnerable to caries.

**TABLE 1****Secretion rate of resting and paraffin-stimulated whole saliva (ml/min)**

	Hyposalivation	Low rate	Normal rate
Resting saliva	<0.1	0.1–0.25	0.25–0.5
Stimulated saliva	<0.7	0.7–1.0	1.0 to >3.0

Certain medication, such as drugs that have a sedative function, antihistamines and tablets against high blood pressure, may also suppress the production of saliva. Individuals who compensate for hyposalivation or xerostomia (the subjective feeling of oral dryness) by sucking sugary sweets and tablets or by frequently sipping beverages containing fermentable carbohydrates are, in addition, at an increased risk of developing dental caries. Hyposalivation can be treated by stimulation of salivary glands or by the use of saliva substitutes. Stimulation of salivary flow can be brought about by gustatory and mechanical stimuli, e.g. by several chewing exercises on sugar-free chewing gum during the day. Occasionally certain salivary flow stimulating drugs such as pilocarpine are prescribed. If stimulation of the salivary glands is not possible, saliva substitutes have to be used.

### ***Who is at risk of developing caries?***

Everyone who has teeth and dental plaque and who consumes fermentable carbohydrates is at risk of developing caries (Figure 5, page 6). However, it is not that simple. Not all of us are diligent in plaque removal, and most of us consume fermentable carbohydrates frequently. But not everybody develops dental caries. Over the last three decades, there has been a huge increase in the number of children and adolescents in western societies who are totally cavity free. In the year

2005 for example, about 70% of the 12-year-old children in Germany had no dental cavities (Figure 3, page 3). The caries distribution is skewed in the population of children and adolescents, with a minority of the children having the majority of the caries burden.

There are many factors that influence the outcome of the alternating de- and remineralisation processes on the tooth surface. On the one hand there are biological factors such as the composition and growth rate of dental plaque, the composition and flow rate of saliva, the retention in the oral cavity of fermentable carbohydrates and the availability of protective substances, importantly fluoride. On the other hand there are personal and behavioural factors that have a major influence on the development of dental caries. The way we eat is important. For example, how often do we eat, do we eat quickly or do we graze? Is the passage of food through the mouth quick or slow? Do we add sugar to our drinks? How effective are we at brushing? Do we follow health advice? Do we believe dental health is important? Do we believe caries is avoidable? Figure 5 summarises all these factors. Those individuals in whom the unfavourable personal and behavioural factors coincide will be at risk of developing dental caries. At a population level, especially in the developed world, many of these compromising factors are present in low socio-economic groups, making them more vulnerable to dental caries.

## DENTAL EROSION

Dental erosion is the irreversible loss of dental hard tissue due to exposure to acids without known bacterial involvement (Figure 8). The typical order of appearance of the signs and symptoms of erosion is: loss of surface anatomy, glassy appearance, darkening of teeth, incisal greying, chipping and fracturing, sensitivity, cupping of molars, dentine exposure, painful teeth, pulpal exposure. The causative acidic substances can be either intrinsic or extrinsic in origin. Factors that cause oral exposure to intrinsic acids can involve certain medical conditions causing vomiting and regurgitation. Examples are eating disorders, gastro-esophageal reflux disorder and alcoholism. Extrinsic acid exposure can result from occupational or dietary factors. Examples of people whose work involves the risk of exposure to high levels of dietary or environmental acids are chefs or wine tasters, personnel with acidic drinks at their workstations, industrial workers involved with plating, galvanising, acid pickling, battery manufacture, sanitary cleanser manufacture, munitions manufacture, acidic drinks manufacture, process engraving, crystal glassworks, dyestuffs-container cleaning or enamel manufacture, and sport swimmers in improperly controlled swimming water.

In the majority of cases, however, dental erosion will be related to acids in food and drinks, mainly fruits and fruit juices, fruit teas, soft drinks, wine and cider, acidic confectionery, pickles, salad dressings and vinegar. Raw-food diets with negligible quantities of soft drinks but containing fruit juices or fruit teas have been shown to increase the risk of erosion. Just as in the case of caries, the erosive potential of the drinks and foods alone does not determine whether erosion develops; it does so

**FIGURE 8**

### Examples of dental erosion



**Erosion of the labial surfaces**

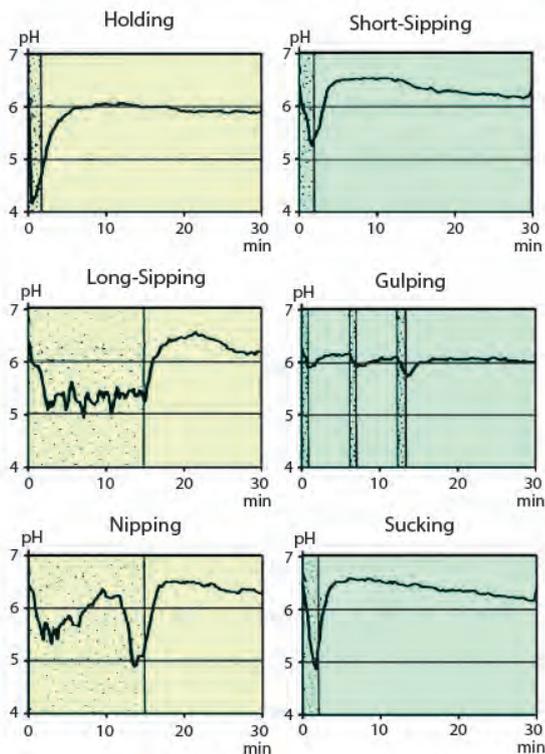


**Erosion of the occlusal surfaces**

together with consumer behaviour. Figure 9 shows how the same type of beverage can have different effects on the pH at the tooth surface, depending on the drinking habit of the subject – either holding, sipping, gulping, nipping or sucking the beverage from a straw. Using a straw does not prevent the beverage from reaching tooth surfaces in the lower posterior region.

FIGURE 9

## Impact of different drinking behaviours on the risk of dental erosion



The *dotted areas* represent the periods of beverage consumption. The *bold lines* show the pH profile at the enamel surface after the consumption of an acidic beverage, comparing different consumption behaviours. Long sipping and nipping exposes the dental enamel to longer-lasting acidic attacks and thus represent more risky consumption behaviour. Sucking from a straw still acidifies the enamel surface. Reprinted with permission from Johansson *et al.*, 2004.

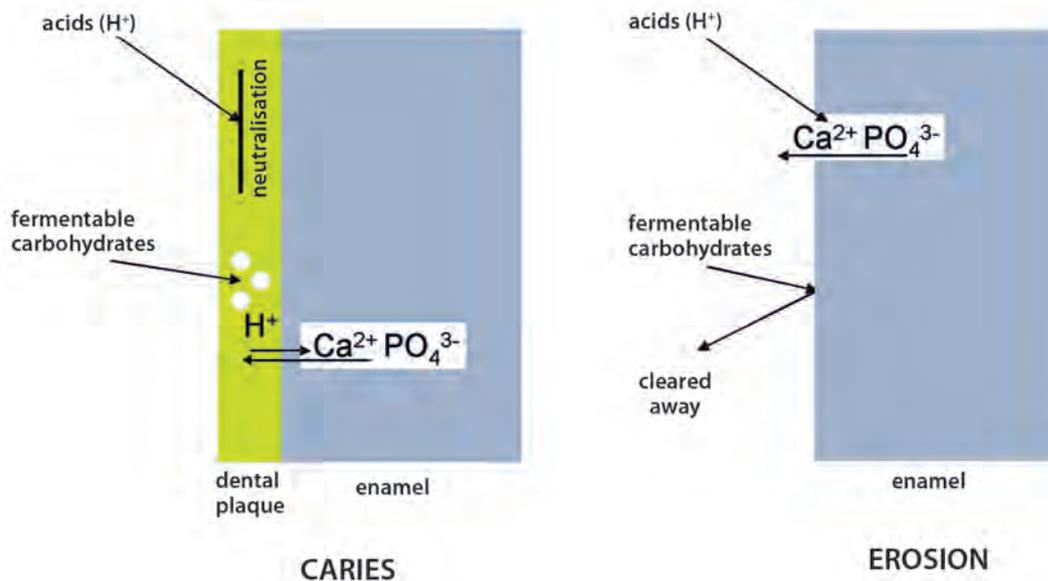
Estimates of the erosiveness of food and drink products are not very precise. Products with a pH  $\geq 5.5$  can be regarded as safe since tooth mineral will not dissolve at these pH values. Products with a pH below 5.5 may cause harm, but erosiveness is not necessarily in proportion to the lower pH. There are several modifying factors inherent in food products and beverages, such as the buffering capacity, the calcium, phosphate, xanthan gum or carboxymethylcellulose content, the organoleptic qualities, the presence of casein-like proteins, and the ability to stick to the tissue. Cooling makes drinks less erosive.

The prevalence of dental erosion seems to be increasing, probably as the result of an increased consumption frequency of acidic foods and drinks. Oral hygiene habits have improved over the last few decades providing plaque-free tooth surfaces, which acids attack more easily than plaque-covered teeth (Figure 10).

After an erosive attack, the tooth surfaces are softened and vulnerable to abrasive forces such as tooth brushing. This is why it is advisable not to brush one's teeth immediately after exposure to a source of acid. Fluoride cannot sufficiently protect tooth enamel because the (micro-)environment of the tooth will remain undersaturated with respect to fluorapatite at the low pH encountered.

FIGURE 10

## Differences between the processes of caries and dental erosion



Caries (*left panel*) may occur when the tooth is covered with dental plaque. Bacteria in the dental plaque ferment fermentable carbohydrates to lactic acid. The acid diffuses into the mineral and causes subsurface demineralisation. When acids are presented to the outer surface of the plaque, buffers present in the plaque will neutralize them; as such the acids will not reach the tooth surface and the tendency to erosion may be reduced.

Dental erosion (*right panel*) occurs when the tooth surface is clean and acids have direct access to the surface. Surface dissolution will occur. When the tooth is not covered with plaque, sugars will be cleared away without causing any damage.

The management of dental erosion depends on whether the erosion is caused by exposure to intrinsic or extrinsic acids. A medical history may reveal conditions that increase exposure to intrinsic acid through frequent vomiting or acid reflux. If the source of acid is of extrinsic origin then the occupational hazard has to be reduced or the diet or the consumption behaviour modified. This may be established by using less erosive products or by

shifting to a less risky consumption pattern. This means reduced consumption frequencies of acidic foods and beverages, especially avoiding sipping throughout the day and consumption at bedtime or during the night when salivary flow is reduced.

## ***PERIODONTAL DISEASES***

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Gingivitis is the mildest form of the periodontal diseases and is reversible. It comprises inflammation of the gingiva induced by the presence of supra-gingival dental plaque. Gingivitis is highly prevalent, but may be restricted to individual sites of the gingival margin and may be temporary until oral hygiene improves.

More advanced stages of periodontal diseases are called periodontitis, which involves loss of connective tissue and bone support. As dental plaque matures to a state associated with periodontitis, the number of gram-negative and anaerobic bacteria increases. Underneath the gum the number of bacteria ranges from  $1 \times 10^3$  in a healthy shallow crevice to more than  $1 \times 10^8$  in a diseased periodontal pocket. A variety of microorganisms can contribute to the development of periodontitis, but the putative pathogens include *Aggregatibacter* (previously: *Actinobacillus*) *actinomycetemcomitans*, *Porphyromonas gingivalis*, *Prevotella intermedia*, *Tannerella forsythensis*, and the spirochaete *Treponema denticola*. Infection of periodontal tissues with these and other organisms is accompanied by the release of bacterial leucotoxins, collagenases, fibrinolysins and other proteases, which aggravate the damage to the gingiva.

Historically, specific overt nutritional deficiencies have been associated with periodontal disease. Vitamin C deficiency leading to scurvy with decreased formation and maintenance of collagen, increased periodontal inflammation, haemorrhage and tooth loss was prevalent amongst sailors in the 17<sup>th</sup> century. But it has to be remembered that the oral hygiene practices in that times were also very poor. A study published in 1963 on 21,559 subjects in eight countries around the world (Alaska, Ecuador, Ethiopia, Chile, Colombia, Lebanon, Thailand

and Vietnam) revealed that periodontitis was mainly associated with lack of oral hygiene (66% of the variance explained) and with age (12%). The effect of the one nutritional influence detected, deficiency of vitamin A, was only 1% of the variance explained. Extensive observational studies in Europe and the USA have failed to show an effect of minor hypovitaminoses on periodontal disease. In a cross-sectional study, a weak but significant association was observed between alcohol consumption and the loss of periodontal support. Prospective studies and studies looking into the mechanisms are needed to confirm the role of alcohol as a risk factor for periodontal disease. All in all, it is generally accepted today that if there is any role of dietary factors in gingivitis and periodontitis, it is only a minor modifying one. Therefore, dietary counselling is not indicated for the prevention of periodontal diseases, but diligent oral hygiene measures are.

Smoking suppresses immune responses, which increases the risk of developing periodontitis. The disease may be more advanced than the clinical appearance suggests. Smoking also strongly decreases the response to periodontal treatment and other oral surgical interventions. Tobacco chewing can also lead to gingivitis and loss of tooth support. Precancerous gingival leukoplakia may develop at the site of quid placement.

### ***Periodontal diseases and systemic health***

Periodontal and general health are associated. Severe periodontal disease with loss of tooth-supporting tissues often occurs when the host response or immune function is impaired. Various systemic diseases such as leukaemia, thrombocytopaenia and leukocyte disorders such as agranulocytosis, cyclic neutropaenia and leukocyte adhesion deficiency may increase the severity of periodontal disease.

A few case-control and cohort studies reported a positive association between common inflammatory periodontal disease and risk of cardiovascular disease. However, the majority of the studies failed to detect a link and questioned the existence of such a relationship because of the possible common effect of cigarette smoking on both diseases. A biological explanation might be found in the fact that periodontitis is associated with raised systemic concentrations of C-reactive protein, fibrinogen and cytokines, all of which are associated with atherosclerosis-induced disease. Standard non-surgical periodontal treatment to reduce periodontal inflammation has been shown to reduce serum inflammatory markers and C-reactive protein, but did not impact on cardiovascular morbidity.

Periodontitis is also a potential complication of diabetes, resulting from increased salivary glucose levels if hyperglycaemia is not well controlled. Therefore diligent oral hygiene measures, thus maintaining good oral health, are especially important for diabetics.

Several case-control and prospective cohort studies have reported an association between poor maternal periodontal health and a risk of preterm birth, low birth-weight and pre-eclampsia, although two large studies in the UK did not find such associations. The research on periodontal disease and chronic diseases is evolving, providing new insights. These possible relationships with general health emphasise the importance of regular oral hygiene measures to minimise the growth of oral microflora and the use of fluorides to decrease demineralisation and to enhance remineralisation, thus maintaining healthy teeth.

## **THE ROLE OF FLUORIDE IN ORAL HEALTH CARE AND CARIES PREVENTION**

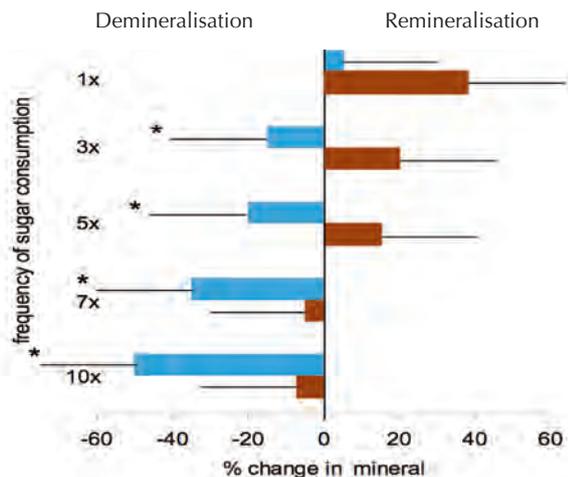
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### *The effects of fluoride*

Fluoride research started in the early 20<sup>th</sup> century (see Box 2). Initially there was emphasis on the pre-eruptive effects of fluoride making erupting teeth less soluble. In later research it became clear that the post-eruptive effects locally in the mouth outweigh the pre-eruptive fluoride effects. The importance of the use of fluoride toothpaste for caries control has been demonstrated in many clinical and *in situ* studies. In the following example of a study *in situ*, the impact of various patterns of consuming 0.5 l of a 12% sugar solution per day over a period of five days was compared in volunteers under two different oral care regimes. The subjects drank the solution either all at once or in 3, 5, 7 or 10 portions a day for a period of five days. During one five-day period they brushed their teeth twice daily with fluoride-containing toothpaste, whereas during another five-day period they brushed with fluoride-free toothpaste. After each period the effects of the different regimes were assessed: demineralisation or remineralisation was measured in enamel pieces that had been built into mandibular removable appliances worn by the volunteer. When subjects had used fluoride toothpaste, some non-statistically significant demineralisation was observed only with regimes of 7 and 10 portions a day (Figure 11). However, in the period with fluoride-free toothpaste, statistically significant demineralisation was observed even when the consumption frequency exceeded once a day. This study demonstrated the importance of brushing with a fluoride containing toothpaste for the control of caries risk. The use of fluoride toothpaste twice a day offsets the effects of frequent consumption of

FIGURE 11

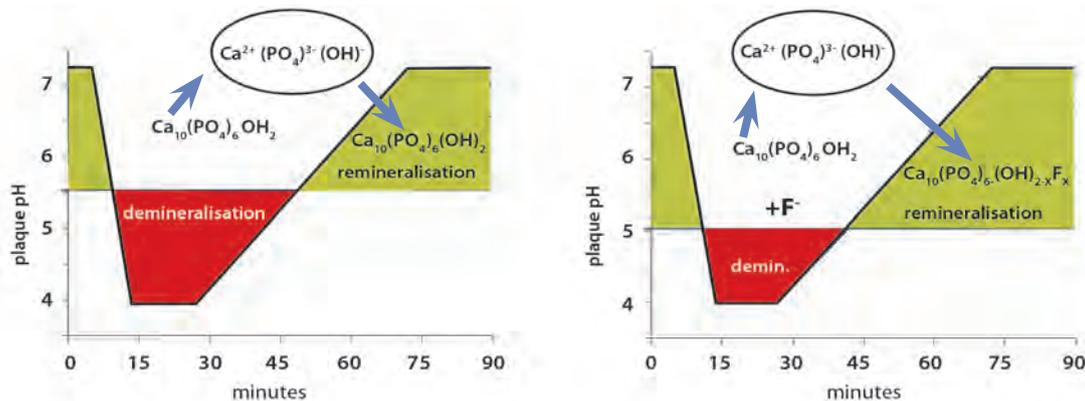
## Caries-protective effect of a fluoride-containing toothpaste



Percentage loss (demineralisation) and gain (remineralisation) in enamel lesions with various frequencies of sugar consumption with the use of a fluoride-containing toothpaste (dark orange) and a fluoride-free toothpaste (blue). The mineral loss with 4 and 10 times of sugar consumption was not statistically significant when the subjects used the fluoride-containing toothpaste. When subjects were using the fluoride-free toothpaste, the mineral loss was significant when the consumption frequency exceeded once a day.

\*Statistically significant demineralisation.  
Adapted from Duggal *et al.*, 2001.

FIGURE 12

Events during a caries attack (pH drop and rise in dental plaque) in the absence of fluoride (*left panel*) and in the presence of fluoride (*right panel*)

In the presence of fluoride the demineralisation period (red) is smaller than in the absence of fluoride as a result of a lower critical pH (pH 5.0 vs. pH 5.5). During remineralisation (green), fluoridated hydroxyapatite is formed, which is less soluble than the hydroxyapatite formed in the absence of fluoride.

fermentable carbohydrates. This has been confirmed in a sample of 1450 pre-school children who participated in the British National Diet and Nutrition Survey. Caries was associated with consumption of sugar confectionery (both in amount and frequency) among children who brushed less than twice a day and not in the children who brushed more frequently (Gibson and Williams, 1999).

## BOX 2

### History of fluoride and dental health

By the second half of the nineteenth century, the use of fluoride was being recommended for hardening teeth against dental decay. The first known fluoride toothpaste dates from 1907. The scientific information used to prove that fluoride protects teeth was gathered in the first half of the twentieth century in observational studies. These studies were actually started to elucidate the cause of mottled enamel. In 1928 McKay wrote: *Mottled enamel is a condition in which enamel is most obviously and unmistakably defective. In fact it is the most poorly calcified enamel of which there is any record in dental literature. If the chief determining factor governing susceptibility to decay is the integrity or perfection of the calcification of enamel, then by all laws of logic this enamel is deprived of the one essential element for its protection... In spite of this, the outstanding fact is that mottled enamel shows no greater susceptibility to the onset of caries than does enamel that may be considered to have been normally or perfectly calcified.*

McKay suspected that something in the drinking water was responsible for mottled enamel. This suspicion was confirmed by decreased prevalence of mottled enamel when cities changed their water supply. When the analytical methods became available, it was confirmed that mottled enamel occurred when the fluoride content of the drinking water was elevated and was absent when the fluoride concentration was low. The recommended fluoride concentration in drinking water was 1 mg/l.

## How does fluoride work?

The main mechanism by which fluoride protects teeth has now been unravelled. Fluoride inhibits demineralisation and speeds up remineralisation. In order to do so effectively, fluoride has to be present in the oral cavity all the time. Fluoride supplementation should achieve this as much as possible. It is not necessary to elevate the fluoride concentration in the oral environment very highly, but it is necessary to ensure its presence in the oral environment over long periods of time.

Inhibition of demineralisation by fluoride is due to the fact that hydroxyapatite in the presence of fluoride behaves as fluoridated hydroxyapatite. Fluoridated hydroxyapatite has a lower pH threshold for demineralisation (about pH 5.0) than normal hydroxyapatite (threshold pH 5.5; see also Figure 6). The promotion of remineralisation is a result of fluoride fitting better in the hydroxyapatite lattice than the  $\text{OH}^-$  ions that it preferentially replaces. Figure 12 illustrates the difference between a caries attack in the absence and in the presence of fluoride and the differences in the relevant critical pH thresholds. The structural formula of hydroxyapatite is given without the impurities of Figure 6, because these impurities are soon lost after caries attacks started.

Fluoride is also known to inhibit the metabolism of oral microorganisms and to affect plaque composition. The concentrations needed for these effects are much higher than those needed for de- and remineralisation. Concentrations of fluoride in the saliva as low as 0.02 mg/l are already beneficial to these processes. Therefore, the interference with the demineralisation process and the promotion of remineralisation are regarded as the predominant ways by which fluoride exerts its cariostatic and anti-caries effects.

After a single application of a fluoride toothpaste or mouthwash fluoride is cleared from the oral cavity in a biphasic manner. Most fluoride is cleared from the mouth within one hour, but there is also a long secondary clearance phase during which the salivary fluoride concentration decreases more slowly. The initial rapid clearance phase is probably the result of fluoride clearance from swallowing due to the continuous salivary flow. The second phase stems from fluoride that was initially retained in oral reservoirs (in particular the soft tissues, which have a relatively large surface area available for retention) and later on released into saliva. Elevated concentrations of fluoride in saliva and plaque fluid were still observed up to 18 hours post-brushing when teeth were being brushed twice daily. So, twice-daily tooth-brushing can be sufficient to ensure the presence of fluoride in the oral cavity for most of the 24-hour period.

### ***The most effective administration of fluoride***

Ideally fluoride is present in the oral cavity 24 hours a day. The best way to achieve this relies as little as possible on the individual's compliance and is affordable. Although water fluoridation perfectly meets these requirements, water is not fluoridated in many countries because opponents regard it as a restraint on their freedom of choice. Therefore, toothpaste is most likely the best choice for administering fluoride to future generations. Many studies have proven the efficacy of different fluoridated dentifrices. There are few problems with compliance since oral hygiene has become more and more socially desirable. Also, fluoridated toothpaste is generally affordable and easily accessible for most people. In addition, tooth brushing combines the application of fluoride with the removal of dental plaque, which not only contributes to caries-prevention but also to the prevention of periodontal diseases.

Until the age of ten, children should be supervised when brushing their teeth, with decreasing intensity as they grow. This is not only to ensure the quality of the brushing procedures but also, for children under five in particular, to avoid the swallowing of an excessive portion of the toothpaste. Up to this age it is advisable to use either fluoride toothpaste with 500–750 ppm fluoride or to use only a pea-size amount of toothpaste with 1000–1500 ppm fluoride. The latter is best achieved when the tube is squeezed perpendicular to the brush head. Toothpastes can contain fluoride in various chemical forms either as sodium fluoride (NaF), sodium monofluorophosphate (Na<sub>2</sub>FPO<sub>3</sub>), amine fluoride, stannous fluoride (SnF<sub>2</sub>) or combinations of these.

The best moment to brush the teeth is when there is time to do it carefully. As saliva flow decreases during sleep, which slows down the rate at which fluoride is washed away, a brushing exercise just before going to bed is expected to be very beneficial. No food, drink or medical syrups should be taken after the last brushing.

### ***Other ways of using fluoride***

There are many other ways of using fluoride in addition to fluoride toothpaste. In general it is best to use these products only upon the advice of a health authority or after consultation with a dental professional.

#### ***Fluoridation of drinking water***

Studies over the last 50 years show that optimally fluoridated domestic water supplies reduce cavities by up to 60 percent, with no significant fluorosis, rendering it a beneficial and inexpensive preventive measure. This method might not be practical where people depend on individual wells for their water supply. Currently more than 360 million people in about 60 countries world wide, including more than 10,000 communities and 145 million

people in the United States alone, benefit from the use of fluoridated water. The optimal concentration is 1 mg/l. In hot climates the population needs to drink more water to stay hydrated, so the fluoride level in drinking water should be lower than that in colder climates.

### ***Fluoridation of salt, milk and beverages***

In countries such as Switzerland, Hungary, France, Germany, Colombia, Costa Rica and Jamaica, the use of fluoridated salt has been shown to reduce caries. Daily consumption of fluoridated milk has also been shown to be protective. Fluoride naturally present in tea can help reduce caries. Some mineral waters contain substantial amounts of fluoride (even >7 mg/l), and can therefore contribute to caries protection. It has been argued that soft and carbonated drinks and fruit juices should be fortified with fluoride. However, in certain countries concerns about excessive ingestion have given rise to legislation prohibiting such initiatives.

### ***Fluoride drops and tablets***

Fluoride drops and tablets are another way to deliver fluoride for caries prevention. Originally these were popular as they were supposed to mimic water fluoridation. The drops and tablets were to be taken several times daily and when swallowed they would give a systemic effect. Both arguments turned out not to be valid. Compliance to the regimen of taking drops and tablets at separate moments during the day was poor in many children, specifically in those children who already showed low compliance with other caries preventive advice. The difficulty in complying with regular doses may explain why studies show such a great variety in effectiveness. Also the contribution of a systemic effect to caries prevention has previously been overestimated. It is now clear that in order to be effective, fluoride has to be present in the oral cavity. Therefore, when fluoride tablets (or drops) are used, they should be allowed to

dissolve slowly in the mouth and be moved about inside the mouth to spread the fluoride around the oral cavity for maximum benefit.

### ***Fluoride mouth rinses***

Fluoride mouth rinses can reduce caries significantly. The combined use of fluoride toothpastes and mouth rinses has a greater effect than either agent used alone.

Despite the effectiveness, the cost-benefit ratio of using fluoride mouth rinses, especially in public health programmes, has been questioned in areas with low caries levels. Mouth rinsing is not recommended for pre-school children because of the risk of swallowing the entire rinse. In public health programmes, the children should always be supervised to ensure that they spit out the rinse solution.

### ***Topical fluoride application in dental surgery***

Three types of topical fluoride applications are used in dental surgery: "paint-on" fluoride solutions, fluoride gels (in a tray) and fluoride varnishes. When applied every six months, the paint-on fluoride solutions and fluoride gels can reduce caries by around 20–25%. The fluoride varnishes seem slightly more effective. For people with severe caries it is advised that the products be applied four times a year. Evidence for a higher efficacy of this increased frequency is lacking. Dentists, hygienists and auxiliaries need to be aware of the hazards of ingesting these products. Therefore, the applications have to be performed carefully. Under specific conditions the dentist may advise high-risk patients, e.g. those undergoing orthodontic treatment or those suffering from severely reduced salivary flow, to apply gels themselves more frequently. The costs of professionally applied fluorides are high. Therefore, these methods may be too costly to be used without selection of caries-active patients; they are more suitable

for use in private practices for individually selected cases rather than as a public health measure.

### ***Slow-release devices for fluoride***

Slow-release devices for fluoride can be cemented on the teeth or can be held in a bracket. The exciting aspect of such a device is that it will continuously (e.g. for a period of one to two years) deliver an appropriate level of fluoride in the oral environment independently of the individuals' compliance with oral hygiene measures. First results are promising, but until now the method is not yet available for routine use in the dental office. Such slow release fluoride devices seem to be ideal for high caries-risk groups that are poorly motivated to improve their oral hygiene habits.

### ***Safety of fluoride***

As many millions of people ingest fluoride by drinking water that naturally contains fluoride, the toxicology of fluoride is well known. For the products used daily in caries prevention, the prescribed amounts are chosen to give the best protection while avoiding health risks even when the products are unintentionally swallowed. Care has to be taken with children under the age of five as they may not effectively spit or rinse their mouths after the use of a fluoride product. If they swallow large amounts over a period of time from toothpaste use, fluorosis might develop. Therefore, it is advisable that the parents supervise the child's brushing and help with it. The use of fluoride supplements is seldom indicated in children under the age of six, except on professional advice.

Fluorosis is a relatively rare condition that might occur when fluoride is ingested at high levels during tooth development. For the front teeth, the period of risk ends around the fifth birthday. In many countries the fifth or sixth birthday is therefore a turning point in the

fluoride advice given by dental professionals, allowing higher fluoride dosages to be used. The use of fluoride supplements was found to be a greater risk factor for fluorosis than the use of fluoride toothpaste.

The appearance of fluorosis ranges from very mild to severe, although severe cases are extremely rare. Very mild fluorosis, the most common form, is hard to detect even for dentists. In fact, when present, it often gives the teeth a pleasing, bright-white look. Mild fluorosis is a surface problem that can easily be treated if necessary. Recently, a study in Ireland, England, Finland, Iceland, the Netherlands, Greece and Portugal showed a low prevalence of mild and severe fluorosis: 0–4%.

## DIETARY FACTORS

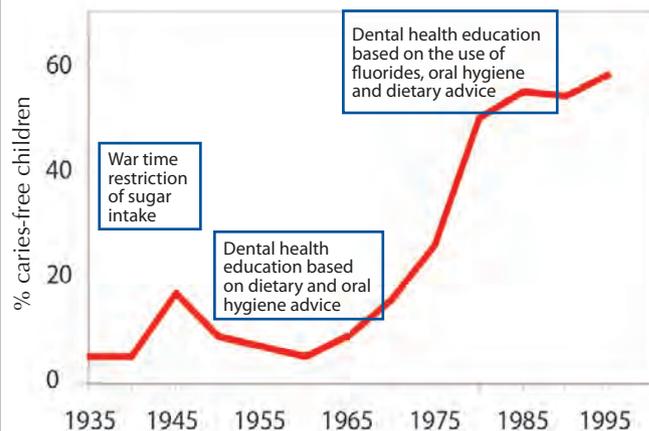
Caries data from shortly after World War II show an increase in the percentage of caries-free children as a result of the wartime diets. Shortly thereafter, in 1954, the Vipeholm studies, named after the institution that conducted the experiments, showed that sugar eaten in the form of large toffees between meals resulted in severe damage to the teeth (Gustaffson *et al.*, 1954). However, consumption of sugar even at high levels (300 g of sucrose, provided in solution) did not significantly increase caries levels when taken in four portions a day with meals, and none between meals. Subjects of the Vipeholm study were unable to perform appropriate oral hygiene and when the study was carried out fluoride was not available to them in any form.

The finding of the Vipeholm study that it was snacking in-between meals that caused most damage to teeth became the most important observation for preventive dentistry at that time. Reduction of the amount of in-between snacking became logically the most important tool in caries prevention. At that time the benefits of topical fluorides were not yet appreciated by the dental profession and oral hygiene was not believed to be effective in reducing caries, because teeth generally were not very well brushed. However, most efforts to limit sugar consumption were not very successful. In the Netherlands the number of children free from caries did not increase in the period when preventive dentistry was aimed at reducing sugar intake (Figure 13). The steep increase in the number of children free from caries occurred when fluoride toothpaste became available. During this period, sugar consumption was continuously high.

In the late 1970s, before fluoride was widely used, Sreebny (1982) compared caries prevalence among 12-year-old

FIGURE 13

### Prevalence of caries-free children in the Netherlands



There was substantial increase in caries-free children only when dental health education was directed to the use of fluorides.

children in 47 nations with the availability of sugar per capita. Of the 47 nations, 21 had sucrose supplies below 18 kg per person per year, 19 had supplies of 18–44 kg per person per year and seven nations had supplies of over 44 kg per person per year. He observed that at sucrose supplies lower than 18 kg per person per year dental caries was less prevalent. The mean number of decayed, missing or filled permanent teeth (DMFT) of the 21 countries with a sugar supply below 18 kg per capita per year was  $1.2 \pm 0.6$ . For nine of the 19 countries, with an average sugar supply between 18 and 44 kg per person per year, the mean DMFT was  $2 \pm 0.7$ , while for the other ten of these countries mean DMFT was  $4 \pm 0.9$ . In the seven countries where sugar supply exceeded 44 kg per capita per year, the mean DMFT was  $8 \pm 2.4$ . However, the relationship between sugar availability and caries was less clearly seen approximately 10 years later in a comparable study in 61 developing countries

and 29 industrialised countries (Woodward and Walker, 1994). In the developing countries approximately 26% of the variation in the caries data was explained by sugar availability. In the industrialized countries less than 1% was explained, suggesting that, where fluoride is available, dietary restrictions may be of lesser importance in caries prevention. The same conclusion was reached at the *Fédération Dentaire Internationale* (FDI) consensus meeting at the *Second World Conference on Oral Health Promotion* in 1999 in London (Consensus Statement on Diet, 2000).

### ***The role of sugars and carbohydrates***

In 1969, Newbrun declared sucrose to be the arch-criminal in dental caries (Newbrun, 1969). The reasons for this statement were that sucrose is the most widely used sugar, that oral bacteria easily ferment sucrose, and that sucrose promotes plaque growth by the formation of extracellular polysaccharides in dental plaque. In a survey conducted in 2001, 18 European experts in preventive dentistry still believed in this paradigm, while 19 other European experts did not (van Loveren *et al.*, 2004). This outcome may reflect our current understanding that there is a relationship between the acidogenic and cariogenic potential of food and the presence of sugars, but their amount or concentration is not as important as we previously believed. For example, the fall in plaque pH after eating a bar of chocolate containing approximately 15 g of sucrose is smaller than that following the consumption of a boiled sweet with 3 g of sucrose. So the sugar content of a product is not a predictor of its cariogenicity. Apart from measuring the sugar content of a product, systems have been proposed to categorise foods into good and bad foods for teeth. Such systems have focussed on: (a) the acid response of the dental plaque, e.g. measured by plaque pH-telemetry or by touch electrodes in volunteers and (b) the determination of the cariogenicity of a food in a rat caries model (Curzon and Hefferren, 2001). However,

these experimental systems do not take account of the most important factor: the local environment modifies the potential cariogenicity of the food. The amount of acid formed in dental plaque, as a response to consumption of a food containing fermentable carbohydrates, is significantly related to the age and thickness of the dental plaque and to oral clearance. Whether acids cause demineralisation also depends on the presence of fluoride. So food may be cariogenic for one individual (having old, thick dental plaque, not using any fluoride) but not necessarily for another person with appropriate oral hygiene.

### ***Are all sugars equally cariogenic?***

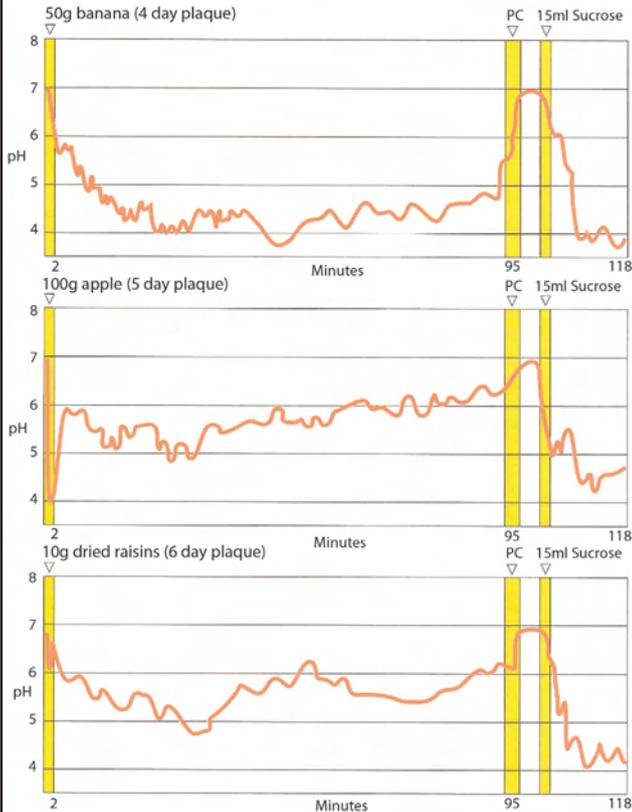
Of the major food sugars, glucose, fructose and maltose are similar (or nearly similar) to sucrose in terms of acidogenicity. It makes no difference whether the sugar occurs naturally or is added. Lactose, however, seems to have a lower acidogenicity. Under specific conditions, lactose may still be cariogenic (see Section 6.5 on early childhood caries). People often think that fructose is a better sugar for dental health. In the so-called Turku sugar studies, performed in the pre-fluoride era, individuals received a diet in which all sucrose was replaced by fructose. These individuals did not develop less caries in the first experimental year but did so in the second experimental year. There was no reasonable explanation for the discrepant findings. It is generally agreed that it cannot be concluded from the Turku study that substitution of sucrose by fructose is a worthwhile caries-preventive measure.

### ***Can fruits or their juices contribute to caries?***

Some fruit juices do carry the claim “no added sugars”. Such a claim may be misleading, since these juices are made from fruits that often contain high amounts of

FIGURE 14

### Intra-oral plaque pH telemetric recordings after consumption of bananas, apples and raisins



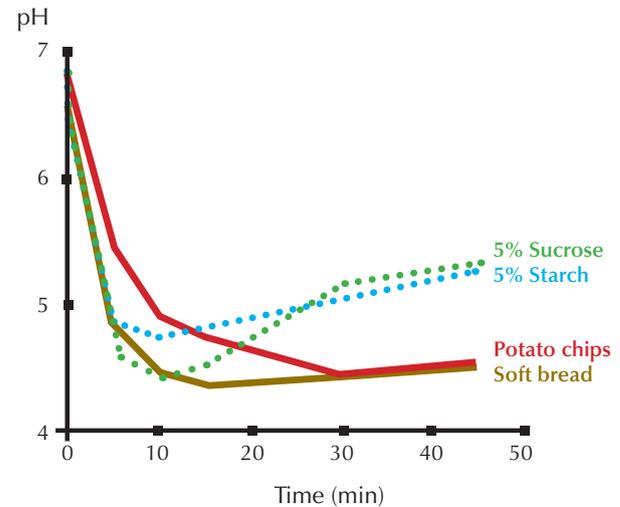
Reprinted and modified with permission from Imfeld, 1983.

PC = Paraffin chewing

naturally occurring sugars. For example, apples and grapes can contain as much as 15% sugars. The Advisory Committee on Medical Aspects of Food Policy to the UK government (COMA, 1991) proposed that intrinsic sugars (those naturally integrated into the cellular structure of a

FIGURE 15

### Acidogenic responses in dental plaque to sucrose, potato crisps, starch and soft bread



Reprinted with permission from Lingström *et al.*, 1993.

food) and extrinsic sugars (those that are free in the food or added to it) should be differentiated. The former were regarded as non-cariogenic, whilst the latter were not. So COMA implied that an apple is non-cariogenic, as it would essentially contain intrinsic sugars, whereas apple juice derived from the same apple would be considered cariogenic by the virtue of the fact that the sugars were now in an extrinsic form. It must be admitted that there are favourable factors in an apple that apple juice lacks: fresh apples are fibrous and crisp which make them self-cleansing, much of the sugar is likely to remain in the bolus until it is swallowed, and the malic acid in the apple is a good stimulant for neutralizing-saliva. Nevertheless, plaque pH-telemetry has shown that, when chewed, apples increase cariogenic acidity levels in plaque.

Rat experiments have also shown that apples may cause caries. Experiments with bananas, grapes and raisins showed that these fruits also possess cariogenic potential (Figure 14). The conclusion from these experiments is that the acidogenicity and cariogenicity of intrinsic and extrinsic sugars is not necessarily different. So fresh fruit can contribute to caries activity, if consumed frequently. This conclusion probably also applies to the erosiveness of fruits.

### ***Can starchy foods contribute to caries?***

Starches are not direct substrates for bacterial fermentation; however, hydrolysis to maltose, isomaltose and glucose, which are substrates, takes place in the mouth. Both salivary and bacterial amylases can accomplish this. Chewing (e.g. of crackers and potato chips) prolongs glucose clearance due to the formation of the intermediary starch degradation products maltotriose and maltose (Linke and Birkenfeld, 1999). Acid formation can start surprisingly quickly after starchy food has interacted with the dental plaque. The acidogenicity of white bread, whole wheat bread, cooked spaghetti, cooked long grain rice and many other starch products with and without added sugar has been tested. The minimum pH in dental plaque measured with indwelling electrodes showed that none of these products were significantly different from 10% sucrose solution. Acid formation in plaque after chewing whole-wheat or soft bread or potato chips may even be more intense and last longer than after the intake of sucrose (Figure 15). Therefore, there is no doubt that, as already observed by Miller in 1889, starches are acidogenic in the mouth. Many animal experiments show that cooked starch is cariogenic. All these experiments indicate a cariogenic potential of processed food starches, depending on the innate make-up of starches and the widely varying conditions of food processing. Studies in humans, however, do not demonstrate unequivocally the

actual cariogenicity of starches. A review on this topic in the year 2000 concluded that, in a sugar-containing diet, starches may increase the cariogenicity as a co-cariogen, for instance by increasing the stickiness of the product (Lingström *et al.*, 2000). Starchy foods in retentive fissures and interdental spaces may give rise to considerable amounts of acid. Therefore, the cariogenic potential of these fermentable carbohydrates must be considered. In addition, in persons with hyposalivation and in those at risk from root caries, the increased cariogenic potential of starches has been recognised.

### ***Early-childhood caries***

Sometimes parents are not aware of the caries risk in very young children. They do not realise that new teeth erupting into the mouth do need cleaning. Additionally the use of bottle-feeding might be quite prevalent and frequent. Sugar may be added into the bottle or the bottle may even be filled with diluted syrups or lemonades. In addition, it is rather common for the comforters to be

**FIGURE 16**

**Early childhood caries (ECC)**



**TABLE 2**

**Commonly used sugar substitutes (polyols, novel disaccharides and intense sweeteners) for the manufacture of tooth-friendly confectionery and for use in oral care products. All substances are non-cariogenic; some of the intense sweeteners can interfere with microbial fermentation.**

Category	Structure	Examples
Polyols (hydrogenated carbohydrates)	Hydrogenated monosaccharides	Sorbitol, mannitol, xylitol, erythritol
	Hydrogenated disaccharides	Isomalt, lactitol, maltitol
	Hydrogenated oligosaccharides	Maltitol syrups, hydrogenated starch hydrolysates
Bulking agent	Polymer	Polydextrose
Novel disaccharides	Disaccharide (isomers to sucrose)	Leucrose, isomaltulose (Palatinose™)
Noncaloric, intense sweeteners		Acesulfame K, Aspartame Cyclamate, Saccharin, Sucralose

sweetened with agents such as sugar or honey. These exposures to fermentable carbohydrates may immediately be followed by a nap or, even worse, the child may be allowed to sleep with the bottle still in its mouth. This poses a serious risk to the teeth because the salivary flow is reduced during sleep and carbohydrates are retained in the mouth for prolonged periods of time. Also other protective mechanisms of saliva are reduced during sleep. This behaviour results in early-childhood caries (ECC), a rampant form of caries, which can affect both the front teeth as well as molars (Figure 16).

Early-childhood caries has been observed in babies breast-fed over longer periods, which is common in African countries. The frequent intake of milk might have allowed the proliferation of high numbers of mutans streptococci and *Lactobacilli* in dental plaque,

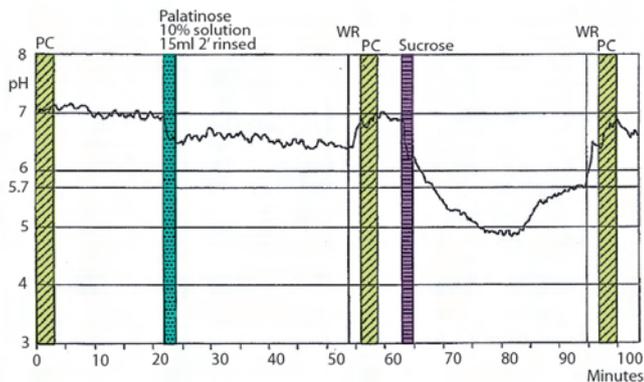
modifying the cariogenicity of human milk (König, 2000). The same may develop when children have access *ad libitum* to nursing bottles with milk. Also, if children are allowed to sleep with the bottle still in their mouth or are fed frequently during the night, milk can be retained in the mouth for prolonged periods allowing lactose to be fermented by oral bacteria to produce acid, thereby causing dental caries.

### ***Are there foods that are safe for teeth?***

Foods that do not contain sugars or any other forms of fermentable carbohydrates and are not inherently acidic are safe for teeth. Foods containing fermentable carbohydrates or that are inherently acidic have a cariogenicity or erosiveness that is difficult to estimate. An indication of the relative cariogenicity and erosiveness of

FIGURE 17

### Intra-oral plaque pH telemetric recordings after consumption of Palatinose™

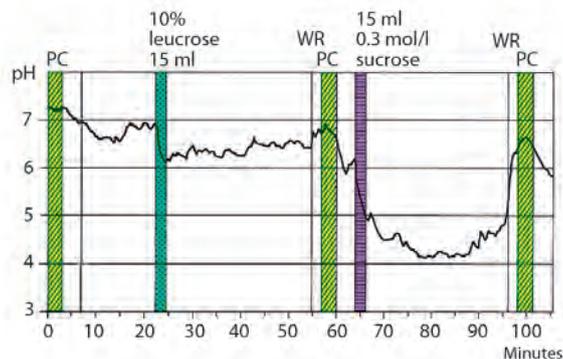


Source: Imfeld, Centre for Dentistry, Stomatology and Orthodontia, University of Zurich (personal communication).

PC = Paraffin chewing WR = Water rinsing

FIGURE 18

### Intra-oral plaque pH telemetric recordings after consumption of leucrose



Reprinted with permission from Ziesenitz *et al.*, 1989.

PC = Paraffin chewing WR = Water rinsing

foods can be obtained through estimation *in vitro* of sugar and acid concentrations, buffer capacity and pH, and by incubation of dissolved products with representative bacterial strains of the oral flora, animal experiments and measurements of acid formation in dental plaque. None of these methods, however, predicts the long-term clinical effect in humans. It is generally agreed that foods can be regarded as safe for teeth when they do not lower the plaque pH or the pH on a clean tooth surface below pH 5.7 during the 30 minutes after consumption. These are mainly sugar-free sweets and confectionery and chewing gum formulated with sugar substitutes and intense sweeteners. The relevant alternative sweeteners, sugar replacers and bulking agents currently used are described in Table 2. All these sugar alcohols and novel disaccharides have been tested and classified as hypo- or non-acidogenic. The relative sweetness of the sugar alcohols compared to sucrose varies from 0.5 to 1. Meanwhile, novel disaccharides of very low acidogenicity and with good gastrointestinal tolerance for manufacturing tooth-friendly sweets have been developed (e.g. Palatinose™ and leucrose) and are on the market (e.g. Palatinose™) (Figures 17 and 18).

Intense sweeteners are not substrates for oral micro-organisms and can thus be classified as non-cariogenic. As a result of their intense sweetness, only very small amounts are needed. Therefore, these sweeteners are mainly used to sweeten beverages such as soft drinks, coffee and tea. Although the light or diet soft drinks may not cause caries, they may contain high amounts of acids that could cause tooth erosion.

For all sugar alcohols it is advised to limit daily intake to 30–40 g. Children with free access to products sweetened with these sugar alcohols may easily ingest an amount in excess of the advised maximum limit. This limit is unlikely

to be reached by using sugar-free chewing gums, as sticks or as pellets. Therefore, regular use of these sugar-free chewing gums fits well in caries prevention.

The rise in dental erosion has increased attention for ways to decrease the erosiveness of certain products. In healthy-volunteer studies the inclusion of calcium in drinks in combination with a pH of between 3.7 and 4.0 reduced enamel loss dramatically (Figure 19). Several acidic beverages with substantially reduced erosive potential have been marketed in Europe in recent years.

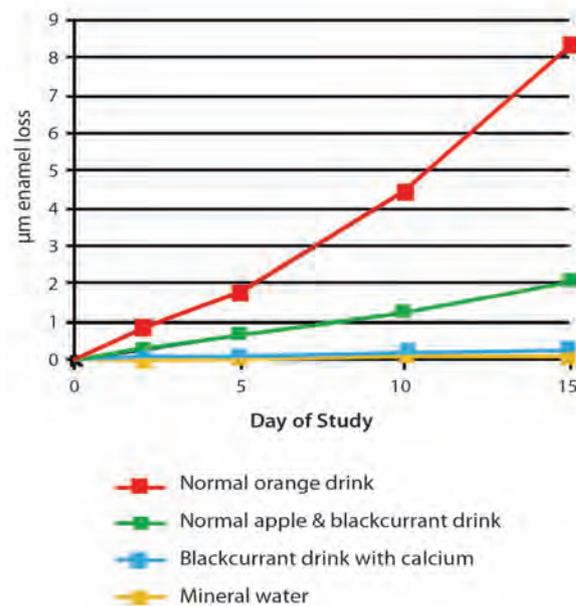
### Protective components in food

When a food promotes salivary flow it may be less harmful, as a result of the cleansing and buffering effects of the saliva. If such food does not contain any fermentable carbohydrates, remineralisation may occur. This effect may be enhanced if the food has additional anticariogenic properties. Milk, for instance, contains the anticariogenic ingredients calcium, phosphate, casein and lipids. Dairy products were recognised to be non-cariogenic in the late 1950s. Casein seems to be protective, but the large amount required and the adverse organoleptic effect involved precludes its use in a food or toothpaste. By tryptic digestion, a caseinophosphopeptide (CPP) can be produced that forms colloidal complexes with calcium and phosphate in solution (CPP-ACP). These complexes inhibit demineralisation and promote remineralisation *in vitro*, in rat caries studies and *in situ*.

Many extracts such as from propolis or liquorice and polyphenols from cocoa and tea may interfere and inhibit processes leading to plaque, gingivitis and malodour. Other substances may reduce the solubility of the mineral. An active constituent of tea is epigallocatechin gallate (EGCG), which inhibits glycosyltransferase and various

FIGURE 19

#### Evaluation of a low erosive blackcurrant juice drink



The erosive responses to drinks were evaluated in human enamel samples retained *in situ* on upper removable appliances. Drinks (normal orange and apple & blackcurrant drink products, modified blackcurrant juice drink with calcium and mineral water) were 250 ml volumes consumed four times per day for 15 days. Enamel loss was measured by profilometry. Erosion by the normal drinks was highly significantly greater than by the modified drink. Erosion by the modified blackcurrant drink only became statistically significantly different to water by day 15. Based on data from Hughes *et al.*, 1999.

salivary proteins and enzymes. However, it is unclear whether the components are sufficiently liberated during consumption to have the desired protective effects; they should be a matter for future study.

## PREVENTION OF CARIES

### *Sensible advice*

It is not realistic to believe that people can totally abstain from consuming cariogenic foods in the interest of better dental health. However, with a diligent practice of oral hygiene (Box 3), a diet rich in whole grains, fruits and vegetables, and with a limited number of in-between meals, most people can enjoy foods traditionally considered “bad” without much risk to their dental health. The following recommendations should be useful:

- First and foremost, people should be encouraged to brush their teeth twice a day with a fluoride containing toothpaste.
- The number of eating and drinking occasions in a day should not exceed seven.
- Food and drinks containing sugars or acids should not be used after the last brushing exercise, at bedtime or during the night.
- Intakes can be combined to reduce the frequency of intakes and thereby the caries and erosion risk.

Dietary counselling, when necessary, should be realistic and positive, and agreement from the patient should be obtained on where consumption can reasonably be curtailed. It should be based on good dietary practice rather than focus on good foods versus bad foods. For the consumption of soft drinks, fruit juices and energy sports beverages, drinking rather than sipping should be recommended. In individuals with tooth erosion, reduction of contact time between the acid food and the teeth should be planned. In order to prevent early-childhood caries and erosion, sugary and acidic foods and drinks should never be used on a dummy as a comforter or served from a nursing bottle.

## BOX 3

### Caries Preventive Measures

- Plaque removal
  - brushing
  - flossing
  - interdental brushes and other aids
  - rinsing with antimicrobials
- Topical fluorides:
  - Individuals
    - toothpaste
    - fluoride mouth-rinses
  - Professionals
    - fluoride solutions
    - fluoride gels
    - fluoride varnishes
    - slow release fluoride
- Blocking plaque build up:
  - antimicrobial agents
- Fissure sealants
- Eating and drinking behaviour:
  - not more than 7 drinking and eating occasions a day
  - avoid constant nibbling and sipping
  - avoid bedtime use of carbohydrate-rich foods and beverages
  - avoid baby-bottles as pacifiers at bedtime (except when used with plain water)
- Regular dental check ups

## What role can the professional play?

The most important preventive assignment is to motivate the patient in good self-care. Dental professionals should give realistic and achievable advice and instructions tailored to an individual's life-style, whilst incorporating all the basic elements of a good preventive regime (Box 3). This will encourage compliance. In addition, there are a few preventive treatments that may be provided when necessary. The most frequent professionally provided preventive treatments are pits and fissure sealants and topical applications of concentrated fluoride solutions, gels or varnishes carried out at intervals determined by the individual's caries risk.

In some cases self-care, in addition to the preventive support provided by the dental professional, is not sufficient for maintaining a caries-free dentition. However, recent research shows little additional benefit from the delivery of intensified programmes. Children who were regarded as being at high risk of developing caries were randomised into two groups. Half were offered an intensive prevention programme (counselling, F-varnish applications, F-lozenges, sealants, chlorhexidine) and the other half were provided the same basic prevention as given to low-risk children (counselling, one F-varnish application per year). After three years there was only a small difference in caries increment between the children receiving the intensified and those receiving the basic programme. One reason for these results may be that the dental professionals, in their enthusiasm to deliver a preventive programme, overlooked patient motivation to take responsibility for their own dental health (Hausen *et al.*, 2000; Hausen *et al.*, 2007).

When attempting to motivate patients, a clear goal should be set and all strategies should be well-defined, for instance:

- Education of parents and children in understanding dental caries as a localised disease
- Intensive training in home-based plaque control
- Early professional non-operative intervention.

The preventive programmes should be individualised by setting an individual recall interval based on clear criteria such as motivation, cooperation, skills, caries-risk and activity of the patients. An example of this is the dental health system in Nexö, Denmark. By applying such strategy, the children in Nexö experienced remarkably fewer caries than other children in Denmark. Transferring this approach to Moscow achieved a tremendous improvement in dental health in Russian children.

## Pit and fissure sealants

The pits and fissures of the teeth may be so small that even the smallest bristle of the toothbrush cannot provide effective cleaning at the depth of the fissures. A way to tackle this problem and to prevent caries from developing in these areas is to seal them with a non-viscous filling material (Figure 20). This prevents colonization of fissures by dental plaque. If dental plaque or even a small caries lesion has been enclosed the method is still effective, since the availability of carbohydrates to the bacteria that are sealed in these areas is effectively prevented. The relative caries risk reduction of resin-based sealants on permanent first molars has recently been estimated to be 33% (relative risk = 0.67; CI = 0.55–0.83). The effect depended on retention of the sealant. The evidence is incomplete for permanent secondary molars, premolars and primary molars.

**FIGURE 20**

**Example of pit and fissure sealants**



Source: Ch. Penning, Department of Cariology Endodontology  
Pedodontlogy ACTA, The Netherlands.

## ***CLOSING REMARKS***

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The last few decades have shown improvements in oral health for many, especially in the developed world. The percentage of children free from caries has increased. Improved oral hygiene with the use of fluoride toothpaste has prevented, retarded and arrested caries lesions. However, the improvements in dental health are less impressive in deprived and lower socio-economic groups in society. It is important to reach the high caries-risk groups with preventive messages, encouraging appropriate oral hygiene in ways that are both acceptable and affordable. For most of us a sensible diet, diligent use of fluoride toothpaste and occasional use of other methods such as mouth rinses are the most important measures that will maintain a caries-free or minimally affected dentition into the future.

### ***Community-based programmes***

One of the challenges for preventive dentistry is to deliver preventive care to the children of families who are not particularly interested in dental health. The parents will not follow preventive advice and they will not visit the dentist. These are usually families who have similar attitudes to other health related issues, not just to dental health. Community-based programmes may reach these children. Programmes like water fluoridation certainly do, but also programmes aimed at increasing the dental awareness and interest can be incorporated whenever the families meet health professionals. Health or life-style lessons, including taking care of your teeth, could be given in school. In many countries such programmes run successfully, but in very locally restricted areas. Health authorities should recognise these challenges and find innovative ways of reaching the populations who have benefited least from advances in preventive dentistry, which have provided so much benefit to large sections of our populations.

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