

A Clinical Overview of Sugarfree Gum in Oral Health





A Clinical Overview of Sugarfree Gum in Oral Health

Contents

Page 4 **Sugarfree Chewing Gum & Oral Health**

An updated review of the positive effects of chewing sugarfree gum on oral health

by Michael Edgar, DDSc, PhD, FDS RCS (Eng).

Page 12 **Saliva**

Saliva - its role in maintaining oral health and preventing dental disease

by Simon Roland, BDS (Lond), LDS RCS (Eng).

Saliva, foods and dental caries

by Michael Edgar, DDSc, PhD, FDS RCS (Eng).

Xerostomia: care and management

by Martin Thornhill, MBBS, BDS (Lond), MSc, PhD, FDS RCS (Edin), FFD RCS (Irel).

Page 18 **Sugarfree gum - The Clinical Support**

Abstracts from key papers on the benefits of chewing sugarfree gum

Page 60 **References**

A review of the positive effects of chewing sugarfree gum on oral health.

Michael Edgar, DDSc, PhD, FDS, RCS (Eng) is Emeritus Professor of Dental Science at the University of Liverpool.

Introduction

Chewing gum is a unique food because it is chewed for a prolonged period (usually around 20 min), while at the same time it contributes relatively few calories. Its effects on the oral tissues – whether harmful or beneficial – have therefore been studied for many years.



Sugared chewing gum may contribute to the cariogenicity of the diet. Chewing sucrose gum causes a moderate fall in plaque pH^{1,2} and some clinical studies have demonstrated an increase in caries incidence with the use of sugared gum, compared with controls who did not chew gum^{3,4} although others did not demonstrate a significant increase in caries in subjects using sucrose gum.^{5,7}

The development of sugarfree gum provided the possibility of a non-cariogenic alternative to sugared gum. Chewing sugarfree gum results in a rise in plaque pH, in contrast to the pH fall observed with sugared gum. This is due to the stimulation of the flow of saliva, with the resulting increase in level of bicarbonate and thus alkalinity. At the same time the plaque microflora do not produce significant amounts of acid.^{1,2,8-10}

Caries incidence is less in chewers of sugarfree compared with sugared gum^{11,12} in agreement with the plaque pH results.

Additionally, other studies have shown that chewing sugarfree gum leads to fewer caries *compared to non-chewing controls*. This implies that the reduction in caries is not due merely to the lack of sucrose from gum in the diet, but that sugarfree products actually inhibit caries' activity due to dietary carbohydrate.¹²⁻¹⁷

Anti-caries mechanisms of sugarfree gum

Many of these beneficial actions of sugarfree gum are due to the activation of the protective effects of saliva by chewing gum, in view of the prolonged stimulation of salivation by gum chewing.

Effects of saliva stimulation

a) Salivary stimulation by chewing gum

When gum is chewed by healthy subjects, the flow of saliva increases from a resting value of 0.4-0.5ml/min, to approximately 5-6ml/min, falling after about 5min to around 2ml/min, and slowly thereafter to 1.2-1.5ml/min at 20min.¹⁸ No significant differences are observed between sugared and sugarfree gum; however, with unflavoured gum base the initial high flow rates are not seen, and the peak flow is around 2ml/min.

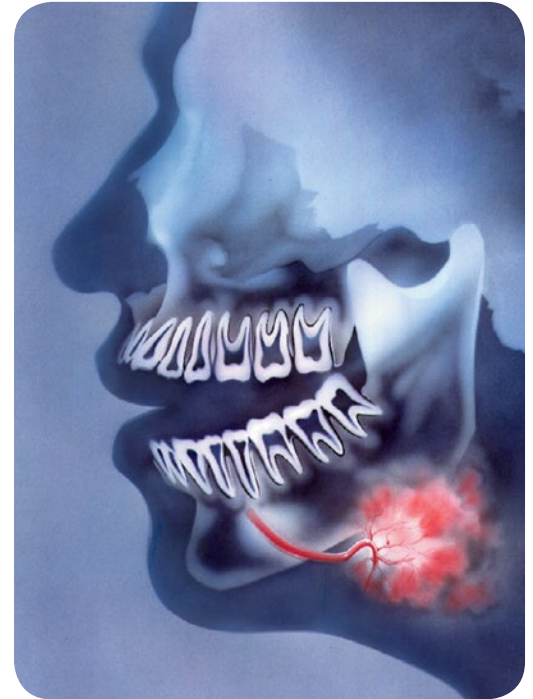
The effect of stimulation is to increase the concentration of bicarbonate in the saliva entering the mouth. This bicarbonate raises the pH of the saliva, and greatly increases its buffering power; the saliva is therefore much more effective in neutralising and buffering food acids and acids arising in plaque from the fermentation of carbohydrate. At the same time, the phosphate of saliva changes as a result of the rise in pH, so that a higher proportion of it is in the form of PO_4^{3-} . The calcium content of saliva rises as well.

b) Salivary protective effects

These changes in the composition of stimulated saliva lead to a greater ability to prevent a fall in pH and a greater tendency to favour hydroxyapatite crystal growth. In addition, the greater volume and rate of flow of stimulated saliva results in an increased ability to clear sugars and acids from around the teeth. These three properties of saliva are related to the caries susceptibility of the individual and are all enhanced by salivary stimulation.

The action of stimulated saliva is most important during the plaque acid attack during the 20-30 min after a cariogenic food intake. However, with most foods, salivary stimulation ceases shortly after swallowing, and salivary composition returns to normal within about 5 min, and so the protective effects are not mobilised when most needed.

In order to enhance salivary protection during the caries attack, a stimulant is needed which is not itself cariogenic.



The major salivary glands source their fluid from the circulating blood, process it and secrete it into the mouth

Consumption of cheese¹⁹ and peanuts²⁰ after sugar intakes showed a dramatic reversal of the plaque pH falls observed with sugar alone. When cheese was administered after a standard cariogenic diet in a programmed feeding experiment in laboratory rats, the development of caries was greatly reduced and the size of the salivary glands increased, presumably due to salivary stimulation by the cheese.²¹

Advice to eat cheese or peanuts after meals and snacks to reduce caries would however lead to an unacceptable increase in dietary fat. The effect of saliva stimulation on plaque pH can be achieved by non-food stimuli such as paraffin wax.²²

To reduce the acid challenge to the teeth sugarfree gum should be chewed as soon as possible after eating fermentable carbohydrate snacks

c) Effects of gum chewing on plaque pH

Sugarfree chewing gum is a much more practical and acceptable stimulus for consumption after carbohydrate foods, and brings no undue calories. The observation by Hein et al.²³ of a 'large and sustained rise in plaque pH' when gum was chewed after a sugar has been thoroughly confirmed in many studies conducted in respected laboratories around the world.²⁴⁻³²

Sugarfree gum chewing for two weeks led to an increase in resting salivary flow rate and pH, and a smaller plaque acid response to sucrose.³³ However, in another study, no difference in salivary flow or plaque acidogenicity was observed after 25 days use of sugarfree gum.³⁴

d) Effects on remineralisation

The concentrations of ions which make up the lattice structure of hydroxyapatite (Ca^{2+} , PO_4^{3-} , OH^-) are higher in stimulated than in unstimulated saliva, thus, stimulated saliva is a more effective medium for remineralising enamel crystals damaged by initial caries attack. In an in situ caries test by Leach et al.³⁵ subjects chewed sorbitol gum for 20 min after meals and snacks (5 times daily). The gain or loss of mineral content of human enamel slabs bearing artificial lesions and mounted intra-orally for three weeks, was then measured and compared with similar periods without gum chewing.

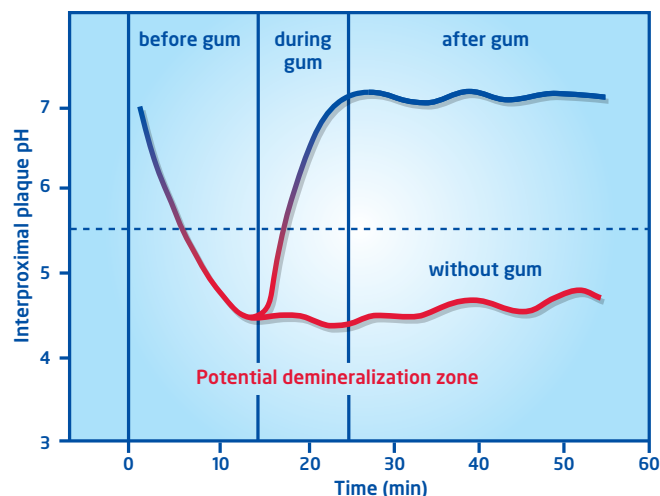
Remineralisation of the enamel lesions occurred both with and without gum, but with gum the remineralisation was approximately doubled. A similar experiment³⁶ showed that, even with sucrose gum, remineralisation was significant with a 30 min chewing period but not after 20 min. These two reports were broadly confirmed by Creanor et al.³⁷ and are consistent with a reduction in enamel demineralisation (measured as iodide penetration) by chewing sorbitol gum found by Kashket et al.³⁸ Also consistent is the finding of Steinberg et al.³⁹ that six-week use of sugarfree gums resulted in an increase in plaque calcium, and a reduction in plaque index, compared with no gum.



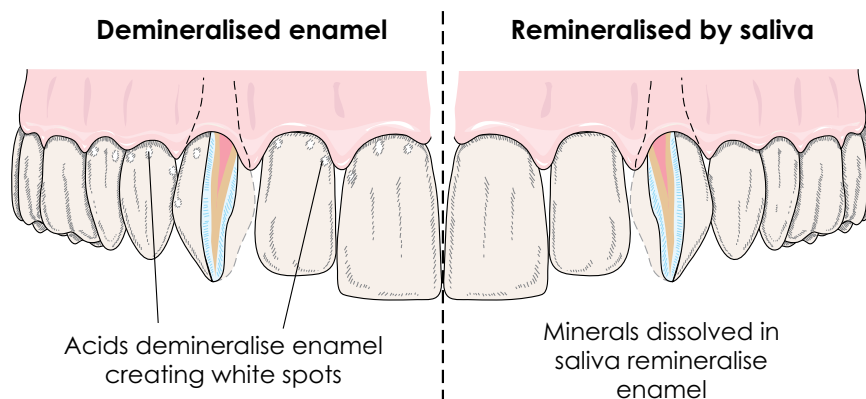
Remineralisation *in vivo* is generally considered to be a slow process⁴⁰ and it is perhaps surprising that significant remineralisation occurred within 3 weeks. A possible explanation is that stimulation of saliva after eating a cariogenic food increases the remineralising effect, as the fall in plaque pH could dissolve CaF_2 deposits on the teeth and free diffusion channels in the enamel to allow inward movement of ions from saliva.

These model experiments imply that gum use can help prevent decay by tilting the equilibrium towards remineralisation and away from demineralisation during the acid attack.

Remineralisation of enamel lesions, and plaque pH raising effects, have also been demonstrated with sucrose gum,^{36,37} consistent with the stimulation of saliva. However, the remineralising and pH raising effects were smaller than with sugarfree gum, required greater subject compliance, and were dependent upon the use of a fluoridated dentifrice; with a non-fluoride dentifrice the same subjects showed an increased demineralisation on chewing sucrose gum.⁴¹ It would not therefore be prudent to recommend the use of sugared gum to patients, but it would be reasonable to recommend that if they refused to switch to sugarfree products, they could minimise any possible cariogenic effect by gum use after meals.



Stimulated saliva is a more effective medium for remineralising enamel crystals



e) Other effects of sugarfree gum

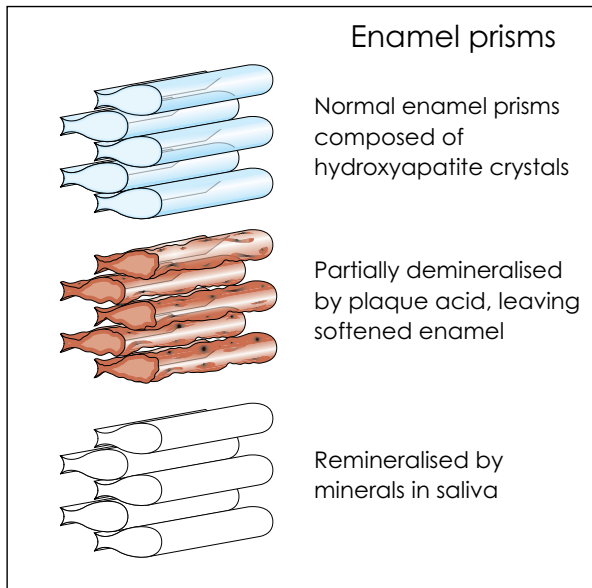
The use of sugarfree gum has been associated with a reduction in the quantity and development of plaque,^{15,42-44} and a reduction in the acid-forming ability of plaque.⁴⁴ These plaque-reducing effects seem marked when the gum is sweetened with xylitol. This sweetener is a sugar alcohol derived from the pentose sugar xylose. It has a sweetness equal to that of sucrose and is not fermented by plaque bacteria to form acid. Moreover, *in vitro* it has bacteriostatic properties; on being taken up by the bacteria it forms an inhibitory phosphorylated intermediate.⁴⁵⁻⁴⁷

Gums sweetened with xylitol or xylitol/sorbitol have, in general, given rise to greater reductions in caries than those with sorbitol alone. More recently, direct comparisons of the effects of sorbitol and xylitol have demonstrated the superiority of xylitol gum.⁴⁸⁻⁴⁹ The effect of xylitol gum persists even after the gum administration ceases.^{50,51} The post-eruptive caries attack rate reaches a plateau at a lower value, caries increment is less and the cost of fillings is reduced in the decade after the start of a three-year trial of xylitol gum; the effect was greatest in teeth erupting during the administration of the gum.⁵² In a recent study, the development of caries

was reduced during the 5 years after gum administration ceased, in children who had received xylitol or xylitol/sorbitol gum (compared with no gum). Sorbitol gum users experienced fewer new caries attacks during the subsequent 5 years, but this reduction was not statistically significant. Again, teeth erupting during the gum period showed the greatest reductions.⁵³

Chewing xylitol gum has been found to reduce the amount of, and the numbers of mutans streptococci in, plaque⁵⁴ and saliva.³⁴ Chewing xylitol gum reduced the pH response of plaque to sucrose,⁵⁵ although other work did show an effect of sorbitol.³³

Chewing sugarfree gum can help prevent tooth decay by tilting the equilibrium towards remineralisation during an acid attack



In view of reports that xylitol may favour remineralisation,⁵⁶⁻⁵⁸ an *in situ* experiment was carried out to compare sorbitol gum with a xylitol/sorbitol gum, similar to that used in the clinical experiment of Kandelman and Gagnon.¹⁵ No difference in remineralising potential was observed;⁵⁹ further work is necessary to decide on this question.

Gum has been used as a vehicle for additives such as fluoride,⁶⁰ dicalcium phosphate¹¹⁻⁶¹ and sodium trimetaphosphate⁶² to reduce the potential cariogenicity of sucrose in gum. In addition, silicates⁶³ and chlorhexidine acetate⁶⁴ have been added to reduce plaque and gingivitis, pancreatic enzymes⁶⁵ have been added for calculus inhibition, and penicillin⁶⁶ for the treatment of acute necrotising ulcerative gingivitis (ANUG). Chewing gum itself may contribute to plaque reduction and some studies have shown beneficial effects on oral hygiene, calculus and/or gingivitis.^{67,68}

How Gum Can Make a Difference

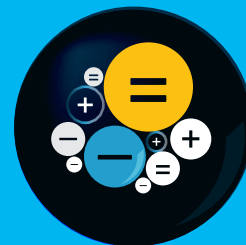
Chewing sugar-free gum can help protect teeth in a number of ways:



Stimulate Saliva Flow



Reduce Plaque



Neutralize Acids



Remineralize Enamel



Clean the Mouth of Food Debris



Relieve Dry Mouth Discomfort*

*Dawes C, Macpherson LM. Effects of nine different chewing gums and lozenges on salivary flow rate and pH. Caries Res. 1992; 26(3): 176-82

Conclusions

The results discussed here and in other reviews^{69,70} provide convincing evidence for the oral health benefits of sugarfree chewing gum, particularly in the control of caries. It is likely that the effects of gum chewing are in addition to those of fluoride, since remineralisation occurs with both preventive agents.

Xylitol or xylitol/sorbitol mixtures as sweeteners in gum have in general proved more effective in caries prevention than sorbitol alone. The concentration of xylitol may be related to the caries reduction: however, it is of interest that there was no difference between the effect of 15% xylitol and 65% xylitol in the study of Kandelman and Gagnon.¹⁵ In the Belize study,⁴⁸ the effects of gums with 15% and 65% xylitol on the development of new carious surfaces over 40 months were only barely significantly different (0.6 and -0.8, compared with 3.8 for sorbitol alone). These effects can be attributed to salivary remineralisation as well as a reduction in plaque cariogenicity.⁴⁹



Although most clinical studies with xylitol gum did not control the timing of gum use, it is likely from the laboratory evidence that it is most effective when chewed after meals and snacks. Controlled administration of sorbitol gum after eating^{16,17} gave reductions in caries of up to 40 per cent in caries increment over two years.

Further research is of course required but, hitherto, the evidence suggests that the use of sugarfree gum (especially after meals and snacks, and preferably containing xylitol) constitutes an important aspect of the advice which can be given to patients to help them prevent caries. The possibility of broadening the oral health benefits of sugarfree chewing gum (e.g. anti-gingivitis effects, low-level fluorides, increased remineralising action, whitening) could prove a significant field for development.

The use of sugarfree gum constitutes an important aspect of oral care advice which can be given to patients



Chewing sugarfree
gum helps contribute
to plaque reduction

Saliva – its role in maintaining oral health and preventing dental disease

Simon M Roland, BDS (Lond) LDSRCS (Eng). Dental Advisor to Oral Healthcare in Action, General Dental Practitioner St John's Wood London.

Saliva has major attributes as follows:

1) Protection to the oral and peri-oral tissues

- **Lubrication** with mucins and glycoproteins
- **Antimicrobial** and *cleansing* activity degrading bacterial cell walls and inhibiting growth
- **Buffering** acid production with bicarbonate and controlling plaque pH
- **Remineralisation** of enamel with calcium and phosphates.

2) Facilitating eating and speech

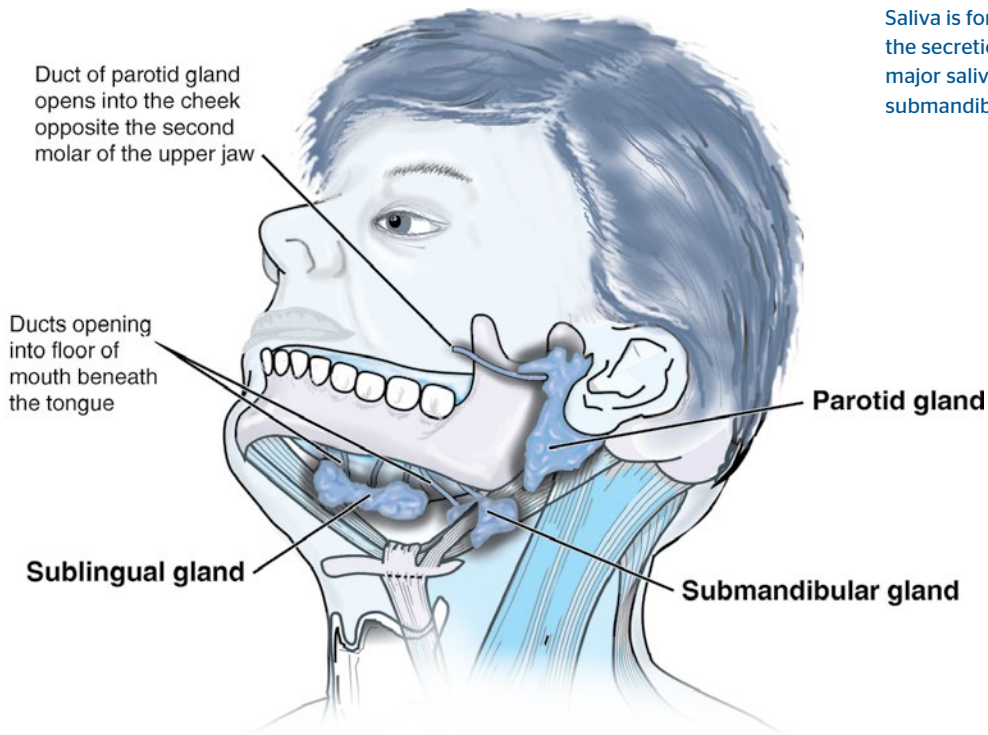
- **Food preparation**, enhancing chewing, the clearing of residues and swallowing
- **Digestion**, initiation with enzymes
- **Enhancing taste**
- **Enabling speech** by lubricating motor functions.

3) Uses in diagnostic testing

- **Bacterial, yeast and viral** counts indicating caries activity and altered immune responses as well as many diagnostic tests for oral and systemic diseases
- **Hormonal balance** to identify steroids, sex hormones.

Saliva consists of 99% water with the remaining 1% for the most part organic molecules (glycoproteins, lipids) and electrolytes (calcium, phosphates).

Saliva is normally secreted continuously at about 600ml per day but can be stimulated by masticatory or gustatory activity. Chewing sugarfree gum can increase the initial salivary flow rate by a factor of 10.¹ It has also been shown that this stimulated saliva is more effective in its ability to buffer and remineralise.²



Saliva is formed primarily from the secretions of the three paired major salivary glands, the parotid, submandibular and sublingual

Three pairs of major glands, the parotid, the sublingual and the submandibular are responsible for the majority of salivary production as well as minor glands distributed around the oral cavity. They are controlled by the autonomic nervous system. Reduction in the amount of saliva is most commonly caused by medication side effects, systemic disease or pathologic changes in the salivary glands. The true value of saliva can best be seen when it is absent. Therefore, patients seriously deficient in saliva with Sjögren's syndrome, or suffering from the effects of irradiation for head and neck cancer, have difficulty in eating and swallowing, increased tooth decay, mouth ulceration and infections. Medications and drugs may cause xerostomia as can various psychogenic, neurologic and hormonal disorders.

Dry mouth can be extremely distressing and with ageing, an increasing proportion of the population is affected.

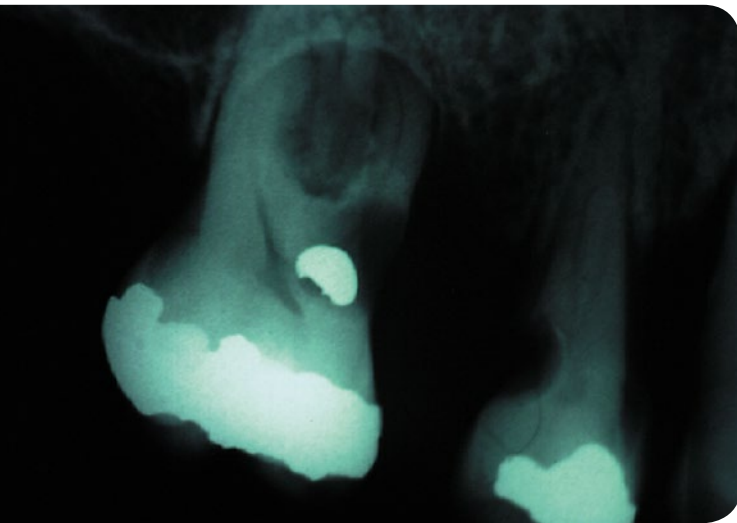
Saliva, foods and dental caries

Michael Edgar, DDSc, PhD, FDS, RCS (Eng) is Emeritus Professor of Dental Science at the University of Liverpool.


The pH of dental plaque is a key factor in the balance between acid demineralisation of the teeth and the remineralisation of the initial caries lesion. Plaque pH falls each time acids accumulate in the plaque due to bacterial acid production following the consumption of fermentable carbohydrates – mainly sugars – in foods and drinks.

On the other hand, the plaque pH rises when the acids are washed out and neutralised by saliva, which contains the important buffer, bicarbonate. The pH also rises when the plaque bacteria either metabolise the acids, or produce alkali, such as ammonia, from nitrogenous compounds found in foods and saliva.

Dental caries is the result of an imbalance between demineralisation and remineralisation



Besides its role in the control of plaque pH, saliva has another function of major importance in caries – remineralising effects. Saliva is 'supersaturated' with the ions which make up the mineral content of the teeth (calcium, phosphate and hydroxyl ions) when the pH is above a 'critical' value, about 5.5. Below this value (e.g. after an intake of sugar) the saliva and plaque is unsaturated, and the tooth dissolves. Above this value, the calcium and phosphate ions in saliva start to repair the damaged mineral crystals in the enamel – the process of remineralisation.



Stimulation of saliva flow results in an increase in the washing out of acids and sugars, as well as an increase in the amount and concentration of bicarbonate buffers and remineralising ions

Dental caries is the result of an imbalance between demineralisation and remineralisation. In health, loss of mineral is balanced by the reparative mechanisms of saliva. This equilibrium can be depicted chemically by the equation:



The solid crystals of the tooth mineral, called hydroxyapatite, dissolve to release calcium, phosphate and hydroxyl ions, only if the latter are below saturation concentrations. If above saturation, the reaction will tend to move to the left and any damaged crystals will be repaired by the acquisition of ions from solution.

Stimulation of saliva flow results in an increase in the washing out of acids (and sugars), and also an increase in the amount and concentration of bicarbonate buffers and of remineralising ions.

It has been shown that salivary stimulation by chewing sugarfree gum after the consumption of sugary foods not only prevents the fall in plaque pH normally seen, but also results in an increased remineralising effect in previously demineralised enamel.^{1,2} It is likely that this salivary stimulation is responsible for the reduction in caries seen in clinical studies of the use of sugarfree gum.^{3,4}

Xerostomia: care and management

Martin Thornhill, MBBS, BDS, MSc, PhD, SDS RCS, FFD RCS, Professor of Clinical Oral Science at St Bartholemews and Royal London School of Medicine and Dentistry.

Xerostomia is the subjective feeling of dryness throughout the mouth. Studies conducted on outpatients and in the general population show that xerostomia affects about one in four people.¹ Salivary flow rate patterns demonstrate both daily and seasonal variation with peaks in mid afternoon and higher flow rates in the spring than in the autumn. During sleep, saliva flow rate is minimal.²



People who complain of dry mouth do not necessarily have a very low flow rate, conversely, those with a low unstimulated flow do not always complain of dry mouth. It is therefore of greater significance to establish whether or not flow rate has changed adversely in a particular individual.³

For general dental practitioners, a reduction in the amount of saliva produced can lead to a variety of clinical problems which include:

- Unexpected increase in caries rate – particularly cervical, cusp regions and lower anterior teeth
- Difficulty in wearing dentures – particularly upper dentures
- Difficulty in swallowing, especially dry foods e.g. biscuits
- Swelling of salivary glands
- Burning discomfort in mouth, soreness and cracking of tongue
- Ulceration of oral mucosa
- An increased susceptibility to oral infections.

Reduced salivary flow rate is due to hypofunction of the salivary glands. This may be reversible, due to anxiety, acute infection, dehydration or the effects of some drugs. There are also some permanent causes of xerostomia such as congenital abnormalities, Sjögren's syndrome, HIV/AIDS and the result of irradiation.

Xerostomia is most commonly associated with the use of xerogenic drugs. More than 400 preparations induce salivary gland hypofunction, including tricyclic antidepressants, antihistamines, certain antihypertensives and drugs with sympathomimetic actions (e.g. some bronchodilators).

The management of xerostomia involves the use of both saliva substitutes and saliva stimulants. Patients may also require referral to a dietitian. Patients with little or no responsive salivary gland tissue will need saliva substitutes. A properly balanced artificial saliva should be of neutral pH and contain electrolytes, including fluoride, to correspond to the composition of saliva. Of the proprietary preparations available Luborant® is licensed for any condition giving rise to a dry mouth.

Gustatory stimuli, such as sugarless sweets containing citric and malic acid, chemically induce saliva production. Care must be taken that the acidic content does not result in the dissolution of tooth enamel.

Controlled studies have shown that pilocarpine is an effective stimulus to saliva production.^{4,5,6} Side effects, mainly the result of generalised parasympathetic stimulation, are the most common reason to discontinue treatment.

There have been a number of studies that have shown that chewing gum increases salivary flow in patients with xerostomia of varying aetiology.^{7,8,9} In some xerostomic patients, the initial stimulated salivary flow rate while chewing sugarfree gum is seven times greater than the unstimulated flow rate.¹⁰ Chewing sugarfree gum has been shown to be one of the most preferred treatments for xerostomia.¹¹

Clinical Abstracts

Benefits of Saliva

Edgar WM, O'Mullane, D.M (2004) *Saliva and Oral Health 3rd Edition. British Dental Journal*, London, Great Britain

A two-day international consensus meeting in 1989 on saliva was the foundation of the original edition entitled *Saliva and Dental Health*, published in 1990. This 140-page A5 second edition, published six years later, updates, and broadens the original edition. The emphasis is not just on dental health but on 'oral' health. Chapters have been radically revised, and the contents confirmed by a two-day meeting in which the international authors discussed their papers. The editors believe the new edition represents a consensus of opinion of world experts.

Chapters in the new edition include: anatomy and physiology of the salivary glands; mechanisms of secretion by salivary glands; factors influencing salivary flow rate and composition; xerostomia (diagnosis, management and clinical complications); clearance of substances from the oral cavity; saliva and the control of plaque pH; salivary influences on the oral microflora; the functions of salivary proteins; and the role of saliva in mineral equilibrium.

Many chapters include a section called 'clinical highlights'. These sections clearly state the clinical implications of the evidence discussed and offer direct advice to dental practitioners. For instance, in the clinical highlights section within the chapter 'Saliva and the control of plaque pH', one conclusion is, 'Continued stimulation of saliva following a meal or snack, e.g. by chewing sugar-free gum, raises plaque pH and thus reduces demineralisation and favours remineralisation'.

Working Group 10 of The Commission on Oral Health Research and Epidemiology (CORE) FDI (1992) *Saliva: Its Role in Health and Disease, International Dental Journal* **42(4)**: 287-304

The Working Group 10 of the Commission on Oral Health, Research and Epidemiology (CORE), Fédération Dentaire Internationale wrote this report in 1991. The Group was chaired by Professor Leo M Sreebny. The ten sections in the supplement are: the fluids of the oral cavity and the factors influencing their composition; the composition of saliva; specific functions of salivary constituents; the collection of saliva; the flow rate of saliva; the diagnosis and causes of (two sections) xerostomia and salivary gland hypofunction; the protective action of saliva against caries; the assay of caries-related constituents of saliva; and the treatment of salivary hypofunction and xerostomia.

The report reviews research on the ability of saliva to prevent dental caries and on the treatment of xerostomia and salivary hypofunction. It highlights important advances in knowledge that have implications for practice. For instance, studies have shown that buffering is enhanced and plaque acidity is neutralized rapidly when sugar-free gum is chewed after eating acidogenic

foods. Furthermore, sugar-free gum increases the remineralising power of the saliva, with the potential to tip the balance against demineralisation. The treatment of salivary hypofunction, whether stimulatory or relieving the symptoms of oral dryness, is determined by a number of factors, including the patient's medical status and the availability of specific therapies. The working party believes that the practitioner must also be able to manage the complications of salivary hypofunction: increased caries, oral candidiasis, altered oral function and pain. The various local stimulatory, systemic stimulatory and symptomatic therapies are reviewed.

[Edgar WM \(1990\) Saliva and Dental Health: Clinical Implications of Saliva: Report of a Consensus Meeting. *Journal of British Dental Association*. 169\(3/4\)](#)

Professor WM Edgar gives a general report of a 2-day international consensus meeting on the clinical implications of saliva. The meeting was held in Ireland in 1989. It took the format of a series of 10 presentations reviewing the role of saliva, and was followed by discussions. The consensus group aimed to identify the future impact in dental practice of the increasing evidence for the protective effects of saliva on oral health. The group concluded that as therapies to treat compromised saliva flow become available, the need to screen patients at risk of compromised flow will become an essential element of dental practice. Evaluation of salivary function could become an everyday part of patient evaluation. The optimisation of the benefits of such a natural protective and defensive mechanism, saliva, is likely to prove attractive to the increasingly sophisticated and ecologically conscious general public, as well as to the dental profession. Diagnostic, preventive and counselling services to exploit the natural benefits of saliva to help prevent dental disease would be a way forward for the profession. The evidence reviewed at the meeting in detail included: protective effects of salivary electrolytes and proteins; saliva and plaque; the problems of dry mouth (xerostomia); and increasing residual salivary gland function.

[Peretz B, Sarnat H, Moss SJ \(1990\) Caries Protective Aspect of Saliva and Enamel, *The New York State Dental Journal*, 56\(1\): 25-27](#)

The authors review the protective aspects of saliva and enamel. Dental caries occur as a localized enamel lesion. The initiation and rate of progress is dependent on environmental conditions. Saliva is a liquid rich in minerals and proteins and supersaturated with respect to calcium and phosphate ions. Saliva surrounds and bathes the tooth. When enamel which has been cleaned is wet by saliva, specific proteins from the saliva are absorbed to the tooth surface and form a delicate membrane referred to as the salivary pellicle or the acquired pellicle. Oral bacteria that come in contact with the pellicle adhere to this membrane and form the foundation on which the dental plaque first develops.



Saliva may be considered as being similar to enamel but in a liquid phase. Saliva, the mechanics of remineralization and demineralization, the acquired pellicle and the enamel surface all act to maintain the status quo, resisting mineral loss from the enamel. Saliva is the first line of protection. The saliva-acquired pellicle is the second line of protection. The surface enamel is the third line of protection.

Edgar WM, O'Mullane D, Dawes C (1989) *Saliva Symposium*, Ashford, Ireland. Collection of speeches and/or topic's by the following authors:

- Dr C Dawes, Winnipeg, Canada - Presenter
- Dr Bruce Baum, Bethesda, Maryland - Presenter
- Dr Denis O'Mullane, Cork, Ireland - Editor
- Dr JS Van der Hoeven, Nijmegen, Holland - Presenter
- Dr I Kleinberg, Sony Brook, New York - Participant
- Dr Donald Hay, Boston, Massachusetts - Presenter
- Dr Bill Bowen, Rochester, New York - Chairman
- Dr Dorothy Geddes, Glasgow, Scotland - Presenter
- Dr MJ Larsen, Aarhus, Denmark - Presenter
- Dr George Dibdin, Bristol, England - Participant
- Dr Ernest Newburn, San Francisco, California - Chairman
- Dr Norman Fleming, Winnipeg, Canada - Presenter
- Dr WM Edgar, Liverpool, England - Editor
- Dr John Featherstone, Rochester, New York - Presenter
- Dr Larry Tabak, Rochester, New York - Presenter
- Dr F Lagerlof, Huddinge, Sweden - Presenter

Izutsu, KT (1989) *Physiological Aspects of Salivary Gland Function. Compendium of Continuing Education in Dentistry. (Supplement 13) S450-456*

Salivary gland secretion is regulated through the activity of the sympathetic and parasympathetic nerves to the gland, and their neurotransmitters. The physiological function of the normally functioning salivary gland was described by Professor Izutsu in this article. He said that the clinical value of comparing normal and pathological models of salivary gland function lies in their ability to suggest the sites of pathological defects for the diseases that affect salivary gland function and explain the mechanism of the disease. In particular, he reviews studies of cystic fibrosis and ageing. He shows how studies of salivary gland function in these diseases have helped our understanding of the different components of the cell-signaling system, and have helped work out more precise sites of pathological defects that produce pathological changes in cell and gland function. This approach should lead to a better understanding of the pathophysiology of other diseases that affect salivary gland function.

Fox PC (1989) Saliva Composition and Its Importance in Dental Health. *Compendium of Continuing Education in Dentistry*. (Supplement 13): S450-456

The importance of saliva composition lies in the ways saliva and its elements support oral functions. Both communication and alimentation may be compromised when salivation is abnormal. The patient with low salivary secretions has difficulty speaking, chewing, forming a food bolus, and swallowing. In addition, there is a rapid and substantial increase in caries and mucosal infection. Taste and soft tissue complaints are also more frequent. As well as discussing the importance of saliva in dental health, the assessment of salivary gland function by different methods was reviewed in this article. It was stressed that meaningful results will be obtained only if great care is given to the collection method. Salochemistry was briefly commented on, with the advice given that dentists should consider salivary chemistries supportive of a specific diagnosis and not diagnostic in themselves. The technique of salivary scintigraphy was described. This provides a means of identifying patients who retain portions of functional parenchyma and may be responsive to treatments to increase salivary output. Sialography was also described as being 'invaluable in demonstrating gland masses or sialoliths', but having several technical disadvantages and the possibility of reactions to the contrast material. Ultrasound, computerized tomography and magnetic resonance imaging were also discussed briefly. The author felt that these techniques to image the gland required further study before pronouncement on their relative utility could be made. The author concludes by listing nine previously published questions to determine individuals with salivary gland hypofunction.



Sreebny LM (1989) Salivary Flow in Health and Disease. *Compendium of Continuing Education in Dentistry* (Supplement 13): **S461-S469**

The source and supply of saliva in health, and its composition when 'resting' or 'stimulated' is reviewed in this article. The mean resting flow rate for whole saliva is about 0.4 mL/min, and the paraffin-stimulated whole saliva is about 2 mL/min. About 5% of the population shows stimulated flow rates of less than 0.7 mL/min. Virtually no saliva is produced during sleep. About 2 hours a day might be assumed to be spent eating, producing stimulated saliva, and 14 hours a day producing resting saliva. The total daily flow of whole saliva is about 600-700 mL. (This contradicts the amount of 1-1.5 L/day cited in many physiology textbooks.) About 50-60% of the daily output is derived from basal saliva.

Saliva flow in disease is also reviewed in this article. Saliva is an indicator of whole mouth dryness and diseases and conditions associated with it. However, its value for the diagnosis of specific diseases is limited. Dry mouth (xerostomia) is a subjective indicator of salivary gland function. Flow rates should be determined to confirm salivary gland hypofunction. The factors that affect salivary flow were discussed in healthy individuals: ageing, circadian rhythm circannual rhythm, body position, light, smoking, olfactory stimulation and previous gustatory stimulation.

Reductions in saliva flow are usually caused by salivary gland hypofunction and xerostomia. The causes include drugs, irradiation, organic diseases; psychogenic disease; and decreased mastication. A variety of methods to increase the flow of saliva are briefly discussed, including citric-acid substances, an electronic stimulator and pilocarpine.

Ferguson MM. (1989) Management of Patients with Xerostomia, *Compendium of Continuing Education in Dentistry*. (Supplement 13): S470-S475

In this review the dentist is advised to consider each patient as an individual, when they present with a history of persistent dry mouth or xerostomia. The disease aetiology should be established by investigation, a diagnosis reached and specific treatment plans can then be developed. Possible aetiologies will include drug therapy, radiotherapy, inflammatory exocrinopathy (Sjögren's syndrome), mouth-breathing, dehydration (e.g. through alcohol abuse), and neurosis. Less common aetiologies include sarcoid, involving the salivary glands. History and investigation are of crucial importance to the diagnosis. Flow rate, sialochemistry, gland biopsy for morphological changes, sialography, and scintiscanning are all relevant. The dentist should also consider investigations for diabetes mellitus and the appropriate haematology and serology for the connective tissue disorders. An aggressive active program of management is advocated for patients with an established diagnosis of persistent dry mouth. The clinician starts by considering routine oral hygiene measures, diet, salivary substitutes, agents that promote saliva flow (sialagogues, chewing gum for example), dental treatment, and psychological treatment. Also discussed were prevention, diet, salivary substitutes and agents that promote saliva flow, including pilocarpine. Dental and psychological treatment may also be relevant.

Mandel IM. (1989) Impact of Saliva on Dental Caries. *Compendium of Continuing Education in Dentistry* (Supplement 13): S476-S481

The widespread use of fluorides in various forms has resulted in a profound change in the pattern of caries. Targeted groups are now: high-risk children; adults with multiple restorations; and older populations with gingival recession and increased susceptibility to root caries. People with xerostomia experience a heightened prevalence of decay and an abnormal distribution of lesions. Their clinical plight dramatically demonstrates the importance of saliva in the protection of the mouth. The salivary protective mechanisms against caries include:

- An ability to clear bacteria via mechanical, immunological, and nonimmunological means; direct antibacterial activity provided by lysozyme, lactoferrin, salivary peroxidase, histatins and their interactions; regulation of oral and plaque pH through constituent buffering systems, through generating ammonia by providing substrates for decarboxylation to form amines, and through ammonia formation from ureas and arginine peptides.
- Maintenance of tooth integrity by post-eruptive maturation, carbohydrate clearance, pellicle formation (a diffusion barrier) and regulation of the ionic environment to encourage remineralisation. People who are naturally resistant to caries (regardless of fluorides and diet) have enhanced salivary protective mechanisms that include increased ability to produce base in plaque, a more effective means of bacterial aggregation and decreased pellicle permeability.

Sreebny LM. (1989) Summation: Conference on the Endogenous Benefits of Saliva in Oral Health. *Compendium of Continuing Education in Dentistry*. (Supplement 13): **S487-S488**

Professor Sreebny presents a summation of a symposium that consisted of presentations from five broad topics on the benefits of saliva: Impact of Saliva on Dental Caries, by Irwin D Mandel. Salivary Flow in Health and Disease by Leo M Sreebny; Physiological Aspects of Salivary Gland Function by Kenneth T Izutsu; Saliva Composition and its Importance in Dental Health by Philip C Fox; and Management of Patients with Xerostomia by Martin M Ferguson.

He prefaces this with an argument that saliva has been neglected. He asserts, 'saliva is a neglected secretion and xerostomia is a neglected symptom'. He argues that, because 'there is no money in it,' for the general practitioner, and because many dentists believe that symptoms associated with salivary gland dysfunction are trivial, the area has been neglected. Professor Sreebny says, 'Forgotten is the significant effect of saliva on the health of the oral tissues and on the quality of life'. He then demonstrates the benefits of saliva in his summary of the other speakers' talks.

Birkhed D, Edwardsson S, Wikesjö U, Ahlén ML, Ainamo J (1983) Effect of 4 days Consumption of Chewing Gum Containing Sorbitol or a Mixture of Sorbitol and Xylitol on Dental Plaque and Saliva. *Caries Research* **17: 76-88**

By aid of a full crossover double-blind design, the effect of a 4-day period with: (1) ten pieces per day or a sorbitol-containing chewing gum, (2) ten pieces per day of a chewing gum containing a mixture of sorbitol and xylitol (sorbitol/xylitol), and (3) no chewing gum, in combination with a controlled diet and no oral hygiene, was studied on dental plaque and saliva in 24 dental students. Sorbitol-containing chewing gum did not enhance the amount (wet weight) compared to no chewing gum. The sorbitol/xylitol period resulted in significantly less plaque in comparison with the two other periods ($p < 0.001$). No clearcut differences were found between the three treatments regarding protein content and peroxidase activity in whole-saliva centrifugates or



total carbohydrate, reducing sugar, protein contents and 'sucrase' activity in soluble plaque extracts. Directly after these 4-day periods, the effect of 3-hour consumption of ten pieces of a sucrose-containing chewing gum was studied. Protein content and peroxidase activity in whole-saliva samples were about the same after this 3-hour period, while plaque wet weight, protein total carbohydrate and reducing sugar content and 'sucrase' activity in soluble plaque extracts increased significantly ($p < 0.001$). The changes in the plaque material after the 3-hour period were about the same and independent of the former treatment with sorbitol- and sorbitol/xylitol-containing chewing gum or no chewing gum.

Jenkins GN (1979) Salivary Effects on Plaque pH. *Saliva and Dental Caries*, New York and London: 307-322

The evidence that acid pH has a major role in caries development is almost overwhelming, although still circumstantial. The main rival concept – that demineralization also occurs by complex formation – must still be considered. This might occur by direct binding of a complexor with the calcium of apatite, but is more likely to be a result from a lowering of the saturation of the plaque with calcium phosphate by complexing calcium already in a soluble form in the plaque. The presence of substances in plaque able to form soluble complexes with calcium cannot be denied; and if they work slowly during the time when the plaque is neutral, their combined effect might be compared with a more rapid effect of acid. However, this paper concludes that although complex formation may contribute to caries and ought never to be ignored, its effect is probably small.

Saliva Stimulation

Dawes C, Dong C (1995) The Flow Rate and Electrolyte Composition of Whole Saliva Elicited by the Use of Sucrose-Containing and Sugar-Free Chewing Gums, *Archs. Oral Biol* **40(8)**: 699-705

On two occasions, 12 adults collected unstimulated saliva and then eight samples of saliva over a 20-min period while chewing 3g of either Wrigley's Spearmint sucrose-containing gum (SCG) or sugar-free gum (SFG) at 70 chews/min. The flow rates peaked initially, then fell with duration of stimulation. With the SFG they were slightly but significantly higher than with the SCG after 4 min of chewing. The sum of the concentrations of cations minus the sum of the concentrations of anions was not significantly different from zero for saliva elicited by the SCG. A second series of saliva collections with SCG and SFG was made by the same 12 participants and these samples were analysed for lactate. For these collections the flow rates with SCG were not significantly less than with the SFG. The lactate concentration in saliva elicited by SCG were not significantly less than with the SFG. The lactate concentration in saliva elicited by SFG had a mean lactate concentration of 0.21 mmol/l. Of the lactate formed during the metabolism of sucrose by the oral bacteria, only 2% or less appeared to be derived from the metabolism of micro-organisms

free in saliva, the balance presumably being formed in dental plaque and entering the saliva by diffusion. All saliva samples were supersaturated with respect to hydroxyapatite but stimulated saliva was significantly more supersaturated than unstimulated saliva. Saliva elicited by use of SFG will also tend to be more anticariogenic than that elicited by SCG because the former does not introduce a source of fermentable carbohydrate into the mouth.

Shern R, Chow S, Takagi S, Chow TH, Takagi KK (1994) Effects of Calcium Phosphates-containing Chewing Gums on Whole Saliva, *Journal Dental Research* **73(1)**: 1314

The present study was aimed at evaluating the feasibility of using chewing gums as a vehicle for enhancing salivation and increasing salivary calcium and phosphate levels. Based on their solubility properties, monocalcium phosphate monohydrate (MCPM) and an equimolar mixture of dicalcium phosphate anhydrous (DCPA) and tetracalcium phosphate (TTCP) were chosen as the experimental chewing gum additives. Each of the six subjects chewed for 16 min a commercial sugarless bubble gum, or the same gum to which MCPM (5 wt %) or a DCPA+TTCP (5 wt %) had been added. The subjects were asked to spit into pre weighed test tubes that were replaced every 2 min. The saliva samples collected were analyzed for weight, pH, and the total Ca and P concentrations. The results were: (1) the MCPM gum produced a significantly greater saliva flow and a lower salivary pH than did the control and DCPA+TTCP gums. (2) Both experimental gums significantly increased the Ca and P concentrations of saliva during the 16 min period. And (3) the degree of saturation with respect to tooth mineral was significantly increased by both experimental gums with the greater increase being produced by the DCPA + TTCP gum. These results suggest that the experimental gums may be useful for inducing salivation and promoting remineralization in xerostomic and other caries-prone patients.

Hoerman K (1992) Kinetics of Parotid Flow in Response to Gum Chewing, *Journal of Dental Research* **71**: 607

Gum chewing should produce combined salivary stimuli; Gustatory (sweet tastant) and mechanical (chewing alone). Gland flow rates should be unique to each effect; intense at first (tastant) and later at a steady state (chewing). We have observed the kinetics of combined stimuli to parotid glands in 4 healthy persons who were fitted with parotid cups. Unilateral chewing was done 2 hrs after meals. Since most chewers retain gum >20 min (Barabolak, et al, *Community Dent Oral Epidemiol*, 1991), volumes were recorded (mL/min) for 20 min. Gum base (GB), sugar (S) and sugarless (SO) gums were tested. Mean flow rates for GB, S and SO were $.17 \pm .05$; $.29 \pm .04$, and $.32 \pm .03$, respectively. The half-life for S retained in gum was 1.2 min. Retained S and flow correlated positively ($r = 0.97$). The mean flow lifetimes (t) due to



Current philosophy encourages dentists to practise maximum prevention and minimum intervention

tastant or chewing alone were separable as exponentials. GB alone showed a near steady state flow with $t_3 = 40.0$ min. S revealed two exponentials due to sweet (tastant) $t_1 = 0.66$ and $t_2 = 3.6$ min $t_3 = 40.0$ min (mechanical). SO showed $t_2 = 3.6$ min and $t_3 = 40.0$ min. A biphasic response to stimuli was observed. Early flow (fast) and volume (high) effects should increase intraoral buffering action. Later (5.0 min), diluent effects on Ca and P ions should subside and a mineralizing domain should be favoured.

Rosenhek H, Dawes C, Macpherson LMD (1992) The Effects of Chewing Gum and Gum-base Stick Size on Salivary Flow Rate, *Journal of Dental Research* **71**: 608

Since chewing gum sticks are made in different sizes, the objective of this study was to determine whether stick size influences salivary flow rate (FR). Ten adult subjects on 10 occasions collected unstimulated saliva and then chewed 1, 2, 3, 6 or 9 g of Wrigley's Spearmint chewing gum or gum-base for 20 min, at 70 chews/min, during which time 8 saliva samples were collected. The final weight of the gum was obtained and expressed as a % of the initial weight. In a separate study 3 subjects chewed each of the 10 gums for 20 min, during which time the gums were weighed at frequent intervals. With all weights of the chewing gum, the gum weight decreased rapidly but plateaued at 27-29% of the initial weight. With gum-base, the weights increased linearly during the 20 min to between 120% (1 g) and 111% (9 g). Salivary FRs peaked at 4-13 times the unstimulated rate (0.49 mL/min) in the 1st min of chewing and, with a given weight of gum, were higher with, chewing gum than gum base. After about 10 min of chewing, the FRs fell to a plateau at 2-4 times the unstimulated FR. With the base, the FRs during both the 1-2 min and 15-20 min of chewing showed a higher linear correlation with log (stick size) (both r values >0.996) than with stick size ($r = 0.971$ & 0.951). In conclusion, salivary FRs, initially and after prolonged chewing, were proportional to stick size.

Dawes C, Macpherson MD (1992) Effects of Nine Different Chewing-Gums and Lozenges on Salivary Flow Rate and pH, *Caries Research* **26(3)**: 176-182

The objectives of this study were to determine how salivary flow rate and pH vary with time during use of chewing-gums and lozenges. Twenty-four young adults collected unstimulated saliva and then, on different occasions, chewed one of six flavoured gums, or gum base, or sucked on one of two lozenges, for 20 min, during which time eight separate saliva samples were collected. Flow rate peaked during the 1st minutes of stimulation with all nine products. With the lozenges, flow rate fell towards the unstimulated rate when the lozenges had dissolved. There were no significant differences in the flow rates elicited by cinnamon-or peppermint-flavoured gums or between sugar-containing or sugar-free gums. With the flavoured gums, the mean flow rate followed a power curve ($r = -0.992$) with time and within about 10 min was not significantly different from that when gum base was the stimulus. The initial stimulated flow rate with flavoured gums was about 10-12 times greater than the unstimulated rate (0.47 ml/min).

After 20 min of chewing, it was still about 2.7 times that rate and about the same as the flow rate elicited by chewing-gum base alone. The pH of unstimulated saliva was about 6.95. With one gum containing about 1.5% organic acids, the salivary pH fell to a minimum of 6.18 in the 1st minute of stimulation, but then rose rapidly to a level about that in unstimulated saliva. With a sucrose-containing and a sucrose-free gum, the pH rose immediately on stimulation and then fell slightly with time to levels which were significantly above the pH of unstimulated saliva.

Macpherson LMD, Dawes C (1991) Effects of Salivary Film Velocity on pH Changes in an Artificial Plaque Containing *Streptococcus oralis* After Exposure to Sucrose, *Journal of Dental Research* **70(9)**: 1230-1234

Results from a computer model suggest that following exposure of dental plaque to sucrose, the rate of clearance of acids from plaque into the overlying salivary film will be greatly retarded at low film velocities. This was investigated with an in vitro technique in which artificial plaque containing *S. oralis* cells was exposed to 10% sucrose for one min. The pH at the proximal (P) and distal (D) undersurfaces of the plaque (0.5 or 1.5 mm thick) was then monitored during the passage of a 0.1 mm thick film of a sucrose-free solution over the surface. Over the range of salivary film velocities that have been estimated to occur in vivo (0.8-8 mm/min), lower minimum pH values and increased times for the pH to recover towards neutrality occurred at the lower salivary film velocity. Lower pH values were also reached with the 0.5- than with the 1.5 mm thick plaque. P/D pH gradients, with a lower pH distally, developed at film velocities of 0.8 and 8 mm/min, and the gradients were much more pronounced at the lower velocity. No P/D pH gradients developed when the film velocity was 86.2 mm/min. Incorporation of dead *S. oralis* cells into the plaque at percentages up to 57% reduced the extent of the pH fall and prolonged the recovery of the pH towards neutrality. The results support the prediction that, other factors being equal, plaque located in regions of the mouth with low salivary film velocity can achieve pH values lower than those of plaque of identical tensions and microbial composition located in areas where salivary film velocity is high.

Dawes C, Macpherson LMD (1991) Salivary Flow Rate and Sugar Clearance from Different Chewing Gums, *Caries Research* **25(1)**: 46

The objectives of this study were to determine (1) whether the salivary flow rate varies with different types of chewing gum, (2) whether the salivary flow rate changes during a 20 min period of gum chewing, and (3) the change in salivary sucrose concentration with time, when sucrose-containing gum are chewed. On seven separate occasions, 5 subjects collected a sample of unstimulated whole saliva and then chewed 1 of 7 different types of chewing gums, 4 of which contained sucrose. The subjects chewed gum for 20 min, during which time eight separate saliva samples were collected over the periods, 0-1, 1-2, 2-4, 4-6, 6-8, 8-10, 10-15, and 15-20 min. The seven chewing gums (2.6-3.2g) did not have significantly different effects on

flow rate. The salivary flow rate peaked at about ten times the unstimulated rate during the 1st min of gum chewing and then fell fairly rapidly to reach plateau values of about three times the unstimulated flow rate after 20 min. The salivary pH increased on chewing gum stimulation, except during the 1st min of stimulation with extra bubble gum. With the sugar-containing gums, the salivary sucrose concentration peaked within the first 1 or 2 min of gum chewing and then fell rapidly, suggesting rapid sucrose clearance, with no significant differences among the four gums. For times greater than 1 min, the sucrose concentration fits much better ($r = -0.995$) to a power curve such that the concentration (mg/ml) = $289.5 (t)^{1.6012}$, where t = time (min), than to an exponential ($r = -0.961$) or logarithmic curve ($r = -0.933$).



Triolo P, Jenson M (1990) Effect of Chewing Gum on Food Clearance from the Dentition, *Journal of Dental Research* **69**: 220

Previous studies have shown plaque reversal and remineralization from chewing gum (Jenson, *JADA* **113**, 1986 and Leach, *JDR* **68**, 1989). While salivary stimulation and buffering are major factors in contributing to this effect, removal of food debris may also be important. Addy et al. (*Clin Perio* **9**, 1982) has shown significant reduction in salivary debris from chewing gum. This study was conducted to examine the effect of chewing gum on clearance of food debris from the dentition. A typical meal was consumed during 30 minutes by 5 subjects. After the meal, subjects either chewed no gum or chewed one of the following: sorbitol chewing gum (Extra), sucrose chewing gum (Doublemint), 1 sucrose-coated pellet (P.K.) or 2 sucrose-coated pellets (P.K.). At prescribed chew times (5, 10, 15, 20 and 30 minutes) debris left after eating was harvested with a Gracey 13-14 curette and an explorer. At 5, 10 and 15 minutes there was significantly less debris with chewing gum compared to no chewing gum. Food debris at each of these time periods was reduced 3- to 4-fold by all gum treatments (e.g., wet weight means at 15 minutes, Doublemint-3.5 ug, No-gum-22 ug). At 20 and 30

minutes there was no significant difference among treatments. ANOVA showed no significant difference between sorbitol and sucrose gums for all time intervals. In conclusion, there was an increase in rate of food debris clearance from teeth with chewing gum, compared to no-gum which was significant from 5 to 15 minutes.

Jenkins JN, Edgar WM (1989) The Effect of Daily Gum Chewing on Salivary Flow Rates in Man, *Journal of Dental Research* **68(5)**: 786-790

Following reports of increased salivary gland size and increased function, induced by increased mastication in animals, the effects of long-term, frequent gum-chewing on resting

and stimulated flow rates were studied in human volunteers in separate experiments in Newcastle upon Tyne and in Toronto. In both experiments, unstimulated and stimulated saliva flow rates were measured in student volunteers at intervals of one or two weeks over a baseline period. Approximately half of the subjects were then given sugarless gum to be chewed (four pieces per day) over the experimental period; controls refrained from vigorous mastication. During (and, in Newcastle, after) the experimental period, salivary flow rates were measured at intervals, as before. In Newcastle unstimulated, but not stimulated, flow rates increased in the gum-chewing group and were still elevated (compared with controls) eight weeks following the experiment. In Toronto, the mean results showed no effect of gum-chewing, but the seven gum-chewers among the 11 subjects with low baseline flow rates (less than 0.3 mL/min) showed a 43% rise in unstimulated flow rate (p approximately 0.05). The results suggest that increased mastication, in the form of gum-chewing, can increase unstimulated flow rates, especially in those with low salivary function. In addition to short-term beneficial effects of sugarless gum, these long-term effects indicate the possibility of a beneficial effect in caries prevention.

Edgar WM (1989) The Clinical Implications of Salivary Stimulation, *General Dental Practitioner*, July/August 9-12

The paper is a review of the most direct evidence of its time for the potential of salivary stimulation in the active repair of the earliest stages of enamel caries. Recent plaque pH results suggest that even sugar-containing gum, if chewed for a prolonged period so that all the sugar is dissolved away, can have a beneficial action in inhibiting the plaque pH fall resulting from prior consumption of sugar. If the use of sugared gum were regulated in terms of duration and timing, then the potential to elicit a reparative salivary response might be revealed.

Council on Dental Therapeutics (1988) Consensus; Oral Health Effects of Products That Increase Salivary Flow Rate, *JADA* **116**: 757-759

The panel met to determine if drug or non-drug products that increase salivary flow provided beneficial effects to organ hard and soft tissues. Ten questions were addressed by this advisory panel of experts: five on the effects of saliva and rate of salivary flow on teeth and the development of caries; five on the effects of saliva and rate of salivary flow on oral soft tissues. The group concluded with respect to dry mouth that most people with dry mouth have the condition because of drug treatment. Because these patients are already taking a larger number of drugs, a non-pharmacological salivary stimulant might be preferred. On the question of whether products that stimulate salivary flow rate enhance soft tissue therapeutic benefits, the group concluded that in individuals with normal salivary flow, it is doubtful that such products would benefit these tissues. In individuals with decreased levels of salivary function, products that can increase the rate of salivary flow may prove beneficial, depending on the cause and extent of the gland hypofunction. Product categories differ in the benefits they provide.

Mouton C (1983) The Efficacy of Gum Chewing and Xylitol to Reduce Oral Glucose Clearance Time, *Journal of Canadian Dental Association* **9**: 655-660

Six teenagers were asked to perform weekly one of the following treatments: chewing for three minutes on a rubber band, or on four different chewing gums with respect to the sweetener, toothbrushing, or no treatment. The oral fluids were collected and the flow rate, pH, and buffer capacity were determined. The time necessary to determinate all detectable glucose (oral glucose clearance time) which was introduced as a mouth rinse before implementation of the treatments was measured. In a second study the oral glucose clearance time was determined in 40 young adults following toothbrushing, xylitol gum chewing or no treatment. Results showed that the resting oral glucose clearance time was significantly reduced by xylitol gum chewing (49 per cent reduction; $p < 0.001$) and by toothbrushing (41 per cent reduction; $p < 0.001$).

Addy H, Perriam E, Sterry A (1982) Effects of Sugared and Sugarfree Chewing Gum on the Accumulation of Plaque and Debris on the Teeth, *Journal of Clinical Periodontology* **9**: 346-354

The aim of this study was to determine the effects of sugar-free-containing gums on plaque formation, established plaque and salivary debris. Plaque accumulating during three 5-day periods was recorded in a group of 10 students who, in the absence of normal oral hygiene methods, chewed sugar-free or sugar-containing chewing gum or did not chew gum. In a second group of 10 students the effect of chewing the two types of gum on 3-day accumulations of plaque was recorded. Finally, the wet weight of liquorice debris present in the saliva with and without gum chewing, was recorded. During the no chewing periods distinct and significant differences in the amounts of plaque accumulating at different sites were apparent. Both types of chewing gum significantly and comparably reduced plaque accumulation during the 5-day period. The chewing gums also significantly reduced by 50% after chewing gum, It was noticed that plaque removal occurred primarily from sites remote from the gingival margin and interdental areas and therefore it was concluded that the observed effects of chewing gum on plaque would not be reflected in a reduction in gingival inflammation.

Soderling E, Rekola M, Makinen KK, Scheinin A (1975) Xylitol, Sorbitol, Fructose and Sucrose Induced Physico-Chemical Changes in Saliva, *Turku Sugar Studies XXI, Acta. Odont. Scand.* **33**: (Supplement 70) 397-403

The aim was to study eventual physico-chemical changes occurring in whole saliva due to sweetened and unsweetened stimulators. The assay was carried out in 10 female subjects with regard to changes of pH, buffering capacity and electrolytes in saliva as influenced by chewing of fructose, sucrose, sorbitol and xylitol gum, gum base and paraffin. The flow rate of saliva was measured in relation to use of xylitol and sucrose chewing gum and unsweetened gum base. These sweeteners increased significantly the salivary flow rate in comparison to the unsweetened gum base. Generally, xylitol and sorbitol on one hand, and sucrose and fructose on the other, behaved in an almost similar way. Increased buffering capacity and elevation of pH saliva was found in the presence of the polyols tested.

Shannon IL, Frome WJ (1973) Enhancement of Salivary Flow Rate and Buffering Capacity, *Journal of Canadian Dental Association* **3**: 77-181

This study compares the salivary flow rate produced by a sugarless gum containing nonfermentable sorbitol and mannitol with that elicited by sugared gums and with unstimulated flow. It was found that chewing either type of gum elicited highly significant increases in rate of flow, pH and buffer capacity values. Flow rate induced by the sugarless gum was significantly higher than that of the sugared gum.

Volker JF, Pinkerton DM (1947) Factors Influencing Oral Glucose Clearance, *Journal of Dental Research* **26**: 225-227

The clearance from the mouth of 500mg portions of glucose sucked, eaten, chewed and rinsed-swallowed has been compared in 6 experimental subjects. The clearance was found to be most rapid for the rinsed-swallowed and slowest for the sucked glucose. When glucose was given in the form of chewing gum, low salivary glucose values were recorded after the first several minutes, despite the fact that the base was still there. The authors note the possible significance of the findings in relation to dental caries. They admit that their findings deal only with the use of sugar in a pure form and do not necessarily forecast what will happen when sugars are combined with other foodstuffs having varying physical properties. However, they feel the results relate directly to some of the more common methods of sugar use (crunched and sucked candies, chewing gum and sweet beverages).

Plaque Acid Neutralisation

Park KK, Schemehorn BR, Stookey GK (1993) Effect of Time and Duration of Sorbitol Gum Chewing on Plaque Acidogenicity, *Pediatric Dentistry* **15(3)**: 197-202

Recent data indicate that salivary stimulation by chewing sugarless gum after snacks or meals can reduce the acidogenic potential of foods significantly. The purpose of this study was to determine the optimal initiation time and duration of post-snack salivary stimulation to obtain the maximum benefits of chewing sorbitol gum on reducing the acidogenic potential of starch-containing snacks. An indwelling plaque pH telemetry system was used on five adults in a randomized block design with four starch-containing snacks - pretzels, potato chips, granola bars, and corn chips. Results indicated that salivary stimulation caused by chewing sorbitol gum initiated after 5 min rather than waiting 15 min significantly reduced the acidogenic challenge induced by the snack foods. This study indicates that when the recommendation to chew sugarless gum following food ingestion is used as an adjunct in caries prevention, it should start within 5 min after food ingestion - the sooner the gum chewing is initiated the better - and should continue for at least 15 min to obtain the maximum benefits.



Chewing sugarfree gum
helps to reduce plaque
acid in the mouth

Manning RH, Edgar WM (1993) pH Changes in Plaque After Eating Snacks and Meals, and Their Modification By Chewing Sugared or Sugar-free Gum, *British Dental Journal* **174**: 241-244

Previous studies have suggested that if either sugar-free or sucrose-containing chewing gum is chewed after acidogenic meals or snacks, the plaque pH response to the latter is reduced and the potential for remineralisation of experimental white spot caries lesions is enhanced. This study has compared the effects of both gums on plaque pH (measured by the sampling technique) in 10 subjects who chewed the gums after standard acidogenic challenges (sucrose rinse, cupcake, and fried chicken dinner). The results showed that while both gums can significantly reduce the acid response, sugar-free gum appears more effective.

Maiwald NJ, Frohlich S (1992) Development of a Simplified Method of Oral Plaque-pH Telemetry. *Journal of Dental Research* **71**: 1308

Previous plaque-pH telemetry studies Imfeld, (Karger Press 1983) and Maiwald (Zahn-Mund-Kieferheilkd, 70: 1982) reported the acidogenicity of various foods and dietary patterns to estimate potential cariogenicity. To avoid patient discomfort, improve compliance, and avoid electrode shorting problems, we simplified our telemetry method and compared it to our previously published model. A removable partial prosthesis with a glass electrode set in the approximal space in the gap left by a missing first molar was used in 2 subjects. In the modified method (MM) subjects suspended oral hygiene for 3 days, the prosthesis was installed on the 3rd day, accumulated plaque was spread on the electrode and covered with gauze to avoid removal. For comparison, the same subjects wore the prosthesis in the mouth (PM) during plaque accumulation. 24 test sessions compared the plaque pH response to 4 treatments: a 10% sucrose rinse (SR), a 10% sorbitol rinse (SO) a snack roll with marmalade and coffee (SN), and SN followed by gum chewing (SN+G). pH curves were similar and selected time intervals (baselines and minimas) showed no differences in mean pH response between the 2 methods:

	Baseline pH	SR = SN pH min	SO pH min	SN+G 1/60 min post chew
MM	6.72 (0.26)	3.98 (0.55)	6.53 (0.28)	6.42 (0.54)/5.97 (0.24)
PM	6.80 (0.27)	4.04 (0.46)	6.36 (0.34)	6.20 (0.23)/6.15 (0.31)

The new method improved participation, reduced the number of “failed” runs and showed Stephan curves comparable to conventional methods.

Maiwald HJ, Frohlich S (1992) A Modified Plaque pH Telemetry Method, *Journal of Clinical Dentistry* **3(3)**: 79-82

Previous plaque-pH telemetry studies reported the acidogenicity of various foods and dietary patterns to estimate potential cariogenicity. To avoid patient discomfort, improve compliance,

and minimize electrode malfunctions, we have simplified our telemetry method and compared it to our previously published model. A removable partial prosthesis with a glass electrode set in the approximal space left by a missing first molar was used in 2 subjects. In the modified method, subjects suspended oral hygiene for 3 days, the prosthesis was then installed on the 3rd day, and accumulated plaque was spread on the electrode and covered with gauze for retention. In comparative tests, the same subjects wore the prosthesis in the mouth during plaque accumulation. Test sessions compared the plaque pH response to 4 treatments: a 10% sucrose rinse, a 10% sorbitol rinse, a snack roll with marmalade and coffee, and the snack followed by gum chewing. Overall, pH curves were similar (mean baselines and minimas) and no significant differences in mean pH response were noted between the 2 methods. The modified method improved subject participation, demonstrated greater reliability, and showed Stephan curves comparable to conventional methods.

Frohlich S, Maiwald HJ (1992) Reversal of Food Induced Plaque Acidity by Chewing Gums, *Journal of Dental Research* **71**:1309

Saliva stimulation by gum chewing has been reported (*Br. Dent. J.* **167**:6:204-2085, 1989) to neutralize plaque acidity. To attempt to confirm these findings with different methodology, we compared the plaque pH response to bread with honey followed by sucrose or sorbitol sweetened gum chewing for 20 minutes. Bread and honey was chosen as previous work in our laboratory found this a “worst case” challenge in terms of the extent and duration of the pH decline. The study design was: 4 subjects x 2 replicates x 3 treatments: food (F), food followed by sorbitol chewing gum (F+SO), and food followed by sucrose chewing gum (F+SU). Subjects accumulated plaque for 3 days, a partial prosthesis with a glass electrode was set in the approximal space in the gap left by a missing first molar, and the accumulated plaque spread on the electrode. pH was monitored for 150 min: Baseline (0-10), food (11-30), ± Gum Chewing (31-50), post chew monitoring (51-150).

x Baseline pH	x pH while chewing	x post chew pH
F 6.69 (0.43)	4.34 (0.37)	5.36 (0.45)
F+SO 6.70 (0.53)	6.68 (0.32)	6.39 (0.23)
F+SU 6.48 (0.26)	6.44 (0.31)	5.97 (0.46)

ANOVA showed: No differences at baseline, $p < 0.01$ for either gum vs. no gum during, and post chew, and $p < 0.06$ for sugar vs. sugarfree gum post chew. PH data was also converted to cH areas F:1703, F+SO:53, F+SU:156. While the post chew pH curves were not identical for sucrose vs. sorbitol chewing gums, both neutralized plaque acidity probably due to a common salivary action.

Frohlich S, Maiwald HJ, Flowerdew G (1992) Effect of Gum Chewing on the pH of Dental Plaque. *Journal of Clinical Dentistry* **3(3)**: 75-78

Saliva stimulation by gum chewing has been reported to neutralize plaque acidity. We compared the plaque pH response to bread with honey followed by sucrose-or sorbitol-sweetened gum chewing for 20 minutes. Bread and honey was chosen as previous work in our laboratory found this a worst case challenge in terms of the extent and duration of the pH decline. The study design was factorial with: 4 subjects x 2 replicates x 3 treatments. Each subject received each of the 3 treatments: food (bread and honey), food followed by sorbitol chewing gum, and food followed by sucrose chewing gum. Subjects accumulated plaque for 3 days on a partial prosthesis with a glass electrode set in the approximal space in the gap left by a missing first molar. Plaque pH was monitored for 150 min: baseline (0-10), food (11-30) ± gum chewing (31-50), post-chew monitoring (51-150).

ANOVA of mean plaque pH showed no difference between treatments at baseline. Significantly higher pH levels ($p < 0.01$) were shown with both gums compared to no gum during the chew and post chew phases. Plaque pH data compared to no gum during the chew and post-chew phases. Plaque pH data were also converted to absolute acid values (cH). Food alone produced 1703 $\mu\text{mol}/\text{min}$; food followed by sorbitol chewing gum produced 53 $\mu\text{mol}/\text{min}$; and food followed by sucrose gum produced 156 $\mu\text{mol}/\text{min}$. While the post-chew pH curves were not identical for sucrose vs. sorbitol chewing gums, both neutralized plaque acidity, probably due to the induced salivary action (*J Clin Dent* 3:75-78, 1992.)

Lee IK, Schachtele CF (1992) Effect of Gum Chewing Following Food Ingestion on the pH of Interproximal Dental Plaque. *Quintessence International* **23(7)**: 455-459

Recent publications have suggested that chewing sorbitol- or sucrose-containing gum after a snack or meal can reduce development of caries by neutralizing dental plaque acids at interproximal sites in the dentition. To confirm these findings four volunteers wore appliances containing a miniature pH electrode. After plaque accumulation, subjects ingested a bowl of sugar-coated cereal with milk and 20 minutes later chewed a sorbitol-containing gum, a sucrose-containing gum, or did not chew anything for 20 minutes. After exposure to the cereal, the plaque pH fell within 20 minutes from approximately 6.4 to 4.8. Sorbitol gum caused the pH to rise to 5.5, while the sucrose gum caused the pH to rise to only 5.1. After cessation of chewing, the pH in all cases dropped to 4.5 or lower. No statistically significant difference could be shown between plaque pH changes with the various protocols. Gum chewing after eating caused only a transient elevation in plaque pH.

Lingstrom P, Imfeld T, Birkhed D (1992) Comparison Between Different Plaque-pH Methods after Starch Consumption. *Journal of Dental Research* **71**: 633

The aim of the study was to compare three methods for pH measurements of human dental plaque, i.e. the telemetric, the microtouch and the sampling method, after consumption of starchy food

products. All volunteers (N=10) were equipped with a partial lower telemetry prosthesis incorporating a miniature glass pH-electrode. They refrained from tooth brushing for 3 days. Four products were tested: 1) soft bread, 2) potato chips, 3) 5% starch solution, and 4) 5% sucrose solution. pH of plaque was measured using the three methods simultaneously at various time points after consumption. The results demonstrated that the pH at the 10-min measurements were in mean 1.5 units lower for the telemetric method than for the sampling method and 1.0 units lower than for the microtouch method. The sampling method was hardly able to differentiate starch from sucrose solution or soft bread from potato chips. The microtouch and telemetric methods were able to rank the four test products. The maximum pH decrease was lower and more quickly reached for the two mouth rinse solutions, while the soft bread and potato chips showed a somewhat lower pH recovery. We therefore conclude that there were big differences in pH level between the three methods and that the two starch food products were easily fermented in dental plaque. This investigation was supported by the Swedish Medical Research Council, - Patentmedelsfonden for Odontologisk Profylaxforskning and the Swedish Dental Association.

Manning RH, Edgar WM (1991) Effects of Chewing Gum on Plaque pH Profiles After a Sucrose-Containing Snack and Rinse, *Caries Research* **25(3): 78**

Chewing gum for 20 min after meals and snacks promotes remineralization of experimental enamel lesions, whether the gum be sweetened with sucrose or sorbitol [*Caries Res* 1990; **24:405**]. Parallel reductions in plaque pH response have been reported using telemetry; this study aims to reinvestigate the potentially beneficial response to chewing gum after acidogenic challenges, using a plaque sampling technique. Plaque pH responses were recorded at intervals over 60 min in 10 volunteers after (A) a sugary snack (cupcake) alone; (B) a snack followed by sugar-free gum (20 min); (C) a snack followed by sugared gum; (D) a 10% sucrose mouth-rinse; (E) a rinse followed by sugar-free gum, and (F) a rinse followed by sugared gum. The mean results (minimum pH, area under the cH curve) were:

	Minimum pH	cH area (µmol/min)
A	5.65	36
B	6.15	-13
C	6.19	11
D	5.32	109
E	5.56	24
F	5.74	22

Analysis of variance showed significant treatment effects with gum for snack, rinse, minimum pH, and cH Area. Significant (p< 0.05) differences were: minimum pH, A vs. B, A vs. c, D vs. E, D vs. F; cH area, A vs. B, D vs. E. The results tend to confirm that with two levels of prior acidogenic challenge, both sugared and sugar-free gum may reduce plaque pH.



After a sugar intake, acid is rapidly generated in the dental plaque. Within 1-2 minutes, plaque pH has fallen to levels which mean that tooth enamel undergoes demineralisation for between 20 minutes and two hours

Park K, Schemehorn BR, Bolton JW, Stookey OK (1991) Effect of Sucrose and Sorbitol Gums on Plaque pH Responses, *Journal of Dental Research* **70**: 1107

This randomized, double-blind, crossover study in 8 subjects compared the effects of sucrose and sorbitol chewing gums on plaque pH, both alone and following an acidogenic challenge. After 3-6 days of plaque accumulation, subjects chewed one piece of sorbitol or sucrose gum for 10, 15, or 20 minutes alone or following consumption of a cream-filled cookie. Plaque pH was recorded for 2 hours using indwelling pH wire-telemetry (Park et al. *Am J Dent*, **3**: #5, 1990), and the area of the curve below pH 5.5 and the lowest pH reached were determined. The results indicated that: (1) chewing sucrose gum alone produced a significantly ($p < 0.05$) greater acidogenic response than sorbitol gum (i.e., 4779 vs. 0 area units below pH 5.5 and 4.62 vs. 5.98 minimum pH); (2) sorbitol gum mediated the pH response significantly more than a sucrose gum (i.e., 84 vs. 3792 area units below pH 5.5. and 5.62 vs. 4.82 minimum pH following gum chewing); (3) the longer both gums were chewed, the greater the reduction in area under 5.5 and the higher the pH minimum.

This study demonstrates that sorbitol and sucrose gums do not affect plaque pH responses comparably.

Lingstrom P, Granfeldt Y, Bjorck I, Birkhed D (1991) pH of Interdental Plaque after Consumption of Starch-Containing Food, *Journal of Dental Research* **70(4)**: 4

The aim was to study the effects of different pasta products on pH of human interdental plaque in vivo and to compare these to in Sweden traditionally used starch-containing food products. The degree of hydrolysis in saliva was also studied.

In total 10 subjects participated, who refrained from toothbrushing for 3 days, pH of plaque was studied, using the so called touch method before and at various points after consumption. Ten products were tested: 1) Spaghetti, 2) macaroni, 3) spaghetti with high content of fibre, 4) pasta porridge, 5) potato, 6) rice, 7) bread, 8) sweetened bread, 9) glucose tablet and 10) 10% sucrose solution. Saliva samples were collected at three occasions after the intake for analysis of low molecular weight carbohydrates (maltose and maltotriose).

The sucrose solution resulted in the greatest pH drop followed, in order, by the glucose tablet, sweetened bread, bread and potato. All pasta products induced smaller pH falls and gave, together with rice, similar results. Comparing the degree of hydrolysis in saliva, significant differences were obtained between bread and spaghetti at 6 min after the intake. Only small differences were found at the other time points.

In conclusion, all starch-containing food products were easily fermented in dental plaque, but the pasta and the rice to a lesser extent than the bread and potato.

Park KK, Schemehorn BR, Bolton JW, Stookey GK (1990) The Impact of Chewing Sugarless Gum on the Acidogenicity of Fast-Food Meals, *Amer. Journal of Dental Research* **3(6)**: 231-235

The objective of this study was to evaluate the effect of chewing sorbitol gum on plaque pH following the ingestion of acidogenic fast-food meals. Plaque pH response was monitored using an indwelling wire-telemetry system in five adult panelists. From a pilot study with 12 fast-food meals, the most acidogenic breakfast, lunch and dinner were selected for this study. In the first test, the fasted, resting plaque pH was recorded for 5 minutes; panelists ingested the selected meals for 10 minutes, rinsed thoroughly with 50 ml of tap water, and the pH response was monitored for the remainder of a 2-hour period. In the second test series, the same procedures were followed through the post-meal ingestion rinse. After the pH response to the meal was monitored for 5 minutes, the panelists chewed a sorbitol gum for 15 minutes in their usual manner and the panelists were encouraged to move the gum around their mouth, however, it appeared as if they favored the side of their mouth without the partial denture. The pH response was monitored for the balance of the 2-hour period. All panelists ate the test foods, with and without the chewing gum, according to a randomized-block test design. The results indicated that the use of sorbitol gum significantly raised the plaque pH, prevented the subsequent pH drops after the fast-food meal ingestion and reduced the pH curve area under 5.5.

Park KK, Schemehorn BR, Bolton JW, Stookey GK (1990) Effect of Sorbitol Gum Chewing on Plaque pH Response after Ingesting Snacks Containing Predominantly Sucrose or Starch, *American Journal of Dentistry* **3(5)**: 185-191

The purpose of this study was to determine the effect of chewing a sorbitol gum (Trident) for 10 minutes on interproximal plaque pH changes following ingestion of selected sucrose- or starch-containing foods. The snacks containing predominantly sucrose (and/or simple sugars) were chocolate bar, cream-filled cupcakes, cream-filled sandwich cookie, cherry pie and raisins. The snacks containing predominantly starch were oat cereal, granola bars, pretzels, potato chips and corn chips. Plaque pH responses were monitored using an indwelling wire-telemetry system in five adult panelists. The test design involved two sets of 5 x 5 Latin square randomization in which each set consisted of two series of tests. In the first series of tests, the fasted, resting plaque pH was recorded for 5 minutes, panelists ingested the designated snacks for 2 minutes, and the pH response was monitored for the remainder of a 2-hour period. In the second series of tests, the same procedure was followed through the snack ingestion. After the pH response to the snack was monitored for 15 minutes, the panelists were asked to chew one stick of sorbitol gum for 10 minutes and the pH response was then monitored for the balance of the 2-hour period. Results indicated that both the sugar- and starch-containing snacks tested in this study caused significant decreases in interproximal plaque pH. Chewing a sorbitol gum after ingestion of the snacks significantly reduced the demineralizing potential of the plaque. The chewing of sorbitol gum following the ingestion of snacks can be recommended as an adjunct to other caries-preventive oral hygiene measures.

Abelson DC, Barton S, Mandel ID (1990) The Effect of Chewing Sorbitol-Sweetened Gum on Salivary Flow and Cemental Plaque pH in Subjects with Low Salivary Flow, *Journal of Clinical Dentistry* **2(1)**: 3-5

The purpose of this work was to study the effect of chewing a sorbitol-sweetened gum on whole and parotid salivary flow rates, and on the cemental plaque pH response to a sucrose rinse challenge, in subjects with low salivary flow. The results show that chewing a flavoured sugarless gum significantly increases salivary flow rates in individuals with dry mouth. Additionally, chewing the sorbitol-sweetened gum effectively prevents the fall in cemental plaque pH generally seen in response to a sucrose challenge. This indicates that chewing a sorbitol-sweetened gum provides a palliative and possibly a protective benefit for people who suffer from dry mouth.

Maiwald HJ, Beu M (1990) The Caries-Preventive Action of Sugar-Containing and Sugarless Chewing Gum, *Ernährungsforschung* **35**: 2-7

Significant advances have been made in the research to establish a reliable means to evaluate the cariogenic potential of foods. Reasonable test systems are being developed, and through collaborative studies it is likely that a scheme for testing foods will be developed. How such a scheme will be used to produce benefits for oral health should and will be the focus of considerable debate. Various issues need to be addressed. The most reasonable immediate goal in this area would be the global acceptance of the interproximal plaque pH test as a means to produce non-acidogenic products.

Bibby BG (1990) Chewing Gum. Food and the Teeth, *Vantage Press New York* **5(4)**: 123-128

After recent modifications in the composition of cariogenic chewing gum, a gum that is non-cariogenic and may even have caries-prevention action has been developed by industry. The authors of this chapter express the hope that it can be taken as an example of what could be done with other cariogenic confections or baked goods by industry. The chapter reviews the history of chewing gums, with discussion of chewing gums containing the following: phosphate reagents; acid-neutralising agents, enzyme blocking agents (vitamin K, chlorophyll, furadoxyl), fluoride. Different non-cariogenic sweeteners are discussed: saccharine; sugar alcohols. The possibility of using chewing gum to prevent or treat diseases of the gums is briefly discussed.

Park KK, Schemehorn BR, Bolton JW, Stookey GK (1989) Impact of Chewing Gum on the Acidogenicity of Meals, *Journal of Dental Research* **68**: 262

The objective of this study was to evaluate the effect of chewing sorbitol gum on plaque pH following the ingestion of acidogenic fast-food meals. Plaque pH response was monitored using an indwelling wire-telemetry system in five adult panelists. From a pilot study with 12 fast-food meals, the most acidogenic breakfast, lunch and dinner were selected for this study. In the first test, the fasted, resting plaque pH was recorded for 5 minutes; panelists ingested the selected meals for 10 minutes, rinsed thoroughly with 50 ml of tap water, and the pH response was

monitored for the remainder of a 2-hour period. In the second test series, the same procedures were followed through the post-meal ingestion rinse. After the pH response to the meal was monitored for 5 minutes, the panelists chewed a sorbitol gum for 15 minutes in their usual manner and the panelists were encouraged to move the gum around their mouth, however, it appeared as if they favoured the side of their mouth without the partial denture. The pH response was monitored for the balance of the 2-hour period. All panelists ate the test foods, with and without the chewing gum, according to a randomized-block test design. The results indicated that the use of sorbitol gum significantly raised the plaque pH, prevented the subsequent pH drops after the fast-food meal ingestion and reduced the pH curve area under 5.5.

Jensen ME, Wefel JS (1989) Human Plaque pH Responses to Meals and the Effects of Chewing Gum. *British Dental Journal* **167(6)**: 204-208

Interproximal plaque pH responses to five different meals were investigated in this study. All meals were found to be acidogenic, with pH challenges lasting well over one hour. The effects of chewing one sorbitol and two different types of sucrose-containing gum for 20 minutes after the meal were examined. All three types of gum reversed the acid challenge of the meal and resulted in an interproximal pH level that is considered 'safe for teeth'. This study indicates that meals can be very acidogenic and that, in addition to normal preventive dental procedures, chewing gum for 20 minutes after meal consumption should be considered, to reduce the cariogenic challenge to the teeth.

Jensen ME, Zibell S, Barabolak R, Triolo PT (1989) Human Plaque pH Responses to Meals With and Without Desserts, *Journal Dental Research* 68: Abstract #264

Indwelling interproximal plaque pH telemetry was used to compare pH responses to five normal meals and one lowly-acidogenic meal with and without a sweet at the end of the meal. Four volunteers with a mean age of 27 years were fitted with wire-telemetric appliances containing glass pH microelectrodes on mandibular first permanent molars. Fasting plaque, age 4-7 days, was used for all tests. Test sessions consisted of meal consumption during a 20-minute period followed by a 600 minute test period. ANOVA* and Duncan's multiple range test was used to analyze pH responses (area, cH minimum) for all meals with and without desserts. Statistically significant differences existed for all measurements at $p < .001$. Duncan's multiple range test however, indicated that the only different group was the low carbohydrate meal without dessert - ham, diet soda and lettuce salad which produced a minimum pH of 5.2 ± 1.2 . All other responses, were not significantly different.

Results of this study indicate that five different normal meals with and without dessert and a low carbohydrate meal with dessert all produce significant interproximal plaque pH responses. Prolonged acid challenges do not differ if dessert is included unless the meal is extremely low in carbohydrate.



Yankell SL, Emling RC (1989) Clinical Study to Evaluate the Effects of Three Marketed Sugarless Chewing Gum Products on Plaque pH, pCa, and Swallowing Rates, *Journal of Clinical Dentistry* **1(3)**: 70-74

The purpose of this study was to determine the ability of three commercially available chewing gums (Extra, Trident, and CareFree) to stimulate saliva flow and reverse the plaque acid and ionized calcium levels induced by a glucose challenge. Electrodes to measure pH and pCa were situated in a Hawley appliance. When the Hawley appliance was in place, the electrodes were inserted into three day old plaque at maxillary interproximal sites. A pressure sensor, located in the posterior center of the Hawley appliance, was used to record swallowing rates. After baseline values were determined, the test procedure consisted of first administering a 5% glucose challenge solution followed by a 10 minute challenge effect period, a 5 minute gum chewing or product period, and finally a 10 minute product effect period after the test gum was discarded. An ANOVA was used to compare the ability of each chewing gum to stimulate saliva and cause a return of the plaque acid and/or ionized calcium to baseline levels following product discard. The three chewing gum products varied in both time and level of pH attained while neutralizing plaque acidity ($p < .05$) induced by the glucose rinse. No significant differences were found between the chewing gums for the pCa data and swallowing rates. All chewing gum products stimulated swallowing and effectively reversed plaque pH and pCa changes caused by the glucose rinse.

Park KK, Schemehorn BR, Bolton JW, Stookey GK (1988) Effect of Gum Chewing on Plaque pH Response After the Ingestion of Snacks Containing Predominantly Sugars or Starch, *Journal of Dental Research* **67**: 1330

The purpose of this study was to determine the effect of chewing a sorbitol gum (Trident) for 10 minutes on interproximal plaque pH changes following ingestion of selected sucrose- or starch-containing foods. The snacks containing predominantly sucrose (and/or simple sugars) were chocolate bar, cream-filled cupcakes, cream-filled sandwich cookie, cherry pie and raisins. The snacks containing predominantly starch were oat cereal, granola bars, pretzels, potato chips and corn chips. Plaque pH responses were monitored using an indwelling wire-telemetry system in five adult panelists. The test design involved two sets of 5 x 5 Latin square randomization in which each set consisted of two series of tests. In the first series of tests, the fasted, resting plaque pH was recorded for 5 minutes, panelists ingested the designated snacks for 2 minutes, and the pH response was monitored for the remainder of a 2-hour period. In the second series of tests, the same procedure was followed through the snack ingestion. After the pH response to the snack was monitored for 15 minutes, the panelists were asked to chew one stick of sorbitol gum for 10 minutes and the pH response was then monitored for the balance of the 2-hour period. Results indicated that both the sugar- and starch-containing snacks tested in this study

caused significant decreases in interproximal plaque pH. Chewing a sorbitol gum after ingestion of the snacks significantly reduced the demineralizing potential of the plaque. The chewing of sorbitol gum following the ingestion of snacks can be recommended as an adjunct to other caries-preventive oral hygiene measures.

Yankell SL, Emling RC, Greco MR (1988) Interruption of a Glucose Challenge with Chewing Gum or Unflavored Gum Base, *Journal of Dental Research* **67**:1646

Paraffin -or unflavoured chewing gum is routinely administered after carbohydrate challenges to stimulate salivary flow which increases clearance and neutralizes plaque acids during *in vivo* pH testing. This study compares an unflavoured gum base (GB) to a peppermint flavored sorbitol chewing gum (Extra) for their effects on stimulating saliva flow and interrupting the increased acidity (pH) and ionized plaque calcium (pCa) induced by a glucose challenge. Six subjects wore maxillary Hawley appliances with pH, and pCa sensors for the study. Swallowing was recorded by pressure sensor or patient signals (*AADR Abs* **998**, 1986). Ten minutes after a sugar rinse, subjects chewed Extra or GB for five minutes. All parameters were then monitored for 10 minutes. During chewing, Extra returned plaque pH to higher mean value than GB ($p < .001$). Post chew Extra elevated plaque pH to above baseline (7.4 vs 6.9 GB) and maintained a pH, which was significantly higher ($p < .001$) throughout monitoring. GB did not return to baseline pH following chewing, and there were no differences in the number of swallows, or pCa levels between the two groups.

A flavoured sweetened chewing gum (Extra) increased saliva stimulation and neutralized plaque acid levels compared to baseline and an unflavoured gumbase.

Yankell SL, Emling RC (1988) Clinical Effects on Plaque pH, pCa, and Swallowing Rates From Chewing a Flavored or Unflavored Chewing Gum, *Journal of Clinical Dental* **1**(2): 51-53

The effects of chewing a flavoured or unflavoured gum after a carbohydrate challenge were studied using saliva stimulation, plaque pH and ionized calcium as monitoring parameters. A commercially available sorbitol chewing gum (CG) stimulated a significant greater number of swallows ($p < 0.05$, $n=6$) than an unflavoured gum base (GB) during five minutes chewing protocol. After chewing was stopped, no differences were observed. Plaque pH measurements were made using multiple iridium oxide electrodes positioned in interproximal posterior sites of the maxillary arch. Results showed that chewing either GB or CG for five minutes after a 5% glucose rinse reversed the acid challenge to plaque. CG was significantly more effective ($p < 0.001$) in its ability to maintain plaque pH during the ten minutes after chewing was stopped. No significant differences in ionized calcium levels were observed at interproximal sites during and after chewing CG or GB. Results suggest that saliva stimulated by chewing limits the duration of acidogenic challenge to maxillary interproximal sites. A flavoured/sweetened masticatory stimulant was more effective in stimulating saliva flow and reversing acidogenic effects of a glucose rinse than an unflavoured stimulant.

Jensen M, Wefel S, Sheth S (1988) Plaque pH Responses to Meals and Effects of Chewing Gums. *Journal of Clinical Research* **67**: 1329

Purpose of this study was to evaluate the acidogenic potential of five different well-balanced meals and assess the effect of chewing gum after a meal. Five representative meals were provided to 3 volunteers for measurement of acidogenic potential using indwelling wire-telemetric appliances containing glass pH microelectrodes. One selected meal was provided to 5 volunteers for comparing the acid challenge with and without the use of sorbitol (Extra) and sucrose (Doublemint) chewing gums for 20 minutes at the end of the meal. Plaque pH responses were continuously recorded for 120 minutes after a 20 minute meal in each recording session. All five meals resulted in plaque responses below pH 4.5 and did not recover to resting values in the next 120 minute period. The grand mean for minimum pH for the 5 meals was 4.21 ± 0.16 . ANOVA for subjects and meals by minimum pH and pH below pH 5.7 showed no statistically significant differences. Comparison of one meal with and without gum chewing after the meal resulted in complete reversal of the acid challenge to the resting pH. These pH values did not drop below pH 5.7 during the remainder of the recording session (100 minutes). The t-test for pH below pH 5.7 with and without gum chewing was significantly different ($p < .001$). The results indicate that all five well-balanced meals produced prolonged acid challenges to the dentition. Using one representative meal, the acid challenge can be rapidly reversed and maintained near the resting pH by chewing sorbitol or sucrose gum for 20 minutes following the meal. This study was supported by the Ctr. for Clin. Stud., NIH/NIDR, grant BP50 DE07010 and the Wm. Wrigley Jr. Company.

Dodds MWJ, Edgar WM (1988) The Relationship Between Plaque pH, Plaque Acid Anion Profiles, and Oral Carbohydrate Retention after Ingestion of Several 'Reference Foods' by Human Subjects. *Journal of Dental Research* **67(5)**: 861-865

The primary aim of this study was to rank several foods (apple drink, caramel, chocolate, cookie, skimmed milk powder, snack cracker, and wheat flake) according to their plaque pH response as monitored in a panel of 12 volunteers by the plaque sampling method for comparison with data previously reported with other methods used to access cariogenicity potential. Secondary experiments (using subsets of the panel of subjects) were undertaken in an attempt to elucidate some of the reasons for the observed plaque pH changes. Oral carbohydrate retention was measured at a single time period after food use as total anthrone-positive carbohydrate material, and as specific acidogenic sugars by gas-liquid chromatography after gel-exclusion chromatography. The concentrations of acid anions in the plaque fluid after food consumption were measured by isotachopheresis eight min after food use. According to the plaque pH response, apple-flavored fruit drink and chocolate were the most acidogenic foods and skimmed milk powder the least acidogenic. There were significant correlations ($p < 0.05$) between plaque pH data and lactate-plus-acetate concentrations in plaque fluid, but the correlations between the pH data and any of the carbohydrate retention parameters were not significant.

Schemehorn BR, Park KK, Bolton JW, Stookey GK (1988) Effect of Time and Duration of Gum Chewing on Plaque Acidogenicity, *Journal of Dental Research* **67**:1328

The purpose of these studies was to determine the effect of changing the time interval between food ingestion and chewing a sugarless gum as well as the duration of gum chewing on plaque acidogenicity.

The indwelling pH electrode model identified by the ADA Foods, Nutrition and Dental Health Program was used. In the first study, the panelists were used in all items (pretzel, potato chips, corn chips and granola bar) in a latin square design. Plaque pH curves of 3-7-day-old plaque were recorded for two hours. The panelists then repeated the test design at a later date except they chewed a sorbitol-containing gum for a 10-minute period 15 minutes following food ingestion. The second study was identical except the gum chewing period was 15 minutes (rather than 10) after a 5-minute (rather than 15) post ingestion period. Parameters monitored included area of the pH curve below 5.5, lowest pH attained, maximum pH drop and time below pH 5.5.

The data indicate that when the gum was chewed for a longer time period and chewing was started 10 minutes sooner after the food-ingestion, the area, under pH 5.5 was reduced by a significantly larger percentage (89 vs 65%). minimum pH attained was significantly higher (1.32 vs 0.38 pH units) and maximum pH drop was significantly reduced (1.18 vs 0.36 pH units). Time under pH 5.5 was numerically but not significantly reduced by reducing the time interval between ingestion of food and the chewing of the sorbitol-containing gum. The results indicate that when the gum was chewed closer to food ingestion and for a longer time period, significantly reduced cariogenic challenge from each of the foods were observed over the two-hour monitoring periods.

Markovic N, Abelson DC, Mandel ID (1988) Sorbitol Gum in Xerostomias: The Effect on Dental Plaque pH and Salivary Flow Rates, *Gerodontology* **7(2)**: 71-75

Adequate salivary flow is important for patient comfort and maintenance of oral health. Xerostomia, or dry mouth, is a common clinical complaint. Masticatory and gustatory activity can stimulate salivary flow from functional salivary tissue and the use of sugarless mints and gums have been recommended to individuals who complain of xerostomia, but there are minimum clinical data. A clinical study assessing the effect of salivary flow rates and dental plaque pH of a sorbitol-sweetened chewing gum in subjects with the complaint of xerostomia. Statistically significant stimulated whole mouth and parotid salivary flow rate increases were found when compared to unstimulated whole mouth and parotid salivary flow rates. Chewing of the sorbitol sweetened gum also effectively reduced the drop in pH seen following the exposure to a fermentable carbohydrate. The findings of this present study indicate that chewing of a sorbitol-sweetened gum may be of benefit to patients with the complaint of xerostomia.

Jensen ME (1988) Effects of Chewing Sorbitol Gum on Human Salivary and Interproximal Plaque pH, *Journal Clinical Dentistry* **1(1)**: 6-27

Interproximal wire-telemetric appliances were used in this study to obtain interproximal plaque pH data from the distal surface and salivary pH data from the buccal surface of lower mandibular molars. Five different test foods – a granola bar, a fruit bar, a Danish pastry, an apple and a jelly sandwich – were used as test food substances. The pH changes from these test foods were recorded continuously for 120-minute periods in each of five human volunteers. Subsequently, two separate series of test sessions were conducted using peppermint flavored sugarless gum or a

grape flavored sugarless gum to observe the effects of chewing these gums for a ten-minute period on both the salivary and interproximal plaque pH. Dramatic pH drops were observed with test foods in interproximal with a varying rate of return toward the resting values. Minimum plaque pH values were similar for all test foods. Salivary pH values, however, differed markedly. Minimum pH values achieved for the salivary responses were not statistically different from the minimum plaque pH values for the fruit bar and apple. The other three test foods showed a higher minimum salivary pH level, which was statistically significant. Both peppermint and grape flavored chewing gums, chewed for a ten-minute period after ingestion of the challenge food, resolved in a relatively rapid pH return, for both saliva and plaque, to the resting values. Salivary and plaque pH remained above a mean pH level of 6.4 during a 30-minute post chew recording period.



For the best effect chewing sugarfree gum should start within five minutes after food ingestion

Hoerman K C, Jensen ME, Barabolak R (1987) Chewing Gum and the Protective Quality of Saliva in Respect to Plaque pH, *Caries Research* **22(2)**: 116

Saliva is known to be involved in plaque pH regulation: however, the degree of effect from stimulated saliva is not fully understood. This paper reports saliva flow rates during the use of chewing gum sweetened with sorbitol (CGsorb) or sucrose (CG Sucr) versus rates with breath mints (Msorb or Msucr). Correspondingly, interproximal plaque pH changes were followed during use of each stimulus alone and 15 min. after ingestion of a sucrose snack.

Parotid glands were cannulated in 4 subjects at about 2h after meals. Duplicate runs were made for each stimulus, and secreted volumes were recorded at 1-min. intervals for 20 min. Flow rates varied by subject and by the physical form of the stimuli. CG The mean flow rates for CGsorb (0.30 ml/min) and CGsucr (0.33 ml/min) were not different, but were greater than flow rates with either breath mint. The corresponding rates for Msorb (0.13 ml/min) and Msucr (0.18 ml/min) were different from each other and higher and resting rates.

Five subjects were fitted with interproximal indwelling pH electrodes. After eating a sucrose snack, and waiting 15 min, the pH drop to -4.00 was reversed to pH 5.5 within 8 min. with CGsorb and within 11 min. with CGsucr. The corresponding pH rises were 40 min. for Msorb

and >60 min for Msucr Gum chewing further neutralized plaque acids to pH 7.0 within 14-18 min, while breath mints were ineffective.

The primary factor contributing to the acid-neutralizing benefit of chewing gum is its ability to deliver the natural protective qualities of saliva interproximally through mastication and the sweet stimuli.

Edgar WM, Geddes AM (1986) Plaque Acidity Models for Cariogenicity Testing Some Theoretical and Practical Observations, *Journal of Dentist Research* **65**: 1498-1502

Plaque acidity studies form a rational basis for providing partial evidence of Cariogenic potential incorporating features not reproduced by other tests (e.g. patterns of food use). Plaque pH data represent the summation of the effects of many microbial and physiological factors of relevance to cariogenic potential. Different plaque pH models may satisfy alternative test requirements, depending upon their performance characteristics, but parallelism exists among models, and between plaque pH test results and findings from other cariogenicity testing methods.

Criteria for selection of subjects and methodology are reviewed, and the validity of the use of pH in statistical procedures discussed. The analysis of organic acid concentration in plaque is relevant to cariogenicity, but the full significance of the acid spectrum is not fully elucidated. The relationship between pH and carious demineralization is no-linear, and plaque pH data should be used to compare foods in relation in internal standards. Finally, the effects of eating frequency, food sequences, and mixtures may modify the response to an individual food item and should be taken into account when one interprets the results of such tests.

Jensen ME (1986) Effects of Chewing Sorbitol Gum and Paraffin on Human Interproximal Plaque pH. *Caries Research* **20**: 503-509

The effects of chewing sorbitol-containing gum and paraffin upon human interproximal plaque pH responses after consumption of a jelly doughnut were investigated in this study. Prolonged plaque pH responses were observed following consumption of the jelly doughnut. Dramatic rises in pH resulted when sorbitol gum and paraffin were chewed 15 min after consumption of the doughnut. Ten minutes of chewing resulted in significantly higher ($p < 0.01$) pH values than 5 min of chewing.

Jensen ME (1986) Responses of Interproximal Plaque pH to Snack Foods and Effect of Chewing Sorbitol-Containing Gum, *JADA* **113**: 262-266

Interproximal wire-telemetric plaque pH data were obtained from five volunteers after they ate milk chocolate bars, raisins, chocolate wafer cookies with cream filling, cupcakes with icing and cream filling, and cherry pies. All the foods produced rapid decreases in plaque pH for extended periods. In a second set of test sessions, volunteers chewed sugarless gum for 10 minutes, starting 15 minutes after they ate the snack food. In all cases, the gum chewing

caused a rapid increase in plaque pH. The pH remained at a level considered safe for teeth for 30 minutes after chewing the gum.

Maiwald HJ (1984) The Role of Sugar in the Etiology of Caries, *Zahnärztliche praxis* **35(5)**: 1-10
It is generally accepted that the carious process is initiated and maintained by the presence of sugar and bacteria which decompose this sugar. The term 'sugar' means not only household sugar, i.e. the disaccharide sucrose, but all low-molecular carbohydrates. They are converted to acids by the microbial metabolism. *Streptococcus mutans* plays the greatest role in this. Food technologists and educators are challenged to reduce the sugar consumption particularly of children but also of adults to a reasonable level.

Schachtele CF, Jensen ME (1983) Can Foods be Ranked According to Their Cariogenic Potential? *Cariology Today, International Congress, Zurich* 136-146

Significant advances have been made in the research to establish a reliable means to evaluate the cariogenic potential of foods. Reasonable test systems are being developed, and through collaborative studies it is likely that a scheme for testing foods will be developed. How such a scheme will be used to produce benefits for oral health should and will be the focus of considerable debate. Various issues need to be addressed. The most reasonable immediate goal in this area would be the global acceptance of the interproximal plaque pH test as a means to produce non-acidogenic products.

Imfeld TN (1983) Dietary sugars and sugar-containing snacks and confectionery products. *Identification of Low Caries Risk Dietary Components, Karger* **5**: 101-116

Main section summary 101-116: The fall in pH of interdental plaque after consumption of certain important dietary carbohydrates shows their highly acidogenic character. A summary of numerous investigations of the relationship of foods and caries reveals that in all reports it is the frequent consumption of sweets and snacks, mostly between meals, that coincides with a higher caries in the subjects. Snack foods certainly have a strong cariogenic potential because they combine the detrimental effects of high sugar content and high frequency of consumption. Representative examples of plaque pH telemetric evaluations of commonly sold confectionery products are presented in the chapter. A considerable reduction of the total sugar consumption would be desirable. A reduction of fermentable sugars contained in between-meal snacks and confectionery products seems a more feasible target to strive for.

Maiwald HJ, Banoczy S, Teitze W, Toth Z, Vegh A (1982) Controlling Plaque pH by Sugar-Containing and Sugar-Free Chewing Gum. *Zahn, Mund u. Kleberheilkd* **70**: 598-604

By measuring the in vitro pH of plaque taken from humans and by applying the method of in vivo plaque pH telemetry, the plaque pH curve was plotted after sorbitol, xylitol and



Chewing sugarfree gum promotes remineralisation of enamel

sucrose flavored gums had been chewed. The sucrose flavoured chewing gum has a slight pH reducing effect, whereas sorbitol and xylitol flavoured chewing gum did not. Furthermore, chewing gum not flavoured with sucrose is appropriate for neutralizing the low pH produced by sugar-containing foods. Thus, we can offer infer that chewing gum not flavoured by sucrose has a preventive effect.

Schachtele CF, Jensen ME (1981) Human Plaque pH Studies: Estimating the Acidogenic Potential of Foods, *Cereal Foods World* **26(1)**: 15-18

The relative cariogenic potential of starch and starchy foods is an issue of importance and controversy. There is little question that starch can be degraded in the oral cavity to substances that can be fermented to acid by plaque bacteria. Starch has been shown to have significant cariogenic potential in the rat-model. The cariogenic potential of starch could be influenced by a large number of factors ranging from the processes used in food manufacturing to the interactions that are involved in salivary amylase production and function. The issue is complex, as starchy foods are usually never eaten in the absence of more rapidly fermentable carbohydrates. Also, the effect on caries of a mixture of highly and moderately metabolisable compounds is totally unknown.

Bibby BG (1975) The Cariogenicity of Snack Foods and Confections, *JADA* **90**: 120-132

A survey of the relationship between caries prevalence and the consumption of sugar and other foods by man has been interpreted to show that snack foods share importance with sucrose in caries causation. Support for this conclusion is found in animal experiments and some in vitro and in vivo tests.

Hein JW, Pramod SM, Gertrude AQ (1961) Changes in Plaque H Following Gum Chewing and Tooth Brushing, *Journal of Dental Research* **40**: 313

Both glass and antimony electrodes were used to measure the effect of gum chewing and tooth brushing on the pH of dental plaques following 25 per cent glucose rinses. Two groups of six subjects abstained from regular oral hygiene procedures from 3 days prior to tests to the end of each series of experiments. Two interproximal and one smooth-surface pH readings were taken in both maxillary quadrants. Chewing (10 minutes) and brushing (30 seconds) were restricted to the right quadrant. Readings were taken in most cases at 10 minute intervals. Brushing with and without water and by three brushing techniques were investigated. While pH values showed considerable variation by surface between subjects and within the same individual on different days, the following conclusions are supported by marked statistical significance. Brushing per se, with or without water, had little effect on plaque pH, In most subjects the chewing of gum* gave a marked, rapid, and sustained rise in pH.

Remineralisation and Demineralisation

Manning RH, Edgar WM (1992) Salivary Stimulation by Chewing Gum and its Role in the Remineralization of Caries-like Lesions in Human Enamel in situ, *Journal of Clinical Dentistry* **11(3)**: 71-74

Salivary stimulation elicited reflexively by taste and mastication leads to an increase in the pH, buffering power, and supersaturation of saliva, which can affect beneficially the balance between enamel de- and remineralisation in early caries. In a previous study in which a sorbitol chewing gum was used as a salivary stimulant for 20 minutes, five times daily after meals and snacks over a three-week period (during which fluoride toothpaste was used daily), significant remineralisation of caries-like lesions in human enamel attached to intraoral appliances in human subjects was observed. In view of the continued public preference for sucrose-sweetened chewing gums, the study was repeated using a sucrose gum. The mean results showed a trend toward remineralization with the use of sucrose chewing gum, which was significant in 10 subjects who chewed for 30 minutes but not in 9 who chewed for 20 minutes. The use of chewing gum after meals and snacks (in the presence of fluoride from toothpaste) can thus enhance the remineralising potential of the mouth, probably as a result of salivary stimulation.

Creanor SL, Strang R, Gilmour WH, Foye RH, Brown J, Geddes DAM, Hall AF (1992) The Effect of Chewing Gum on in situ Enamel Lesion Remineralization, *Journal of Dental Research* **71(12)**: 1895-1900

Two independent cross-over studies investigated the possibility of enhanced early enamel lesion remineralisation with the use of chewing gum. The first study involved a sorbitol-containing chewing gum, and the second, which had an identical protocol, tested a sucrose-containing chewing gum. In each study, 12 volunteers wore in situ appliances on which were mounted enamel sections containing artificial caries lesions. Subjects brushed twice daily for two min with a 1100-ppm-F (NaF) dentifrice (control and test) and in the test phase chewed five sticks of gum per day for 20 min after meals and snacks. Microradiographs of the enamel lesions were made at baseline and at the end of the seven-week experimental period. In the sugar-free gum study, the weighted mean total mineral loss (delta z) difference [(wk7-wk0) x (-1)] was 788 vol.% min. x micron for the gum, corresponding to remineralisation of 18.2%, vs. the control value of 526 vol.% min. x micron, 12.1% remineralisation (p = 0.07). There were no significant differences for the surface-zone (p = 0.20) and lesion-body (p = 0.28) values. In the sucrose-containing gum study, the delta z difference was 743 vol.% min. x micron for the gum, corresponding to a remineralization of 18.3%, vs. the control value of 438 vol.% min. x micron, 10.8% remineralisation (p = 0.08). The surface-zone values were not significantly different (p = 0.55). For the lesion body, however, the sucrose-containing gum value of 6.11 vol.% min. was significantly different (p = 0.01) from that of the control (2.81 vol.% min.).

Manning RH, Edgar WM, Agalmanyi EA (1992) Effects of Chewing Gums Sweetened with Sorbitol or a Sorbitol/Xylitol Mixture on the Remineralization of Human Enamel Lesions in situ, *Caries Research* 26: 104-109

Intra-oral remineralisation of experimental caries-like lesions in human enamel, as determined by polarised light microscopy and quantitative microradiography, was promoted to a similar extent (% fall in delta Z, 18.6 and 19.0) by chewing a sorbitol or sorbitol/xylitol (3:1)-sweetened gum for 20 min after each of three meals and two sugary snacks daily. The results suggest that reported differences in the properties of the two sweeteners do not affect their ability to enhance remineralisation due to salivary stimulation.

Edgar WM, Manning RH (1991) Xylitol and Sorbitol Gum; Comparative Enamel Remineralization Potential in situ, *Journal of Dental Research* 70: 1496

Previous work (Leach et al., *J. Dent Res*, 1989, 68, 1064) has demonstrated significant remineralisation of artificial lesions in human enamel in situ when sorbitol gum is chewed for 20 min after meals and snacks. Since xylitol is a sweetening component of many chewing gums and has been suggested to have unique remineralisation properties, it was of interest to compare the stimulus to remineralisation by use of sorbitol and xylitol sweetened chewing gum.

Nine subjects chewed one piece of gum each for 20 mins after 3 meals and 2 snacks daily for two successive periods, during which a piece of enamel containing an artificial subsurface lesion was attached to the buccal surface of the lower molar tooth (Leach et al., op cit). After 21d the enamel lesions were sectioned and their mineral content assessed by quantitative microradiography and polarizing microscopy. Xylitol and sorbitol gums were used in randomised crossover design. Fluoride dentifrice was used throughout. Compared with the baseline lesion, integrated mineral loss (ΔZ) values for lesions after chewing either gums were significantly ($p < 0.05$) reduced, but were very similar for sorbitol and xylitol gums (mean ΔZ : baseline, 2318; xylitol, 1877; sorbitol, 1888). This finding was confirmed by birefringence measurements. The results indicate that sorbitol and xylitol gums are nearly identical in their ability to enhance remineralisation of experimental early enamel lesions.

Leach SA, Lee GTR, Edgar WM (1989) Remineralization of Artificial Caries-like Lesions in Human Enamel in situ by Chewing Sorbitol Gum, *Journal of Dental Research* 68(6): 1064-1068

The objective of the study was to determine quantitatively the effect on the potential for in situ remineralization of artificial caries-like lesions in human enamel when sugar-free gum containing mainly sorbitol as sweetener was chewed after meals and snacks. Artificial whitespot lesions were created in extracted human premolars and divided into three parts. One part was used as reference and the other two worn consecutively for two 21 day periods by 10 volunteers in a cast silver band cemented on lower molar teeth and covered with gauze to promote plaque formation. During the experimental periods, the subjects used fluoridated toothpaste twice



Chewing sugarfree gum after eating significantly reduces the incidence of dental caries

daily, and consumed three meals (breakfast, lunch and dinner) and two snacks (selected from chocolate bar, raisins, chocolate wafer, and iced cupcake). Sorbitol gum was chewed for 20 min immediately after each meal or snack during one of the experimental periods. The three parts of the enamel lesions were then sectioned (80 μ m) and examined together by means of quantitative microdiagraphy and by polarized light microscopy.

All estimates of mineral content indicated that significant remineralization occurred and was approximately doubled with gum-chewing. It is suggested that sorbitol gum stimulates salivation, which is responsible for the significantly enhanced remineralization, thus contributing to a therapeutic, caries-preventive effect. Because the gum was chewed immediately after meals and snacks, inhibition of demineralization may also have occurred.

Kashket S, Yaskell T, Lopez LR (1989) Prevention of Sucrose-Induced Demineralization of Tooth Enamel by Chewing Sorbitol Gum, *Journal of Dental Research* **68(3)**: 460-462

Measurements were made of the effect of chewing sorbitol gum on the intra-oral demineralization induced by rinsing with 10% sucrose solutions. Blocks of bovine enamel were covered with a layer of *Streptococcus mutans* IB1600, and mounted on palatal appliances that were worn by five subjects for defined periods of time. Enamel demineralization was determined by following changes in iodide penetrability (delta Ip) of the enamel surfaces. Delta Ip increased to a maximum of about 15 units between 30 and 45 min, while the pH of the *S. mutans* plaque dropped to below 4 by 15 min. Plaque pH returned to 4.9 by 60 min. Chewing sorbitol gum after the sucrose rinse minimized further increases in delta Ip and brought about a more rapid return of the *S. mutans* plaque pH toward neutrality. The effect of chewing gum was greater when chewing was initiated earlier so that, when gum was given at five min after the sucrose rinse, demineralization was only 37% of that obtained without gum. The findings confirm earlier reports on the effect of gum on plaque pH, and directly demonstrate the profound protective effects that chewing sorbitol gum can have on tooth enamel.

Caries Prevention

Szoke J, Banoczy J, Proskin HM (2001) Effect of After-Meal Sucrose-Free Gum-Chewing on Clinical Caries, *Journal of Dental Research* **80(8)**: 1725-1729

Previous in situ and in vitro studies have demonstrated that the chewing of sucrose-free gum after eating reduces the development of dental caries. To investigate the extrapolation of these findings to the clinical setting, we conducted a two-year study on 547 schoolchildren in Budapest, Hungary. Subjects in the "Gum" group were instructed to chew one stick of a commercially available sorbitol-sweetened chewing gum for 20 minutes after meals, three times daily. The "Control" group was not provided with chewing gum. After two years, the "Gum" group exhibited a 38.7% reduction in incremental caries, excluding white spots, compared with the "Control" group. Including white spots, a corresponding 33.1% reduction was indicated.

These results clearly suggest that even in a moderate caries population practising normal oral hygiene, including the use of fluoride dentifrices, an after-meal gum-chewing regimen can significantly reduce the rate of caries development.

Edgar WM (1998) Sugar Substitutes, Chewing Gum and Dental Caries – A Review, *British Dental Journal* **184(1)**: 29-32

The prevalent use of chewing gum has prompted interest in its dental effects. Important defining aspects are the ability to use sugar substitutes in gum manufacture and the prolonged stimulation of a protective flow of saliva. The main sugar substitutes used are sorbitol and xylitol. Because it is not fermented by oral bacteria, xylitol is considered to be non-cariogenic, and while sorbitol in solution can be fermented slowly by mutants streptococci, chewing sorbitol-sweetened gum does not cause a fall in plaque pH. Effects of chewing sugar-free gum on the ability of plaque to form acid from sucrose are equivocal, although the tendency is for the plaque acidogenicity to be reduced with the use of xylitol gum for 2-3 weeks, due to its inhibitory effects on mutants streptococci. Gum-chewing also stimulates a protective salivary flow when used after an acidogenic stimulus, and may enhance salivary function, especially in subjects with low flow rates. Sorbitol and xylitol gums have similar beneficial effects in promoting enamel remineralisation in short-term in-situ experiments. Clinical trials indicate that xylitol gum has a useful anticaries role, superior to the effects of sorbitol gum. In conclusion, both sorbitol and xylitol chewing gums are non-cariogenic in contrast to sugared gum, and exhibit beneficial anticaries properties through salivary stimulation. In addition, xylitol's antibacterial properties seem likely to lead to caries reductions superior to the more modest reductions with sorbitol gum.

Beiswanger BB, Boneta AE, Mau MS, Katz BP, Proskin HM, Stookey GK (1998) The Effect of Chewing Sugar-free Gum After Meals on Clinical Caries Incidence, *JADA* **129**: 1623-1626

Edgar WM, Higham SM, Manning RH (1994) Saliva Stimulation and Caries Prevention, *Adv Dental Research* **8(2)**: 239-245

The protective role of saliva is demonstrated by the rampant caries seen in human subjects with marked salivary hypofunction, and in desalivated animals. In normal cases, however, the relationship between saliva flow and coronal or root caries experience is doubtful, and to examine the concept that stimulation of saliva might have protective effects against caries, one must look beyond a simple correlation between caries and flow rate. Protective properties of saliva which increase on stimulation include salivary clearance, buffering power, and degree of saturation with respect to tooth mineral. These benefits are maximised when saliva is stimulated after the consumption of fermentable carbohydrates, by reducing the fall in plaque pH leading to demineralisation and by increasing the potential for remineralisation. Plaque acid production is neutralised and experimental lesions in enamel are remineralised, when gum is chewed to stimulate saliva after a carbohydrate intake. The pH-raising effects are more easily explained by

the buffering action of the stimulated saliva than by clearance of carbohydrates. The remineralisation action depends upon the presence of fluoride. These findings suggest that the protective actions of saliva can be mobilized by appropriate salivary stimulation, and that in addition to established procedures such as tooth cleaning and fluoride regimens, eating patterns which lead to saliva stimulation to increase the potential for saliva protection might be included in recommendations for caries prevention. Confirmation of this concept in clinical tests is required.

Kandelman D, Gagnon G (1990) A 24-Month Clinical Study of: the Incidence and Progression of Dental Caries in Relation to Consumption of Chewing Gum Containing Xylitol in School Preventive Programs, *Journal Dental Research* **69(11)**: 1711-1775

The effect of chewing gum containing xylitol on the incidence and progression of dental caries was tested in a sample of 274 children, aged eight and nine years, of low socio-economic status and high caries rate. They were divided into two experimental groups (15% and 65% xylitol chewing gum distributed three times a day at school) and one control group (without chewing gum). The three groups were exposed to the same basic preventive program. Children who chewed gum had a significantly lower net progression of decay (progressions-reversals) over a 24-month period than did the controls. Results for the two groups chewing gum were similar. Chewing xylitol gum had a beneficial effect on the caries process for all types of tooth surfaces, and especially for bucco-lingual surfaces. The two experimental groups had a DMF(S) increment of 2.24 surfaces, compared with 6.06 surfaces for the control group. For this indicator, there was no difference between the two experimental groups. Results for the plaque index were in agreement with those of the DMF(S) increment and the net progression of decay.

Kandelman D, Gagnon, G (1987): Clinical Results After 12 Months from a Study of the Incidence and Progression of Dental Caries in Relation to Consumption of Chewing-gum Containing Xylitol in School Preventive Programs, *Journal of Dental Research* **66(8)**: 1407-1411

This study was designed to evaluate the effect of chewing-gum containing xylitol on the incidence and the progression of dental caries.

A sample of 433 children, aged 8 and 9 years, of low social-economic status and high caries rate, was divided into two experimental groups (15% or 65% xylitol chewing-gum distributed three times a day at school) and one control group (without chewing-gum). The three groups were exposed to the same basic preventive program.

Children who chewed gum had a significantly smaller DMF (S) increment over a 12-month period than did the control group. The former had increments of 1.58 surfaces, compared with 3.28 for the latter. No statistically significant difference, however, was demonstrated between the two experimental groups.

The net progression of decay (progressions-reversals) showed a significant difference between the two experimental groups and the control group. In addition, 65% xylitol chewing-gum produced better results than did that containing 15% xylitol, suggesting a dose response relationship. Chewing xylitol gum had a beneficial effect on the caries process for all types of tooth surfaces, but chewing gum with a higher xylitol content had an additional positive effect on buccolingual surfaces.

A questionnaire asking the participants about stomach pain indicated that there was no difference between the experimental and control groups.

The feasibility of such a preventative measure has been demonstrated by the excellent level of participation of both children and teachers. This activity could easily be integrated into existing preventive public health programs.

Glass RL (1983) A Two-Year Clinical Trial of Sorbitol Chewing Gum, *Caries Research* **17**: 365-368

A clinical trial of sorbitol chewing gum was carried out in 540 children aged 7-11 in a non fluoride area. Subjects were assigned at random to one of two groups, a no chewing group and one which chewed gum twice daily. Mean caries increments over the 2-year study period were 4.6 new DF surfaces (SD=4.8) for the sorbitol gum group and 4.7 new DF surfaces (SD=5.8) for the no gum group. Differences between groups were non significant. As daily chewing of as much as 2 sticks of gum is unusually high, these findings demonstrate that sorbitol gum is noncariogenic.

Moeller IJ (1977) Sorbitol Containing Chewing Gum and its Significance for Caries Prevention, *Disch Zahnarz* **32(1)**: S66-S70

The purpose of the present study was to determine the effect of long-term systematic chewing of sugarless, sorbitol-containing chewing gum on the incidence of dental caries, plaque and gingivitis. Eight to 12 year old children from two schools in the town of Norresundby, Denmark, participated in the study, which took place from 1968 bis 1970. A total of 174 Children from School T were given 3 pieces of sorbitol-containing chewing gum daily (Sor-bits*, identical to Ben-bits*) to be chewed after breakfast, lunch and supper; 166 children from School K acted as a control group. After two years the caries increment in children from School T was 5.6 decayed and filled surfaces per child as compared to 6.2 in children from School K. This difference (approx 10%) was statistically significant. The results indicate a depression in the caries progression rate in children who have been chewing sorbitol-containing chewing gum for 2 years. On the other hand, a series of practical circumstances in connection with the planning and performance of the study would suggest that the results obtained cannot with certainty be attributed to Sor-bits alone, but only taken as an indication thereof. Chewing of Sor-bits did not cause any change in the occurrence of gingivitis and plaque.





Richardson AS, Hole LW, McCombie F, Kolthammer J (1972) Anticariogenic Effect of Dicalcium Phosphate Dihydrate Chewing Gum: Results After Two Years, *Journal of Canadian Dental Association* **6**: 213-218

This study evaluates the clinical anticariogenic effectiveness of a sugar chewing gum containing calcium phosphate dihydrate. The 850 children who participated in the study were divided into three groups. After two years there were no statistically significant differences between the mean total caries increments of the group of children who chewed a sugar phosphate gum when compared with either the group who chewed sugarless gum or the non-chewing group.

Finn SB, Jamison HC (1967) The Effect of a Dicalcium Phosphate Chewing Gum on Caries Incidence in Children: 30 Month Results, *JADA* **74**: 987-995

A total of 606 school-age deaf and blind children at the Alabama State School for the Deaf and Blind participated in a study of the effect of a dicalcium phosphate dihydrate chewing gum on the incident of dental caries. The children were randomly assigned to groups that chewed a sugar, a sugar-phosphate, or a sugarless gum. After 30 months, 416 students were in the study, but the number of students remaining in each group was almost equal. Results after six examinations indicated a significant reduction on DFS and DMFS increments in the group that chewed the sugar-phosphate gum compared with the group that chewed the sugar gum, but there was a more significant difference and reduction in dental caries in the former group compared with the latter group when only proximal lesions were considered. The significant reduction in caries increment in the group that chewed the sugar-phosphate gum compared with the group that chewed the sugar gum was greater than the reductions in the group that chewed the sugarless gum compared with the group that chewed the sugar gum. Differences between the groups that chewed sugarless and sugar-phosphate gum were not significant.

Management of Xerostomia

Thornhill MH (1998) Care of the Dry Mouth. *Dentistry Monthly* 46-9

Dry mouth, or xerostomia, is a common complaint and dentists are often the first health professional with whom patient discuss the problem. It is important, therefore, for practitioners to be aware of both the causes and treatments of this disturbing condition.

Jensen JL, Langberg CW (1997): Temporary hyposalivation induced by radiation therapy in a child. *Tidsskr Nor Loegeforen* 21: 3077-9

This case report deals with the diagnosis and treatment of hyposalivation after radical radiation therapy and cytotoxic treatment of a metastasising nasopharyngeal carcinoma in an eight-year old girl. After cancer treatment the patient suffered from xerostomia, and pronounced hyposalivation was demonstrated. Frequent chewing of sugar-free gum and use of lozenges was recommended, and the patient was followed up for one year. During this time, the values

for unstimulated whole saliva increased by a factor of five, and stimulated whole saliva values increased as well, but less so. Two years after cancer treatment, the patient no longer suffers from xerostomia.

Odusola F (1991) Chewing Gum as Aid in Treatment of Hyposalivation, *NYSMJ* **57(4)**: 28-31

Chewing gum is more than a popular confection. In people suffering from dry mouth, chewing gum produces transitory relief from symptoms by stimulating residual functional salivary glands to increase flow rates. Chewing sugarless gum also reduces the risk of carious attack secondary to dry mouth by neutralizing acid production in plaque.

Abelson DC, Barton J, Mandel IR (1990) The Effect of Chewing Sorbitol-Sweetened Gum on Salivary Flow and Cemental Plaque pH in subjects with low Salivary Flow, *Journal of Clinical Dental* **2**: 3-5

The purpose of this work was to study the effect of chewing a sorbitol-sweetened gum on whole and parotid salivary flow rates, and on the cemental plaque pH response to a sucrose rinse challenge, in subjects with low salivary flow. The results show that chewing a flavoured sugarless gum significantly increases salivary flow rates in individuals with dry mouth. Additionally, chewing the sorbitol-sweetened gum effectively prevents the fall in cemental plaque pH generally seen in response to a sucrose challenge. This indicates that chewing a sorbitol-sweetened gum provides a palliative and possibly a protective benefit for people who suffer from dry mouth.

Markovic N, Abelson DC, Mandel ID (1988) Sorbitol Gum in Xerostomics: The Effect on Dental Plaque pH and Salivary Flow Rates, *Gerodontology* **7(2)**: 71-75

Adequate salivary flow is important for patient comfort and maintenance of oral health. Xerostomia, or dry mouth, is a common clinical complaint. Masticatory and gustatory activity can stimulate salivary flow from functional salivary tissue and the use of sugarless mints and gums have been recommended to individuals who complain of xerostomia, but there are minimum clinical data. A clinical study assessing the effect on salivary flow rates and dental plaque pH of a sorbitol-sweetened chewing gum in subjects with the complaint of xerostomia was conducted. The chewing of the gum in this present study stimulated salivary flow in the subjects with xerostomia. Statistically significant stimulated whole mouth and parotid salivary flow rate increases were found when compared to unstimulated whole mouth and parotid salivary flow rates. Chewing of the sorbitol-sweetened gum also effectively reduced the drop in pH seen following the exposure to a fermentable carbohydrate. The findings of this present study indicate that chewing of a sorbitol-sweetened gum may be of benefit to patients with the complaint of xerostomia.

Whitening

Kleber CJ, Moore MH, Putt MS, Milleman JL (2000) Effect of Carbamide/Baking Soda Chewing Gum on *in vitro* Stain Removal/Formation, *Journal of Dental Research* **79**: 2602

Dirol White (also called V6) chewing gum containing carbamide and baking soda (C/BS) was evaluated *in vitro* for both stain removal and inhibition of stain formation. Stain removal was conducted starting with stained bovine teeth (*JDR* **61**:1236, 1982) which were treated for 20, 60, and 120 minutes with the C/BS gum using a mastication device to stimulate human chewing (*JDR* **60**:109, 1981). Stain inhibition was tested using stain-free teeth which were treated with water (control) or C/BS gum 20 min/day for 7, 10, and 14 days using the mastication device. Between daily treatments, the teeth were subjected to a coffee/tea/red wine/bacteria stain formation process. In both studies, stain was measured colorimetrically using the L*a*b* colour scale and **DE**. Mean \pm SD changes and significant differences in the **DE** stain scores (n=8 teeth/group) were:

Treatment	Stain Removal by C/BS Gum		Number of days	AE Stain Formation (p>0.001)	
	AE	Significance		Control	C/BS Gum
Time (min)					
20	0.8 \pm 0.3	p< 0.001	7	26.1 \pm 3.3	19.5 \pm 2.2
60	1.1 \pm 0.4	p< 0.001	10	29.8 \pm 2.0	21.1 \pm 1.7
120	1.3 \pm 0.5	p< 0.001	14	30.6 \pm 2.2	21.9 \pm 1.5

The C/BS gum significantly removed extrinsic stain from the teeth after 20, 60 and 120 minutes of mechanical chewing. Compared to the control, the C/BS gum significantly inhibited stain formation by over 25%. The changes in both studies were visually obvious. In conclusion, a carbamide/baking soda chewing gum was significantly effective in removing existing stains from teeth and inhibiting the formation of new stains *in vitro*.

Kleber CJ, Milleman JL, Putt MS, Nelson B.J, Proskin HM (1998) Clinical Effect of Baking Soda Chewing Gum on Plaque and Gingivitis, *Journal of Dental Research* **77** Spec. Issue A, p. 290: 1473

The effect of a 5% baking soda chewing gum (BSCG) on plaque and gingivitis when used as an adjunct to daily toothbrushing was investigated in a 1 month clinical trial. At baseline, 88 subjects with mild gingivitis were stratified into 4 groups balanced for gingivitis and 24 hour plaque. They then chewed 2.6 g of the BSCG for 20 min either 0 (control), 1, 2, or 3 times a day In addition to once daily brushing. Subjects were examined for oral health, plaque, and gingivitis after 1, 2, and 4 wks. Compared to the control, the BSCG significantly reduced plaque after 1 wk, with increased reductions at 2 and 4 wks. Gingivitis also decreased over time, but significant effect was observed only after 4 wks.

Although increasing BSCG usage from 1 to 2 or 3 times daily provided no further improvement in 24-hr plaque, a trend towards reduced gingivitis was observed. In conclusion, a sugarless chewing gum containing 5% baking soda was safe and effective in removing plaque and reducing gingivitis and may serve as a significant complement to daily toothbrushing, especially for people with gingivitis.

Yankell SL, Emling RC (1997) Efficacy of Chewing Gum in Preventing Extrinsic Tooth Staining, *Journal of Clinical Dentistry* **8**: 169-172

The purpose of this six-week clinical study was to determine the efficacy of sugar-free chewing gum versus no chewing on preventing Peridex (0.12% chlorhexidine)-associated stain. One-hundred and fifty healthy adult subjects, categorized by tea or coffee intake and smoking, were randomly assigned to a chewing or no chewing gum group. All subjects were given Peridex and an ADA-approved toothbrush and fluoride toothpaste to use twice a day. Gum was chewed for 20 minutes five times each day, after toothbrushing and Peridex rinse in the morning and evening, and after each meal. At baseline, all subjects received a professional cleaning to remove all supragingival deposits and extrinsic strain. At three and six weeks, safety and stain intensity and area were monitored on the anterior teeth and posterior Ramfjord teeth using the Lobene stain scoring method. Seventy-two subjects in each group completed the study. Attrition was unrelated to product use. No untoward reactions were reported or observed at any time in the study. At the six-week evaluations, the chewing gum group exhibited significantly lower ($p < 0.05$ - 0.001) total stain scores on both anterior and posterior areas evaluated compared to the no chewing group scores. In addition to the stain evaluations, a randomly selected subset of 60 subjects was evaluated for gingivitis at baseline prior to cleaning, and at three and six weeks, on the buccal and lingual surfaces of the Ramfjord teeth. Both the chewing gum and no chewing gum subset subjects had a significant decrease in gingivitis scores from baseline to three weeks ($p < 0.001$) and from baseline to six weeks ($p < 0.05$ - 0.001). There were no significant statistical differences between the two groups at anytime during the study on gingivitis levels. Chewing gum, after product use, did not reduce the efficacy of chlorhexidine on gingivitis scores.



Calcium

Zero D (1999) Evaluation of the New Sugar Free Chewing Gum Using an *in situ* Remineralization Model. Data on file

This study evaluated a new 'Test Gum' with calcium for its ability to remineralize early caries lesions beyond that which could be obtained with a fluoride dentifrice alone. The experiment using the 6-hour partially demineralized human enamel blocks in the *in situ* model permitted evaluation



Positive oral health
promotion in practice
should be encouraged

of the ability of the Test Gum treatment to remineralize early enamel lesions. The experiments using the 24-hour partially demineralized human enamel blocks in the in situ model permitted evaluation of the ability of the new Test Gum to remineralize more advanced enamel lesions. This type of lesion represents the extent of demineralization possible over a longer period of exposure.

The findings suggest that the Test Gum with calcium lactate was significantly more effective in remineralizing the enamel blocks than the 'Placebo Gum' for the 6-hour model. For the 24-hour model, the Test Gum treatment effect was numerically higher than the Placebo Gum, although the difference was not statistically significant. This beneficial effect is attributed to the delivery of additional calcium to dental plaque and the enamel surface thus inhibiting enamel demineralization through a common ion effect and enhancing remineralization

by providing mineral substrate. Importantly, this effect was achieved in the presence of background use of fluoride dentifrice.

[Schemehorn B \(1998\) Calcium Bioavailability and Remineralization Study R/D173, Data on file.](#)

The purpose of this study was to determine the efficacy of calcium from chewing gum to promote lesion remineralization under dynamic conditions simulating in vivo caries formation. Apart from a few exceptions, the model used is the one used for testing dentifrice remineralization potentials for the American Dental Association seal of acceptance. Enamel specimens were removed from extracted human teeth and prepared. Twelve specimens were prepared for each group. Artificial lesions were formed in the enamel specimens to an average depth of 40-70 μ . One half of each specimen was covered with nail varnish to serve as a control.

Pooled wax-stimulated saliva from five humans was used as the remineralization medium in most of the cases.

It was found that addition of calcium to the remineralization system (artificial saliva) will improve the remineralization potential of enamel lesion. It was noted that the addition of calcium to some of these test systems resulted in remineralization in two weeks that is approaching the level promoted by 1000 ppm fluoride dentifrice in four weeks in the same model. These dentifrice systems typically show clinical caries reduction in the 30-40% range. Although not a replacement for fluoride therapy, the use of a calcium-containing gum has the potential to aid in remineralization of early enamel lesions.

Richardson AS, Hole LW, McCombie F, Kolthammer J (1972) Anticariogenic Effect of Dicalcium Phosphate Dihydrate Chewing Gum: Results After Two Years, *Journal of Canadian Dental Association* **38(6)**: 213-218

This study evaluates the clinical anticariogenic effectiveness of a sugar chewing gum containing dicalcium phosphate dihydrate. The 850 children who participated in the study were divided into three groups. After two years there were no statistically significant differences between the mean total caries increments of the group of children who chewed a sugar phosphate gum when compared with other the group who chewed sugarless gum or the non-chewing group.

Pickel FD, Bilotti A (1965) The Effects of a Chewing Gum Containing Dicalcium Phosphate on Salivary Calcium and Phosphate, *Ala. Journal of Medical Science* **2(3)**: 286-287

Solutions of calcium and phosphate ions have previously been shown to reharder acid-softened enamel of human teeth in vitro. In vivo, one method of supplementing the salivary calcium and phosphorus might be to use a chewing gum containing dicalcium phosphate. This study describes the release of calcium and phosphate from a dicalcium phosphate-containing chewing gum formulated as a potential anticaries agent. The study involved two subjects chewing a gum containing 7.5% dicalcium phosphate dehydrate, 30% gum base, and 62.5% corn syrup and sucrose. Ten serial samples of their saliva were collected separately during a 20 to 30 minute period of chewing the gum. Each sample was analysed.

It was found that chewing the dicalcium phosphate gum produced a higher concentration of salivary calcium and phosphate than when chewing the control gum. The investigators found that the elevations in calcium and phosphate while chewing the gum were due to suspension of the dicalcium phosphate in the saliva rather than an increase in ionic calcium and phosphate. The researchers conclude that they have established that chewing gum can be used to maintain elevated calcium and phosphate levels in saliva in humans. The anticaries potential of such gum should be explored by clinical trial.

References

A review of the positive effects of chewing sugarfree gum on oral health - pages 2-9

- 1 Edgar WM, Bibby BG, Mundorff S, Rowley J (1975) Acid production in plaques after eating snacks: modifying factors in foods. *J Amer Dent Assoc* 90: 418-25
- 2 Rugg-Gunn AJ, Edgar WM, Jenkins GN (1978) The effect of eating some British snacks upon the pH of human dental plaque. *Br Dent J* 145: 95-100
- 3 Glass RL (1981) Effects on dental caries incidence of frequent ingestion of small amounts of sugars and stannous EDTA in chewing gum. *Caries Res* 15: 256-62
- 4 Baron HJ (1981) Modifying the cariogenicity of foods with dicalcium phosphate. *Foods, Nutrition and Dental Health*. Hefferen JJ, Koehler HM, Eds., Pathotox Publishers Inc., Illinois, pp. 61-8
- 5 Volker JF (1948) The effect of chewing gum on the teeth and supporting structures. *J Amer Dent Assoc* 36: 23-7
- 6 Toto PD, Rapp G, O'Malley J (1960) Clinical evaluation of chewing gum in gingivitis and dental care. *J Dent Res* 39: 750-1
- 7 Slack GL, Duckworth R, Scheer B, Brandt R, Allanon MC (1972) The effect of chewing gum on the incidence of dental diseases in Greek children. *Brit Dent J* 133: 371-7
- 8 Graf H (1971) The glycolytic activity of plaque and its relation to hard tissue pathology - recent findings from intraoral pH telemetry research. *Internat Dent J* 20: 426-35
- 9 Schneider P, Mühlemann HR (1976) Sckerfreie zahnschonende Kaugummis und Bonbons. Stand nach 7 jährigen Untersuchung. *Schweiz Monatschrift Zahnheilkund* 86: 150-66
- 10 Maiwald HJ, Banoczy J, Tietze W, Toth Z, Vegh A (1982) Die beeinflussung des plaque-pH durch zuckerhaltigen und zuckerfreien Kaugummi. *Zahn-Mund-Kieferheilk* 70: 598-60.
- 11 Finn SB, Jamieson HC (1967) The effect of a dicalcium phosphate chewing gum on caries incidence in children: 30 month result. *J Amer Dent Assoc* 74: 987-95
- 12 Scheinin A, Mäkinen KK, Tammsalo E, Rekola M (1975) Turku sugar studies XVIII. Incidence of dental caries in relation to 1-year consumption of xylitol chewing gum. *Acta Odont Scand* 33: 269-78
- 13 Möller U, Poulsen S (1973) The effect of sorbitol-containing chewing gum on the incidence of dental caries, plaque and gingivitis in Danish schoolchildren. *Community Dent Oral Epidemiol* 1: 58-67
- 14 Isokangas P, Alanen P, Tieckso J, Mäkinen KK (1988) Xylitol chewing gum in caries prevention: a field study in children. *J Amer Dent Assoc* 117: 315-20
- 15 Kandelman D, Gagnon G (1990) A 24-month study of the incidence and progression of dental caries in relation to consumption of chewing gum containing xylitol in school preventive programs. *J Dent Res* 69: 1771-5
- 16 Beiswanger BB, Boneta AE, Mau MS, Katz BM, Proskin HM, Stookey GK (1998). The effect of chewing sugar-free gum after meals on clinical caries incidence. *J Amer Dent Assoc* 129: 1623-6
- 17 Szóke J, Proskin HM, Banoczy J (1999) Effect of after-meal sugar-free gum chewing on clinical caries. *J Dent Res* 78(Special Issue): (Abstract # 3118)
- 18 Dawes C, Macpherson LM (1992) Effects of nine different chewing gums and lozenges on salivary flow rates and pH. *Caries Res* 26: 176-182
- 19 Rugg-Gunn AJ, Edgar WM, Geddes DAM, Jenkins GN (1975) The effect of different meal patterns upon plaque pH in human subjects. *Brit Dent J* 139: 351-6
- 20 Geddes DA M, Edgar WM, Jenkins GN, Rugg-Gunn AJ (1977) Apples, salted peanuts and plaque pH. *Brit Dent J* 140: 317-9
- 21 Edgar WM, Bowen WH, Amsbaugh S, Monell-Torrens E, Brunelle J (1982) Effects of different eating patterns on dental caries in the rat. *Caries Res* 16: 384-9
- 22 Higham SM, Edgar WM (1989) Effects of paraffin and cheese chewing on human dental plaque pH and metabolism. *Caries Res* 23: 42-8
- 23 Hein JW, Soparkar PM, Quigley GA (1961) *J Dent Res* 40: 753-4 (Abstract)
- 24 Soparkar PM, Newman MB, Hein JW (1978) Comparable effects of saccharine and aspartame sweetened sugarless chewing gums on plaque pH. *J Dent Res* 57: 196 (Abstract)
- 25 Jensen ME (1986) Responses of interproximal plaque pH to snack foods and effect of chewing sorbitol-containing gum. *J Amer Dent Assoc* 113: 262-6
- 26 Jensen ME (1986) Effects of chewing sorbitol gum and paraffin on human interproximal plaque pH. *Caries Res* 20: 503-9
- 27 Jensen M E, Wefel JS (1989) Human plaque pH responses to meals and the effects of chewing gum. *Brit Dent J* 167: 204-8
- 28 Park KK, Schemehorn BR, Bolton JW, Stookey GK (1990) Effect of sorbitol gum chewing on plaque pH response after ingesting snacks containing predominantly sucrose or starch. *Amer J Dent* 3: 185-92

- 29 Park KK, Schemehorn BR, Bolton JW, Stookey GK (1990) The impact of chewing sugarless gum on the acidogenicity of fast-food meals. *Amer J Dent* 3: 231-5
- 30 Maiwald HJ, Beu M (1990) Die kariespräventive wirkung von zuckerhaltigem kaugummi. *Ernährungsforschung* 35: 46-8
- 31 Fröhlich S, Maiwald HJ, Flowerdew G (1992) Effect of gum chewing on the pH of dental plaque. *J Clin Dent* 3: 75-8
- 32 Manning RH, Edgar WM (1993) pH changes in plaque after eating snacks and meals, and their modification by chewing sugared- or sugar-free gum. *Brit Dent J* 174: 241-4
- 33 Dodds MW J, Hsieh SC, Johnson DA (1991) The effect of increased mastication by daily gum-chewing on salivary gland output and dental plaque acidogenicity. *J Dent Res* 70: 1474-8
- 34 Wennerholm K, Arends J, Birkhed D, Ruben J, Emilson CG, Dijkman AG (1984) Effect of xylitol and sorbitol in chewing-gums on mutans streptococci, plaque pH and mineral loss of enamel. *Caries Res* 28: 48-54
- 35 Leach SA, Lee GTR, Edgar WM (1989) Remineralization of artificial caries-like lesions in human enamel in situ by chewing sorbitol gum. *J Dent Res* 68: 1064-8
- 36 Manning RH, Edgar WM (1992) Salivary stimulation by chewing gum and its role in the remineralization of caries-like lesions in human enamel in situ. *J Clin Dent* 3: 71-4
- 37 Creanor SL, Strang R, Gilmour WH, Foye RH, Brown J, Geddes DAM, Hall AF (1992) The effect of chewing gum use on in situ enamel lesion remineralization. *J Dent Res* 71: 1895-1900
- 38 Kashket S, Yaskell T, Lopez LR (1989) Prevention of sucrose induced demineralisation of tooth enamel by chewing sorbitol gum. *J Dent Res* 68: 460-2
- 39 Steinberg LM, Odusola F, Mandel ID (1992) Remineralising potential, antiplaque and antigingivitis effects of xylitol and sorbitol sweetened chewing gum. *Clin Prev Dent* 14: 31-4
- 40 Gelhard TBFM, Arends J (1984) In vivo remineralization of artificial subsurface lesions in human enamel. I. *J Biol Buccale* 12: 49-57
- 41 Manning RH, Edgar WM (1998) In situ de- and remineralisation of enamel in response to sucrose chewing gum with fluoride or non-fluoride dentifrices. *J Dent* 26: 665-8
- 42 Mouton C, Scheinin A, Mäkinen KK (1975) Effect of a xylitol chewing gum on plaque quantity and quality. *Acta Odontol Scand* 33: 251-7
- 43 Topitsoglou V, Birkhed D, Larsson L-Å, Frostell G (1983) Effect of chewing gums containing xylitol, sorbitol or a mixture of xylitol and sorbitol on plaque formation, pH changes and acid production in human dental plaque. *Caries Res* 17: 369-78
- 44 Söderling E, Mäkinen KK, Chen C-Y, Pape HR, Loesche W, Mäkinen P-L (1989) Effect of sorbitol, xylitol and xylitol/sorbitol chewing gums on dental plaque. *Caries Res* 23: 378-84
- 45 Assev S, Vegarud G, Rølla G (1980) Growth inhibition of *Streptococcus mutans* strain OMZ 176 by xylitol. *Acta Pathol Microbiol Scand Sect B* 88: 61-6
- 46 Assev S, Rølla G (1984) Evidence for presence of a xylitol phosphotransferase system in *Streptococcus mutans* OMZ 176. *Acta Pathol Microbiol Immunol Scand Sect B* 92: 89-92
- 47 Trahan L, Bareil M, Gauthier L (1985) Transport and phosphorylation of xylitol by a fructose phosphotransferase system in *Streptococcus mutans*. *Caries Res* 19: 55-63
- 48 Mäkinen KK, Bennett CA, Hujoel PP, Isokangas PJ, Isotupa KP, Pape HR, Mäkinen PL (1995) Xylitol chewing gums and caries rates: a 40-month cohort study. *J Dent Res* 74: 1904-13
- 49 Mäkinen KK, Mäkinen P-L, Pape HR, Allen P, Bennett CA, Isokangas PJ, Isotupa KP (1995) Stabilisation of rampant caries: polyol gums and arrest of dentine caries in two long-term cohort studies in young subjects. *Internat Dent J* 45: 93-107
- 50 Isokangas P, Tieckso J, Alanen P, Mäkinen KK (1989) Long-term effect of xylitol chewing gum on dental caries. *Community Dent Oral Epidemiol* 17: 200-3
- 51 Isokangas P, Mäkinen KK, Tieckso J, Alanen P (1993) Long-term effect of xylitol chewing gum in the prevention of dental caries: a follow-up 5 years after termination of a preventive program. *Caries Res* 27: 495-8
- 52 Virtanen JI, Bloigu RS, Larmas MA (1996) Timing of first restorations before, during and after a preventive xylitol trial. *Acta Odontol Scand* 54: 211-6
- 53 Hujoel PP, Mäkinen KK, Bennett CA, Isotupa KP, Isokangas PJ, Allen P, Mäkinen P-L (1999) The optimum time to initiate habitual xylitol gum-chewing for obtaining long-term caries prevention. *J Dent Res* 78: 797-803
- 54 Loesche WJ, Grossman NS, Earnest R (1984) The effect of chewing xylitol gum on the plaque and saliva levels of *Streptococcus mutans*. *J Amer Dent Assoc* 108: 587-92
- 55 Aguirre-Zero Q, Zero DT, Proskin HM (1993) Effect of chewing xylitol chewing gum on salivary flow rate and the acidogenic potential of dental plaque. *Caries Res* 27: 55-9
- 56 Arends J, Christoffersen J, Schuthoff J (1984) Influence of xylitol on demineralisation of enamel. *Caries Res* 18: 296-301
- 57 Smits MT, Arends J (1985) Influence of xylitol- and fluoride-containing toothpaste on the remineralisation of surface softened enamel defects in vivo. *Caries Res* 19: 528-35

- 58 Smits MT, Arends J (1988) Influence of extraoral xylitol and sucrose dippings on enamel demineralisation in vivo. *Caries Res* 22: 160-5
- 59 Manning RH, Edgar WM, Agalarnyi EA (1992) Effects of chewing gum sweetened with sorbitol or a sorbitol/xylitol mixture on the remineralisation of human enamel in situ. *Caries Res* 26: 104-9
- 60 Lind V, Stelling E, Nystrom S (1961) Fluorhaltigt tuggummi som kariesprofylaktikum. *Odont Revy* 12: 341-7
- 61 Richardson AS, Hole LW, McCombie F, Kolthammer J (1972) Anti-cariogenic effect of dicalcium phosphate dihydrate chewing gum: results after two years. *J Can Dent Assoc* 38: 213-8
- 62 Finn SB, Frear RA, Liebowitz R, Morse W, Manson-Hing L, Brunnelle J (1978) The effect of sodium trimetaphosphate as a chewing gum additive on caries increment in children. *J Amer Dent Assoc* 96: 651-5
- 63 Kleber CJ (1986) Plaque removal by a chewing gum containing silicate. *Compend Cont Educ Dent* 7: 681-5
- 64 Ainamo J (1987) Prevention of plaque growth with chewing gum containing chlorhexidine acetate. *J Clin Periodontol* 14: 524-7
- 65 Ennever J, Sturzenberger OP (1961) Inhibition of dental calcula formation by use of an enzyme chewing gum. *J Periodontol* 32: 331-8
- 66 Emslie RD, Cross WG, Blake GC (1962) A clinical trial of an ascorbic acid-peroxide preparation and penicillin chewing gum in the treatment of acute ulcerative gingivitis. *Br Dent J* 112: 320-3
- 67 Ainamo J, Sjoblom M, Ainamo A, Tainen L (1977) Growth of plaque while chewing sucrose and sorbitol flavored gum. *J Clin Periodontol* 4: 151-60
- 68 Addy M, Perriam E, Sterry A (1982) Effects of sugared and sugar-free chewing gum on the accumulation of plaque and debris on the teeth. *J Clin Periodontol* 9: 346-54
- 69 Edgar WM (1998) Sugar substitutes, chewing gum and dental caries - a review. *Brit Dent J* 184: 29-32
- 70 Itthagarun A, Wei SH (1997) Chewing gum and saliva in oral health. *J Clin Dent* 8: 159-62

Saliva - its role in maintaining oral health and preventing dental disease - pages 10-11

- 1 Dawes C, Macpherson LMD (1992) Effects of Nine Different Chewing Gums and Lozenges on Salivary flow rate and pH. *Caries Res*. 26: 176-182
- 2 Edgar WM (1990) Saliva and Dental health. *Br Dent J*. 169: 96-98 Saliva, foods and dental caries - pages 12-13
- 1 Manning RH, Edgar WM (1993) pH changes in plaque after eating meals, and their modification by chewing sugared or sugar-free gum. *Brit Dent J*. 174: 241-244
- 2 Leach SA, Lee GTR, Edgar WM (1989) Remineralisation of artificial caries-like lesions in human enamel in situ by chewing sorbitol gum. *J Dent Res*. 69: 1064-1068
- 3 Moller U, Poulsen S (1973) The effect of sorbitol-containing chewing gum on the incidence of dental caries, plaque and gingivitis, in Danish school children. *Community Dent Oral Epidemiol*. 1: 58-67
- 4 Makinen KK, Bennett CA, Hujoel PP et al (1995) Xylitol chewing gums and caries rates: A 40-month cohort study. *J Dent Res* 74: 1904-1913

Xerostomia: care and management - pages 14-15

- 1 Billings RJ (1989) Studies on the prevalence of xerostomia. Preliminary results. *Caries Res*. 23: Abstract 124, 35th ORCA Congress
- 2 Whelton H (1996) The Anatomy and Physiology of Salivary Glands. In: Edgar WM, O'Mullane DM. ed. *Saliva and Oral Health*. BDA, London. pp 1-8
- 3 Dawes C (1996) Factors influencing Salivary flow rate and composition. In: Edgar WM, O'Mullane DM. ed. *Saliva and Oral Health*. BDA, London. pp 27-41
- 4 Greenspan D, Daniels TE (1987) Effectiveness of pilocarpine in postradiation xerostomia. *Cancer*. 59: 1123-1125
- 5 Rieke JW, Hafermann MD, Johnson JT et al (1995) Oral pilocarpine for radiation-induced xerostomia: integrated efficacy and safety results from two prospective randomised clinical trials. *Int J Rad Onc Biol Phys*. 31: 661-669
- 6 Fox PC, van der Ven PF, Baum BJ, Mandel ID (1986) Pilocarpine for the treatment of xerostomia associated with salivary gland dysfunction. *Oral Surgery, Oral Medicine, Oral Pathology*. 61: 243-248
- 7 Olason H, Axell T (1991) Objective and subjective efficacy of saliva substitutes containing mucin and carboxymethylcellulose. *Scand J Dent Res*. 99: 316-319
- 8 Aagaard A, Godiksen G, Teglers PT, Schindt M, Glenert U (1992) Comparison between new saliva stimulants in patients with dry mouth: a placebo-controlled double blind crossover study. *J Oral Path and Med*. 21: 376-380
- 9 Rishheim H, Arneberg P (1993) Salivary stimulation by chewing gum and lozenges in rheumatic patients with xerostomia. *Scand J Dent Res*. 181: 40-43
- 10 Dawes C, Macpherson LMD (1992) Effects of Nine Different Chewing Gums and Lozenges on Salivary Flow Rate and pH. *Caries Res*. 26: 176-182
- 11 Bjornstrom M, Axell T, Birkhed D (1990) Comparison between saliva stimulants and saliva substitutes in patients with symptoms related to dry mouth. A multi-centre study. *Swed Dent J*. 14: 153-161

A Good Oral Care Routine

Maintain a healthy oral care routine by following these five easy steps:

1

CHECK-UP



Visit your dentist regularly

2

BRUSH



Brush your teeth twice a day

3

FLOSS



Floss daily

4

CHEW



Chew sugarfree gum after eating and drinking when “on-the-go”

5

HEALTHY DIET



A balanced diet that is low in sugars is essential to maintaining healthy teeth

.....

*Working for better
oral healthcare*

The Wrigley Company PTY. LTD

Locked Bag 3355
Hornsby NSW 1630
Australia

www.wrigley.com.au