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.

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.

Introduction: 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 1.2-1.5 at 20ml/min.[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 PO4-. 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.

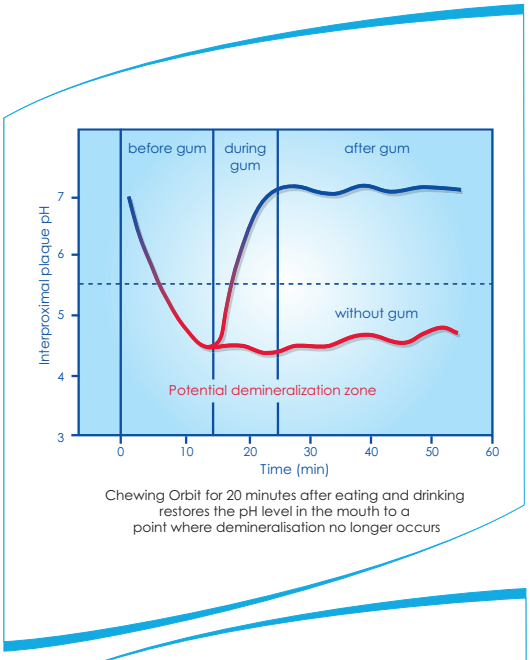
The action of stimulated saliva is most important during the plaque acid attack during the 20-30 min after a cariogenic food intake. However, with most foods, salivary stimulation ceases shortly after swallowing, and salivary composition returns to normal within about 5 min, and so the protective effects 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.

Consumption of cheese[19] and peanuts[20] 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]

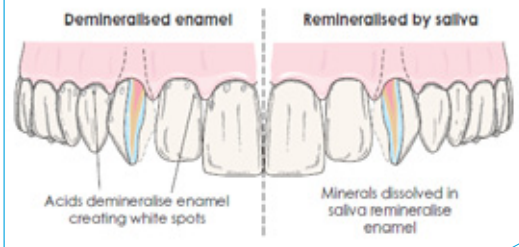
Advice to eat cheese or peanuts after meals and snacks to reduce caries would however lead to an unacceptable increase in dietary fat. The effect of saliva stimulation on plaque pH can be achieved by non-food stimuli such as paraffin wax.[22]

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.



Chewing Orbit for 20 minutes after eating and drinking restores the pH level in the mouth to a point where demineralisation no longer occurs



Remineralisation of the enamel lesions occurred both with and without gum, but with gum the remineralisation was approximately doubled. A similar experiment[36] 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 process[40] 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.

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 plaque[54] 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] 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 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 phosphate[11-61] and sodium trimetaphosphate[62] to reduce the potential cariogenicity of sucrose in gum. In addition, silicates[63] and chlorhexidine acetate[64] have been added to reduce plaque and gingivitis, pancreatic enzymes[65] 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]

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.

References:

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 Healt. Hefferen J J, Koehler H M, 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, Ailianon 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. Schweitz Monatschrift Zahnheilkund 86: 150-66
10. Maiwald HJ, Banoczy J, Tietze W, Toth Z, Vegh A (1982) Die beeinflüssung des plaque-pH durch zuckerhältigen 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, Tammisalo 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 IJ, 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
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. 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
28. 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
29. Maiwald HJ, Beu M (1990) Die kariespräventive wirkung von zuckerhaltigem kaugummi. Ernärungsforschung 35: 46-8
30. Fröhlich S, Maiwald HJ, Flowerdew G (1992) Effect of gum chewing on the pH of dental plaque. J Clin Dent 3: 75-8
31. 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
32. 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
33. 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
34. 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

35. 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
36. Creanor SL, Strang R, Gilmouur 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
37. 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
38. 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
39. Gelhard TBFM, Arends J (1984) In vivo remineralization of artificial subsurface lesions in human enamel. I. J Biol Buccale 12: 49-57
40. 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
41. Mouton C, Scheinin A, Mäkinen KK (1975) Effect of a xylitol chewing gum on plaque quantity and quality. Acta Odontol Scand 33: 251-7
42. 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
43. 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
44. Assev S, Vegarud G, Rölla G (1980) Growth inhibition of Streptococcus mutans strain OMZ 176 by xylitol. Acta Pathol Microbiol Scand Sect B 88: 61-6
45. Assev S, Rölla 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
46. 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
47. 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
48. 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
49. 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
50. 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
51. 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
52. 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
53. 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
54. Aguirre-Zero O, 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
55. Arends J, Christoffersen J, Schuthoff J (1984) Influence of xylitol on demineralisation of enamel. Caries Res 18: 296-301
56. 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
57. Smits MT, Arends J (1988) Influence of extraoral xylitol and sucrose dippings on enamel demineralisation in vivo. Caries Res 22: 160-5
58. Manning RH, Edgar WM, Agalamanyi 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
59. Lind V, Stelling E, Nystrom S (1961) Fluorhaltigt tuggummi som kariesprofylactikum. Odont Revy 12: 341-7
60. 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
61. 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
62. Kleber CJ (1986) Plaque removal by a chewing gum containing silicate. Compend Cont Educ Dent 7: 681-5
63. Ainamo J (1987) Prevention of plaque growth with chewing gum containing chlorhexidine acetate. J Clin Periodontol 14: 524-7
64. Ennever J, Sturzenberger OP (1961) Inhibition of dental calculusa formation by use of an enzyme chewing gum. J Periodontol 32: 331-8
65. 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
66. 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
67. 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
68. Edgar WM (1998) Sugar substitutes, chewing gum and dental caries - a review. Brit Dent J 184: 29-32
69. Itthagarun A, Wei SH (1997) Chewing gum and saliva in oral health. J Clin Dent 8: 159-62