Milk and oral health

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Introduction

Milk is an important part of the human diet: for infants, it is the only source of essential nutrients. Its importance declines after weaning, so that young adolescents in the UK in 1997 consumed about 178g of milk per day (Gregory and Lowe, 2000). Worldwide, using data from the Food and Agriculture Organisation, dairy production and supply (total and per capita) have increased since 1980 (Wang and Li, 2008). Consumption in China has tripled since 1982. In most countries, cow’s (or ‘bovine’) milk is the most common milk consumed, and there is little information on dental properties of milk other than bovine. Human milk will not be considered in this review, but has been considered elsewhere (Rugg-Gunn, 1993).

Some of the earliest investigations regarding milk and dental caries were carried out by Sprawson (1932a,b,c, 1934, 1947), who concluded that milk improved oral health. Mellanby and Coumoulos (1944) attributed an improvement in children’s teeth in London between 1929 and 1944 to improved diet, notably the introduction of cheap milk in 1934. In 1958, the Research Committee of the Canadian Dental Association (1958) reviewed the evidence that milk consumption was associated with a reduction in caries incidence. Since then, much research, both clinical and non-clinical, has been published and, almost uniformly, milk is not seen as a cause of dental caries. The Department of Health (1989) COMA report on dietary sugars and human disease concluded that: “Although lactose alone is moderately cariogenic, milk also contains factors which protect against dental caries, so that milk without added sugars may be considered to be virtually non-cariogenic” (report section 6.7). In a review of diet, nutrition and chronic diseases, the World Health Organisation (2003) classified evidence linking diet to several diseases including dental caries: the strength of the evidence for a ‘decreased risk’ of dental caries from milk was classified as ‘possible’. Other reviews of the relation between milk consumption and oral health have been published in the USA (Tinanoff, 2000; Merritt et al., 2006; Dietrich et al., 2009), UK (Moynihan, 2000) and Sweden (Johansson, 2002) and these comment on the favourable role of milk in the control of oral disease. In a briefing paper in the British Dental Journal, Levine (2001) concluded that “milk and milk products, if unsweetened by added sugars, are safe and possibly beneficial for teeth.” Milk is considered a suitable vehicle for substances beneficial to oral health, principally fluoride (Bánóczy et al., 2009), probiotics (Meurman, 2009; Stecksén-Blicks et al., 2009; Petersson et al., 2010) and possibly vitamin D (Dietrich et al., 2009).
**Review of evidence – milk and oral health.**

Eighty per cent of carbohydrate in milk is lactose. Various other components of milk have been considered to be protective against dental caries, namely the minerals, casein and other proteins and lipids. Cow's milk contains about 4.8g lactose per 100g milk. This amount could be sufficient to classify milk as cariogenic, but there is much evidence that lactose is the least cariogenic of the common dietary sugars (Rugg-Gunn, 1993). In addition, the high concentrations of calcium and phosphorus in milk will help to prevent dissolution of enamel (which is largely calcium and phosphate) and other factors, principally casein, is likely to be protective as well. Thus, it is possible that milk could be caries-promoting (due to the lactose content), caries-preventing, or somewhere between these two. This review considers these aspects.

Since yoghurt is sometimes drunk in place of milk in school programmes, the dental effects of yoghurt will be considered. Likewise, probiotics are sometimes added to milk and their dental effects will also be examined. Recently, the possibility that milk consumption may be beneficial to the prevention of dental enamel erosion and periodontal health has received attention – this evidence will be reviewed.

**Milk and dental caries – epidemiological studies.**

There appears to have been just one intervention study and about 18 observational studies into the relationship between milk consumption and dental caries. While the majority of studies identify bovine milk as such, in a few studies ‘dairy products’ are listed: these studies have been included where it is clear that the majority of the ‘dairy products’ was bovine milk. As elsewhere in this review, formula milks are not included or considered.

The one intervention study was conducted a long time ago – in 1929 – and involved 319 4-16 year-old children, living with their parents in New York, USA (Brodsky, 1933). The three intervention groups received supplements of milk, fresh fruit and vitamin D, and caries development over 13 months was compared with that of a control group. To quote the author: “With the cooperation of the parents and the supplementation of the diets with milk and fruit, we have proved that an adjustment of the diets and the arrest of caries are possible even under the poorest conditions.” “Our good diets which contained a minimum of two or three pieces of fruit daily gave similar results to the diets which contained 1 quart [~ 1.14 litres] of milk without the additional fruit.”

Two large-scale observational studies were conducted many years ago – one in the UK (Read and Knowles, 1938) and the other in New Zealand (Hewat, 1948). The UK study involved 2,894 6-13 year-old children, while the New Zealand study involved over 2,000 children aged 7-16 years. In both studies, no relationship was found between milk consumption and caries experience, although a
positive relation was recorded between consumption of sweets (sugar confectionery) and caries experience in the UK study. Read and Knowles (1938) stated “The value of milk in preventing caries does not appear to be supported.” Lacto-vegetarian children in Australia were reported by Gillman and Lennon (1958) to have lower caries experience than other children although, in their Finnish study, Linkosalo and Markkanen (1985) reported no difference in caries experience.

In observational studies of diet and disease, the potential for confounding factors to influence relationships between diet and disease is substantial and should be taken into account in study design and data analysis. There have been four observational studies where the analyses of relationships between milk consumption and dental caries experience or increment were not controlled for possible confounding factors. Of these studies, Rugg-Gunn et al. (1984) recorded a positive, but not statistically significant, relation between consumption of cow’s milk and dental caries increment: the difference between the daily milk consumption in the high caries children (269ml) and the caries-free children (242ml) was small. Mattos-Graner et al. (1998) reported that, in young Brazilian children, milk in bottles was not associated with dental caries unless sugar, or sugar and cereal, was added to the milk when the relationship became positive. Zita et al. (1959) reported a weak negative correlation (-0.08) between milk intake and caries experience, in contrast to a positive correlation of 0.77 between meal sugar intake and caries experience, while Potgieger et al. (1956) reported “a marked and consistent drop in DMF [caries] rate with increase in the number of cups of milk consumed”, in 864 10-16 year-old children in Connecticut, USA.

More recently, greater computing power has allowed routine use of multivariate statistical analyses where the relationships between diet, namely milk consumption, and caries experience can be controlled for effects of possible confounding variables. Eleven such studies have been published. In ten of the eleven studies milk consumption was associated with lower caries experience, although the results were not always clear-cut (Serra Majem et al., 1993; Petridou et al., 1996; Petti et al., 1997; Levy et al., 2003; Sohn et al., 2006; Kolker et al., 2007; Levine et al., 2007; Llena and Forner, 2008; Lim et al., 2008; Johansson et al., 2010). In the tenth study (Marshall et al., 2003) “milk had a neutral association with caries” in their study of 642 pre-school children in Iowa, USA. In the study of Serra Majem et al. (1993), 893 5-14 year-old Spanish children were examined: while skimmed milk had a “protective effect”, the effect of whole milk was neutral. Petridou et al. (1996) reported that milk and dairy products were negatively associated with dental caries in 380 Greek adolescents aged 12-17 years. The study of Petti et al. (1997) of 890 6-11 year-old Italian children who, on average, drank about 209ml of milk per day, reported an inverse relation between milk and caries that was strongest in children with the highest frequency of sucrose consumption. Levy et al. (2003) examined the relationship between caries experience of children in Iowa, USA, at age 5 years with diet during the previous years: higher milk consumption at 24-36 months was related to reduced risk of caries at 5
years. Sohn et al. (2006) analysed data from the US NHANES III study (1988-1994) which involved nearly 6,000 children aged 2-10 years: “children with a high milk consumption pattern had a tendency toward lowest caries experience”. The study of Kolker et al. (2007) of 436 3-6 year-old children in Detroit, USA, revealed that increase in milk consumption was associated with lower caries experience. Levine et al. (2007) related diet of 317 children in the UK at ages 7-11 years and at 11-14 years with caries experience at 11-14 years: moderate consumption of ‘dairy products’ was associated with less caries, although higher as well as lower frequency of consumption tended to be associated with greater caries experience. Llena and Forner (2008) examined 369 6-10 year-old Spanish children: “in general, more frequent consumption of sugary foods was associated with a rise in the caries indices, while the children who consumed non-sugared milk and dairy products more often suffered lower caries rates”. Lim et al. (2008) analysed data from the Detroit Dental Health Project: 369 children aged 3 years or over were examined in 2002-3 and 2004-5. The authors concluded: “Children who consumed more soft drinks, relative to milk and 100% fruit juice, as they grew older were at a greater risk of developing dental caries.” Johansson et al. (2010) studied 1,206 1-5 year-old children in Boston, USA: caries prevalence was considerably lower (6%) in children who consumed milk with snacks compared with children who consumed a sugared drink with snacks (29%). A number of studies have reported that consumption of milk in children to be inversely related to consumption of added sugar drinks (Harnack et al., 1999; Skinner et al., 1999; Marshall et al., 2005).

One study has examined the relationship between consumption of ‘dairy products’ and root caries experience (Papas et al. (1995). This study involved 141 adults aged 47-83 years living in Boston, USA: while there was a strong relation between consumption of ‘dairy products’ and root caries, only cheese consumption was significantly associated with lower occurrence of root caries.

In summary, while there is an absence of controlled clinical trials, on balance the results of the above epidemiological studies suggest that milk consumption is associated with lower experience of dental caries. This is especially so for the more recent studies which have examined their data using multivariate analysis. In ten out of these eleven studies, higher milk (or dairy) consumption was associated with lower caries experience. The one study to examine root caries also recorded lower caries experience with higher consumption of dairy products, although the major effect in this study would appear to be from consumption of cheese.

**Milk and dental caries – animal experiments.**

Evidence from animal experiments not only indicates that cow’s milk is non-cariogenic, but also strongly suggests an anti-cariogenic effect. The extensive studies of Schweigert et al. (1946), Shaw (1950), Dreizen et al. (1961) and Stephan (1966) labelled milk as non-cariogenic. Early indications that milk was not only non-cariogenic but anti-cariogenic (Sperling et al. 1955; Shaw et al. 1959)
were followed up by Reynolds and Johnson (1981). They found that supplementation of a cariogenic diet with cow's milk reduced substantially dental caries incidence and, importantly, that this was not due to reduced consumption of the cariogenic diet. In a review, Bowen and Pearson (1993) came to the same conclusion. The study of Shaw et al. (1959) also showed that the caries protective effect of milk was a post-eruptive effect (an intra-oral effect on erupted teeth) and not a pre-eruptive effect (an effect during tooth development). The caries-preventive effect of milk, in comparison with water, has also been reported by König (1960), Bánóczy et al. (1990) and Stösser et al. (1995). A severe test of the cariogenic or cariostatic properties of milk was developed by Bowen et al. (1991), using desalivated rats which are therefore much more caries-susceptible. In these experiments, the rats given milk or lactose-reduced milk remained essentially caries-free, while those given sucrose or lactose in water developed caries. Using the same model a few years later, Bowen et al. (1997) found a similar result regarding the very low caries potential of milk. The authors concluded “that [cow’s] milk does not promote caries, even in the highly caries-conducive environment engendered”, and “that milk or lactose-reduced milk can be used safely by hyposalivatory patients as a saliva substitute”. In a study in Brazil, Perez et al. (2002), also using desalivated rats, “concluded that cow’s milk was not cariogenic”. Bowen and Lawrence (2005) compared the cariogenicity of cow’s milk, human milk, Cola and honey in desalivated rats, once again concluding “that cow milk is essentially noncariogenic.”

Caution is always needed when extrapolating the findings of animal experiments to the human situation, and this may be important when dietary phosphate is thought to play a caries-preventive role (Harper et al., 1987; Rugg-Gunn, 1993) as is the case with milk. Although some caution is advisable, the results of the above numerous animal experiments give considerable weight to the evidence that bovine milk is non-cariogenic and may be anti-cariogenic.

**Milk and dental caries – plaque pH experiments.**

Several studies have shown that the fall in plaque pH after drinking milk is negligible (Jenkins and Ferguson, 1966; Frostell, 1970; Edgar et al. 1975; Mor and McDougall, 1977; Rugg-Gunn et al. 1985; Saeed and Al-Tinawi, 2010). In the studies of Rugg-Gunn et al. (1985), 14 volunteers rinsed their mouths with cow's milk, human milk, lactose solution, or sucrose solution. Sucrose solution caused substantial falls in plaque pH, while the milks depressed plaque pH only slightly. An exception is the report by Birkhed et al. (1993) which showed that dental plaque microflora may adapt to lactose in milk leading to a greater ability to ferment this lactose following frequent milk consumption. In their experiment, 10 subjects rinsed six times a day for four weeks with low-fat milk: the fall in plaque pH with milk consumption was substantially greater at the end of the four weeks compared with measurements before the four weeks of rinsing. The authors speculate that adaptation may have been less if the milk had been full-fat due to the protective effect of the higher casein content in full-fat
milk.

While plaque pH studies are a useful guide to the fermentability of sugars in foods, such experiments do not consider the presence and effect of substances in foods which may protect against dental caries, such as, for example, calcium, phosphate and casein in milk.

Milk and dental caries – *in vitro* studies.

Artificial caries-like lesions can be induced in small slabs of enamel by exposing their surface to acid buffers. This method has been used both in the laboratory (*in vitro*), and in the mouth (*in vivo*) where they are held by a purpose-built intra-oral appliance, to examine the ability of foods to demineralise or remineralise tooth enamel. Weiss and Bibby (1966) examined the effect of bovine milk (raw, pasteurised, whole or skimmed) and found that all the milks “reduced the solubility of enamel.” Bibby *et al.* (1980), using an artificial mouth test system (Orofax), found that the inclusion of milk solids reduced the cariogenicity of sugar-containing foods, while similar results were obtained by Thomson *et al.* (1984) using an enamel slab intra-oral device. The same team reported that flavoured milk, containing 5% sucrose, caused considerably less demineralisation compared with an apple-based fruit juice (Dever *et al.*, 1987) and bovine milk caused little demineralisation, much less than human milk (Thomson *et al.* (1996). Jensen *et al.* (2000), using a similar system which held slabs of enamel or dentine in the mouth of volunteers, showed that milk (with three levels of fat content) was not cariogenic for enamel or dentine; Gedalia *et al.* (1991) reported that milk remineralised enamel previously softened by Cola. More recently, Walker *et al.* (2006, 2009) reported that milk remineralised previously demineralised enamel slabs *in vivo*.

In addition to the plaque pH studies mentioned above, Jenkins and Ferguson (1966) conducted *in vitro* comparisons of 4 % lactose solutions and cow’s milk. They concluded that, within the limits of their experiments, their results “gave no grounds for suggesting that milk has a local effect on the teeth which would favour caries”, and suggested that the negligible fall in plaque pH was partly due to milk’s high buffering power, and the low level of dissolution of test enamel was due to the protective action of milk’s high levels of calcium and phosphate. Rugg-Gunn *et al.* (1985) also reported that both cow’s milk and human milk protected enamel from dissolution in *in vitro* experiments, compared with sucrose or lactose, but that human milk was less protective than cow’s milk, as would be expected from their different calcium and phosphorus content. Five further *in vitro* studies have investigated the caries inducing or caries preventive effect of milk. McDougall (1977) showed that (a) demineralisation of enamel in an acid buffer was reduced by intermittent exposure to milk, and (b) that milk aided the remineralisation of demineralised enamel. Mor and Rodda (1983) reported that milk remineralised artificially-induced lesions in enamel slabs. Arnold *et al.* (2003) showed that milk inhibited enamel demineralisation compared with saline or a remineralising solution, and Ivancacova
et al. (2003) reported that milk reduced the rate of root caries progression. Recently, Prabhakar et al. (2010) incubated enamel slabs with human or bovine milk in vitro, and concluded that plain bovine milk was relatively cariogenic in the absence of saliva, in contrast to the findings of many of the studies listed above.

Taken as a whole, both in vitro and in vivo enamel slab experiments suggest that bovine milk has little ability to cause demineralisation and, indeed, is capable of remineralising previously demineralised enamel.

The actions of the constituents of milk.

The principal constituents of bovine milk, other than carbohydrate (4.8%), are fat (up to 3.8%), protein (3%), calcium (118mg/100g) and phosphorus (96mg/100g), with some small variation. It has been mentioned previously that lactose is the least cariogenic dietary sugar. When Thomson et al. (1996) increased the concentration of lactose in bovine milk from 5% to 7% (the level in human milk) no increase in the already low cariogenic potential was observed.

Oral clearance is influenced by the ingredients of foods as well as by salivary flow, action of the tongue, cheeks and lips, and by other factors. One ingredient which accelerates oral clearance is fat (Bibby et al. 1951; Swenander Lanke, 1957; Frostell, 1969; Brudevold et al. 1990). This is probably due to a physical action of holding all the particles together.

Proteins are adsorbed well onto enamel surfaces. Weiss and Bibby (1966) investigated the ability of milk to reduce demineralisation in an in vitro enamel slab experiment. They found that milk components adhered to the enamel surface and that these could not be removed by washing with water. The protective effect was, however, removed by a protein solvent, and restored by application of casein alone. Muhler (1957) showed that the protective effect, which he surmised was organic, was removed by heat-treating the milk powder prior to feeding to the experimental animals: this would denature proteins. Pearce and Bibby (1966) tested 11 proteins and found that casein and globulins were adsorbed in greatest amounts, and albumins the least.

Casein is a phosphoprotein and represents about 87% of all proteins present in milk: it is considered to be one of the main ingredients responsible for the caries-protective action of milk. Decreases in the development of dental caries in rats have followed increases in the casein content of their diet (Bavetta and McClure, 1957; Holloway et al. 1961; Reynolds and Black, 1987a,b). The reasons for the caries preventive effect of caseins have been studied by Vacca-Smith et al. (1994), Vacca-Smith and Bowen (1995, 2000) and Danielsson Niemi et al. (2009). Casein appears to prevent adherence of
salivary components and bacteria to enamel and pellicle, and to reduce the activity of glucosyltransferase, thus reducing glucan formation and plaque adherence. In a series of rat caries experiments, Guggenheim et al. (1999) demonstrated the marked caries preventive effect of ‘milk micellar casein’ when incorporated into a cariogenic diet. Large reductions in the proportion of S. sobrinus were observed leading the authors to conclude that the micellar casein interfered with the adhesion of some plaque bacteria. Research into the favourable properties of casein, particularly its ability to concentrate calcium and phosphate in plaque, has led to the development of a compound known as CPP-ACP: since this is much discussed, it will be reviewed briefly in a separate section below.

Harper et al. (1987) questioned whether casein was the most caries-protective constituent in milk, following experiments in rats which tested the caries-reducing potential of three mineral-rich milk concentrates with various levels of whey protein, calcium, and phosphorus, but negligible levels of casein. The results suggested that considerable protection could be afforded by calcium and phosphate compounds in the absence of casein. In another animal experiment, Beighton et al. (1979) concluded that the increased cariogenic potential of freeze-dried milk compared with whole milk could be due to lower calcium and phosphate concentrations in their freeze-dried milk. While the caries protective effect of phosphates in the diets of rats needs to be interpreted with care (Harper et al., 1987; Rugg-Gunn, 1993), the results agree with other in vitro studies which demonstrate the favourable role of the high concentrations of calcium and phosphorus in milk (Jenkins and Ferguson, 1966; Rugg-Gunn et al. 1985). Using the in vivo enamel slab model, Thomson et al. (1996) reported that when the calcium and phosphorus concentrations in human milk (~ 22mg/100g and 10mg/100g, respectively) were raised to the concentrations found in bovine milk (~114mg/100g and 96mg/100g, respectively) demineralisation of enamel was reduced by 70%. In agreement with these findings, Bowen and Lawrence (2005) stated: “It seems, therefore, that the difference in the cariogenicity of the milk resides for the most part in the mineral content. Clearly, other factors such as casein content cannot be discounted.” Support for the protective role of the mineral content of milk came from the studies of Grenby et al. (2001) which showed, in in vitro experiments, that the “removal of lactose, fat, casein and other proteins had little influence on the protective effect of the milk fractions. Besides calcium and phosphorus, milk contains other more powerful protective factors against demineralisation, which they identified as proteose-peptone fractions 3 and 5.” The protective role of other components of milk has received some attention. Oho and workers reported that lactoferrin reduced binding of Streptococcus mutans to other cells (Mitoma et al., 2001) and to saliva-coated hydroxyapatite beads (Oho et al. 2002), while Aimutis (2004) discusses possible roles of lysozyme and lactoperoxidase, present in milk, in reducing cariogenic oral microflora.

The non-cariogenic and protective properties of bovine milk would appear to be due to several factors.
First, the lactose content is low and of limited cariogenic potential; second, the high calcium and phosphate content resists demineralisation and aids remineralisation of enamel and dentine; third, casein reduces demineralisation of tooth tissue and, finally, other components of milk may reduce the ability of plaque microflora to adhere to enamel and produce acids.

**Casein phosphopeptide-amorphous calcium phosphate (CPP-ACP).**

The preventive role of casein in milk has been discussed above. It would appear to have a dual role of being ready adsorbed onto dental enamel providing a physical barrier and as an accumulator of calcium and phosphate; its possible anti-microbial role would now seem less important. While the addition of casein to foods, such as chocolate confectionery, was successful in reducing caries in animals, the taste was unacceptable at concentrations required for effectiveness. Reynolds and colleagues, who had carried out the more recent work on casein described above, identified a casein derivative which had an acceptable taste and yet retained very strong affinity for calcium and phosphate. This derivative, casein phosphopeptide, is basically a trypsin digest of casein, after purification by ultrafiltration. Further development led to casein phosphopeptide-amorphous calcium phosphate (CPP-ACP) nanocomplexes, which are patented as Recaldent. CPP-ACP has a remarkable ability to stabilise high concentrations of calcium and phosphate under neutral and alkaline conditions, which would otherwise precipitate ready as insoluble calcium phosphate. The high concentrations of calcium and phosphate provided by CPP-ACP are available within plaque and at the tooth surface to resist tooth hydroxyapatite dissolution and aid remineralisation (Reynolds, 1997; Cochrane and Reynolds, 2009).

The favourable properties of CPP-ACP have led to the development of a wide variety of products ranging from foods and chewing gums to toothpastes, mouthrinses and dental mousses (Reynolds, 1998; Aimutis, 2004; Cochrane and Reynolds, 2009). There have been two systematic reviews of research into the clinical effectiveness of these products. The first (Azarpazhooh and Limeback, 2008) identified 10 studies of caries prevention, eight of which were randomised clinical trials. Nevertheless, they concluded that the evidence was “inconclusive”. The second systematic review (Yengopal and Mickenautsch, 2009) concluded that the results were “promising”. A very interesting commentary on these two reviews came from Bader (2010) pointing out imperfections in both reviews. Neither review considered the extensive research other than clinical trials.

Of relevance are studies of the effect of adding CPP-ACP to milk (Walker et al., 2006, 2009). Both were in vivo enamel slab experiments. While milk alone remineralised previously demineralised enamel, remineralisation was increased by the addition of CPP-ACP; both studies showed a dose-response with different concentrations of CPP-ACP. Also of relevance are studies showing that the
caries-preventing actions of CPP-ACP and fluoride are additive (Reynolds et al., 1995; Cross et al., 2004).

**Yoghurts and Probiotics.**
These will be considered briefly since yoghurt may be provided in school to some children.

Fermentation of milk leads to production of lactic acid and the resulting fall in pH inhibits growth of many pathogenic organisms. One of the most important of these fermented foods is yoghurt which has been traditionally fermented with *Lactobacillus bulgaricus*. The lactose content reduces substantially during fermentation although some galactose remains: other constituents are unchanged (Southgate, 2000).

Two observational epidemiological studies have been identified. Marshall *et al.* (2003) compared the diets of children in Iowa, USA, between the ages to 1 and 5 years with caries experience at about 5-6 years. While milk consumption had a neutral relation with caries, “high intakes of non-milk dairy foods were associated with an increased extent of caries.” However, they continue: “Yoghurt and dairy desserts are made with added sugar (eg, sucrose and/or high-fructose corn syrup) which is a substrate for oral bacteria.” The second study was by Tanaka *et al.* (2010) who compared the diets of 2058 3-year-old Japanese children with their caries experience. They reported: “compared with yogurt consumption in the lowest tertile, its intake at the highest level was significantly associated with a lower prevalence of caries, showing a clear dose-response relationship.” There is no record of these products being sweetened. An *in vivo* enamel slab study by Jensen *et al.* (2000) found that consumption of sweetened (strawberry) yogurt increased demineralisation of enamel and dentine compared with milk. However, the sugars content of this (sweetened) product was not given. In an *in vitro* enamel slab experiment, Ferrazzano *et al.* (2008) reported that yogurt with enhanced levels of naturally-occurring casein phosphopeptides inhibited demineralisation and promoted remineralisation of dental enamel.

Probiotics are defined as ‘live microorganisms which, when administered in adequate amounts, confer a health benefit on the host’ (Guarner, quoted by Meurman, 2009). Probiotic strains mainly belong to the genera *Lactobacillus* and *Bifidobacterium*. Probiotics may well have beneficial effects within the mouth (as well as in the gut) and this is likely to be due to “displacing of *Streptococcus mutans* or other cariogenic microorganisms from their binding sites on dental surfaces or in oral biofilms” (Meurman, 2009). He continues: “In future, probiotic products will inevitably be developed specifically for oral health benefits.” Research to identify which probiotic microorganisms might have the best potential for preventing oral diseases has been reviewed by Meurman (2009).
month randomised double-blind controlled clinical trial, Näse et al. (2001) reported that consumption of milk containing *Lactobacillus rhamnosus*, five days a week in day-care centres, reduced caries risk in children aged 1 to 6 years in Helsinki, Finland. In this short trial, caries risk was assessed from clinical and microbiological information.

Two randomised controlled trials of the effectiveness of adding probiotics and fluoride to milk in reducing caries increment in Swedish children and adults have been published (Stecksén-Blicks et al., 2009 and Petersson et al., 2010, respectively). In both studies, substantial caries reductions were recorded (in the adults it was rehardening of root caries). While in the four-group adult study, benefit from the addition of probiotics and fluoride, both independently and combined, was observed, in two-group child study, it was not possible to allocate the substantial effect (prevented fraction = 75%) to the probiotics, the fluoride, or both. In an *in vivo* enamel slab experiment, Lodi et al. (2010) reported that two commercial probiotic products caused demineralisation of enamel.

Information on the dental effects of yoghurt is limited. As long as sugars are not added, there is no reason for suspecting that the effects of yoghurt would differ from that of milk. Information regarding probiotics is also limited; nevertheless, published research is encouraging.

**Addition of sucrose and cocoa to milk.**

In some countries, it is common to flavour milk, especially milk for children, with sugar and other flavourings. The use of flavoured, fluoridated milk has been reported in Hungary (Bánóczy et al. 1983, 1985; Gyurkovics et al. 1992), the USA (Rusoff et al. 1962; Legett et al. 1987) and China (Bian et al. 2003). In most countries, the sugar added is sucrose and the most common other flavouring is cocoa. Current UK regulations allow only flavoured low fat milk (defined as milk with a fat content of not more than 1.8%), to be provided in schools as long as the milk component of the drink is at least 90% by volume, and contains less than 5% added sugar or honey.

It is reasonable to assume that adding sucrose to milk will increase cariogenicity, but at what concentration will the added sucrose overcome the caries-protective properties of plain milk, is an interesting question. The question is complicated by the knowledge that cocoa itself has caries-protective properties (Gustaffson et al. 1954; Rugg-Gunn, 1993).

The effect of adding sucrose to cow’s milk has been investigated in a variety of studies. In an uncontrolled, observational epidemiological survey, Mattos-Graner et al. (1998) recorded that children who had drunk milk with added sugar had higher caries experience than children who had drunk milk with no added sugar. Dunning and Hodge (1971) reported results of a two year clinical
trial in American children and young adults. Caries increment was slightly higher (of borderline statistical significance) in children drinking milk with 6% sugar added, compared with children drinking plain milk. In a case-control study of rampant (extensive) caries in young Chinese children, Ye et al. (1999) reported that, after multivariate analyses, bottle feeding with sugar-containing bovine milk was associated with rampant caries.

Shaw et al. (1959) conducted animal experiments on milk-based products including ‘chocolate drink’ and ‘chocolate milk’. There was a ‘general trend’ for chocolate drink and chocolate milk to be less protective than plain milk. The authors do not give the sugars content of these products, but comment: “one would tend to suspect the reason for these differences to be the increased carbohydrate content of the preparations containing chocolate rather than chocolate itself.” Also, using the rat caries model, Bowen and Pearson (1993) studied the effect of adding 10% sucrose or 10% fructose to milk. There was little difference in cariogenicity between these two sugars and both were more cariogenic than milk alone but less cariogenic than 10% sucrose in water. A further aspect of this experiment showed that 4% lactose in water was of very low cariogenicity. In a second series of experiments, these authors found that caries development was similar in groups of animals receiving 2%, 5% or 10% sucrose in milk. This caries development was greater than that recorded for rats receiving milk alone and less than that recorded for rats receiving 10% sucrose in water. From these results they concluded “..that lactose has little capacity to promote caries.”; “It is clear that the addition of as little as 2% sucrose to milk enhances the caries activity of milk even though the milk-sucrose solutions are significantly less cariogenic than the water-sucrose solutions.” and “..the practice of adding any sugar to milk should be discouraged.”

A small and statistically non-significant increase in enamel softening was recorded by Thomson et al. (1984) when 5% sucrose was added to cow’s milk, in an intra-oral enamel slab experiment. However, softening was much greater when the enamel was exposed to 5% sucrose in water. Two plaque pH studies have indicated that the addition of 5% and 10% sucrose to cow’s milk increased acidogenicity, but this increase was less than when sucrose was added to water (Mor and McDougall, 1977; Moynihan et al. 1996). In an in vitro enamel slab experiment, Bibby et al. (1980) reported that “commercial chocolate milk was much more destructive” than whole milk. The chocolate milk contained 6% added sugars (75% corn syrup, 25% sucrose). In another, more recent, in vitro study, Prabhakar et al. (2010) compared the cariogenicity of various milks. They commented that: “It is a common household practice to add sugar to bovine milk to make it more palatable for infants [in India]”. They concluded that “it was clearly evident that supplementation with an external carbohydrate source (sucrose) enhanced the cariogenic potential of milk.”
There appears to be only one study comparing the effect on caries development of cocoa with any other flavouring. In the intra-oral enamel slab experiment of Thomson et al. (1984) mentioned above, there was a hint that milk flavoured with cocoa caused less enamel softening than milk flavoured with strawberry; both milks also containing about 5% sucrose.

Thus, it is probable that adding sugar to milk increases risk of caries development: this evidence comes from several types of study – human, animal and laboratory. The concentration of added sugar at which caries development might begin is uncertain but may be as low as 2%. There is too little information on the effect of cocoa flavouring to draw conclusions.

**Milk and dental erosion.**

“Erosive tooth wear is becoming an increasingly important factor when considering the long-term health of the dentition. There is some evidence that the presence of this condition is growing steadily.” (Lussi, 2009a). Although there are many causes, acidic drinks and foods are the most important. There is much research published on the protective role of calcium (and phosphate) additives to drinks (West et al., 2003; Hooper et al., 2004; Lussi, 2009a,b) and there are now several calcium-enriched drinks on the market. There are few studies on milk and dental erosion, although CPP-ACP technology (see above) has been exploited as an anti-erosive system (Cochrane and Reynolds, 2009). Syed and Chadwick (2009) showed, in an *in vitro* laboratory experiment, that the addition of milk to a low pH carbonated beverage (6.25ml milk, 25ml beverage) reduced the erosive capacity of the beverage by about 50%. Lussi (2009a) states that although yoghurt has a low pH (~4.0), “yet it has hardly any erosive effect owing to its high calcium and phosphate content, which makes it supersaturated with respect to apatite.” It should be noted that fluoride is protective against dental erosion (Lussi, 2009a,b): currently, there are no studies on the protective potential of fluoridated milk.

**Milk and periodontal disease.**

The publication by Al-Zahrani (2006) reported an inverse association between intake of dairy products and prevalence of periodontal disease. The data source was the US NHANES III (1988-1994) involving 12,764 adults over the age of 18 years. Following multivariate analyses, “individuals who were in the highest quintile of intake of dairy products were 20% less likely to have periodontitis than individuals in the lowest quintile independent of major risk factors for periodontitis.” The classification ‘dairy products’ covered ‘various milk and milk products’ – the study did not consider milk alone. It was speculated that calcium and vitamin D might explain the findings, partly because
of the link between osteoporosis and susceptibility to loss of alveolar bone support. Nishida et al. (2000) had shown an inverse relation between calcium intake and risk of periodontitis, and Dietrich et al. (2004) an inverse relation between vitamin D and periodontitis. However, the above suppositions were called into question by the study of Shimazaki et al. (2008). They examined 942 Japanese adults aged 40-79 years and found no relation between intake of milk (which was high in this population) and periodontal disease after multivariate analyses. Instead, there was a strong inverse relation between intake of ‘lactic acid foods’ and periodontal disease. They questioned the view that calcium intake was important, stating that “The plausible hypothesis that lactic acid foods may have a beneficial effect on periodontal disease might be based on the probiotic effect of lactobacilli in these foods.” Thus, further clarification of the relation between milk consumption and periodontal disease is needed.

Conclusion

Bovine milk can be considered non-cariogenic; its anti-cariogenic role is possible. Evidence from several types of study contributes to these conclusions. While early epidemiological evidence is equivocal, more recent observational studies which have used multivariate analyses have strongly favoured milk being associated with lower caries experience. Information from animal experiments shows clearly milk’s non-cariogenicity as well as the caries-protective role of milk – some caution in interpreting this latter finding in animal experiments is needed. In vivo and in vitro demineralisation and remineralisation (enamel slab) experiments also indicated the low cariogenic potential of milk and also demonstrated its caries-protective role. These actions would appear to be due to (a) lactose being the least cariogenic of dietary sugars, (b) the protective role of casein and possibly fats, and (c) the protective role of calcium and phosphorus. There would appear to be no reason why yoghurt should be any different from milk with regard to dental effects. Although evidence is at present limited, the addition of probiotics to milk may benefit oral health. Milk may have a role in the control of dental erosion but evidence is, at present, limited. The role of milk in the control of periodontal disease is unclear. The favourable dental profile of milk and yoghurt is likely to be compromised by the addition of sucrose.

References


Al-Zahrani, M. S. (2006). Increased intake of dairy products is related to lower periodontitis


