

## articles

## Can infants catch caries? A review of the current evidence on the infectious nature of dental caries in infants

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### INTRODUCTION

Despite a decline in its prevalence worldwide, dental caries remains one of the most common chronic diseases in childhood (Mouradian, 2001). Moreover, its distribution has become more polar with most of the disease being experienced by certain vulnerable groups, such as pre-school-aged children, for whom reductions in caries experience have not been as marked as in other age groups (Pitts and Palmer, 1994). There is strong evidence to indicate that the experiences and health status of young children directly influence their health, development and wellbeing throughout life (Weinstein, 1998). Dental problems in early childhood have been shown not only to be predictive of future dental problems but also to impact upon general growth and cognitive development by interfering with sleep, appetite, eating patterns, poor school behaviour and negative self-esteem (Ayhan et al, 1996; Acs et al, 1999; Low et al, 1999; Edelstein, 2000; Thomas & Primosch 2002). It is important to understand why very young children develop dental caries, to be able to identify those most at risk, and to instigate effective preventive strategies.

Although dental caries is a complex chronic disease with a wide range of biological, environmental and behavioural determinants that complicate its aetiology, its pathogenesis is relatively well understood. There are three essential factors in its aetiology: the host or susceptible tooth, the plaque or oral microflora and the frequent presence of a fermentable carbohydrate. The development and progression of dental caries depend on the presence of cariogenic micro-organisms to metabolise fermentable carbohydrates and produce organic acids that in turn demineralise dental hard tissues (Seow, 1998). The microbiology of dental caries in humans has been well described elsewhere (Loesch, 1986; van Houte, 1994; Tanzer et al, 2001; Kleinberg, 2002). The principal group of bacteria responsible for dental caries has been identified as being mutans streptococci (van Houte et al, 1982; Milnes and Bowden, 1985). Mutans streptococci (MS) possess many characteristics that facilitate caries development, including the ability to adhere to tooth surfaces and to synthesize certain glucans from sucrose (Freedman et al, 1978). MS are also able to synthesize intracellular polysaccharides, which supports continual acid production, which in turn fosters demineralization of the dental hard tissues (Spatafora et al, 1995).

Early Childhood Caries or ECC (historically also referred to as nursing bottle caries, baby bottle decay and many other terms) is a particular form of dental caries affecting pre-school-aged children. It has been defined in many different ways over the years, however currently the American Academy of Pediatric Dentistry defines ECC as "the presence of one or more decayed (non-cavitated or cavitated lesions), missing (due to caries) or filled tooth surfaces in any primary tooth in a child 71 months of age or younger. In children younger than three years of age, any sign of smooth-surface caries is indicative of severe early childhood caries. From ages three through five, one or more cavitated, missing (due to caries), or

filled smooth surfaces in primary maxillary anterior teeth, or a decayed, missing, or filled score of equal or more than 4 (age three), equal or more than 5 (age four), or equal or more than 6 (age five) surfaces, constitutes severe early childhood caries" (American Academy of Pediatric Dentistry, 2003). It can affect children as soon as their teeth erupt. Although there is strong evidence that MS cause dental caries, MS are not found in the mouths of newborn babies (Berkowitz et al, 1975). This implies that MS have to be acquired from elsewhere, therefore dental caries can be considered to be a transmissible disease.

The aim of this paper is to review the current literature in order to answer the following questions:

1. What is the evidence that MS are associated with caries in infants?
2. When do infants acquire MS?
3. From where or from whom do infants acquire MS?
4. What factors influence the risk of infection with MS in infants?

Finally, we will speculate on some potential methods for preventing the initiation and development of dental caries in this young age group.

### WHAT IS THE EVIDENCE THAT MS ARE ASSOCIATED WITH CARIES IN INFANTS?

As with caries in general the main bacteria associated with ECC are of the group now termed "mutans streptococci" of which *Streptococcus mutans* and *Streptococcus sobrinus* are the species most commonly isolated in human dental caries (van Houte, 1994). There is an abundance of evidence showing that MS are the principal organisms isolated from the carious teeth of children with ECC (van Houte et al, 1982; Berkowitz et al, 1984; Boue, 1987). A high salivary count of MS may be predictive of caries activity (Klock and Krasse, 1979). Kohler and Bratthall (1979) developed a method to estimate MS levels in saliva. If the number of colony forming units (CFU) greater than 10µm in diameter was 0-20 it represented a "low" MS score, 21-100 was a "moderate" score, and a "high" MS score was assigned if there were over 100 CFUs. Children with active caries have been shown to have high MS scores (Brown 1985). In another study, MS and lactobacilli were isolated from the dental plaque of children aged between 1 and 2.5 years, irrespective of their caries status; however, the mean counts of MS and lactobacilli were 100-fold higher in those with rampant caries than in those who were caries-free (Matee et al, 1992). In a study by van Houte and colleagues (1982) children with "nursing bottle caries" were consistently shown to have very high concentrations of MS in the cultures taken not only from the carious lesions themselves but also from both the white spot margins of these lesions and clinically sound tooth surfaces.

### WHEN DO INFANTS CATCH MS?

MS could persist in the oral cavity by either forming adherent colonies on the mucosal surfaces, or by living and

multiplying in saliva. The action of swallowing means that only a minority of MS will remain in the saliva and it is well accepted that bacteria must become attached to an oral surface in order to proliferate. However early studies suggested that MS requires a non-shedding surface for colonization and hence it has been hypothesized that they are unable to colonize the mouth of a healthy pre-dentate infant (Carlsson et al, 1970; Berkowitz et al, 1975; Catalanotto et al, 1975). Furthermore, several studies have failed to demonstrate any MS in pre-dentate infants, although the researchers did find MS in the oral cavity once the primary dentition had commenced to erupt (Carlsson et al, 1975; Berkowitz et al, 1980).

Caufield and co-workers (1993) followed 46 mother-and-infant pairs from the child's birth to between 3 and 6 years of age. Seventy percent of the infants in the study initially acquired MS at the mean age of 26 months, and MS was detected in 25 percent of these infants by 19 months and in 75 percent by 31 months of age. Caufield and colleagues went on to suggest that the critical time for oral colonization by MS lay within a well-delineated age range of 19 to 31 months of age, a period designated as the "window of infectivity". They hypothesized that this window of infectivity was associated with the increasing surface area of the erupting teeth. From these findings arose the hypothesis that the acquisition of MS depended upon newly-exposed tooth surfaces and that MS colonized the mouth of infants only during the period when teeth emerge into the oral cavity. However these results and their interpretation should be viewed with some caution, as no account was taken of the possibility of other biological or environmental factors that might be changing at the same time as the primary dentition is erupting, such as dietary preferences, feeding practices and oral hygiene measures, all of which may influence the ability of MS to colonize the oral cavity of these infants.

Caries has also been reported in infants under the age of 19 months (Croll, 1988), suggesting that very young children with teeth may also become colonized (Berkowitz et al, 1980; Mohan et al, 1998), raising questions about the accuracy of the "window of infectivity" theory. Yet more doubt was

thrown on this theory following the detection of MS in the pre-dentate oral cavity (Wan et al, 2001a,b; Tanner et al, 2002). In one study 50 percent of full-term and 60 percent of preterm babies were found to harbour MS in the oral cavity by 6 months of age (Wan et al, 2001b). In another study the same researchers found evidence of MS colonization in three-month-old pre-dentate infants. They also demonstrated that the MS colonization was positively correlated with the number of developmental (Bohn's) nodules present (Wan et al, 2001a).

Whilst contemporary evidence would suggest that colonization of the oral cavity with cariogenic MS may occur much earlier than previously thought, there is also a substantial amount of evidence that colonization is more likely with increasing age and number of teeth (Mohan et al, 1998; Thorild et al, 2002; Wan et al, 2003). Furthermore, longitudinal studies have shown that the younger the child acquires MS, the more caries they experience (van Houte et al, 1981; Alaluusua and Renkonen, 1983; Burt et al, 1983). By contrast, children with no detectable MS over a study period of seven years did not have any dental caries (Lindquist and Emilson, 2004). Therefore, the timing of first MS detection appears to be a strong determinant of future caries experience.

#### WHERE DO THE MS COME FROM?

Most of the available evidence points to the mother (as the primary care-giver in most cases), or more rarely another person with intimate physical contact with the infant, as the source of the MS inoculation. This makes evolutionary sense because the transfer of indigenous biota from mother to offspring is a recurring theme in the lower invertebrates for which research data are available (Baumann et al, 1995). There is evidence that mothers with high caries levels have infants with more caries (Alaluusua et al, 1989); see Table I. High MS counts have been measured in the saliva of mothers of children with high caries experience (van Houte et al, 1981). A high maternal salivary MS count has been shown to be a strong factor influencing MS detection in infants (Kohler and Bratthall, 1978; Brown, 1985; Caufield et al, 1988). Recent studies have confirmed a positive correlation between maternal

TABLE I - Summary of studies investigating mother-child transmission of MS

Authors and dates	Number of subjects	Method of study	Outcome
Berkowitz et al, 1981	156 mother-child pairs	MS scores	Mother MS > 10 <sup>5</sup> 58 percent of infants infected with MS Mother MS < 10 <sup>5</sup> , 12 percent of infants infected with MS
Berkowitz and Jordan, 1975	4 mother-child pairs	Bacteriocin typing	Per 10 isolates, there were 5/10 to 10/10 matching strains within each mother-infant pair.
Van Houte et al, 1981	48 caries free mother-child pairs and 41 caries positive mother-child pairs	MS scores	For caries-free children, MS detected in 62 percent of their mothers For caries-positive children, MS detected in 100 percent of their mothers
Brown, 1985	112 mother-child pairs	MS scores	High MS score in both caries active infants and their mother's
Kohler and Bratthall, 1978	36 children and their mothers and fathers	MS scores	When both parents have <10 <sup>5</sup> MS CFU per mL of saliva, their children not infected. When both parents were >10 <sup>6</sup> MS CFU per mL, 11 out of 15 of their children are infected.
Davey and Rogers, 1984	10 children and their mothers and fathers	Biochemical characterization of MS, Bacteriocin typing	All infected children shared at least 1 common strain with the mother, fathers did not share strains with others in the family.
Thorild et al, 2002	200 mother-child pairs	MS scores	Mother with high MS score, 52 percent of their children were infected. Mother with low MS score, their children has no MS detected.

and infant salivary MS levels (Wan et al, 2001a; Thorild et al, 2002). Fifty-two percent of children whose mothers had high salivary MS counts were colonized with the bacteria (Thorild et al, 2002), while none of those whose mothers had values below the detection level harboured MS.

In the landmark studies carried out by Kohler and co-workers (Kohler et al, 1983; Kohler et al, 1984), the children of selected mothers with high levels of MS in their saliva were monitored for initial acquisition of MS and caries activity over a three-year period. A strong relationship was demonstrated between the maternal MS levels and those of their infants. Kohler and Bratthall (1978) had suggested that infection of a child's oral cavity with MS can occur via salivary contacts between the mother and child. Large numbers of MS may be introduced into the infant's oral cavity by an infected mother when engaging in practices such as using her own spoon to feed the child. This would be even more significant if the mother had a high saliva MS count. If the mother were feeding the child with sucrose-containing foods this could further support the implantation of MS in the child (Kohler and Bratthall, 1978).

During the last decade, the methods applied in the characterization of oral bacteria have become much more sophisticated. DNA analysis techniques based upon genotype offer rapid and reliable identification of the bacteria compared with methods based upon phenotypic characterization. Bacteriocins (mutacin) are proteinaceous antibacterial substances that some bacteria produce to interfere with the growth of other bacteria (Gronroos et al, 1998), and their typing has been used as an epidemiological tool for tracing bacterial infections in humans. Bacteriocin typing has previously been used to demonstrate human transmission of bacteria. In Berkowitz and Jordan's study (1975), the bacteriocins produced within four mother-and-child dyads were compared to determine the likelihood of maternal transmission of MS, and between 50 and 100 percent of the bacteriocins found in the children matched those of their mothers. This similarity of the bacteriocin types from the children, compared with those of their mothers, strongly implied maternal transfer of this group of bacteria. When data from the infants' fathers were included in the analysis, it was found that they did not share bacterial strains with the other family members, although all infected children shared at least one common strain with their mothers (Davey and Rogers, 1984). Further supportive data came from another study that 95 percent of the mother and baby pairs shared the same MS serotypes (Berkowitz and Jones, 1985). Homology between maternal MS genotypes and those of their infants has also been demonstrated, which strongly suggests that MS strains are transmitted from mother to infant (Li and Caufield, 1995; Emanuelsson and Wang, 1998). Li and Caufield (1995) carried out a study to determine the commonality of MS genotypes between mothers and infants at the time of initial acquisition, in which the oral bacteria of mothers and their infants were monitored from the child's birth for approximately three years at three-month intervals. The genotypes of MS in infants appeared identical to those present in mothers in approximately 71 percent of the 34 mother-infant pairs that were studied.

Using an alternative approach, Gronroos and co-workers (1998) studied the ribotyping and mutacin typing of MS isolates from young children and their mothers. Ribotyping is a form of DNA fingerprinting in which the genetic fingerprint comes from genes that code for ribosomal ribonucleic acids (rRNA). Ribosomal genes are highly conserved in microbes,

which means there will be much less variation of the genetic information coded for the same strain than between different strains. Transmission can be inferred if the mother and her child harbour MS with identical ribotype profiles. The results of this study showed the presence of identical ribotypes in 9 out of the 14 MS-positive mother-child pairs. Maternal transmission of MS was present in 64 percent of these MS-positive children.

Mutacin is a proteinaceous antibacterial substance that MS produce to interfere with the growth of other closely related bacteria, and mutacin typing (including production or activity of mutacin) has been used for epidemiological typing of isolates and to examine the role of maternal transmission of MS to the child (Berkowitz et al, 1975). To complement the result of the ribotype study, mutacin activity was investigated in the 10 ribotypes that were shared between mother-and-child pairs (Gronroos et al., 1998). The activity profiles of the ribotypes were all similar, which further suggested the likelihood of mother-to-child transmission of MS.

Whilst all the evidence points to the mother as the primary source of the MS infection, the parameters that facilitate successful transfer are not clearly defined. Theories as to how the MS are transferred include contact with mother's saliva, for instance at feeding time. Infection transfer media that have been suggested include feeding spoons, kisses on the mouth, or the mother's "cleaning" the infant's pacifier by putting it into her own mouth (Aaltonen and Tenovu, 1994). Speaking and food tasting may also promote direct salivary contact (Kononen et al, 1992). It has been postulated that the more frequently a mother transmits MS-harboring saliva to her infant's mouth, the earlier the colonization of MS in the baby's oral cavity is likely to occur (Kohler and Bratthall, 1978; van Houte et al, 1981). However, no studies have been performed to quantify the effects of mothers and their children sharing food and eating utensils on the subsequent MS colonization in the infants.

Evidence that the father is a source of MS infection in an infant has rarely been reported. Emanuelsson and colleagues (1998) studied 11 families in which MS was detected in all three family members (mother, father, and child). Six of the children showed MS genotypes identical to those of their mother, five harboured MS genotypes different from those of either parent, and none harbored MS genotypes similar to those of their father. This was in spite of the fact that two-thirds of the fathers had high or very high MS levels. Similar results have been reported elsewhere, and it has been suggested that working fathers' absence from home during the day in the child's first three years of life may limit the occasions for close contact (Davey and Rogers, 1984; Li et al, 1995). On the other hand, similar MS strains with identical genotypes have been found among all family members (fathers and children, mothers and children, and mothers and fathers), suggesting that any member of a family may be able to acquire MS from other family members (Emanuelsson and Wang, 1998). Taking into account the ability of MS to survive outside the oral cavity for several hours (Kohler and Bratthall, 1978), cultural differences within families such as eating with the same cutlery or from the same plate, using the same toothbrush, and ineffective utensil-washing routines, may increase the likelihood of transmission. While the mother is usually the primary caregiver and consequently may have frequent close salivary contact with her infant, it is also possible that a child can acquire MS from both inside and outside the family (Caufield et al, 1993, Emanuelsson et al, 1998; Emanuelsson and Wang, 1998).

## WHAT FACTORS AFFECT COLONIZATION OF THE ORAL CAVITY OF AN INFANT?

Maternal MS levels have been shown to be associated with the colonization of infants by MS. As has already been discussed there is a positive correlation between maternal levels of MS and the likelihood of infection being found in the infant (Kohler and Bratthall, 1978; Berkowitz et al, 1980; van Houte et al, 1981; Wan et al, 2000b). It can therefore be postulated that factors resulting in an increase in maternal MS levels will increase the risk of colonization of the infant's oral cavity.

Maternal sugar consumption may also influence the colonization of the infant. A diet high in sucrose predisposes an individual to high levels of oral MS (Kristofferson and Birkhed, 1987), so placing mothers at higher risk of infecting their children (Berkowitz et al, 1981, Kohler et al, 1983). Conversely, children of mothers who consume sucrose less frequently and have low MS levels may themselves exhibit low MS levels (van Houte, 1981). However, the confounding factor associated with this discussion is whether mothers, whose influence over an infant's dietary habits is known to be significant (King, 1978), can promote higher sugar consumption in their infants even from birth if they themselves have a high sugar consumption.

The degree of maternal involvement in infant care may influence the implantation of bacteria in the young child. DNA patterns or "fingerprints" can be used to identify strains of MS, and inferences can be drawn concerning its mode of transmission, by comparing DNA patterns; if two or more isolates demonstrate a similar pattern, they are believed to be genetically related. In one urban Chinese cohort, only 44.7 percent of children acquired MS strains identical to their mother's strain (Li et al, 2000). However, the same authors, in an earlier study of DNA fingerprinting in an American cohort, found 70.5 percent of children with identical strains of MS to their mother's strain (Li and Caufield, 1995). They speculated that the Chinese children surveyed started their regular day-care life at a younger age (around 20 months of age) and spent an estimated 8 hours per day there while their mothers worked outside of their homes. Lack of contact with their mothers might reduce the risk of transmission from mother to infant but may of course allow transmission from other caregivers.

The nature and type of infant feeding practices may influence the establishment of MS. Lactose is present in breast milk (7.4g lactose per 100mL breast milk) and is an essential sugar and energy source in infant nutrition. Neither human nor bovine milk is cariogenic (Weiss and Bibby, 1966; Bowen et al, 1991; Bowen and Pearson, 1993; Jenkins and Ferguson, 1996; Thomson et al, 1996; Erickson and Mazhari, 1999), unless it is taken simultaneously with a cariogenic challenge such as sucrose (Bowen and Pearson, 1993). Evaluation of the cariogenic potential of human milk as opposed to bovine milk and sucrose solution has shown that human milk has a greater acidogenic potential than bovine milk, but is less acidogenic than sucrose solution (Thomson et al, 1996). It is interesting to note however that even though the addition of sucrose to milk renders it cariogenic, milk with a 10 percent sucrose solution causes less caries than a simple 10 percent aqueous sucrose solution (Bowen and Pearson, 1993), which supports the notion that milk contains a protective factor. The relationship between various infant formulae or indeed breast milk, and the rate of colonization of the infant oral cavity with MS, has not been explored.

The relationship between breast-feeding and caries remains controversial. There are many, usually somewhat anecdotal,

reports in the historic literature that have promoted the idea that prolonged and excessive breast-feeding is associated with rampant caries in infants (Gardner et al, 1977; Kotlow, 1977; Abbey, 1979). Infants who breast-feed at will during the night can present with higher MS counts in their dental plaque (van Houte et al, 1982; Matee et al, 1992). Similarly, children who breast-feed on demand have been shown to have high caries experiences (Harrison et al, 1997). However controversy still exists. Li and co-workers (2000) carried out a cross-sectional study of 50 two- to three-years-old Chinese children in which 35 (70 percent) of the subjects were breast-fed. The results suggested that breast-feeding *per se* was not associated with higher caries rates. Fifty-three percent of the non-breast-fed group were caries-active, compared with 43 percent of the breast-fed group. Furthermore, whilst 65 percent of the caries-active children were breast-fed, 74 percent of the caries-inactive group were also breast-fed. Other studies have failed to demonstrate any link between duration of breast-feeding and caries development. In one study only 9 percent of the 96 children who were breast-fed for a prolonged period of time developed caries (Weerheijm et al, 1998), whilst in another study children who either did not breast-feed at all or did so for just a few months actually developed more caries than those who continued breast feeding for many months (Mattos-Graner et al, 1998). It has been suggested that dietary intakes other than breast-feeding could be a confounding factor in these breast-feeding related caries studies (Hackett et al, 1984).

## HOW CAN WE PREVENT THE TRANSMISSION OF MS FROM MOTHER TO CHILD?

Table II summarizes the studies that have reported on the effectiveness of preventive measures targeted at mothers as regards the oral health of their children. Children whose oral cavities are colonized early by MS show greater caries occurrence than children with later or no MS colonization (Alaluusua and Renkonen, 1983; Kohler et al, 1988). In the primary dentition, the presence of MS has been shown to be the most accurate indicator of caries risk as indicated in the review by Powell (1998). Salivary MS counts can be expected to have a sensitivity of 93 percent and a specificity of 57 percent for identifying caries in 3- to 6-years-old children (Edelstein, 1995). Once MS has been detected in a child, its presence is often persistent. Stable levels are even observed until adulthood (Kohler and Andreen, 1994). Preventing or delaying the transmission of MS from mother to child has the potential to reduce the caries experience in the next generation.

Three broad strategies aimed at reducing maternal transmission of MS have been studied: the use of chlorhexidine digluconate, chewing xylitol gum, and oral health education.

Expectant mothers with high salivary MS who used one percent chlorhexidine digluconate (CHX) varnish with special applicators, for five minutes once daily for two weeks, not only reduced the levels of MS in their saliva but also delayed the subsequent acquisition of MS in their infants (Kohler et al, 1983). A follow-up study confirmed that the reduction of MS level in saliva had a long-lasting effect on the MS colonization and caries experience of the children (Kohler and Andreen, 1994). In a more recent study (n=16), professional cleaning of the mothers teeth followed by CHX application led to fewer infants exhibiting MS colonization infants at 2 years of age than in a control group of untreated mother-child pairs (n=13), both groups had high initial maternal MS levels (Gripp and Schlagenhauf, 2002).

TABLE II - Summary of studies investigating preventive measures on mothers and its effects on transmission of MS to their children

Authors and dates	Number of subjects	Study period	Prevention method given to mothers	Outcome
Kohler et al, 1983	87 first-time mothers	23 mth	Dietary counseling, professional tooth-cleaning, OHI, F tx, excavate large cavities	E: 19 percent of infants infected with MS C: 63 percent infants infected with MS
Kohler et al, 1984	77 first-time mothers	Nearly 3 yrs	Prophylactic programme repeated at 2-monthly intervals	E: 41 percent of infants infected with MS C: 70 percent infants infected with MS
Tenovuo et al, 1992	151 mother- and-children- pair	3 yrs	CHX-NaF gel every 6 monthly	E: 16 percent of infants infected with MS C: 38 percent infants infected with MS
Kohler and Andreen, 1994	27 first-time mothers	Started when infants were 15 mth until 7 yrs old	Dietary counseling, professional tooth-cleaning, OHI, F tx, excavate large cavities and 1 percent CHX repeated every 4 mth	At 7 yrs old: E: 46 percent of carried MS C: 95 percent carried MS
Isokangas et al, 2000	195 mothers	Started when infants were 3 mth, until 24 mth	E: regular xylitol gum chewing C: F tx or CHX application	At 2 years of age: E: 10 percent infected with MS C (CHX): 32 percent infected with MS C (F): 50 percent infected with MS
Gomez and Weber, 2001	241 mothers	4 yrs	Preventive Dental Programme (PDP)	E: 97 percent caries free C: 77 percent caries free
Soderling et al, 2001	195 mothers	Started when infants were 3 mth, for 21 mth only. MS levels in children were checked at age 3, then at age 6 again.	E: xylitol C: 6- mthly F tx or 6- mthly CHX	At age 3: E: 28 percent have MS C (F): 65 percent have MS C (CHX): 37 percent At age 6: E: 52 percent C(F): 84 percent C(CHX): 87 percent
Gripp and Schlagenhauf, 2002	29 mothers	10 weeks after delivery until children reach 2 yrs old	Professional clean and 40 percent CHX every 3 mth	At age 2: E: 19 percent MS positive C: 69 percent
Brambilla et al, 1998	65 mothers at the start of second trimester with MS >10 <sup>5</sup> cfu	Treatment for 6 mth during pregnancy and follow up for 24 mth after delivery	Both E and C: dietary counseling, prophylaxis for once and OHI, systemic F (1mg daily) E received extra daily 0.05 percent NaF rinse and 0.12 percent CHX rinse	Incidence of MS acquisition in E is 34 percent lower than C Mean age of colonization in E: 23 mth old C: 18 mth old

## Abbreviations:

OHI – oral hygiene instructions; F tx – fluoride treatment; E – experimental group; C – control group; CHX – chlorhexidine; NaF – sodium fluoride; mth – months; yrs – years

The use of xylitol in a chewing-gum has also been shown to reduce the MS levels in saliva and in plaque (Soderling et al, 1989). It also reduces the amount of plaque and can inhibit the metabolism of MS (Trahan, 1995). It is possible therefore that xylitol could affect the transmission of MS from the mother to her child. This hypothesis was tested in a 2-year study in which mothers regularly chewed xylitol gum for 21 months, starting 3 months after delivery of the baby, the control group receiving either CHX or fluoride varnish treatment at 6, 12, and 18 months after delivery (Soderling et al, 2001). As can be seen from Table II, MS was detected in significantly fewer infants in the xylitol group (28 percent) compared to those in the fluoride group (65 percent) and the CHX group (37 percent). The researchers suggested that the xylitol altered the adhesive properties of the MS and thus inhibited colonization, so allowing MS to be flushed away by saliva (Soderling et al, 1991; Trahan et al, 1992). In a follow-up study the same cohort of children was reviewed at the age of 6 years (Soderling

et al, 2001). The intervention (CHX, xylitol or F) had been discontinued when the children were two years of age, and the MS colonization percentages increased in all groups over the follow-up period. Nevertheless, the results suggested that a xylitol-associated reduction in the mother-child transmission of MS during the emergence of the primary teeth did have a long-term influence on MS colonization (see Table II). Further support for the efficacy of xylitol gum has been demonstrated in a study involving 195 women who were asked to start xylitol chewing gum three months after the birth of their babies and to discontinue its use 24 months after delivery. The habitual consumption of xylitol gum was associated with a statistically significant reduction in the likelihood of MS being transmitted between mother and child when assessed at two years of age. The same group of subjects was assessed again at the age of five years, when it was found that the children whose mothers had used xylitol showed significantly less total caries experience than the control group (Isokangas et al, 2000).

Specific preventive programmes (see Table II) targeted at expectant mothers have been shown to reduce significantly maternal salivary MS levels and delay colonization by these bacteria in their children (Kohler et al, 1983; Brambilla et al, 1998). In one of the earlier studies half the cohort of first-time mothers with high salivary MS counts ( $>10^6$  CFU per ml) were exposed to a special preventive programme in an attempt to reduce their salivary counts to under  $3 \times 10^5$  CFU per ml (Kohler et al, 1983). Where such a reduction was achieved, the establishment of MS in the respective infants was shown to be prevented or delayed. Only 11 percent of the infants from the “successfully” treated mothers were infected with MS after 2 years, as compared to 45 percent of the control group. The between-group difference was maintained at three years of age (Kohler et al, 1984).

Over a four-year period, Gomez and Weber (2001) evaluated a free Preventive Dental Program (PDP) for 241 mothers in their fourth month of pregnancy and in 180 controls who were not involved in the same programme. The PDP involved oral hygiene and dietary instruction to expectant mothers, with emphasis being placed on teaching them to avoid or minimize infecting their children with their own MS. Ninety-seven percent of the PDP group were caries-free after 4 years compared to 77 percent in the control group. The targeting of women early in their pregnancies and continuing after the birth of their children may not only be highly effective in preventing and delaying the development of dental caries in the children but may also improve the mothers' overall oral health and attitudes to dental care (Weinstein, 1998). Studies have shown that if MS are not established in the dental plaque during the first two years of life, or if they are established later, there will be minimal caries in the young primary dentition (Alaluusua and Renkonen, 1983; Kohler et al, 1988). It was also suggested that if a child has a low caries potential in the primary dentition, there will be minimal caries in the permanent dentition (Alaluusua et al, 1987), and it is possible that the caries and microbial patterns seen in early childhood will reflect the oral health of individuals as they grow to be teenagers and beyond (Alaluusua et al, 1989).

## CONCLUSIONS

So, can infants catch caries? The answer is yes, and there is strong evidence to suggest that they do so primarily from their mothers. The earlier MS is detected in the oral cavity the more likely the child will be to develop dental caries, with those at highest risk being those in whom MS is detected before two years of age. Infants are susceptible to MS infection before as well during the eruption of their primary teeth. Colonization is more likely when the maternal salivary MS levels are high, when there is frequent salivary contact between mother and child, and when the diet is high in sucrose. From the literature, we can conclude that mothers are the most common but not the exclusive source of MS infection. However, with the use of new technologies in recent research we can be confident that the MS cultured from infants and children are often the same as those found in their mothers. With this knowledge, oral health professionals should be promoting measures to improve maternal oral health as a means of preventing or inhibiting MS colonization in infancy. Such measures may include antenatal advice on personal oral hygiene for expectant mothers as well as proactive treatment of carious lesions and periodontal disease to reduce the bacterial load. At a community level it would be possible to identify high-caries-risk expectant mothers in maternity clinics. Appropriately trained dental auxiliaries working with maternal child health nurses and lactation consultants could screen mothers and provide oral hygiene and dietary advice with an emphasis on the future health of their children. The use of antibacterial varnishes and xylitol confectioneries may be of value in reducing maternal MS counts and thus delaying transmission to their offspring, but further research is required in this area.

In summary, oral health professionals ought to be promoting oral health awareness not only in expectant mothers but in their associated healthcare professionals. In this way we may contribute to an improvement in the oral health of young children, since it is never too early to prevent caries.

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