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DENTAL CARIES – AN INFECTIOUS DISEASE OF CHILDHOOD – A REVIEW

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ABSTRACT

Dental Caries is a common, chronic disease of childhood. Most studies that have assessed risk factors for dental caries focused on non-modifiable risk factors such as previous caries experience and socioeconomic status. It is also important to investigate modifiable risk factors that can be used in developing guidelines for risk assessment and prevention. This review discusses about dental caries in childhood and it's association with Streptococcus mutans, discusses the colonization of Streptoccus mutans, Origin of Streptococcus mutans, factors affecting colonisation of oral cavity in children, & prevention of transmission of Streptoccus mutans from mother to child in detail.

Keyword: Dental caries, Streptococcus mutans, Children

INTRODUCTION

Dental caries is widely recognised as an infectious disease induced by diet. Aetiology of the disease are a) cariogenic bacteria b) fermentable carbohydrates c) susceptible tooth & host d) time.(Seow, 1998).Dental caries remains one of the most common chronic diseases in childhood (Mouradian, 2001). Dental problems in early childhood was not only predictive of future dental problems but it also had an impact on general growth & cognitive development by interfering with sleep, appetite, eating patterns, poor school behavior & negative self-esteem (Ayhan et al, 1996; Acs et al, 1999;Low et al, 1999; Edelstein, 2000; Thomas & Primosch 2002). The principal group of bacteria which causes dental caries are mutans streptococci (van Houte et al, 1982; Milnes and Bowden, 1985). Mutans streptococci had many characteristics that facilitate caries development, including the ability to adhere to tooth surfaces & synthesize certain glucans from sucrose (Freedman et al, 1978). Mutant streptococci ability to synthesize intracellular polysaccharides, that induces continual acid production, results in demineralization of the dental hard tissues (Spatafora et al, 1995). Mutans streptococci participate in the formation of biofilms on tooth surfaces. These biofilms are known as dental plaque(s) (Martin *et al.* (2006)). Early Childhood Caries or ECC is a particular form of dental caries affecting pre-school aged Children. ECC is defined as "the presence of one or more decayed, missing(due to caries) or filled tooth surfaces in a infant. In children younger than three years of age, any sign of smooth-surface caries is indicative of severe early childhood caries. From ages three to five, one or more cavitated, missing (due to caries), or filled smooth surfaces in primary maxillary anterior teeth, or 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). The aim of this review is to identify dental caries in childhood predominantly associated with Streptococcus mutans & recommend suitable methods for prevention.

Evidence for Streptococcus mutans associated with dental caries in childrens:

ECC causing bacteria grouped as "Mutans streptococci" of which *Streptococcus mutans* and *Streptococcus sobrinus* are the species most commonly isolated in human dental caries (van Houte, 1994). Many evidences shows that Streptococcus mutans 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 streptococcus mutans may be predictive of caries activity (Klock and Krasse, 1979). . (Kohler & Bratthall (1979) developed a method to estimate streptococcus mutans 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). 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(van Houte et al (1982)). In another study, MS and lactobacilli were isolated from the dental plaque of children aged between 1and 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).

Colonisation of streptococccus mutans

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 predentate infant (Carlsson et al, 1970; Berkowitz et al, 1975; Catalanotto et al, 1975). Several studies have failed to demonstrate any MS in predentate infants, but the researchers found MS in the oral cavity once the primary dentition had commenced to erupt (Carlsson et al, 1975; Berkowitz et al, 1980). But in fact MS could persist in the oral cavity by either forming adherent colonies on the mucosal surfaces, or by living & multiplying in saliva. The action of swallowing allows only small percentage of streptococcus mutans to remain in the saliva but bacteria must become attached to an oral surface in order to proliferate. Caufield et., al., 1993 study involves 46 mother & infant pairs from the child's birth to between 3 and 6 years of age. 70% of the infants in the study initially acquired MS at the mean age of 26 months, and MS was detected in 25% of these infants by 19 months and in 75 % by 31 months of age. They suggested 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". 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) In one study 50 % of full-term and 60% of preterm babies were found to colonize MS in the oral cavity by 6 months of age (Wan et al, 2001b).Further longitudinal studies have shown that the younger the child acquires MS, the more caries they experience (van Houte et al, 1981; Alaluusua& Renkonen, 1983; Burt et al, 1983). In contrast, children with no detectable MS over a study period of seven years did not have any dental caries (Lindquist and Emilson, 2004). So 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, which may influence the ability of MS to colonize the oral cavity of children.

Origin of Streptococcus mutans

Mostof 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); Recent studies have confirmed a positive correlation between maternal and infant salivary MS levels (Wan et al, 2001a; Thorild et al, 2002). In the landmark studies carried out by Kohler and coworkers (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. Large numbers of MS may be introduced into the infants 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 & Bratthall, 1978**).** Bacteriocins (mutacin) are proteinaceous antibacterial substances that some bacteria produce to interfere with the growth of other bacteria (Gronroos et al, 1998). Their typing has been used as an epidemiological tool for tracing bacterial infections in humans. The bacteriocins produced within four mother-child combo were compared to determine the likelihood of maternal transmission of MS, and between 50 and 100 % of the bacteriocins found in the children matched those of their mothers(Berkowitz and Jordan's study (1975),). 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). Infection transfer media that have been suggested include feeding spoons, kisses on the mouth, or the mother's "cleaning" the infants pacifier by putting it into her own mouth (Aaltonen and Tenovuo, 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-harbouring saliva to her infants 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 et.al., (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 father's 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 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 & Wang, 1998).

Factors affecting colonization in oral cavity of children:

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 infants, 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 (Kristoffersson & 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). 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% sucrose solution causes less caries than a simple 10% 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). In one study only 9% 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).

Prevention of streptococci mutans transmission from mother to 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). 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). 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). 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). 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). 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% 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). Also 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), targeting preventive measures as early as possible to play a vital role in overall wellbeing of the child.

CONCLUSION

The earlier MS is detected in the oral cavity, the earlier children develop dental caries. The highest risk being those in whom MS is detected before two years of age. 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 it is clear 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 can promote measures to improve

maternal oral health as a means of preventing or inhibiting MS colonization in children. Advice on personal oral hygiene for expectant mothers as well as proactive treatment of carious lesions and periodontal disease to reduce the bacterial load are a few measures. The use of antibacterial varnishes and xylitol confectioneries may be of value in reducing maternal MS counts and thus delay transmission to their offspring, but further research is required in this area. Oral health professionals should promote oral health awareness in expectant mother & associated healthcare professionals. In this way dental caries can be prevented in younger children

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