

V. DENTISTRY

CARIES RISK FACTORS IN CHILDREN

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Abstract

The preoccupation for maintaining dental health, resistant to the action of microorganisms starts from the 5-6 week of intrauterine life, when the embryogenesis of the child's teeth begins. Related to that, each stage has to be well differentiated regarding dental care measures and an evaluation of the oral health is necessary because of several facts: it is the period of temporary and mixed dentition when the frequency of the dental lesions is very high with negative impact on the development of the cranio-facial system but also with an impact on the development of the whole body. The understanding of the complex nature of caries depends on the multiple risk factors that have to be taken in consideration and the prophylactic and therapeutic measures are based on the accuracy of the evaluation of all these factors.

Key words: caries risk, children, fluoride, diet, oral health.

The preoccupation for maintaining dental health, resistant to the action of microorganisms starts from the 5-6 week of intrauterine life, when begins the embryogenesis of the child's teeth. Related to that, each stage has to be well differentiated regarding dental care measures and an evaluation of the oral health is necessary because of several facts: it is the period of temporary and mixed dentition when the frequency of the dental lesions is very high with negative impact on the development of the cranio-facial system but also with an impact on the development of the whole body.

Caries can be viewed as an infectious disease and mutans streptococci are considered to be important bacteria for its development (Emilson & Krasse 1985, Loesche 1986), although no single type of micro-organism has been identified as the primary cause of either enamel, root or crown caries (Nyvad & Kilian 1987). The bacteria attach to the first primary teeth to erupt, especially to the fissures of the molars in 2-3-year-old children (Alaluusua & Renkonen 1983). However more recent studies indicate that the infection may occur at a younger age and those mutans streptococci can colonize the oral cavity of predate children as young as 6 months of age.

The time of contamination is of a certain importance, as the later a child is infected, the less caries lesions develop in early childhood and later on (Alaluusua &

Renkonen 1983, Köhler *et al.* 1988). Mutans streptococci are transmittable from the primary dentition to the permanent dentition (Gibbons 1984, Alaluusua *et al.* 1987), and also between individuals (Köhler & Bratthall 1978, Rogers 1981). The transmission occurs through contamination of the saliva (Rogers 1981) so that mothers are considered to transfer the infection to their child (Köhler & Bratthall 1978, Aaltonen *et al.* 1990). High levels of mutans streptococci in the mother's mouth contribute to maternal transfer as does maternal dietary habits and poor oral hygiene. It has been found that 20-50% of mothers in the Scandinavian countries have high counts of salivary mutans streptococci (Berkowitz & Jones 1985, Paunio *et al.* 1988). Habitual xylitol consumption by mothers has been shown to lead to a significant reduction in mother-child mutans bacteria transmission when assessed in two-year-old children (Söderling *et al.* 2000).

Cariou lesions increase the counts of mutans streptococci, while reductions can be achieved by restriction of sucrose-sweetened products (Rugg-Gunn & Edgar 1984, Birkhed *et al.* 1990) and the use of xylitol (Isokangas *et al.* 1989, Söderling *et al.* 2000), together with anti-microbial preventive procedures (Loesche *et al.* 1989, Tenovuo 1992).

Dietary habits and dental caries have shown to be of importance for caries development (Kleemola-Kujala & Räsänen 1979, Birkhed 1990), but the frequency of brushing the teeth was more related to caries than were dietary factors in some studies (Schröder & Granath 1983, Stecksén-Blicks 1985a, Stecksén-Blicks & Holm 1995). The frequency of consumption of sugar-containing products relates to caries, and the ingestion of fermentable carbohydrates is associated with its prevalence (Rugg-Gunn & Edgar 1984, Holbrook *et al.* 1995, Gibson & Williams 1999). Significant correlations between sugar consumption and caries increment have also been observed by Rugg-Gunn & Edgar (1984), while a clear correlation was observed between the occurrence of "rampant" caries in young children and the use of sweetened dummies and prolonged use of "dinky feeders" containing sugar (Walker 1987).

Social and demographic factors such as race, knowledge, schooling and financial status have all been linked with the occurrence of caries (Hunt 1990, Powell 1998, Gibson & Williams 1999). In addition, the time needed for the development of caries is also an important

consideration (Alalusa & Renkonen 1983, Köhler *et al.* 1988).

Today the biological mechanisms of dental caries are well established. It is a disease with a number of important etiological factors – each of which must be simultaneously present to initiate and progress the disease. The factors are: fermentable carbohydrates (substrate); cariogenic microorganisms; susceptible tooth surface/host and the fourth factor time.

Bacteria (mutans streptococci) in dental plaque metabolize sugars and produce acids, which lowers the pH in the mouth and promotes loss of minerals from the tooth surface. Minerals in the oral cavity including fluoride are redeposited on the tooth surface once the neutral pH is restored (normally after approximately 20min). This process is dynamic and as long as minerals are replaced the tooth surface remains sound and intact. However, a prolonged pH drop and frequent net loss of minerals lead to a weakening and eventual break down (cavity) in the tooth surface.

Early childhood caries (ECC) appears to be a particularly virulent form of dental caries causing extensive destruction of the deciduous teeth, often very rapidly. This may be due to extremes in one or more of the three factors above. Much research into the etiology and prevention of ECC has focused on the dietary substrate component leading to the terms ‘baby bottle tooth decay’ and ‘nursing caries’. More recent research confirming the relative role of the microbial(plaque) and tooth resistance factors has fostered a better appreciation of the biological risk and protective factors in ECC.

There is overwhelming evidence that sugars (such as sucrose, fructose and glucose) and other fermentable carbohydrates (such as highly refined flour) play a role in the initiation and development of dental caries. Sucrose is the most common sugar and is the only one that, when metabolized, produces dextrans which promote superior bacterial adhesion to teeth. Because of this it is considered the most important substrate in the establishment of cariogenic bacteria.

It now appears that the frequency of intake of sucrose is more important than the total amount consumed. A review of the role of substrate in ECC by Reisine and Douglass found that the total weight of sugar in children’s diet was not predictive of dental caries; however, the frequency of sugar intake was. Frequent consumption of sugar favors the establishment of cariogenic bacteria and provides continuous substrate that influences the initiation and progression of the caries.

Controversy exists as to whether infant formulas or bovine milk in bottles and breast milk given frequently to infants contribute to the development of ECC. The evidence of a relationship between bottle use and caries risk is weak and it is likely that the risk of caries may be sensitive to the interaction of multiple factors including other (non-bottle) dietary practices.

Prolonged or on demand breast-feeding has been implicated in the development of ECC. The American Academy of Pediatric Dentistry’s policy (AAPD) on breast-feeding states that: “Although breast-feeding is essential in

providing the best possible nutrition to infants, the AAPD cautions that frequent breast-feeding at night and on demand after eruption of teeth may be implicated in contributing to the development of early childhood caries (ECC)”.

There are controversies about this issue. The international dietary guidelines recommend exclusive breastfeeding until 6 months and then continuing breast-feeding with complementary foods until 2 years or more.

More recent and methodologically stronger studies have suggested that breast-feeding per se is not significantly associated with ECC. Laboratory studies have noted that human breast milk does not appear to cause the drop in plaque pH required for the initiation and progress of dental decay and may in fact promote the deposition of calcium and phosphate ions on the tooth surface.

Several factors can predispose an individual or indeed a particular tooth to dental caries. These may include immunological factors, reduced saliva flow, immature enamel and defects of the tooth tissues.

Because enamel is immunologically inactive, the main immune defence against mutans streptococci is provided largely by Immunoglobulin A (IgA) or serum and gingival crevicular fluid. As children become infected with oral microorganisms, they develop salivary IgA antibodies. In addition to providing specific immunological factors, the saliva acts as an important protective factor. Saliva buffers plaque acids, aids in oral clearance and acts as a reservoir for minerals to assist in the re-mineralization of enamel.

Teeth erupt into the mouth with immature enamel. The process of enamel maturation continues following tooth eruption, so that teeth become less susceptible to decay over time. The enamel matures incorporating orally available ions including fluoride.

The ingestion of the fluoride, irrespective of the available form, acts preeruptive, during the mineralization period of the teeth, which is developed in 2 phases:

- the mineralization of dental hard tissues
- the preeruptive maturation of the enamel.

The period of time for this 2 phases, for both temporary and permanent dentition is 13 years and a half (without the third molar).

The mineralization of the temporary incisors starts at 3-4 months intrauterine life and is finished 4-5 months after birth. The mineralization of the permanent incisors starts at 3-4 months after birth, excepting the lateral incisor which begin the mineralization at 10-12 months and it ends at 4-5 years.

The mineralization of the temporary canine starts at 5 months of intrauterine life and it ends at 9 months, while the mineralization of the permanent canine starts at 4-5 months and it ends at 6-7 years.

The mineralization of the premolars starts at 1 ½ -2 ½ and it ends at 5-7 years.

The temporary molars begin their mineralization at 5 months of intrauterine life and it ends at 6 months for the first molar, while the second molar stars at 6-7 months and ends at 10-12 months.

The permanent first molar starts the mineralization at birth and ends at 2 ½ -3 years and the second molar achieve mineralization between 2 ½ -3 and 7-8 years.

The intervals presented are medium values for a certain population. There are variation conditioned by climacteric condition, race, region.

From this data it has to be revealed the fact that the preeruptive mineralization of the first molar is the shortest when it is compared with the other teeth. This explains the great susceptibility of this tooth to dental caries.

On the other hand, the time of mineralization of the temporary teeth is 6 time shorter comparative to the permanent teeth, fact that can explain the susceptibility to caries.

Regarding the utilization of prenatal fluoride the American Dental Association asked during a debate the following questions:

- Is the passing of fluoride through the placenta safe?
- Does the child benefit from the fluoride?
- Is it a proven fact that fluoride in intrauterine is involved in the maturation of enamel?

The answer to the first question is that fluoride passes easily through the placenta from the 5th – 6th month of pregnancy.

Regarding the second question, the answer is given indirectly through the epidemiologically studies that revealed a decrease of carious lesions in temporary teeth at children who had benefit from an optimum fluoride income during intrauterine life.

At last, the answer to the third question is that fluoride is incorporated during the maturation of the enamel of temporary teeth, but at a lower concentration than in adult teeth because the mineralization stages are shorter. It looks like the incorporation of fluoride is not just the result of absorption but also of the concentration of preexistent fluoride in the bony tissue.

Ingestion of fluoride during the second half of the pregnancy, increasing the concentration of fluoride in the skeletal tissue of the child, is susceptible of influencing the ulterior incorporation of fluoride in the teeth. We can practically say that the ingestion of 1 mg F/day at young mothers is recommended from the fourth month of pregnancy.

Therefore, a tooth is most susceptible to caries immediately after eruption until final maturation.

Many studies have found a significant relationship between developmental defects of the tooth surface and dental caries. Developmental disturbances to the tooth germ during embryological development can result in loss of integrity of the surface enamel which in turn allows additional plaque accumulation on what would otherwise be a smooth surface. Such developmental disturbances may include premature birth or low-birth weight, pre- and postnatal infection/illness, nutritional deficiency and a variety of environmental pollutants including maternal smoking.

Much of the literature uses terms (including nursing caries and baby bottle tooth decay) or case definitions that imply that the inappropriate use of the baby bottle plays a central role in the development of dental decay in infants and young children, however, supporting epidemiological data is difficult to find. For this reason the term early childhood caries (ECC) is the term now used to

collectively refer to dental decay in infants and preschool children.

In reality most babies are fed with a nursing bottle for at least some of the time and yet as Horowitz points out most of them do not develop ECC. Two bottle-related behaviours have attracted most interest in ECC research – the use of bottles at night/nap time and the use of the bottle beyond 12 months.

Reisine and Douglass found little strong evidence to support either of these ideas and suggest that this paucity of evidence may be due to the use of retrospective parental self-reports. The alternate explanation they offer is that the critical period may be soon after the eruption of teeth into the mouth and that early use of the bottle containing sweet fluids supports the early establishment and dominance of cariogenic microflora. This may be more important than bottle use after 12 months.

Litt *et al.* found that the use of the bottle at night-time was associated with sugar intake. The mothers who reported night-time bottle use were also more likely to have children with a higher sugar intake.

Because of this aspect is very important that the treatment of the pregnant mother should include education regarding preventive oral care of the infant and toddler.

Too often a child's first dental examination occurs after the deciduous dentition or even much of the permanent dentition has erupted. By this time, much opportunity for the prevention of dental pathology has passed. The expectant mother and family should be instructed in the importance of early dental examinations for the child. A child should receive the first dental examination between 6 to 12 months of age. Early examination enables the oral health professional to identify detrimental feeding habits, educate the parent about oral hygiene procedures, determine fluoride intake status, introduce the child to dentistry in a non threatening manner, and prepare the parents for the child's future dental needs (Goepferd & Garcia-Godoy, 1999).

Expectant mothers and their families should also be instructed in the care of the infant's mouth beginning at birth. The infant's mouth should be gently cleansed daily with a damp washcloth. As soon as teeth have erupted, they should be brushed daily with a soft toothbrush.

The deciduous dentition is critical to proper phonetic development, space maintenance for permanent teeth, and the child's self-image.

The number of teeth and surfaces at risk varies with age (Hausen *et al.* 1983, Nordblad & Larmas 1985a, Vehkalahti *et al.* 1991, Virtanen 1997), as also does the maturation age of tooth (Nordblad & Larmas 1985b). Nowadays the eruption of teeth occurs at an earlier chronological age than earlier (Helm 1969, Virtanen 1994, Eskeli *et al.* 1999). Most caries attacks on fissures occur during the first three years after eruption, and the survival of the first and second permanent molars immediately after eruption and filling increments in the upper incisors are good indicators of dental health. A distinction should be made between chronological and dental age, however. The post-eruptive filling placement curves for individual teeth,

obtained using the survival analysis method, evidently follow the pattern of caries attack. (Virtanen 1997)

The occlusal surfaces of permanent teeth are those most frequently attacked by caries (Nordblad & Larmas 1985b, Vehkalahti *et al.* 1990, Li *et al.* 1993, Virtanen & Larmas 1995), and more caries lesions have been demonstrated in pits and fissures of posterior teeth than on other surfaces (Dummer *et al.* 1990, Kingman 1993). The highest caries experience of all has been found in permanent molars (Nordblad & Larmas 1985b, Greenwell *et al.* 1990, Vehkalahti *et al.* 1990), whereas caries is seldom seen in teeth, such as canines, lower incisors and premolars (Nordblad 1986, Greenwell *et al.* 1990, Vehkalahti *et al.* 1990, Virtanen & Larmas 1995). The risk of occlusal caries is highest during and after tooth eruption (Härkänen *et al.* 2002), between 6 and 9 years of age for the first permanent molars, and after the age of 13 for the second permanent molars (Nordblad 1986, Ripa *et al.* 1988, Vehkalahti *et al.* 1991, Larmas *et al.* 1995). Approximal surfaces of permanent molars have been found to become carious after 12 years of age (Nordblad 1986, Ripa *et al.* 1988, Virtanen & Larmas 1995), and a correlation has been shown between past caries on approximal surfaces and the developing of new approximal caries lesions (Mejäre *et al.* 2001). Fifteen is an important age because of newly erupted second molars and the increasing role of approximal decay in the dentition (Vehkalahti *et al.* 1990).

The occurrence of caries has been declining in communities with and without organized preventive programs or fluoridation (Hargreaves 1987, Seppä *et al.* 2000). It is assumed that the reasons are related to the use of fluorides, to improvements in oral hygiene, or to a change in microbial, host and salivary factors, or to dietary changes (Marthaler 1984). It is suggested, however, that the most probable reason is related to the increased use of fluorides (Marthaler 1984, Bratthall *et al.* 1996), while according to Renson *et al.* (1985), the organized availability of dental resources and oral health education programs may be one explanation. The decline in caries may also have been due in part to new diagnostic and treatment criteria (Nadanowsky & Sheiham 1995). More recently, it has been reported that the decline in caries seems not to be associated with professional preventive measures performed in dental clinics (Seppä *et al.* 1998), but there is nevertheless a good deal of agreement on the preventive effect of fluoride toothpastes in this respect (Bratthall *et al.* 1996). Probably the most effective caries prevention treatment available today is fluoridation of municipal water supplies and the use of

fluoride toothpastes (Winston & Bhaskar 1998). According to Mandel (1996), the protective properties of fluoride dominate and host resistance wins out as an explanation for the decline in caries.

The use of the fluoride compounds on a large scale modified the evolution and progression of the carious lesions. For example, the occlusal caries become cavitory lesions much later and as a consequence the lesions that were cavitory sometimes ago, today is present only a slight modification in the enamel colour. This phenomenon of hidden caries was described in the early 80 and it was demonstrated in various clinical trials.

In the case of the caries localised on approximal surfaces there are modification in the relation between the depth of the lesion and the presence or absence of the cavity. If the lesion was considered cavitory at the moment that the radiotransparency reached the enamel-dentin junction, today much of the lesions with this depth are not cavitated lesions. This suggests that the disponibility of the fluorid modifie the radiografic image of the approximal lessions (Pitts, 1992).

When preventive procedure is planned it must be taken in consideration a few risk indicators: age risk, the period of risk, the teeth and surfaces with risk, the medical and social risk. (Bader *et al.* 1986, Nordblad & Larmas 1986, Virtanen 1997, Vehkalahti *et al.* 1997, Meurman 1997, Powell 1998).

Because of that, the coloboration with the pediatrician, wich has to oversee the geneal heath condition, is very important, any change in the general equilibrium could affect the oral health status.

The use of fluoridation, the sealing of pits and fissures together with oral hygiene implamantation and healty dietary habits are the 4 methods indicated by WHO(World Health Organiyation) for prevention of dental caries. The dentist has to aply the prophylactic measures, has to implement an adequate toothbrush teqniques contributing to the consolidation of oral health.

A healthy diet which must contain all the necessary nutrient elements is important from the pediatrician point of view for an adequate groth and development and also for the dentist in order to prevent the risk factor represented by the existense of a cariogenic diet –carbohydrate substrate.

The understanding of the complex nature of caries depends on the multiple risk factors that have to be taken in consideration and the prophylactic and therapeutic measures are based on the accurency of the evaluation of all this factors.

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