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DENTAL CARIES AS AN INFECTIOUS DISEASE AND ITS RELATION WITH A WINDOW OF INFECTIVITY

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ABSTRACT

Dental caries is known as behavior disease, but now its known as infectious disease that can be spread. The transmission can occur horizontally and vertically. Most of transmission occurs vertically, the mother or primary caretaker has been identified as the principal source of the bacterial infection, there is transmission of *Streptococcus mutans*. Children with low or non-detectable levels of *Streptococcus mutans* at an early age are at lower risk of caries in their primary teeth. From the study in Northern Mariana Islands,USA found that the colonization of *Streptococcus mutans* occurred in the period 6 to 12 months of age(53%) and 13 to 24 months (72%), whereas according to a study in Alabama, the colonization occurred in the period 19 to 31 months of age. This is called "the window of infectivity". The earlier the *Streptococcus mutans* colonization in a child, the higher is his/her caries risk.

Key words: Dental caries, *streptococcus mutans*, window of infectivity

INTRODUCTION

Dental caries, also known as tooth decay or cavity, is a disease where bacterial processes damage hard tooth structure (enamel, dentin, and cementum). Tooth decay is caused by specific types of acid-producing bacteria that cause damage in the presence of fermentable carbohydrates such as sucrose, fructose, and glucose.[1]

According to Keyes and Jordan in 1960, caries is a multifactorial disease, that is the existance of several factors that cause the formation of caries. These factors are the presence of cariogenic microorganisms (Microbes), fermentable carbohydrate in the diet (Substrate), susceptible tooth (Host&Teeth),and time. The development of caries depends on the interaction between them.[1]

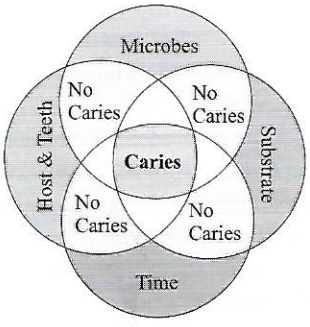

Figure 1. Caries is a multifactorial disease.[2]

As we all know, the main cause of caries is the acid produced by bacteria in plaque. Thus the occurrence of caries is often associated with the bad behavior which is can not maintain the oral hygiene. Therefore, to prevent the occurrence of caries is always associated with changing behaviors. Now, there's a changing paradigms concepts for dental caries. New paradigm is caries as an infectious disease and window of infectivity.

Dental Caries as an Infectious Disease

The use of the concept 'an infectious disease' immediately signals that a disease is caused by a particular microorganism or agent, which has 'infected' an individual. As defined by Last, who uses infectious disease synonymously with communicable disease: "an illness due to a specific infectious agent or its toxic products that arises though transmission of that agent or its toxic products from an infected person, animal or reservoir to a susceptible host" .[3]

Mutans streptococci (Streptococcus mutans and *Streptococcus sobrinus),* acidogenic and aciduric micro-organisms which colonize the oral cavity, are associated with the development of caries.[4] It is occasionally claimed that because *Streptococcus mutans* cannot be detected in some patients with no caries increment this is a 'proof' that you need to be 'infected' with *Streptococcus mutans* to get a lesion.[3] *Streptococcus mutans*, a major pathogen of dental caries, can colonize the mouth of children at an early age, depending on the presence and intensity of factors that favour its transmission and establishment in the oral cavity. [5] The transmission of Streptococcus mutans can occur horizontally and vertically, which is:

a. Horizontal transmission is the transmission of

microbes be-tween members of a group (eg, family members of a similar age or students in a classroom). More recent reports indicate that vertical transmission is not the only vector by which *Streptococcus mutans* are perpetuated in human populations. Hori-zontal transmission also occurs.[6]

b. Vertical transmission is the transmission of microbes from mother/caregiver to child. The major reservoir from which infants acquire *Streptococcus mutans* is their mothers. Transmission usually occurs from mother to child during feeding and nurturing .[7] The early evidence for this concept comes from bacteriocin typing studies where *Streptococcus mutans* isolated from mothers and their infants demonstrated identical bacteriocin typing patterns. More advanced technology that utilized chromosomal DNA patterns or identical plasmids provided more compelling evidence to substantiate the concept of vertical transmission. [6]

However, several factors might influence the initial age of *Streptococcus mutans* acquisition, for example the extent of sucrose intake and the frequency of salivary contact with *Streptococcus mutans*-colonized subjects have been defined as the main source of *Streptococcus mutans* transmission to children), but other familial and non-familial sources of transmission may also exist salivary contact between nursery children during a phase of initial establishment of their oral microbiota and maturation of the immune response might also contribute to the transmission of oral micro-organisms. The identification and understanding of the sources and course of *Streptococcus mutans* transmission are important to define strategies to control the initial colonization.[5]

Window of infectivity

Oral microbes start to colonize an infant's mouth soon after birth. Successful colonization *Streptococcus mutans* is likely to depend on the presence of a favourable environment of the dental microbial biofilm. Pioneer microbes entering the oral cavity can influence the colonization of later species and microbes arriving later will need to compete for colonization sites and nutrients. As earlier-arriving species have an ecological advantage over those arriving later, it is likely that early arrival of *Streptococcus mutans* will promote their persistance in the mouth compared to later arrival.The numbers of oral bacteria increase gradually from exposure with microbial sources from the external environment .

As is the case regarding timing of development and maturity of the dental biofilm in children, the timing of initial *Streptococcus mutans* acquisition in children is not yet established. Most studies suggest that the bacteria can be acquired at any time from under 6 months to over 3 years of age. Until recently, it was thought that *Streptococcus mutans* require a nonshredding surface to colonize and multiply.

DISCUSSION

Initial acquisition and transmission of *Streptococcus mutans* have been widely studied. Mother-child transmission has been identified as the major route for early-infancy *Streptococcus mutans* acquisition.

A small study by Caufield *et al.*, 1993 suggested that there is a "window of infectivity" for initial acquisition of *Streptococcus mutans* at a mean age of 26 months. Thirty-eight of the 46 infants initially acquired *Streptococcus mutans* at a median age of 26 months. These infants came from a population predicted to be at high risk for acquiring *Streptococcus mutans* based on their mothers' high levels of *Streptococcus mutans* in saliva as well as past dental experience. *Streptoccoccus mutans* was detected in 25% of these 38 infants by 19 months of age and in 75% by 31 months of age. Eight children remained free of *Streptococcus mutans* for a period greater than 56 months. This finding was based upon repeated negative samples for *Streptococcus mutans* taken at three-month intervals. The non-*Streptococcus mutans*-colonized children were mainly black (seven of 8; 87.5%) compared with the *Streptococcus mutans*-colonized cohort (20 of 38; 52.6%) ($p = 0.07$). Neither gender nor mother's past dental history (DMFS or DMFT indices) was s ignificantly different between the *Streptococcus mutans*-colonized and non-*Streptococcus mutans*-colonized cohorts.[8]

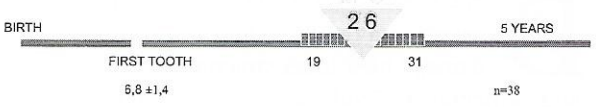

Figure 2. The window of infectivity (Caufield, 1993)[8]

The median time to initial acquisition of mutans streptococci for 38 infants is depicted as afunction of infant age in months. The average age and standard deviation at emergence of the first primary tooth are also shown for these 38 infants.

On the other hand, longitudinal studies by Wan and co-workers, Law and Seow in 2001 showed that there was increasing *Streptococcus mutans* colonization with increasing ages of the children, without any discrete windows of infectivity. There is now clinical evidence that *Streptococcus mutans* can be detected in mouths of predentate children prior to the eruption of the first tooth. Wan and co-workers showed more than 30 per cent of predentate children at the age of 3 months were infected with *Streptococcus mutans* and over 60 per cent showed presence of the bacteria by the age of 6 months.[9]

Karn *et al.* in 1998 also reported a prevalence of 25 per cent of *Streptococcus mutans* infection in children aged 12 months, in the 15-month age group, 60 percent were colonized.[10,11] Thorild *et al,* 2002 detected *Streptococcus mutans* in 30 per cent of 18 month old

children.[12] Higher infection rates were reported by Roeters *et al* in 1995., who found *Streptococcus mutans* in 43 per cent of preschool children aged 2 to 5 years[13], and Milgrom *et al.*,2000 who reported *Streptococcus mutans* in 53 per cent of children aged 6 to 12 months and 72 per cent in those aged 13 to 24 months. Other investigators also reported similarly high levels in 24 month old children.[14]

The timing of *Streptococcus mutans* colonization is of clinical significance as there is evidence that the earlier the *Streptococcus mutans* colonization in a child, the higher is his/her caries risk. A few longitudinal studies have reported that children who had acquired *Streptococcus mutans* before 2 years of age showed greater caries experience in both primary and permanent dentitions compared to children who were colonized at later ages. The first report by Alaluusua and Renkonen, 1983 found that children who had *Streptococcus mutans* by the age of 2 years showed higher caries experience compared to children who were colonized later in childhood. This observation, together with the fact that *Streptococcus mutans* is usually transmitted by the mother, led to the concept that prevention of caries in children may be possible by reducing maternal *Streptococcus mutans* counts and delaying *Streptococcus mutans* colonization in the children.[15]

In later clinical studies, Kohler *et al.*, 1988 investigated children who were colonized at different times due to differing levels of maternal *Streptococcus mutans*, and correlated the colonization times with caries experience.The authors reported that 4 year old children who were colonized at ages below 2 years had significantly lower caries experience compared with children who were colonized after 2 years of age. When the same cohort were followed-up at 7 years of age, the same group of children who were colonized after 2 years of age had the lowest levels of *Streptococcus mutans* and significantly lower caries risk compared to those who were colonized earlier. In other studies which attempted to reduce *Streptococcus mutans* transmission by reducing maternal levels.[16] Isokangas *et al.* , 2000 also reported that colonization with *Streptococcus mutans* before 2 years of age is associated with significantly earlier caries attack in the primary dentition compared to colonization after 2 years of age.[17]

Early colonization and high levels of Streptococcus mutans in infants and young children is promoted by a range of factors and is associated with high caries experience : a high carbohydrate intake, sweetened pacifiers, fruit juices, cordials, soft drinks are a source of carbohydrate, a grazing pattern of eating, poor pre natal nutrition and peri-natal illnesses may cause enamel hypoplasia and predispose to early childhood caries.[18] And according to study by Milgrom et al, 1999 there's three behaviors that have the propensity to increase colonization have been studied extensively (baby bottle use, sugar consumption, and toothbrushing). Bottles were given to the children until after 2 years of age in most of the cultures. Length of time given a bottle past 12 months of age was related to enamel cavitation but not white spot lesions or level of microbial infection[14]

Future prospects to prevent the caries

In deciding what detection levels of dental caries should be used in planning dental public health problem[19]. Public health programs have been organized to prevent deadly infectious diseases promote health by programs designed to reduce the exposure to risk factors in populations. The basic view of public health should be modified to incorporate all activities that the public or health professionals undertake to promote health, prevent disease or manage the damage caused by a disease. These guidelines are expected to be published in late 2005 as a New York State Health Department publica-tion. The currently drafted recommendations for dental providers addressing the issue of interfering with early acquisition of *Streptococcus mutans* are as follows:[5]

1. Reduce the *Streptococcus mutans* reservoir in the mother, sibling(s), and all of the infant's caretaker(s) by eliminating active dental caries lesions and using agents such as fluorides and chlorhexidene.
2. Alter saliva-sharing activities, such as tasting food before feeding and sharing toothbrushes.
3. Twice daily tooth-brushing of the dentate infant with an appropriate amount of an American Dental Association-approved fluoridated toothpaste.
4. Avoid decay-promoting feeding behaviors.
5. Oral health evaluation of the infant by a dental profes-sional should occur before the first birthday.

The paradigm caries as a transmittable, infectious disease: even one caused by specific microorganisms would require caries prevention by vaccination, but there is evidence that caries is not a classical infectious disease. The concept of vaccination against dental caries has existed almost from the time that this disease was recognized to result from colonization of the teeth by acidogenic bacteria. [20,21]

CONCLUSIONS

Based on this paper's findings, the following conclusions can be made:

dental caries is an infectious and transmis-sible disease. Streptococcus mutans are important organisms in the initiation and pathogenesis of dental caries. Recent evidence demonstrates that these bacteria are found in the mouths of predentate infants and are acquired via vertical and horizontal transmission from human reservoirs. Improvements in the prevention of dental caries may likely be realized through intervention strategies that focus on the natural history of this infectious disease. This information should facilitate the focusing of clinical interventions that prevent or delay

infant infection, thereby reducing the prevalence of dental caries

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