

The relationship between diet, saliva and baby bottle tooth decay

Stephen J Moss
New York, USA

Summary

The problem of early infant caries (Baby Bottle Tooth Decay or Nursing caries) occurs worldwide. This paper examines reasons for this occurrence and suggests that it is the quality and quantity of saliva wetting the maxillary incisors that is the primary cause. Applying this thesis, the author indicates two relatively novel, non-dietary approaches that practitioners and parents can take to prevent this disease. Additionally, the author proposes that in order to place focus on obtainable prevention, the name of this disease might be changed to Tooth Cleaning Neglect.

Today the condition known as Baby Bottle Tooth Decay (BBTD) or nursing caries occurs and is recognised worldwide. The prevalence of this condition in various populations has been estimated as ranging from 3 to 45 percent¹. Many paediatric dentists believe that it is the most common cause for caries occurring in infants and pre-school children. This paper explores some of the reasons why infants' teeth, particularly the maxillary incisors, are susceptible to this form of rapid and destructive demineralisation. The issues raised herein challenge the concept that the primary cause for the development of BBTD is prolonged feeding from either the nursing bottle or the breast. It is the author's intent to direct some focus away from the dietary aspects of this unique pattern of demineralisation and teach toward a more primary cause – the quality and quantity of the saliva wetting the infant's maxillary incisors. It is hoped that such an approach might provide practitioners and parents with insights leading to more logical and effective methods for both treating and preventing BBTD.

The combined effects of saliva, fluoride and diet

Surveys in a dozen countries worldwide have shown a dramatic increase in the number of children reaching their teens totally caries-free. Studies confirm that the single factor common to all these countries is the widespread daily use of dentifrices containing fluoride².

Caries is not prevented by fluoride toothpaste acting alone. The true workhorse is saliva. Fluoride, from dentifrices, plays the important role of a chemical catalyst, enhancing saliva's ability to do its job of protecting, maintaining and repairing tooth enamel³.

Saliva is to tooth enamel what blood is to the cells of the body. Just as body cells depend on the bloodstream to supply nutrients, remove waste, and protect the cells, enamel depends on saliva to perform similar functions. Saliva, working with fluoride, can neutralise and overcome dietary caries challenges and inhibit the caries process. The eventual understanding of the interrelations between diet, fluoride and saliva should enable dental health professionals to better understand the clinical phenomena that caries occurs only on specific locations (sites) on the teeth. It is helpful to recall some of saliva's numerous beneficial actions:

- Saliva protects hard and soft tissue from desiccation
- Saliva enhances taste
- Saliva lubricates the food bolus, which facilitates chewing and swallowing and prevents scratching of the soft tissues and the oesophagus
- Saliva speeds oral clearance of food particles and dissolves sugars
- Salivary enzymes facilitate the removal from the mouth of insoluble carbohydrates
- Salivary buffers neutralise organic acids produced by plaque bacteria
- Salivary minerals act on tooth structure to inhibit demineralisation and enhance remineralisation

- Saliva recycles ingested fluoride into the mouth
- Salivary elements discourage the growth of bacteria, which inhibits infection
- Salivary proteins absorb onto tooth surfaces, inhibiting mineral loss and bacterial adhesions.

Saliva is supersaturated with calcium and phosphate, the principal minerals that make up human enamel⁴. Some scientists think of saliva as a form of liquid enamel. In the mouth, while the enamel surface is awash in this supersaturated mineral bath, a continuous interchange of calcium and phosphate ions occurs between the saliva and the enamel. An equilibrium is established between the minerals in the enamel surface and those minerals present in the plaque fluid and saliva.

For substrate, plaque bacteria depend on ingested sugars and cooked starches. Too often some practitioners fail to think of cooked or processed starch foods as potential substrate for bacterial metabolism. Intra-orally, the salivary enzyme amylase quickly breaks down cooked starches into the simple sugars maltose and glucose. This is a mixed blessing, beneficial because it acts to clear insoluble starches from the mouth, but a problem because the maltose and glucose are metabolised by plaque bacteria leading to dangerous levels of organic acid production. Bacteria can also metabolise starches without requiring salivary enzymes to first turn the starches into sugar. Recent research has shown clearly that starch products trapped in plaque can be metabolised directly to organic acids with or without salivary amylase⁵.

Understanding demineralisation and remineralisation, we realise that early lesions, where the enamel surface has not been broken, can be remineralised using the saliva as the remineralisation agent. Also, many clinicians now realise that carious lesions develop on the certain tooth locations where there is an insufficient flow of saliva to provide buffer against the acid condition. In the presence of mature plaque, all foods containing even small quantities of sugars or cooked starches have the potential to lead to all acid condition. This phenomenon is one of the reasons why it is difficult to make judgements between the relative cariogenicity of different foodstuffs, and why we should not be overly concerned that any particular food is either 'good' or 'bad' for teeth. When making dietary suggestions for caries prevention, many practitioners focus on daily frequency of ingestion (no more than 3 or 4 snacks) as well as on the nutritional aspects of what is eaten.

Baby Bottle Tooth Decay

Unfortunately, the bottle alone is mistakenly charged as being the villain here, hence the name: Baby Bottle Tooth Decay. This is far from the complete story. Considering the inter-relationships between saliva and diet helps us to better understand how and why the rapid tooth destruction of the maxillary incisors, known as Baby

Table 1 Salivary secretions relevant to caries (after C. Dawes 1994).

Glands	Parotid	Submandibular sublingual	Minor mucous
Viscosity	Very low	Medium	Very high
Inorg. P.	10.8	3.6	0.6
HCO ₃	1.0	2.2	0.0



Figure 1

- This mother is demonstrating an effective position to use while cleaning her infant's teeth. She is using a small piece of gauze between her fingers and wiping her child's teeth clean. This cleaning should be done twice a day.
- Cette mère présente une posture efficace lors du nettoyage des dents de son nourrisson. Elle utilise un petit morceau de gaze entre ses doigts et nettoie les dents de son enfant. Ceci doit être fait deux fois par jour.
- Diese Mutter demonstriert die optimale Position zum Säubern der Zähne ihres Kindes. Mit einem um die Finger gewickelten Mullstück nimmt sie die Zahnreinigung vor. Dies sollte zweimal täglich erfolgen.
- La madre demuestra una posición efectiva para limpiar los dientes del infante. Utiliza un pequeño trozo de gasa entre sus dedos para limpiar los dientes del niño. Esta limpieza debe efectuarse dos veces al día.

Bottle Tooth Decay, develops in infants. What are the other factors that make maxillary incisors more susceptible to caries than other tooth sites and an infant's mouth susceptible to bacterial infection?

The nature of saliva and its flow around an infant's maxillary incisors make those teeth highly susceptible to bacterial colonisation⁴. Because of gravity and the location of the salivary ducts, salivary flow around the maxillary anterior teeth is notoriously low and slow. The sucking action during nursing hinders salivary flow. Also, because infant lip muscles are not yet sufficiently developed to remain 'sealed', saliva on the upper incisors tends to evaporate and the incisors become dry.

Sugars in the bottle or in breast milk merely act as substrate for the bacteria. Any carbohydrate will do, even the lactose in mother's milk. The rapid and extensive tooth breakdown is associated with an overgrowth

of *Streptococcus mutans*. *Streptococcus mutans* is a bacteria that has very particular nutritional requirements and frequent feeding is necessary for it to colonise. An infant using either the bottle or breast as a pacifier fulfils this important biological requirement.

Further complicating the process is the fact that saliva does not mix well. This complication matters because the compositions of parotid, submandibular, sublingual, and minor mucous gland secretions are so different in many respects. *Table 1* shows that, relevant to caries, submandibular and sublingual saliva is the better buffer and contains more phosphorous for remineralisation. Note also that the viscosity of the minor mucous glands is very high⁶.

Why is a buccal surface more susceptible to caries than the lingual surface of the same tooth? Studies of salivary flow show that salivary film velocity is much slower buccally than lingually. The buccal surfaces of the infant's maxillary incisors are wet mainly by saliva from the highly viscous minor mucous glands⁶. Saliva from these lip glands is viscous and low in both mineral and buffer.

The situation is also complicated by the fact that babies sleep more than adults, and less saliva is produced during sleep. Finally, as mentioned above, in the infant's mouth there is little mixing of saliva. The characteristics of the saliva that wet the infant's maxillary incisors are poor buffering capacity, low phosphorous content, and high viscosity. The nature of an infant's saliva is also different than that of an adult.

Whereas adults are protected by the immunoglobulin secretory IgA in their saliva, infant saliva contains only half the concentration of secretory IgA found in adult mouths⁷. Because an infant's salivary antibody titre is so low, microorganisms are more likely to flourish in the infant's mouth. Babies are continuously

inoculated with bacteria by their parents through kissing, sharing of food and other contacts. Bacterial genetic typing indicates that it is most often the mother that passes the acidogenic organisms to the child. It appears certain that the infant's oral flora is established quite early during tooth eruption, most likely before the second birthday⁸. It seems impossible to stop such a transmission however, to keep the number of bacteria in the inoculum low, parents should be encouraged to clean their own teeth during the time their infant's teeth are erupting. The infant's teeth, when appearing in the mouth also should be cleaned twice a day (*Figure 1*).

As for the cleaning of babies teeth, perhaps 'Baby Bottle Tooth Decay' should be renamed 'Tooth Cleaning Neglect', so as to shift the emphasis away from the bottle toward the need for cleaning. After all, consider the parents who did not change a baby's diaper. They would be neglectful. So, too, are the parents who do not take the time to clean the infant's teeth, to prevent the colonies of microorganisms from establishing themselves on tooth surfaces that are relatively unprotected by saliva. Twice daily cleaning not only prevents the establishment of colonies of the particular organism responsible for this disease (*mutans streptococci*) but can also serve to retard and/or arrest the development of early caries. Since fluoride tends to inhibit demineralisation and favour remineralisation, when early lesions are discovered it is helpful to place on the tooth cleaning gauze a tiny amount of a properly constituted fluoride dentifrice. After cleaning care should be taken to remove remaining dentifrice in order to prevent ingestion.

In conclusion, to prevent the development of early infant caries, infant caretakers should be: taught to clean their infant's teeth daily, simultaneously encouraged to clean their own teeth, and cautioned about not allowing the infant to use either the nursing bottle or the breast as a pacifier.

F

Rapport entre le régime alimentaire, la salive et la carie dentaire consécutive au biberon

Résumé

Le problème de la carie dentaire consécutive au biberon ou carie d'allaitement existe dans le monde entier. On étudie dans cet article les raisons de son apparition et il y est suggéré que la déficience salivaire en est la cause principale. On présente les démarches que peuvent suivre les praticiens et les parents pour essayer d'enrayer ce problème.

D

Interdependenz: Diät, Speichel und "Fläschchenkaries"

Zusammenfassung

Das "Nursing-Bottle-Syndrom", d.h. die Kariesentstehung im Kleinkindalter durch gesüßte Getränke aus der Flasche, ist ein weltweites Problem. Im vorliegenden Beitrag, der die Gründe hierfür untersucht, wird als Primärursache Speicheldefizienz angeführt. Es wird aufgezeigt, welche Maßnahmen Zahnärzte und Eltern ergreifen können, damit es nicht zur Entstehung dieses Kariestyps kommt.



La relación entre la dieta, la saliva y la caries de biberón

Resumen

El problema de la caries de biberón existe en todo el mundo. Este trabajo examina las razones por las cuales ocurre esta condición y sugiere que la deficiencia salival es la causa principal. Se presentan métodos que los dentistas y los padres pueden adoptar para tratar de prevenir este problema.

References

1. National Center for Health Statistics. *Healthy People 2000 Review, 1993 - Oral Health*. Hyattsville, Maryland: Public Health Service, 1994. Chapter 13, 80-81.
2. Moss S J, Sarnat H, Koch G. Dental Caries: Disease in Decline. Slide Programme. Education Committee - International Association of Dentistry for Children, the London Hospital Medical College, London, United Kingdom, 1985.
3. Nikiforuk G. Understanding Dental Caries - 2. Prevention, basic and clinical aspects. In Karger A G (ed) *Mechanism of Cariostatic Action of Fluoride*. Chapter 3, 45-62. Switzerland: Basel, 1985.
4. Moss S J, Sarnat H, Koch G. *et al.* Insights into Saliva Action. Slide Programmes. Education committee. International Association of Dentistry for Children.
5. Granath L, Schroder U, Poulsen S, *et al.* Dental Caries: Analysis of disease factors. In (Eds) Koch G, Modeer *et al.* *Pedodontics - A Clinical Approach*. pp 143-151. Copenhagen: Munksgaard, 1991.
6. Dawes C. The ebb and flow of the salivary tide. In Bowen W, Tabak L. (eds) *Cariology for the Nineties*. pp 133-141. Rochester, New York: University of Rochester Press, 1993.
7. Oppenheim F G. Salivary Antimicrobials: where are we now? In Bowen W H, Tabak L A. (eds) *Cariology for the Nineties*. pp 117-131. Rochester, New York: University of Rochester Press, 1993.
8. Thibodeau E A, O'Sullivan D M. Salivary Mutans streptococci and incidence of caries in pre-school children. *Caries Res* 1995 **29**: 1 148-53.

Correspondence to: Professor S.J. Moss, New York University, Department of Paediatric Dentistry, 345 East 24th Street, New York, New York 10010, USA.