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NATURE OR NURTURE: DIET, DENTAL CARIES AND ORAL IMMUNOLOGY

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While fluoride has inarguably been the single most important therapeutic factor in the decline of tooth decay, especially among school-aged children in industrialized countries, its true role in preventing caries has been mythicized into oversimplification.

Caries is not prevented by fluoride dentifrice acting alone, nor is it caused by the absence of fluoride alone. The true workhorse of the mouth, in fact, is saliva. Fluoride does play the important role of chemical catalyst, augmenting saliva's ability to do its job of maintaining, protecting and repairing tooth enamel.

THE INTRA-ORAL FLUORIDE RESERVOIR

Fluoride accumulates in residual plaque, on the enamel surfaces and on soft tissues. It is stored intra-orally on the teeth and in plaque as calcium-fluoride-like precipitates covered by salivary phosphates. **1**. Since saliva is supersaturated with respect to phosphate the precipitates do not readily dissolve and resemble calcium-fluoride-like particles that accumulate in the mouth. This occurs before the enamel dissolves, releasing fluoride ions at the saliva-enamel interface, precisely where and when they are needed. **(Figure 1)**

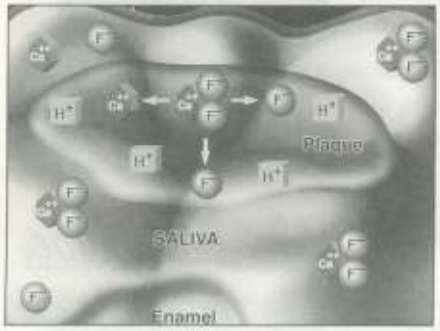

Figure 1. Fluoride is stored intra-orally on the teeth and in plaque as calcium fluoride-like precipitates. Since saliva is supersaturated with calcium and phosphate, the precipitates do not dissolve until the plaque become slightly acidic. This occurs before enamel begins to dissolve, releasing fluoride ions at the saliva-enamel interface to prevent demineralization.

Fluoride and diet are related and inseparable in the caries process. Whenever fluoride in a properly-constituted toothpaste is used twice daily as recommended, diet is certainly less of a factor in the caries process because fluoride augments the components and defensive action of saliva. **(Figure 2A and 2B)**

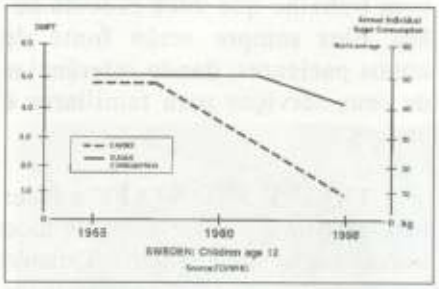

Figure 2 A. Sweden.
In the past two decades, annual per-capita sugar consumption in Sweden has remained relatively constant. Caries rates, however, have dropped precipitously during the same period, demonstrating through epidemiology that with the use of fluoride dentifrice, diet and dietary factors become far less significant in the caries process.

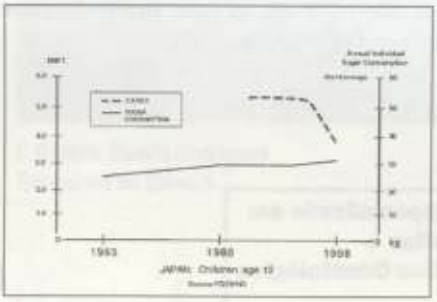

Figure 2B. Japan. Despite a high dentist to population ratio and consumption of sugar at less than half the world average, Japan has had one of the highest caries rates among industrialized nations in the past 10 years. This phenomenon is attributed to the absence of fluoride dentifrice on the Japanese market until the early 1990's. A sharp drop in caries for Japanese 12-year-olds has been documented since fluoride dentifrice was introduced.

When the enamel surface remains bathed in saliva, there is a continuous exchange of calcium and phosphate ions between the saliva and the surface of the enamel. These are typically in equilibrium, or homeostasis. Fluoride offers additional help *when* present at the tooth surface bu providing ions, which inhibit demineralization and promote remineralization, as opposed to rebuilding the tooth structure itself as was long believed. **2. (Figure 3)**

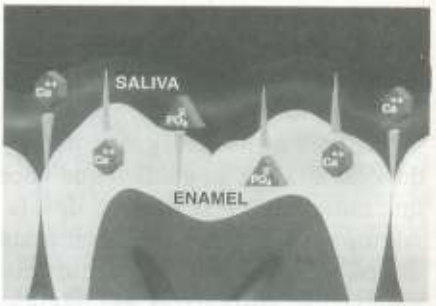

Figure 3. With the enamel surface bathed in supersaturated saliva, calcium and phosphate ions constantly interchange. They establish and equilibrium with the enamel surface and the minerals present in plaque fluid and saliva. Caries occurs when equilibrium on particular sites of the teeth is disturbed, resulting in more mineral being lost *than* replaced.

Saliva is to tooth enamel what blood is to the cells of the body; just as the body depends on the bloodstream nutrients, remove waste and protect the cells; tooth enamel depends on saliva to perform similar tasks. The importance of saliva is obvious in the rampant caries that can occur in individuals with xerostomia.

LESS SUGAR EQUALS FEWER CARIES?

The story of how caries has decreased in recent years is not black and white. Caries researchers believe the decrease is multi-factorial, or a phe-

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nomenon that has relied on the synergy of several factors. Therefore, attributing the decrease in caries to less sugar in the diet is not only simplistic, but inaccurate. In fact, industrialized countries consume sugar in and everincreasing array of foods, including many processed foods in which it is "hidden". In the last 10 years, the true role of diet in dent health has become clearer, as the line between foods are potentially cavity-causing because most contain some type of carbohydrate in the form of sugar, starch or a combination of the two. **3.**

Natural sugars such as fructose in fruit and fructose in fruit and lactose in milk have just as much potential to cause decay as processed sugar, as do a number of starches found in products such as crackers and potato chips. **(figure 4)** To highly evolve and sophisticated oral bacteria that mediate tooth decay, the ingredients are virtually all the same. Oral bacteria have evolved over thousands of years to become extraordinarily efficient at adapting to and utilizing the various foods we eat.

Recently, Boston's Forsythe Dental

Figure 4. *When plane is mature, all foods containing even minuscule amounts of sugars or cooked starches (carbohydrates) have the potential to generete acid prodution. This makes it virtually impossible to compare the realtive cariogenicity of varius foods and supersedes lists os "good" and "bad" foods. All of the foods pictured have some potential for entering into the caries process.*

Center reported findings that support the view that "high-starch foods contribute to the development of caries lesions". **4.**

The study found a critical difference in the way that high-starch and high-sugar foods contribute to the caries process, but the and results could wellbe the same. While confectionery foods delivered high levels of sugars to the oral bacteria immediately after in- gestion, those levels lasted only a short time. High-starch food started out slower, but delivered progressively higher concentrations of sugars over a considerably longer period.

In may 1989, the American Academy of Pediatric Dentistry board of trustees acknowledged the changing concept of caries when it commented that "dramatic reductions in caries following widespread access to fluorides have resulted in decreased emphasis on dietary counseling as a preventive strategy for all children... frequency of carbo hydrate consumption should be restricted for cavity-susceptible children (those who develop decay in other than chewing surfaces). This traditional advice may be relaxed for ca- vity-free and low-cavity children who are regularly exposed to fluoride and comprehensive dental care".

The board's point: while there is no question that dietary restrictions or modification word to limit tooth decay other preventive methods, in light of new scientific evidence, seem more affective and today have a better chance of success. Put another way, we have seen the potential enemy, and it is us, not sugar or starch. **5.**

Other than stimulating salivary flow and providing the micronutrient fluoride, nutritional status actually plays little direct role in the prevention of tooth decay. From the standpoint of diet, in the case of frequent snacking, three to four eating "events" a day appear to be in the safe average range regardless of what is eaten if fluoride dentifrice is used twice daily to augment saliva's protective properties. More important is the absence of the fluoride ion at the saliva-enamel interface.

Only small amounts of carbohydrate are needed for the maximum production of lactic acid in the mouth. And that acid, the predominant factor in the decay process, is produced for up to 30 minutes whether or not the carbohydrate remains in the mouth. This knowledge is paving the way for a more progressive perspective of tooth decay and the caries process. **(Figure 5)**

The Forsythe study offers further

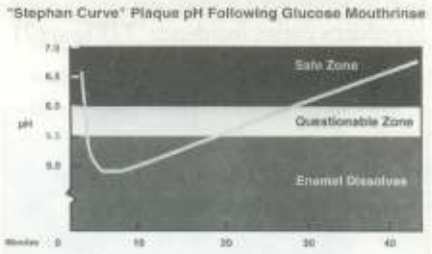

Figure 5. *Using a Stephan Curve, or the typical intraoral pH response to fermentable carbohydrates, researchers can show that even a short exposure will produce and immediate pH drop into the enamel dissolution zone and will rise only gradually back to neutral. Even when a solution that leaves the mouth quickly is used, an acid condition will exist for another 30 minutes. This demonstrates that the longer carbohydrate containing food stays in the mouth, the longer the pH will remain depressed.*

confirmation that the theoretical line between good and bad foods, dentally speaking, is just that. Adding to the difficulty in making food compa-risons are food-related factors that can determine cariogenicity. Those include how much saliva a certain food stimulates and whether that food contains natural buffers against the acid produced by oral bacteria. Or, a certain food may be naturally acidic or may take longer to clear the mouth. **6. (Figure 6)**

Model and insitu research has found

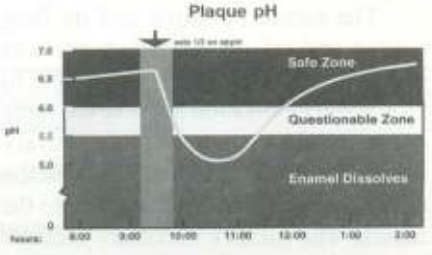

Figure 6. *This chart tracks the depression of oral pH after ingestion of an apple, residual pieces of which can depress the pH for more than 2 hours. This illustrates the point that identifying good and bad foods is futile. More important than what is eaten is how many times we eat between meals and how long residual food material remains in the mouth after eating.*

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that chewing sugarless gum after eating stimulates saliva to neutralize plaque acids and promote remineralization, for example. A recent study further supports the finding that chewing sugarless gum after eating augments the remineralization of white spot lesions and significantly reduces caries incidence. **7.(Figure7)**

Saliva Is The Key

Figure 7. Saliva flow is always increased by physical movement of the mandible, such as in chewing, and its production is greatest on the chewing side. This "stimulated" saliva has a higher buffering capacity, which makes chewing sugarless gum after eating a practical and useful recommendation.

This inter-relationship be-tween saliva, its components and diet helps better explain the how and the why of the rapid tooth destruction in infants known as Baby Bottle Tooth Decay (Early Infant Caries), for instance. Is the sweetened liquid in a baby bottle the villain? Or are there other mediating factors involved in the process?

The nature of saliva and its flow around and infant's maxillary incisors make those teeth highly susceptible to bacterial co-lonization because the force of gravity and the location of the salivary ducts provides little salivary flow to the anterior teeth. Saliva does not mix in the mouth. The infant's maxillary incisors are wet by saliva from the minor mucus glands. These glands lack buffer and have little mineral content. **(Figure 8)**

Also, because infant lip muscles are not developed enough to remain "sealed", saliva on the upper incisors tends to evaporate and the incisors become try. Nutrients in the bottle simply act as fuel for the bacteria. **8.**

Salivary Secretions Relevant to Caries

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

(after, C. Davies, 1993)

Figure 8. This chart shows that, relevant to caries, saliva produced in different parties of the mouth has different composition and properties. Submandibular and sublingual saliva has been shown to be better buffers and contain more phosphorous, which aids saliva in the remineralization process. In the mouth, these various salivas do not mix well. The viscosity of saliva the minor glands is very high. The minor salivary glands are those responsible for protecting the infant's maxillary anterior teeth.

The modern model of tooth decay involves the constant interaction of carbohudrates, saliva, salivary components, serum antibodies, fluoride and oral microorganisms. Recent scientific evidence has revealed that the synergy between foods, saliva, fluoride and our immune systems is highly variable from person to person and is far more complex and important than once imagined or understood. All those factors, under normal conditions, exist in and oral equilibrium that has evolved in human over thousands of years. **9.**

THE GENESIS OF CARIES

Tooth decay begins only when that natural oral stasis is disrupted, which could be prompted by environmental or hereditary factors or by a change in the quality of salivary flow.

Like a terrarium, the mouth is a complex, delicately balanced and self-contained environment. Microorganisms such as the bacteria involved in the dental diease process

are not transients but permanent residents that have evolved over thousands of years and which live in harmony with the structures of the mouth. Therefore, dental caries can be viewed as a breakdown or chance in homeostasis at specific sites in the mouth that can lead to and ecological advantage for the microorganisms involved in dental disease. **10.**

The interactions of several forces also may help explain why some people are more resistant to caries than others, even when their food intake is virtually the sama. Familiar and twin studies a have established that there is a strong genetic component in caries development. Subsequent comparative studies of nonrelated caries-resistant and caries-susceptible people has shed further light on the phenomenon, providing a basis for the existence of immune-host factors that may be genetically regulated and can reside in the teeth, plaque or saliva, or any combination thereof. Or, perhaps, the difference lies in the relative virulence of various strains of S. mutans passed from mo-ther to infant child during a caries-susceptible period that extends to about the fourteenth month of life. **11.**

A consistent and significant difference between caries-resistant and-susceptible adults lies in plaque pH, for example. When examined, plaques from caries-resistant adults have a higher initial pH, a significantly smaller drop in pH and a more rapid return to normal. **12.**

In summary, the last 30 years has seen major progress in the understanding of dental caries and nutrition, yet even now the true nature of the relationship if often misunderstood and frequently ignored. Research findings have shown that preventive programs should be expanded to include not only food consumption patterns, oral hygiene and fluoride, but the amount and composition of saliva, the immunologic response of the individual and the bacterial composition of the individual's plaque biofilm. **13.**

Note: All of the figure in this article were taken from the educational slide program, Insights Into Saliva Action, produced by the International Association of Pediatric Dentistry and support by the Colgate-Palmolive Company.

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REFERENCES

1. MOSS SJ et. alli. Insights into Saliva Action (educational program). ***International Association for Pediatric Dentistry***, London, England,1996

2. MOSS SJ et. alli. Insights into Saliva Action (educational program), ***International Association for Pediatric Dentistry,*** London, England , 1996.

3. KASHKET S et. alli. Accumulation of Fermentable Sugars and Metabolic Acids in Food particles that Become Entrapped in the Dentition, ***Journal of Dental Research***, 75 (11): 1885-1891,1996.

4. KASHKET S et. alli. Accumulation of Fermentable Sugars and Metabolic Acids in Food particles that Become Entrapped in the Dentition, ***Journal of Dental Research,*** 75 (11): 1885-1891,1996.

5. **Mandel** I.D., Nature Versus Nurture in Dental Caries, ***Journal of the American Dental Association,*** October, 1345-1351,1994.

6. Linke HAB et. alli. Intra-oral Kactic Acid Production During Clearance of Different Foods Containing Various Carbohydrates, ***Journal for Oral Science***, 36:191-197,1997.

7. Szoke J et. alli. The Effect of Sorbitol-Based Chewing Gum Use After Eating on Caries: A Longitudinal Clinical Trial, ***Caries Research*** , 31:281-328 (abstract 111),1997.

8. Moss S.J. The Relationship Between Diet, Saliva and Baby Bottle Tooth Decay, ***International Dental Journal,*** 46 (Supplement 1), 399-402, 1996.

9. Ferguson D.B. Physiological, Pathological and Pharmacologic Variations in Salivary Composition Frontiers of Oral Physiology: ***The Environment of the Teeth;*** 3, 138-153,1981.

10. MOSS S.J. et. alli. Insights into the Mouth (educational program), ***International Association for Pediatric Dentistry,*** 1997.

11. Mandel I.D. Nature Versus Nurture in Dental Caries, ***Journal of the American Dental Association,*** October, 1345-1351,1994.

12. Mandel I.D.Nature Versus Nurture in Dental Caries, ***Journal of the American Dental Association***, October, 1345-1351, 1994.

13. Navia J.M. Nutrition and Dental Caries: Tem Findings to be Remembered ***International Dental Journal,*** 46 (supplement 1): 381-387, 1996.

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