Caries in the Primary Dentition: A Spectrum Disease of Multifactorial Etiology

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Dental research – are we going in circles?

1950 – 2010

Risk Assessment

Relationship to sugar

Antimicrobial approach

Fluoride

Vaccine

Sealants?

Oral hygiene

Mutans streptococci
Working Model for Etiology of Rampant Caries from Phoenix meeting
But, the missing link seems to be...

Enamel hypoplasia is what defines S-ECC
Discussion Points

• Natural history of ECC and S-ECC (rampant caries) are vastly different and should not be considered as a permutations of the same disease

• The major antecedent to S-ECC is enamel hypoplasia from perinatal insult

• Effects of Western diet on Native Americans

• S. mutans may be prominent factor in some forms of rampant caries
What is dental caries?
Dental caries

• An infectious, transmissible disease
• Caries is not a single disease but several depending upon:
  – Location – pit and fissure vs smooth vs root surface
  – Time to onset – chronic vs acute
  – Composition of bacterial biofilm
  – Primary or secondary
ECC and S-ECC seem to be clearly defined and delineated.

Caries is a biofilm (plaque)-induced acid demineralization of enamel or dentin, mediated by saliva. The disease of early childhood caries (ECC) is the presence of 1 or more decayed (noncavitated or cavitated lesions), missing (due to caries), or filled tooth surfaces in any primary tooth in a child 71 months of age or younger. In children younger than 3 years of age, any sign of smooth-surface caries is indicative of severe early childhood caries (S-ECC). From ages 3 through 5, 1 or more cavitated, missing (due to caries), or filled smooth surfaces in primary maxillary anterior teeth or a decayed, missing, or filled score of ≥4 (age 3), ≥5 (age 4), or ≥6 (age 5) surfaces constitutes S-ECC.
Are ECC and S-ECC different diseases?

Yes!
ECC can be a particularly virulent form of caries, beginning soon after dental eruption, developing on smooth surfaces, progressing rapidly, and having a lasting detrimental impact on the dentition. Children experiencing caries as infants or toddlers have a much greater probability of subsequent caries in both the primary and permanent dentitions.\(^5\)\(^-\)\(^8\) Not only does ECC affect teeth, but consequences of this disease may lead to more widespread health issues.\(^9\)\(^,\)\(^10\) Infants with ECC grow at a slower pace than caries-free infants. Some young children with ECC may be severely underweight because of associated pain and the disinclination to eat. ECC also may be associated with iron deficiency.\(^10\)
Adding to the confusion

Early Childhood Caries
California Dental Association
MOVING FORWARD. TOGETHER.

Early childhood caries (ECC), also known as baby bottle tooth decay (BBD), is a preventable, infectious disease caused by certain types of bacteria (bugs) that live in your mouth. Bacteria stick to the film on your teeth called plaque. The bacteria feed on what you eat, especially sugars (including fruit sugars) and cooked
Misconceptions about Childhood Caries

✓ ECC ≠ S-ECC. These are distinct, different dx
  ✓ i.e., S-ECC is not just a more severe form of ECC
✓ Traditional therapeutics and approaches can prevent/manage ECC will not work with S-ECC
Rampant caries (S-ECC)
Etiological Triad of Dental Caries
Etiological Triad of Rampant Caries

High carbohydrate:low protein diet

Early colonization Of cariogenic bacteria

Enamel hypoplasia
Psoter, Zhang, Pendrys, Morse, Mayne. Classification of dental caries patterns in the primary dentition: a multidimensional scaling analysis 2003

5171 Arizona preschool children ages 5-59 months
Hypoplasia – Caries Connection
The concentration of MS in saliva was assayed for each child. Nutritional status was deduced from body height and weight. Birth weight, prematurity, and nursing history were also determined.
Malnutrition as leading cause of enamel hypoplasia in the primary dentition

- >1300 Chinese children, 3 - 5 yo
- Case-control study ~ 200 children per group
- Population of Miyun chronically malnourished
- High percentage of EHP
- Children with EHP had significantly higher colonization levels of MS than non-EHP
- The more severe EHP, the higher the counts of MS

Li, et al., 1994
Key to the Li study

children did not have access to sugar so less aggressive caries to mask hypoplasia
Linear enamel hypoplasia on primary incisors **without** caries overlay

Miyun, China, 1994
Tooth Emergence
Neonatal line

• FIGURE 12-9
• Microscopic view of the neonatal line (arrow), a pronounced line of Retzius, that corresponds to the birth of the individual. Thus it demarcates the enamel formed prenatally (P) and after birth (B). (Courtesy of Dr. James McIntosh, PhD, Department of Biomedical Sciences, Baylor College of Dentistry, Dallas, TX.)
• (Bath-Balogh, Mary. Illustrated Dental Embryology, Histology, and Anatomy, 2nd Edition. W.B. Saunders)

The neonatal line is an accentuated incremental line of Retzius (Figure 12-9). The neonatal line marks the stress or trauma experienced by the ameloblasts during birth, again illustrating the sensitivity of the ameloblasts as they form enamel matrix. Microscopically, the darkened neonatal line marks the border between the enamel matrix formed before and after birth. As one would expect, the neonatal line is found in all primary teeth and in the larger cusps of the permanent first molars.
Surfaces of Primary Teeth with S-ECC
The *Strep mutans* story
Koch’s Approach to Infectious Disease

“one bug, one disease, one bullet”
Mutans streptococci

Dental Caries

- Bullets
  - Mechanical
  - Immunological
  - Chemical
Natural History of MS

- Window of infectivity
- Fidelity of transmission
- Clonality and virulence factors
- Multiplicity of infection
Window of Infectivity

Birth

First Tooth
6.8 ± 1.4 mo.

mutans streptococci

26

19 31

N = 38

5 y
Initial Colonization of MS

- Correlated to the emergence of primary teeth
- Correlated to total surface area of teeth, specifically fissures in molar teeth
- Newly emerged, “virgin” surfaces
- Time to colonization function of many factors, including perinatal history, race, antibiotics
- Supported by six longitudinal studies
Fidelity of Transmission

Mother --> Infant
DNA Chromosomal Fingerprinting
Infants Acquire MS from Mothers

<table>
<thead>
<tr>
<th>mother's strains</th>
<th>DNA Fingerprints of Infant</th>
</tr>
</thead>
<tbody>
<tr>
<td>mother's strains</td>
<td>Match 24 (71%)</td>
</tr>
</tbody>
</table>

Li and Caufield, JDR, 1995
Fidelity of Transfer - mutans streptococci

Mother’s genotypes

Female: 88%  
Male: 53%

N = 34  
MITS  
p ≤ 0.02
Fidelity of Transfer - mutans streptococcci

Mother’s genotypes

Black: 88%  
White: 56%  
N = 34  
MITS  
p ≤ 0.04
Summary

- Perinatal events sets the stage for infectivity and possibly future caries outcomes in children
- Mothers are the major source of MS to their infants
- Colonization is stable over a lifetime
- Only a few genotypes per individual
- Fathers are not the source of MS to infants
- Breastfeeding, c-section, antibiotics alter infectivity
How does this apply to rampant caries seen among Native American Indian populations?

• Unusually high prevalence of rampant caries
• Mothers and infants are likely to have risk factors such as poor nutrition predisposing infants to enamel hypoplasia
• The prevalence and mode of transmission of cariogenic bacteria may be different from other populations
• Treatment/prevention needs to address these antecedent conditions to treat or attenuate this disease
• 25 percent of this racial group lives at the poverty level.
• American Indian/Alaska Native infants are 3.7 times as likely as non-Hispanic white infants to have mothers who began prenatal care in the 3rd trimester or did not receive prenatal care at all.
• American Indian/Alaska Native adults were 2.3 times as likely as white adults to be diagnosed with diabetes.
• American Indian/Alaska Native adults were 1.6 times as likely as White adults to be obese.
NUTRITION, GROWTH AND DEVELOPMENT OF NORTH AMERICAN INDIAN CHILDREN

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Public Health Service
National Institutes of Health
What can we do now?

• Put down the needles and drills, step back, look at situation
• Put out the fire – early dx and treatment
  – AgDMF
  – GIC sealants over hypoplasia
  – Varnishes/CHX?
• Retrospective case-control of perinatal history relationship to rampant caries