

2010 Symposium on Early Childhood Caries in American Indian and Alaska Native Children



Summary

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Introduction

Caries in the primary dentition—often referred to as early childhood caries or ECC¹—is a chronic disease that has a relatively low prevalence and minimal morbidity among many populations of U.S. children. In contrast, for many American Indian and Alaskan Native (AI/AN) communities, ECC prevalence for young children is extremely high—approximately 300 percent higher than the U.S. all races prevalence. Of far more importance than simple prevalence, caries in the primary dentition among AI/AN children results in a very high burden of disease, with over 25 percent of children in some communities requiring full mouth restoration under general anesthesia prior to entering kindergarten. This represents a rate that is about 50 to 100 times that of U.S. all races children. In addition to the considerable short term morbidity from severe caries early in childhood, it also often predisposes a child to a lifetime of dental disease.

Decades of programmatic efforts by Indian Health Service (IHS)—the federal agency whose mission is to provide preventive and curative health services to AI/AN people—and tribal health programs have resulted in little or no long-term improvement in ECC prevalence or severity in most AI/AN communities. These interventions include community water system fluoridation, attempts to improve children's diets and oral hygiene and, more recently, application of fluoride varnish and use of xylitol-containing products. Strategies found effective in preventing ECC in other populations have shown no demonstrable long-term or sustainable benefit in most AI/AN communities. In the words of a senior IHS dental officer, "At best, we have had minor, transient victories from our efforts."

There continues to be a pressing need to examine ECC in light of the current scientific understanding of the disease and identify new research and programmatic strategies that are based on the best available science. The overall purpose of this Symposium, co-hosted by the American Dental Association (ADA) and the American Academy of Pediatrics (AAP) with support from Oral Health America, was to bring together a group of some of the most experienced caries researchers in the U.S., representing many of the most prestigious caries research centers, to review the state of the science of prevention of caries in the primary dentition, identify gaps in our current understanding of this disease and formulate strategies to close the existing gaps in knowledge.

The objectives of the Symposium were to:

- establish a common knowledge base among Symposium participants of caries in the primary dentition of children in tribal communities, including past programmatic

and research efforts to control ECC, the current ECC epidemiology in AI/AN and other U.S. children, and preliminary data from ECC research in progress;

- review the current state of the science about the initiation and progression of severe ECC, with special emphasis on the bacterial etiology of the disease; review the current state of the science on prevention of ECC, with understanding of the limitations in generalizing the results of ECC prevention research done in non-Indian communities to AI/AN communities;
- identify the most important and relevant topical areas which will inform progress to control ECC among AI/AN children;
- identify the knowledge gaps which, if they remain unanswered, will significantly impede progress toward the control and prevention of severe ECC among AI/AN children;
- develop a prioritized list of specific questions, issues, uncertainties that need to be addressed, including recommendations on who should be involved in addressing each issue;
- develop research questions to fill in gaps in the understanding of why ECC is so severe among AI/AN children; and
- make recommendations to the American Dental Association (ADA) and other relevant organizations for the next steps to be taken to improve ECC prevention and research.

Symposium Format

Day 1 included scientific presentations by distinguished caries researchers who were selected by the Symposium planning group. The presentations began with a broad perspective in the keynote address, "Caries in the primary dentition: A spectrum disease of multifactorial etiology." Subsequent presentations were grouped into the topical areas considered most important by the Symposium planning group, including microbiology, epidemiology, enamel hypoplasia, caries research in AI/AN children and new intervention products. The morning of Day 2, participants divided into small workgroups to discuss each of the major topical areas. The afternoon of Day 2, the workgroups reported to the larger group on conclusions and recommendations for each topic. Summary workgroup reports are found at the end of these proceedings.

¹ The generic terms, "ECC" and "S-ECC" (for "severe ECC"), are widely used in the literature and have official definitions from the ADA, American Association of Pediatric Dentistry and other national organizations. However, as was extensively discussed in the Symposium, there is great variation in the way these terms are used. There is also concern that the current terminology and definitions may be inadequate for describing the severe end of the spectrum of caries in the primary dentition. Nonetheless, for convenience in this report, "ECC" is often used generically to represent "caries in the primary dentition."

Presentation Summaries

Don Marianos, DDS, MPH

An historical perspective of early childhood caries in American Indians and Alaska Natives: There and back again

I was the Dental Director at an Indian Health Service dental program in a Southwest American Indian tribal community for seven years starting in the late 1970s. The prevalence of caries was approximately 90 percent in Head Start age children when I first arrived. We utilized every available recommended ECC prevention modality, including intensive oral health education, community water system fluoridation, fluoride supplements, etc. At the end of six years of intensive effort, the prevalence and severity of ECC were essentially unchanged.

I left the IHS in the late 1980s and worked with CDC for the next 10 years, including being the Director of the Division of Oral Health. During this time I was aware that the overall oral health of American children was continually improving, with only a very tiny proportion of children in most population groups in the U.S. having severe forms of ECC.

After retiring from CDC, in 2006 I began working as a consultant with the same Southwest American Indian community for which I had been the Dental Director in the 1980s. I was both surprised and very discouraged to see that the prevalence and severity of caries in the primary dentition were about the same as it had been when I first arrived there over 25 years ago. This was in spite of all the caries prevention efforts that had been continued after I had moved to CDC. I am convinced there is still a lot we do not understand why these children are at such extraordinarily high risk for the early and very destructive ECC they so often develop. It is incumbent on us to work collectively to better understand and control this disease.

Page W. Caufield, DDS, PhD

Caries in the primary dentition: A spectrum disease of multifactorial etiology

- Dental caries is an infectious, transmissible disease. Its initiation depends upon the presence of a bacterial biofilm comprised of indigenous oral bacteria rather than from an exogenous pathogen. For many decades *Streptococcus mutans* and *Streptococcus sobrinus*—collectively referred to as ‘mutans streptococci’ (MS)—have been considered the primary cariogenic bacteria and represent the paradigm for indigenous biota. Yet as methods for surveying the vast assortment of bacterial species present on the human body expands, the leading role for MS diminishes somewhat. Nonetheless, a substantial body of literature supports the correlation between early colonization by and increased levels of MS

associated with early childhood caries. Even at increased levels, MS usually represent less than 1% of the total flora, yet MS levels have been reported as high as 40% of total flora in some cases of severe ECC.

- Caries is not a single disease but several, depending upon: (1) location site of the lesion; (2) time to onset (chronic vs. acute); and (3) composition of bacterial biofilm.
- Definitions of, and differentiation between, early childhood caries (ECC) and severe early childhood caries (severe ECC) continue to be updated by the American Academy of Pediatric Dentistry (AAPD). But these definitions are not consistently used in the caries research literature, nor have they been uniformly adopted even in publications by the AAPD, and are not clearly correlated with actual morbidity from caries in the primary dentition. The AAPD definition of ECC—i.e., at least one lesion in a child under 6 years of age—should not be confused with the presentation of severe ECC or rampant caries. The two diseases are clearly different with different antecedents, severity, acuteness, bacterial composition and sites of attack. In my opinion, the comparatively mild form of “garden variety” ECC seen in most populations of U.S. children and the severe, rampant, destructive ECC seen mainly in those children living in poverty, represent two different and distinct diseases rather than a continuum of the same disease.
- The working model for the causation of rampant or severe ECC includes three critical antecedent events: (1) Prenatal insult to the fetus during the 3rd trimester of pregnancy or early neonatal period resulting in enamel hypoplastic lesions affecting mainly the anterior incisors, canines and first molars; (2) Early colonization by mutans streptococci, perhaps other as yet unknown microbes within hypoplastic sites; (3) a cariogenic diet in the infant further promotes a rapid increase in the level of acidogenic bacteria such as MS, leading to destruction of the primary dentition.
- The Hypoplasia-Caries Connection: The necessary antecedent to severe ECC is enamel hypoplasia from perinatal insult. Microscopic defects on the surface of primary teeth provide the ecological niche by which mutans streptococci and other early oral bacterial colonizers become established. The rapid onset and destructive nature of severe ECC often masks the underlying hypoplasia. Moreover, even though enamel hypoplasia can be seen in primary incisors shortly after emergence from 6–24 months of age, it is often missed by health care workers.
- Perinatal history from birth trauma is recorded in the primary dentition resulting in what is known as the neonatal line. This is a normal permutation and evidently does not manifest as ECC. However, premature birth and other forms of birth trauma are linked to enamel hypoplasia, with or without severe ECC (Needleman, Seow and others).

- Severe ECC—dietary, socio-economic connection. Severe ECC affects almost exclusively children at or below national poverty standards. The link between poverty and malnutrition, while seemingly obvious to most, is often overlooked as a major contributor to enamel hypoplasia even though dozens of studies have clearly established this link. Other perinatal insults resulting in prematurity or underweight birth weight are also linked to enamel hypoplasia. The incidence of these conditions is particularly high among the impoverished children of this country. Poor infant diets, high in carbohydrates including sucrose and high fructose corn syrup and low in protein, further exacerbates the rapidity and extent of destruction.
- The *Streptococcus mutans* Story: Concepts include:
 1. Window of infectivity. (Influenced by # of sites and surfaces available for initial colonization of cariogenic bacteria with *S. mutans* as the prototype). Hypoplastic defects on smooth surfaces foster early colonization of *S. mutans*, altering the window period to an earlier colonization period.
 2. Fidelity of transmission. (generally thought to be transmitted from mother to child but, in the case of severe ECC, horizontal spread of infection has been suggested as a means of spread within a closed or endemic population).
 3. Clonality and virulence. Studies are underway (Drake and Caufield) examining whether populations of *S. mutans* found in severe ECC are different from those found in caries-free children. One concept we published shows the total microbial biota of severe ECC children is less diverse than the biota of caries-free children. This implies, among other things, that as caries severity increases, diversity (i.e., the number of species in the biofilm) decreases. One implication of this concept is that mothers with heightened caries experience may transmit a less diverse oral biota to their children. So far, only select bacteria such as *S. mutans* and *S. sanguinis* have been monitored, so the universality of this concept remains unproven.
 4. Multiplicity of infection. Published research suggests infants and very young children usually harbor only a single strain of *S. mutans*. Some papers, however, suggest multiple strains of *S. mutans* present in severe ECC children in contrast to non-severe ECC children. This raises the question whether severe ECC might be a mono-infection by a particularly virulent, hence dominant, microbe or set of microbes.

Noel K. Childers, DDS, MS, PhD

Maternal transmission of mutans streptococci in severe early childhood caries

- The study population was children in Alabama who were to undergo full mouth restoration under general anesthesia for rampant caries in the primary dentition.
- Plaque samples collected from mothers, children and cariogenic lesions.
- Only 26% of the children had *S. mutans* isolates that all matched their mother; 15% had a partial match; and 59% of the isolates did not match any of the mother's.
- Overall, 74% of the children had at least some isolates that did not match their mothers. All four sibling-pairs matched with each other, but only one matched with their mother.
- Maternal transmission was not the main source of the *S. mutans* in the children with very severe early childhood caries in this small sample of children.
- Conclusion: practices to minimize maternal transmission may have limited effectiveness in control of S-ECC in at least some populations of children.

David R. Drake, MS, PhD

Etiologic factors for early childhood caries

- *S. mutans* can adhere to the tooth enamel in the absence of sucrose (sugar-independent adhesion), but it is not very efficient.
- Sucrose promotes a more efficient bacterial adhesion to the tooth surface.
- Greater adhesion leads to a greater concentration of cariogenic bacteria.
- Greater adhesion leads to greater acid production
- This promotes the growth of bacteria that will tolerate a higher level of acidity (lower pH)
- This results in caries.
- Usually *S. mutans* does not colonize infants and very young children
- When this happens, *S. mutans* becomes dominant leading to higher rates of caries.
- As *S. mutans* becomes the dominant member of the oral flora, it becomes much more cariogenic.

Wenyaun Shi, PhD

Ecological considerations of *S. mutans*

- There is a lot about caries and Severe Early Childhood Caries that is not clear.
- What is getting clear is that acid-producing cariogenic bacteria are the final common pathway required for initiation of caries.
- Dental plaques containing cariogenic bacteria are very resistant to antimicrobials. Plaque grows back in 8 hours

after it has been mechanically removed. Therefore, the mechanical approach is unlikely to provide lasting benefit.

- There are new findings about *S. mutans* in plaque and acid profiles in plaque. *S. mutans* provides 60% of acid in plaque and is suspected to be the leader
- The presence of *S. mutans* is correlated with the presence of the disease of caries.
- The absence of *S. mutans* is correlated with an absence of caries.
- In an insured population of children in Washington state, >95% of the children had <10% *S. mutans* as a percentage of their total oral flora.
- The solution lies in modulating the flora such that it maintains a healthy balance.
- A targeted approach is more likely to be an effective intervention than broad spectrum killing of *S. mutans*.

Yihong Li, DDS, MPH, DrPH; Timothy Bromage, PhD and Page W. Caufield, DDS, PhD

Enamel hypoplasia as a prerequisite to rampant caries

- Various forms of Developmental Enamel Defects (DED) are seen
 1. Linear
 2. Pitted
 3. Opaque (most commonly called enamel hypoplasia, or EHP)
- Decreased and flawed mineralization resulting in surface irregularities
- Surface irregularities provide easy sites for colonization and retention of cariogenic bacteria
- Hypoplastic areas are less resistant to acid attack
- The position and extent of defect correspond to the time of insult prenatally or neonatally
- Known risk factors for EHP (from Seow et al and Needleman, et al)
 1. Malnutrition
 2. Low birth weight
 3. Prematurity
 4. Maternal illness
 5. Smoking
 6. Drug abuse
- Linear EHP on the incisors is followed by caries that develops over the same area (pattern) as the EHP
- We are unlikely to control the rampant ECC seen so often in AI/AN children until we can prevent enamel hypoplasia or find a product to protect the hypoplastic areas of the primary dentition.
- We do not know the prevalence of enamel hypoplasia among AI/AN children, but both retrospective and longitudinal studies would seem to be relatively easy to design and conduct.

Steve Holve, MD

Early childhood caries in American Indians/ Alaska Natives: A pediatric infectious disease with dental manifestations

As the Director of the pediatric program at a large Southwest American Indian community, I believe there is compelling evidence that early childhood caries in AI/AN children should be considered a pediatric infectious disease with dental manifestations rather than a strictly dental disease.

- A dental survey of Navajo Nation in 1933 found almost no caries; the diet was largely meat based at that time. Children living in remote camps had much lower rates of caries than children in boarding school.
- The change from a traditional diet to a “modern” diet was a necessary but not sufficient factor. There is no evidence that the current diet in AI/AN is worse than the general US diet, merely the same.
- The cause of the disparity is likely to be that the children acquire *Streptococcus mutans* at an earlier age and at a higher load. We will not reduce the ECC disparity in AI/AN until we treat the infectious component.
- The regions with the highest rates of poverty and crowding are the regions with the highest rates of ECC.
- Like severe ECC, other common pediatric infections (such as *H. pylori* and *H. influenzae*) occur at rates 2-10 times higher than the general US population, and like severe ECC they occur at younger ages:

| Prevalence of <i>H. pylori</i> | | |
|--------------------------------|--------------------------|--------|
| | Children <2 years of age | Adults |
| U.S. | 3% | 30% |
| Apaches | 54% | 77% |
| Canadian Inuits | 67% | NA |

| <i>Haemophilus influenzae</i> type b: Pre-vaccine era incidence per 100,000 | | |
|--|----------------------------|----------------|
| | Southwest American Indians | U.S. all races |
| Cases <5 years of age | 500 | 60 |
| Nasopharyngeal colonization | 7 % | 2 % |
| Peak age of illness onset | 2–6 months | 6–12 months |

Summary: other common pediatric infectious diseases have been proven to have very different attributes among AI/AN populations. Similarly, severe ECC appears likely to have different attributes among AI/AN children, which may explain why the usual recommended interventions have not been effective.

Ananda P. Dasanayake, BDS, MPH, PhD, FACE

Epidemiology of early childhood caries and effectiveness of interventions

- What's in a name? Caries in the primary dentition has been historically and still is referred to by many different names. There is huge variability in the way it is defined by different groups and in different studies. Are we capturing the same disease under these many different names and definitions?
- Do we need a different metric to define and measure the most severe end of the spectrum of caries in the primary dentition? A new metric could possibly include a composite of parameters such as number of lesions, age of onset and rate of progression. Any new metric needs to have a correlation with actual morbidity instead of simply counting lesions.
- Beyond the problem of definition, the primary challenge to measuring the disease is the relatively poor sensitivity and specificity of the methods for detecting lesions. Until we have better metrics, we are limited in our ability to determine prevalence, severity and the effectiveness of interventions.
- Although collected from clinical encounters, the best available nationwide data on the rate of caries in the primary dentition among AI/AN children indicate;
 1. There is a marked disparity in both prevalence and severity of ECC.
 2. The most recent survey data from 1999 showed no improvement from 1991.
 3. In the 1999 survey approximately 45% of AI/AN children had a history of caries on at least two maxillary incisors.
- The HP 2010 health objectives for the nation have a goal that <12% of 2–4 year old children have caries experience; the best available data indicate the current prevalence of caries experience among AI/AN children is approximately 70%.
- Efficacy of various interventions: To date, there is no convincing evidence that any of the currently available ECC-prevention products or strategies substantially reduce the prevalence or severity of the most severe end of the spectrum of caries in the primary dentition in the highest risk populations. Some of the recently published studies on the efficacy of various agents in prevention of ECC are likely to have limited applicability among AI/AN populations.
- Dr. Holve has provided provocative data showing that several common pediatric infectious diseases have markedly different epidemiological attributes among AI/AN children compared to the US all races. We need to understand better whether this paradigm applies to the severe end of the spectrum of caries in the primary dentition among AI/AN children.

Conclusions:

1. We need to re-visit our current definitions. Using a 'new definition,' we need to get a valid estimate of the disease burden.
2. We need additional research to better understand the real causal factors.
3. One-Size-Fits-All prevention approaches are unlikely to work and there are no Silver Bullets.
4. The best solution will be culturally appropriate, innovative prevention strategies based on the population-specific patho-physiology and the common risk factor approach.

James Irvine, MD; Robert Schroth, DMD, PhD and Rosamund Harrison, DMD

Early childhood caries and Aboriginal children in Canada

- Indigenous or Aboriginal children in Canada include First Nations, Métis and Inuit. Approximately 700,000 persons in Canada are Indigenous, representing about 4 percent of the Canadian population. In the provinces of Manitoba and Saskatchewan, First Nations constitute about 15% of the population.
- First Nations children in Canada have similarities of socio-economic and health status circumstance to AI/AN children.
- Prevalence of ECC for various Canadian Aboriginal communities is as follows: Northwest Territories – 66% (Inuvik Region); Manitoba – 98% (Garden Hill First Nation), 59% (Northern First Nation), 23% (Winnipeg); Quebec – 81% (Cree Territory); Ontario – 67–78% (District of Manitoulin), 79–92% (Sioux Lookout Zone); and British Columbia – 31% (Hartley Bay).
- Children living in the most isolated communities are reported by parents to have approximately twice the prevalence of ECC compared to non-isolated communities.
- In June 2007 a forum, Oral Health and Aboriginal Child, sponsored by a range of funders was held in Winnipeg, Manitoba. A goal of the forum was to build Aboriginal oral health research capacity; a knowledge transfer website (<http://oralhealth.circumpolarhealth.org/>) was subsequently developed. This Web site is a repository for oral health research and health promotion information for Indigenous populations in Canada, the US, Australia and New Zealand.
- The Children's Oral Health Initiative (COHI) sponsored by Health Canada has been underway for over 6 years in many native communities. COHI focuses on children, 0 – 7 years of age, and their mothers and includes oral health counseling, fluoride varnish, interim restorative techniques, sealants and xylitol therapies.
- In some provinces and territories, dental therapists provide clinical treatment and preventive services to First Nations, Inuit and other Northern children.

Therapists refer children to other dental professionals when more advance treatment is needed.

- Several ECC-prevention projects have been conducted by Canadian researchers in partnership with First Nations emphasizing capacity-building to assure sustainability.
 - A project in 4 Manitoba communities relied on community development principles to promote preschool oral health and prevent ECC through community action. Significant reductions were found after 6-years in both the age adjusted mean (SD) deft rate: 4.2 (5.0) at baseline vs. 3.9 (5.0) at follow-up, $p < .001$ and the age adjusted prevalence for S-ECC: 45.0% (baseline) vs. 38.6% (follow-up), $p = .026$.
 - A randomized controlled trial (Lawrence HP et al, CDOE 2008) in the Sioux Lookout region of Ontario compared the effectiveness of fluoride varnish (FV) plus counseling compared to counseling alone. FV + counseling resulted in a 26% reduction in the 2-year caries increment measured at the d1 and d3 level. A secondary outcome was that “proportion of children who had general anesthesia dental care was 25% lower in test group.”
 - A 5-year randomized controlled trial nearing completion in northern Quebec analyzes the effectiveness of a Motivational Interviewing counseling approach with Cree mothers in controlling caries in their young children. Preliminary analysis has compared test and control communities; risk ratios are significantly different for most “tooth level” indices.

Sara DeCoteau

Tribal community perspective on ECC-prevention research

- The Sisseton-Wahpeton Oyate (SWO) of the Lake Traverse Reservation of NE South Dakota currently has a Cavity-Free in 2-0-1-3 initiative. This was implemented under the guidance of the SWO Tribal Council via resolution in 2007 expressing the goal that *all SWO children enter kindergarten cavity-free in the year 2-0-1-3*.
- Although we have a wonderful new medical and dental facility, we agree with Dr. John Zimmer (our pediatric dentist) that we will never drill and fill our way out of our problem with childhood caries.
- From the SWO Tribal Perspective ECC Prevention Research must meet three criteria:
 1. The services and activities must be sustainable after the study has concluded.
 2. The services and activities must not depend on having a high level of staffing of dentists or hygienists.
 3. The intervention strategies must be replicable in other American Indian communities as well as non-Indian communities in which the children experience high levels of early childhood caries.

- The conceptual basis for Cavity-Free in 2-0-1-3 is that ‘it takes all three to be caries-free’:
 1. Good diet and oral hygiene
 2. Remineralization therapy to strengthen the tooth enamel
 3. Control of cariogenic bacteria to promote a healthy balance of oral flora.
- Ms. DeCoteau concluded her presentation by showing a video documentary recently completed on different aspects of the community involvement in Cavity-Free in 2-0-1-3, including the full support of the community Head Start and Early Head Start staff.

Steve Holve, MD

Applying fluoride varnish in primary care settings

- All children 9–30 months of age seen at pediatric well child checks were offered fluoride varnish
- Pediatric clinic staff provided oral health education and an oral screening, followed by application of fluoride varnish at the conclusion of the visit
- Children were examined approximately 2–3 years later in Head Start
- Results: children who received ≥ 4 fluoride varnish applications had 35% lower caries scores than historical controls
- This project was done as a public health practice rather than as controlled research, so potential confounding variables were not examined
- This project showed the feasibility of providing fluoride varnish to a large proportion of the under two years of age population in an Indian health care pediatric clinic setting

Dee Robertson, MD, MPH

Using chlorhexidine dental varnish to prevent early childhood caries in Indian children

This study was conducted in four Indian health dental clinics—two in the Pacific Northwest and two in Arizona. The conceptual basis for this study was:

- Acid producing oral bacteria (mutans streptococci) are the final common pathway for caries
- Chlorhexidine has an extensive record as an effective and safe antimicrobial
- Most studies have shown most children acquire their mutans streptococci by vertical transmission from the mother
- Children who acquire mutans streptococci later have lower rates of ECC

The study protocol included:

- 600 mother-child pairs to be recruited in and randomized to receive either active or placebo treatments
- Mother-child pairs enrolled when child is 4–6 months old
- Weekly applications x 4 of CHX varnish to the mother only, followed by repeat single applications 6 months and 12 months later
- End of study exam for caries when child is 24 months old.

The primary outcome variable was caries increment over 19 months in the child. The study was conducted under FDA Investigational New Drug permit with approval from each IRB of record and the tribal health authority and tribal council of each participating community.

Results:

- There was no difference in mean dmfs or mean dmft in active vs placebo children
- There was no difference in the proportion of active vs placebo children who were caries-free at the end of study exam
- When the children who developed any caries during the study were grouped into tertiles by the number of new carious surface, compared to the Placebo children there was a 26% reduction in the proportion of Active children who were in the most severe category

Discussion: By shifting the distribution of caries from more severe to less severe, there *may have been* a clinical benefit to children in the Active arm of the study. Failure to reduce the mean *dmfs* and *dmft* may be partly explained by a recently published report (Mitchell et al.) showing that a high proportion of children with the most severe end of the spectrum of caries in the primary dentition did not acquire their *S. mutans* from their mothers. This obviously would limit the effectiveness of any intervention that treats only the mother, as was done in this study.

David R. Drake, PhD

Streptococcus mutans and dental caries in Native American children

- The overall goal of this study is to identify risk factors for ECC in American Indian children.
- Our hypothesis that there are particularly virulent genotypes of *Streptococcus mutans* (SM) that are readily transmitted from mother to child
- Objectives:
 - To determine the temporality and fidelity of transmission of SM from mother to child
 - To characterize expression of virulence determinants of different genotypes of SM
 - To determine the composition of total acid flora in mothers and children

- To determine the incidence and progression of ECC in American Indian children
- To identify behavioral, environmental, growth, and dietary risk factors in American Indian children
- Study protocol: Baseline when the baby is approximately one month old, followed by follow-up visits approximately every 4 months to age 36 months.
- Preliminary results:
 - Approximately 90% of mothers have had *S. mutans* detected and 70–80% have had lactobacillus
 - Through 8 months of age, less than 10% of children have had *S. mutans*, while 40–60% have had lactobacillus; at 12 & 16 months of age, 40–60% of children have *S. mutans*.

Dee Robertson, MD, MPH

Cavity-free in 2-0-1-3

Cavity-Free in 2-0-1-3 is a multimodal caries prevention program developed as a follow-up to a similar pilot project conducted in 2008. At the request of the Steering Committee of community members, Cavity-Free in 2-0-1-3 is both a study (for children age 0–35 months) and a project (children 36–60 months). It addresses ECC-prevention from three different perspectives at the same time:

- Promoting good diet and oral hygiene
- Enhancing the resistance of the tooth enamel to caries
- Promoting a healthy balance of oral bacteria
- Study protocol:
 - Enrollment when the children are 6–24 months
 - Treatment provided quarterly to mothers and children (licorice extract for the mother and child, provided by C3 Jian, Inc.); 5% sodium fluoride varnish for the child
 - Levels of *S. mutans* measured for study children and their parents seven times during the 1-year study using a monoclonal antibody salivary test by C3 Jian
- End of study caries scores will be compared to historical controls

Results to date:

- Children 6–11 months of age already have substantial levels of *S. mutans*
- Children 12–24 months of age have mean *S. mutans* levels as high as their mothers and higher than Head Start children age 37–60 months in the same community.
- For 77% of participating children, *S. mutans* constitutes more than 10% of their total oral flora. By comparison, the same monoclonal antibody test was used for a large sample of insured Washington state children, and *S. mutans* constituted less than 10% of the total oral flora for over 95% of these children.

Conclusion: Similar to the report earlier in the Symposium by Dr. Holve concerning AI/AN children having much earlier acquisition of pathogenic bacteria, SWO children seem to acquire a surprisingly high level of *S. mutans* very early in life. For many of these children *S. mutans* rapidly becomes the dominant member of their oral flora.

Joel Berg, DDS, MS

Glass ionomers to prevent early childhood caries

- Over recent years there has been a shift in the focus of dentistry from (1) treatment of the results of dental disease, to (2) prevention of dental disease, to (3) reducing the risk factors for dental disease.
- Likewise there has been a continuum in development of dental adhesives and sealing materials, from composite → compomer → RMGI → glass ionomer.
- Glass ionomers have a number of attributes that make them superior to other products:
 1. Glass ionomer form a physico-chemical bond with the tooth enamel.
 2. Unlike other sealing products, glass ionomers do not require a separate adhesive to be applied.
 3. The thermal coefficient of expansion of glass ionomer is about the same as dentin, which is obviously advantageous.
 4. Glass ionomer releases fluoride ions to the adjacent tooth structure which provides additional protection to this caries prone interface.
 5. Glass ionomer can be used as a transitional sealant or for very deep grooves.
 6. Glass ionomer sealants can arrest existing caries lesions and are also effective in preventing new caries lesion development in the surfaces covered.
- There is great potential for further enhancement of the properties of glass ionomers in the future:
 - Improved properties
 - Improved handling and delivery
 - More research—particularly in further elucidation of ion exchange mechanism and chemical adhesion
 - Perhaps as a protective barrier for hypoplastic enamel

David R. Drake, MS, PhD

1% chlorhexidine gel

- Developed in collaboration by Cindy Marek, PharmD, and David R. Drake, MS, PhD, University of Iowa College of Dentistry
 - 1% chlorhexidine gel prepared from ultra-pure stock
 - Para-chloroaniline levels carefully determined and minimized
 - Alcohol-free
 - Anhydrous raspberry flavoring that children like

- Phase I trials conducted in an Investigational New Drug permit from the U.S. FDA
 - 21 Eastern Iowa Head Start classrooms
 - 217 children randomized to 1.0% flavored chlorhexidine gel or placebo
 - children received one 1-minute application monthly
 - *S. mutans* tested using semi-quantitative MSKB RODAC plates
- Results:
 - Feasibility: high participation levels by children without complaints about the taste
 - Reduction of 2–4 orders of magnitude of *S. mutans* in 1.0% flavored chlorhexidine gel group compared to placebo group

Conclusion: In many ways the 1.0% flavored chlorhexidine gel seems to be an ideal intervention product to control cariogenic bacteria among children at high risk for caries in the primary dentition. Chlorhexidine has a long history of safety in both adults and children, with hundreds of CHX-containing products available world-wide. This formulation could easily be used in any group setting for high risk children such as Head Start, and could also be safely used on a scheduled protocol at home.

Noel K. Childers, DDS, MS, PhD

Application of caries vaccine to humans

- Mechanisms involved in *S. mutans* colonization and pathogenesis
 - Sucrose-independent attachment (Ag I/II)
 - Sucrose-dependent reaction (glucosyltransferase)
 - Bacterial metabolic activities with lactic acid production
- Preclinical (Animal) Studies
 - Numerous animal studies have shown a variety of antigens delivered to different mucosal sites have been immunogenic against *S. mutans*.
 - Mucosal immunization is enhanced using liposomes as delivery system.
 - UAB had done seven Phase 1 FDA sanctioned studies: 3 Oral Immunization and 4 Nasal (with one topical tonsillar) with no Adverse Effects
- Potential caries vaccine strategies
 - topically applied vaccines (intranasal, palatine tonsil, salivary glands)
 - use of adjuvants (cholera toxin subunits, monophosphoryl lipid A)
 - recombinant vaccines (cloned antigens, recombinant vector)
- Summary of Clinical Phase 1 Immunization studies with *S. mutans* antigens
 - Oral, nasal, tonsillar (topical) antigen administration were safe.

- Nasal spray vaccine induced antigen specific mucosal IgA responses. Response appeared to be dose specific (three separate studies).
- Re-immunization (18 months after first immunization) resulted in higher responses compared to newly immunized group.
- IgA response appears to be associated with delayed recolonization of oral cavity with *S. mutans*.
- Clinical approach to further development of a caries vaccine
 - Adult Phase 1 studies
 - Pre-adolescent Phase 1 and 2 studies
 - Pre School Children Phase 1 and 2 studies
 - Infant Phase 1,2,3 studies
- Important Considerations and Challenges: “A vaccine for dental caries”
 - FDA approvals: Safety concerns for a disease that is usually not life-threatening
 - Funding: NIH resources, Patent-ability issues for private funding
 - Efficacy studies:
 - ♦ Chronic disease, therefore beneficial outcomes are not immediately observed
 - ♦ Long-lasting immune mucosal response is most desirable

Richard Niederman, DMD

Silver diamine fluoride

- Silver compounds have been used for over 100 years to prevent infections
 - 1890: Silver sutures + silver nitrate: prevent infections
 - 1910: States began to mandate that silver nitrate drops be put in the eyes of newborns to prevent ophthalmia neonatorum (bacterial infection of the eyes)
 - 1920: Silver nitrate: caries prevention (Howe)
 - 1960: Silver diamine fluoride: caries prevention (Yamaga Japan, Aron Mexico, Gotjamanos Australia)
- Current FDA-approved uses of silver compounds
 - Silver topical ointments for burns
 - Slow release silver wound dressing
 - Colloidal silver water purification
 - Silver fabric surgical gowns and drapes
 - Silver sutures
- Compared to other potential caries control products such as glass ionomer sealants, fluoride varnish, chlorhexidine products, etc.:
 1. Silver diamine fluoride is one of the most efficacious agents available anywhere in the world.
 2. Silver diamine fluoride is also the most cost-effective agent known at this time.
- Below is a comparison of caries arrest, prevention, and cost.

| Delivery | Caries | | Cost per Mouth |
|-------------------------|------------|--------|----------------|
| | Prevention | Arrest | |
| Diamine silver fluoride | >70% | >90% | \$ |
| Fluoride varnish | 45% | 25% | \$ |
| F-Toothpaste | 25% | ? | ¢ |
- At the present AgFI is not FDA-approved for marketing and distribution in the U.S., but the FDA is currently reviewing an application for licensing in the U.S.

Wenyaun Shi, PhD

Targeted antimicrobial therapy against dental caries

- Various mechanical and antimicrobial therapies have been and are currently being used to control cariogenic bacteria.
 - Mechanical removal with frequent cleaning; High concentration of fluoride; CHX; Listerine; Triclosan; Povidone-iodine; Bleaching agents; Antibiotics;
- The current antimicrobial therapies are all broad spectrum and kill the normal, beneficial oral flora as well as the bad guys.
- A better approach would be an agent that selectively controls the *S. mutans* while not affecting the other oral flora, such as:
 - Vaccination (active or passive)
 - Replacement with non-acidic *S. mutans*
 - Enhancement of base-producing bacteria
 - Targeted phage therapy
- C3 Jian is developing three selective *S. mutans* control products:
 1. Targeted quorum sensing therapy
 2. Smart bomb (STAMPs)
 3. Natural herbal products
- STAMPs: Antimicrobial peptide – Linker peptide – Targeting peptides
 - The mode of action of the antimicrobial peptide is pore formation in the cell wall of gram positive bacteria.
 - The targeting molecules contain: Species-specific antibody; Species-specific minibody; Species-specific signaling peptides; Phage landing gears; Phage display peptides; Sex pheromones
 - Our *in vitro* data show complete elimination of viable *S. mutans* in a sample with 100,000 *S. mutans* treated with STAMPs, while *S. sanguinis* levels are unaffected.
 - Other finds with STAMPs:
 - ♦ Elimination of *S. mutans* greatly reduces other cariogenic bacteria in dental plaque

- ♦ Elimination of cariogenic bacteria greatly reduces or stops demineralization
- ♦ Targeted killing of cariogenic bacteria re-balance microbial ecology, achieving long term protective effects
- Glycyrrhizol A (derived from the licorice plant *Glycyrrhiza uralensis*)
 - Is approved by the FDA as a food flavoring under the generally recognized as safe designation.
 - This product does not contain the glycyrrhizic acid that is known to cause side effects when licorice is consumed in large quantities over an extended period of time.
 - Shows in vitro selective inhibition of *S. mutans* with minimal effect on most other species of the oral flora.
 - Limited small clinical studies have confirmed a decrease in *S. mutans* levels of adults and children.

Ming Tung, PhD

ACP technology: Amorphous calcium phosphate forming fluoride varnishes

- Why use amorphous calcium phosphate (ACP)?
 - Fastest formation and dissolution
 - Solid solution: Incorporates other beneficial ions readily.
 - Therapeutic agents and also as the carrier for long term releases
 - Transform to apatite: Put back lost tooth mineral
 - Fill and obstruct dentin tubules (one ACP unit is 0.96 nanometer).
 - Easy to prepare and use
- Why use varnish as a carrier?
 - Long contact time with Enamel
 - Lasting effect with an annual application.
 - Easy to apply on the tooth
 - Only safe topical fluoride treatment for young children
 - Recommended by American Dental Association based on evidence.
- Other delivery methods of ACP
 - Solution, Gel, Toothpaste, Prophylaxis paste, Composite, Chewing gum, Mouth rinse, Floss
- Advantages
 - Remineralizes the tooth: Puts back the tooth mineral and increase the hardness.
 - Increases fluoride efficacy: more release and more uptake
 - Obstruct the dentin tubules: Decrease hypersensitivity
- Second generation ACP under development
- ACPF Varnish with Chlorhexidine provides an antimicrobial and remineralization agent in the same product.
- ACPF Varnish with Arginine & Chlorhexidine is Antiacidic, Antimicrobial and Remineralizing
- Conclusion:
 - ACP Technology is able to remineralize the tooth in clinical applications.
 - ACP can incorporate and deliver beneficial ions: F, Chlorhexidine, and Arginine.
 - Varnishes readily deposit ACPs which act as therapeutic agents and also as the carriers for long term releases.
 - Some products are ready for clinical study.

Timothy Bromage, PhD

Coupled microbial and human systems: The importance of complexity and integrative thinking

- Waddington's epigenetic landscape: At the early stages of the development of a complex entity, there are many paths that can be followed and are influenced by the environment. As development continues, there are progressively fewer and fewer options, and the energy required to move back to an earlier path not taken is progressively larger. ECC is an example of a health problem canalized by social and environmental factors that draw the disease further along its deleterious path.
- The science of complexity is about revealing the principles that govern the ways in which new properties appear. These principles include, self-organization, self-adaptation, rugged energy landscapes, and scaling (e.g. power-law dependence) of the parameters and the underlying network of connections. ECC is a complex problem.
- Complexity results in stable scale-free ecological relationships
 - Example: For the human economic production system and the role of microbes, the main elements are:
 - ♦ Energy, metabolism and growth
 - ♦ Microbes and metabolic adaptation
 - ♦ Economic stoichiometry
 - When comparing the health of countries, there is a clear direct relationship between the risk of having a major infectious disease and the birth rate: the higher the risk, the higher the birth rate, which diminishes the amount of energy available for national production and the demographic transition. This example demonstrates that the complexity of a problem must be understood in order to find a stable-state solution.

- Regarding severe, destructive caries in the primary dentition, despite the very substantial efforts to treat the disease, administer both preventive and curative programs well, and to conduct investigations into the problem, so far in many AI/AN communities these energies have not translated into improved health for the children. That is, the complexity of this problem has not been fully mapped.
- Wacantognaka, the Sioux word for generosity, means to contribute to the well-being of one's people and all life by sharing and giving freely. This sharing is not just of objects and possessions, but of emotions like sympathy, compassion, kindness. It also means to be generous with one's personal time. The Sioux must come to express this sentiment and to own their role in finding a solution to ECC.
- This suggests that it is likely that the missing part of all the ECC-prevention prevention efforts has been to incorporate the social networks that will provide the cultural support needed for parents to be more effective in their health promoting behaviors. The task is to define who are the individual's real 'neighbors' and how they can help influence positive health behavior.

Dee Robertson, MD, MPH

Barriers to ECC control in AI/AN children

Collectively we have made little if any progress over the last three decades in controlling rampant caries in the primary dentition of AI/AN children. I believe the primary barrier to progress has been, and continues to be, attitudes that are based in perception rather than fact:

- Barriers to ECC Control: Myth #1: We already know what needs to be done to control ECC among AI/AN children—we just need to do a better job with the products and strategies we currently have.
- Barriers to ECC Control: Myth #2: If there are enough dentists, we can resolve this problem
- Barriers to ECC Control: Myth #3: We can't control ECC until we eliminate poverty
- Barriers to ECC Control: Myth #4: We can't do anything to control ECC because of the parents won't change their behavior
- Barriers to ECC Control: Myth 5: If the mom said she followed our recommendations and her child still got ECC, the mom was probably lying
- Barriers to ECC Control: Myth 6: ECC is self-limiting disease of the primary dentition
- Barriers to ECC Control: Myth #7: Indian parents don't care

Leaving the world of mythology and coming back to the world of reality, we have eliminated polio, measles, mumps, rubella, whooping cough, Hib meningitis among AI/AN children. We did this by a combination of technology and

public health infrastructure We have the public health infrastructure to control ECC, but we don't have the technology. The reason we don't have the technology is that unlike the other infectious diseases referenced above, rampant caries in the primary dentition is not an equal opportunity disease: it usually affects only the most disadvantaged children.

Conclusion: If a child:

- Is born with a substantial amount of enamel hypoplasia, and
- Has early and heavy exposure to *Streptococcus mutans*, and
- Has a moderate exposure to simple sugars in the diet,

you would expect a Perfect Storm of caries in the primary dentition. This is exactly what AI/AN children have experienced for decades, and what we continue to see at very high rates in many AI/AN communities.

Workgroups

Based on the recommendations of the Symposium planning group, five key topical areas were specified for extended discussion and recommendations by small workgroups on the morning of day #2 of the Symposium:

1. The Epidemiology of caries in the primary dentition among AI/AN, including issues of definition, nomenclature, prevalence and severity, adequacy of existing data sources.
2. The Microbiology of caries in the primary dentition among AI/AN, including the primary pathogens of importance, accuracy and validity of different methods of assessment of cariogenic bacteria, and correlation between assessment of cariogenic bacteria and clinical outcomes.
3. The role of Enamel Hypoplasia in rampant caries in the primary dentition, including evidence on the etiologic role in rampant caries in the primary dentition, methods of diagnosis, classification of severity, and correlation with morbidity.
4. New Prevention Products And Strategies, including those available but not being utilized, those expected to be available in the foreseeable future, expected efficacy in controlling rampant caries in the primary dentition, and feasibility for use in public health practice settings as opposed to research.
5. Planning the Next Steps, including building coalitions and funding support.

The following guidelines were given to the workgroups to help structure their discussion and their reports that were presented in the subsequent plenary session to conclude the technical aspects of the Symposium:

- **Workgroup Goal:** Use information from Day #1 to identify knowledge gaps which, if they remain unanswered, will significantly impede progress toward the control and prevention of rampant caries in the primary dentition among AI/AN children.
- **Format:** The workgroups consisted of 5–10 Symposium participants each. Each group had a leader chosen by the Symposium planners who is an expert in the broad area to be covered, but not necessarily a subject expert in the specific topics to be covered. The leaders guided and focused the discussion in order to produce a brief report for the plenary session to follow consisting of the following:
 - a. The specific issues in their broad topical area most relevant to advancing the overall goal of reducing the burden of disease from rampant caries in the primary dentition among AI/AN children.
 - b. Specific areas that are deemed sufficiently defined at the present and, thus, not priority areas for additional research.
 - c. Specific knowledge gaps that will impede progress unless resolved.
 - d. A prioritized list of specific questions, issues, uncertainties that need to be addressed.
 - e. Recommendations on who should be involved in addressing each issue.

Workgroup Topic Reports

1. Epidemiology

In order to move ahead we need:

- New case definitions of what have previously been called ‘early childhood caries’ and ‘severe early childhood caries,’ including working definitions useful for research and surveillance. This new case definition should include a measure of severity with a correlation to clinical morbidity.
- A better descriptive epidemiology, including baseline data on prevalence (using the case definition), and representation at the national and tribal community level
- A better way to quantify risk factors, including generalizeability across population groups and a community-based participatory approach
- A better surveillance system, including integration with national surveillance efforts, determining what needs to be measured and the instruments to be used, frequency of measurement, and getting population-based data (as opposed to clinical encounter data)
- Prognostic markers that will predict early in life whether a child will suffer excessive morbidity (longitudinal studies)
- Cost effectiveness research for interventions
- Prenatal risk studies, focusing on identifying prenatal risk factors and developing interventions
- Training AI/AN Human Resources to enhance their research capacity
- Central coordination and funding to avoid the “silo effect” and duplication of efforts among tribes and advocates for maximizing efficiency in research and surveillance

2. Microbiology

What is already defined

- *Streptococcus mutans* and *Streptococcus sobrinus* (collectively called mutans streptococci or MS) are the primary organisms driving rampant caries in the primary dentition
- Interaction between diet and MS drives concentration and dominance of these cariogenic bacteria (Ecologic Plaque Hypothesis)
- Early age of acquisition correlates with increased risk of rampant caries in the primary dentition
- Culture-based methods lack sensitivity and specificity and have limitations, but remain acceptable for some epidemiological and intervention study designs (scientific and funding perspective)
- The FDA requires use of real-time PCR as the approach for licensing new MS assays
- Microbial community in plaque correlates well with saliva by species presence and probably percentage
- Total elimination of MS is not necessary to achieve reduction in new caries development—the goal being a reduction of density or percent of total flora.

Knowledge gaps

- Need to confirm and better define the preliminary finding of very high percentages of MS of total oral bacteria in some indigenous populations.
- Need agreement on optimal methods of MS detection and quantification
- Determine the optimal sample method for specimen collection and handling for MS studies
- Conduct cross-lab comparisons of MS count and percent of total oral bacteria quantification to allow comparison of reported data
- Further characterize transmission patterns of MS to infants and children and factors related to transmission.
- Identify strain characteristics of MS and relationship to virulence and disease outcome
- Develop a library resource for strain typing with standard nomenclature and documentation
- Further characterize oral flora and association with caries risk
- Epidemiology of antibiotic resistance of MS and impact of resistance on colonization and virulence or disease
- Host immunity: Is IgA response protective against MS (age of acquisition, density, caries)?
- Identify whether there other correlates of immune protection

3. Enamel Hypoplasia

- Recognizing the importance of enamel hypoplasia (EHP) in the etiology of rampant caries in the primary dentition among AI/AN children:
 - Can change the current perception of rampant caries in the primary dentition as being an easily preventable disease.
 - Can eliminate much of the “blame game”
 - Can provide one of the “missing measures” of oral health and perhaps even general health
- Previous research suggestions there are many risk factors for EHP, but we don’t know if there are any dominant factors and therefore can’t make preventive recommendations beyond the current prenatal care standards. Therefore, at present we should:
 - Assume EHP is a good predictor of risk for rampant caries in the primary dentition
 - Apply classic interventions earlier or at higher frequency (don’t have any other options at this time)
 - Include well baby clinical visits and non-clinic based visits by public health nurses to serve as the primary screening and referral sources
- The most immediate need is:
 - A good working definition of “enamel defects” or enamel hypoplasia. This could be developed by modifying existing definitions used by experts but with input from clinicians serving this community
 - A user-friendly screening protocol that can be used by non-dental professionals outside of the dental clinic. The purpose of screening include, research, determining the prevalence in different populations, correlation with rampant caries in the primary dentition (predictive value), role in causation
- Gaps and recommendations
 - Develop age and user appropriate materials specific to EHP
 - Determine the suitability of and effectiveness of glass ionomers applied to areas of EHP
 - Assess the efficacy of remineralizing products containing amorphous calcium phosphate
 - Assess the efficacy of polymer or glass ionomer infiltration materials for smooth surface sealing
 - Do what we can to expedite approval of any efficacious new interventions available in other countries, such as silver diamine fluoride.
- available, (b) delivered at optimal time (before age 1), (c) by low cost personnel
- Use of placebo control groups in AI/AN research may be problematic in many AI/AN communities who are suspicious of health research
- We need better case definition of rampant caries in the primary dentition
- Risk assessment tools
 - Need both diagnostic and prognostic tools
 - The tools need to be specific to rampant caries in the primary dentition (not just risk for any caries)
 - Need to evaluate tools such as laser fluorescence or microbial metrics
- Microbial Intervention Knowledge Gaps
 - The efficacy of silver diamine fluoride (at this point not FDA-approved or IRB approved) in the children at the very highest risk for rampant caries in the primary dentition
 - Most available interventions have not been adequately tested in the populations with highest risk for rampant caries in the primary dentition
 - All interventions need to be evaluated not only for cavitation endpoint, but also for quality of life, economic outcomes (e.g., lowered need for treatment under general anesthesia or other high cost treatment)
 - Need more research to evaluate multimodal interventions
 - The efficacy of fluoride varnish in reducing rampant caries in the primary dentition. Would it be more effective if combined with CaPO₄, chlorhexidine, arginine, or all of the above (Dr. Ming’s ‘super varnish’)
 - What is the most efficacious dosing (amount and frequency) for each intervention?
 - Are probiotic interventions effective? Substitution strategies?
 - Is the newly-developed specifically targeted antimicrobial peptides (STAMPs) technology effective in this population?
 - FDA label claims are an issue that should be resolved for direct to consumer advertising (motivation)
- Structural Interventions Knowledge Gaps
 - Improving the efficacy of fluoride in rampant caries in the primary dentition by combining it with other products such as CaPO₄ in different preparations, such as varnish, rinse, gel, toothpaste, or as a stand-alone companion product?
 - Restorative Knowledge Gaps
 - Glass ionomer effectiveness as part of a program to prevent rampant caries in the primary dentition, including recurrent caries rate effects, efficacy when combined with other interventions, and frequency of “recharging”

4. New Intervention Products

- Starting assumptions
 - Rampant caries in the primary dentition is a multifactorial disease
 - It is likely that not all risk factors are equal
 - Effective interventions need to be (a) widely

- Behavioral Knowledge Gaps
 - Efficacy of motivational interviewing efficacy in rampant caries in the primary dentition in different settings
 - Efficacy of other educational strategies
 - How to get an effective message to the right receiver at the right time
- Prioritized Recommendations
 - Conduct research on the efficacy of silver diamine fluoride in populations at highest risk for rampant caries in the primary dentition population
 - Develop and validate a risk assessment tool for rampant caries in the primary dentition
 - Answer the above fluoride varnish questions
 - Answer the above glass ionomer questions
 - Answer probiotic questions (when products are available)

5. The Next Steps

Broad issues that need to be addressed to reduce rampant caries in the primary dentition of AI/AN children:

- Implementation and Advancement of Research Agenda. A wide range of issues were identified, including governmental relationships with tribes, Indian Health Care Improvement Act, congressional earmarks, NIDCR core funding, CDC & HRSA (title v), philanthropic oral health specific support from Kellogg, Pew, tribal gaming, DentaQuest, and others with an interest in children's health such as the national oral health alliance.
- Specific knowledge gaps that will impede progress
 - The disease burden from rampant caries in the primary dentition in AI/AN communities
 - Understanding the differences among and within AI/AN communities in terms of rampant caries in the primary dentition burden
 - Are there specific social determinants that are making rampant caries in the primary dentition so prevalent in AI/AN communities?
 - The long term sequelae of rampant caries in the primary dentition, including co-morbidities, economic success and educational attainment
 - How do we work with Indian communities to develop infrastructure such as community review boards to partner with researchers
 - Evaluating successful models and disseminating best practices
 - In developing a research agenda, look at models such as the Community Health Representative (CHR), Promotora, CDHC which may make the most sense in oral health messaging
 - Identify how IHS should fit into this research agenda

Collectively the different workgroups identified the following entities as the parties that should be responsible for carrying out the issues identified:

- Broad based group of epidemiologists and researchers
- Indian Health Service (IHS)
- Tribes
- Centers for Disease Control (CDC)
- Academic researchers and institutions
- HRSA
- NIDCR

Conclusion

The Symposium met each of its stated objectives. On the first day, 16 experienced caries researchers, representing 10 caries research institutions, reviewed and discussed the most current state of the science of caries in the primary dentition. On second day, the Symposium participants split into five small workgroups to summarize the relevant information from Day 1, identify aspects of each topical area that are insufficiently understood and make recommendations on how to fill those gaps. The first workgroup report on Day 2 (from the Epidemiology group) summed up why there may be such a limited understanding of this disease: "The terminology, definitions and classification of caries in the primary dentition is deficient as a metric for measuring disease burden and improvement from effective interventions." Carrying out the recommendations of the Symposium participants may greatly enhance future efforts to understand and control this disease. Although the bacterial etiology of ECC has been known for decades, the methods of assessment of bacterial levels and correlation with clinical outcomes are largely inconsistent and may be a substantial barrier to understanding and controlling the severe end of the spectrum of caries in the primary dentition. A method for developing a much more standardized and reliable measure was proposed. The relatively new and unexplored area of enamel hypoplasia was posited as a primary risk factor for severe caries in the primary dentition, and recommendations were made about how to verify or refute this as a risk factor. Last, highly promising new intervention agents were reviewed, with recommendations on how to proceed to utilize these with AI/AN populations.

Appendix A

Agenda: Symposium on Early Childhood Caries in American Indian/Alaska Native Children

ADA Council on Access, Prevention and Interprofessional Relations
in cooperation with
American Academy of Pediatrics

Hotel Alex Johnson, Rapid City
October 20–22, 2010

Wednesday, October 20

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|------------|--|
| 7:00 a.m. | Registration and Breakfast |
| 8:00 a.m. | Symposium Welcome Dr. Monica Hebl |
| 8:10 a.m. | Traditional Blessing Tim White and Delbert Pumpkinseed (Sisseton-Wahpeton Oyate) |
| 8:20 a.m. | Review Agenda, Format and Objectives Dr. Katherine O'Brien |
| 8:45 a.m. | An historical perspective of early childhood caries in American Indians and Alaska Natives There and back again Dr. Don Marianos |
| 9:00 a.m. | Caries in the primary dentition: A spectrum disease of multifactorial etiology Dr. Page Caufield |
| 9:35 a.m. | Maternal transmission of mutans streptococci in severe early childhood caries Dr. Noel Childers Etiologic factors for early childhood caries Dr. David Drake Ecological considerations of <i>S. mutans</i> Dr. Wenyan Shi |
| 10:50 a.m. | BREAK |
| 11:00 a.m. | Enamel hypoplasia as a prerequisite to rampant caries Drs. Yihong Li, Timothy Bromage and Page Caufield |
| 11:30 a.m. | Early childhood caries in American Indians/Alaska Natives: A pediatric infectious disease with dental manifestation Dr. Steve Holve |
| 11:45 a.m. | Epidemiology of early childhood caries and effectiveness of interventions Dr. Ananda P. Dasanayake |
| Noon | Working Lunch |
| 12:20 p.m. | Questions and Answers for All Morning Presentations |
| 1:00 p.m. | Early childhood caries and Aboriginal children in Canada Drs. James Irvine, Robert Schroth and Rosamund Harrison |
| 1:20 p.m. | ECC Research among American Indian and Alaska Native Children <ul style="list-style-type: none">• Tribal Community Perspective on ECC-prevention Research Sara DeCoteau• Applying Fluoride Varnish in a Pediatric Clinic Dr. Steve Holve• Applying 10% CHX Varnish to the Mothers' Dentition Dr. Dee Robertson• Transmission of <i>S. mutans</i> in a Tribal Community Dr. David Drake• Multimodal Intervention using a Community-based Participatory Research Model Dr. Dee Robertson |
| 2:35 p.m. | BREAK |

| | |
|-----------|--|
| 2:50 p.m. | New Interventions for ECC Prevention in High-risk Populations <ul style="list-style-type: none"> • Glass ionomer sealants Dr. Joel Berg • 1.0% flavored chlorhexidine gel Dr. David Drake • Caries vaccine Dr. Noel Childers • Silver diamine fluoride Dr. Richard Niederman • STAMPS Dr. Wenyan Shi • ACP Technology: Amorphous Calcium Phosphate forming Fluoride Varnishes Dr. Ming Tung |
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| 5:00 p.m. | Selection of Day 2 Workgroups Dr. Katherine O'Brien |
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Thursday, October 21

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| 7:00 a.m. | Breakfast |
| 8:00 a.m. | Opening <ul style="list-style-type: none"> • Traditional Blessing Tim White and Delbert Pumpkinseed (Sisseton-Wahpeton Oyate) • Summary of Day 1 Discussion and Day Agenda Dr. Katherine O'Brien |
| 8:30 a.m. | Holistic Perspectives of ECC and Barriers to ECC Control <ul style="list-style-type: none"> • Coupled Microbial and Human Systems: The Importance of Complexity and Integrative Thinking Dr. Timothy Bromage • Barriers to the Control of ECC among AI/AN Children Dr. Dee Robertson |
| 9:30 a.m. | Topical Workgroups <ul style="list-style-type: none"> • Epidemiology of ECC among AI/AN • Microbiology of ECC among AI/AN • Enamel Hypoplasia • New Prevention Products and Strategies • Planning the Next Steps |
| Noon | Working Lunch |
| 1:00 p.m. | Workgroup Reports <ul style="list-style-type: none"> • Introduction • Epidemiology of ECC among AI/AN • Microbiology of ECC among AI/AN • Enamel hypoplasia • New Prevention Products and Strategies • Planning the Next Steps |
| 3:30 p.m. | BREAK |
| 3:45 p.m. | Prioritization of Research Topics <ul style="list-style-type: none"> • Research Agenda Development Dr. Katherine O'Brien |

Friday, October 22

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| 7:00 a.m. | Breakfast |
| 8:00 a.m. | Learning Visit to Nearby Reservation |
| 6:00 p.m. | Dinner and Presentation of Draft Symposium Recommendations |
| 7:00 p.m. | Research Agenda Presentation |

Appendix B

Participants

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| Max Anderson, DDS, MS, MEd | Co-founder; C3 Jian, Inc. |
| Abdullah Baqui, DrPH, MPH, MBBS | Professor; Johns Hopkins Bloomberg School of Public Health |
| Eugenio Beltran, DMD, MPH, DrPH | Team Leader and Lead Epidemiologist; Centers for Disease Control and Prevention, Division of Oral Health |
| Joel Berg, DDS, MS | Professor and Lloyd and Kay Chapman Chair for Oral Health; University of Washington, Department of Pediatric Dentistry |
| Winifred Booker, DDS | Member; Society of American Indian Dentists |
| Tim Bromage, PhD | Professor, Department of Biomaterials and Biomimetics, and Director, Hard Tissue Research Unit; New York University, College of Dentistry |
| Page Caufield, DDS, PhD | Professor, Cariology and Comprehensive Care; New York University, College of Dentistry |
| Noel Childers, DDS, MS, PhD | Joseph F. Volker Professor and Chair, Department of Pediatric Dentistry; University of Alabama at Birmingham, School of Dentistry |
| Amanda Ciatti | Manager, Program Research & Development; Oral Health America |
| Ananda Dasanayake, BDS, MS, PhD | Professor, Epidemiology and Health Promotion; New York University, College of Dentistry |
| Sara DeCoteau | Tribal Health Coordinator; Sisseton-Wahpeton Oyate of the Lake Traverse Reservation |
| David R. Drake, MS, PhD | Professor; University of Iowa, College of Dentistry |
| Frederick Eichmiller, DDS | Vice President and Science Officer; Delta Dental of Wisconsin |
| Steve Geiermann, DDS | Senior Manager, Access, Community Oral Health Infrastructure & Capacity; American Dental Association |
| Nicole Glines, RDH, MPH | Dental Hygienist; Indian Health Service |
| Lori Goodman, RDH | Dental Hygienist; Indian Health Service |
| Connie Halverson; Vice President | Delta Dental South Dakota |
| Rosamund Harrison, DMD, MS, MCRD | Associate Professor and Chair, Division of Pediatric Dentistry; University of British Columbia |
| Monica Hebl, DDS | Member, Council on Access, Prevention and Interprofessional Relations; American Dental Association |
| Thomas Hennessy, MD, MPH | Director, Arctic Investigations Program; Centers for Disease Control and Prevention |
| Steve Holve, MD | Chief Clinical Consultant in Pediatrics; Indian Health Service |
| James Irvine, MD, MSc | Professor, Department of Family Medicine; University of Saskatchewan, College of Medicine |

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| Sara Jumping Eagle, MD | Member, Committee on Native American Child Health; American Academy of Pediatrics |
| Lew Lampiris, DDS, MPH; | Director, Council on Access, Prevention and Interprofessional Relations; American Dental Association |
| Don Marianos, DDS, MPH | Independent Consultant |
| Richard Niederman, DMD | Director, Center for Evidence-Based Dentistry; The Forsyth Institute |
| Kate O'Brien, MD, MPH | Professor, International Health; Johns Hopkins Bloomberg School of Public Health |
| Kathy Phipps, DrPH | Independent Consultant |
| Gary Podschun | Manager, Community Outreach and Cultural Competence; American Dental Association |
| Dee Robertson, MD, MPH | Independent Consultant |
| Lindsey Robinson, DDS | President; California Dental Association Foundation |
| R. Gary Rozier, DDS, MPH | Professor, Health Policy and Management, and Director, Dental Public Health & Residency Training Program; University of North Carolina at Chapel Hill, Gillings School of Global Public Health |
| Shelli Ryczek, RDH | Independent Consultant |
| Bob Schroth, DMD, MSc | Assistant Professor, Department of Oral Biology, and Faculty of Dentistry; University of Manitoba |
| Wenyuan Shi, PhD | Professor and Chair, Section of Oral Biology; University of California Los Angeles, School of Dentistry |
| Delores Starr, RDH | Dental Hygienist; Indian Health Service |
| Timothy Thomas, MD | Medical Officer, Arctic Investigations Program; Centers for Disease Control and Prevention |
| Ming Tung, PhD | Senior Project Leader; Paffenbarger Research Center |
| John Warren, DDS, MS | Professor, Preventive and Community Dentistry; University of Iowa, College of Dentistry |
| Bob Weyant, DMD, DrPH | Associate Dean, Office of Public Health and Outreach; University of Pittsburgh, School of Dental Medicine |
| John Zimmer, DDS | Pediatric Dentist; Indian Health Service |

Contact Information

Gary Podschun
Council on Access, Prevention and Interprofessional Relations
American Dental Association
211 East Chicago Avenue
Chicago, IL 60611
312.440.7487
PodschunG@ada.org

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