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Characterization of maternal mutans streptococci transmission in an African American population

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Dental caries is an infectious and transmissible disease in which mutans streptococci (MS) is regarded as a contributing etiological agent [1,2]. The species of MS most commonly isolated from dental plaque in humans is *Streptococcus mutans* (serotypes *c*, *e*, and *f*) and *Streptococcus sobrinus* (serotypes *d* and *g*). Both have been isolated from populations worldwide [3]. Epidemiological and clinical studies have demonstrated the relationship between MS in dental plaque and the development of caries. More specifically, MS are universally found in caries-active individuals [2] and strongly correlate with the prevalence of caries, including rampant caries and early caries lesions in children [4–8]. Children who acquire MS at an earlier age are at greater risk for developing caries than those who acquire MS at a later

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age [9–13]. Kohler and Andreen [6] reported that reducing the salivary levels of MS in mothers during the emergence of the primary teeth in her child could delay MS colonization and influence subsequent caries experience.

Although a substantial basis of knowledge exists concerning the etiologic association of MS and other microorganisms with dental caries, the natural history of the disease is not well understood. For example, one might predict that MS levels in plaque would be a good predictor for caries; however, this is not the case. The presence or absence of MS levels alone only moderately improves the fitness of most caries prediction models [14–16]. This is because most humans harbor MS, but not all manifest caries. Sigurjóns et al [17] monitored a group of 0- to 40-month-old children for 3 years to study the changes in cariogenic bacteria, including *S mutans*, and the development of caries. They concluded that bacterial counts alone could not predict caries with sufficient reliability. Perhaps key questions concerning the association between MS and caries are not only when and in what numbers MS colonize the infant's mouth, but also the source and the cariogenic potential of the individual strains present.

Previously, we demonstrated that African American mothers transmitted MS to their infants with greater fidelity than did white mothers [18]. This finding was coupled with the observation that African American children experience higher incidence of dental caries than do US white children [19,20]. Socioeconomic status has been named as a contributing factor to the difference [21]. Few of the previous studies, however, have evaluated the variation in caries among population subgroups from a microbiological perspective. During the past few years, our research group has conducted a number of investigations in the area of the natural history of MS infection and transmission in an African American mother–child cohort. The following discussion summarizes our previous study findings as well as how some of main maternal attributes affect the MS transmission in children.

Epidemiology of MS

Prevalence of MS in different ethnic populations

Epidemiologic studies of MS in different populations have shown that this organism is frequently isolated in children living in many parts of the world with different ethnic and socioeconomic backgrounds. The prevalence estimates significantly vary depending on the bacteriologic methods used. A summary of reported MS prevalence among preschool children under the age of 6 is presented in Table 1. The prevalence of MS among children 1 to 2 years of age ranged from 7% to 86%. The discrepancy in detecting MS among 5-year-old children could be as much as a twofold difference when comparing populations. Using different samples and techniques, Dasanayake et al [22] examined the recovery of MS from all subjects. They found that plaque samples provided a slightly higher MS recovery than did

Table 1
Prevalence of mutans streptococci in different ethnic groups

Age (years)	Sample size	Prevalence (%)	Ethnic group
1	1095	6	White (Sweden) [59]
1–2	60	85	Puerto Rican [60]
1–3	356	40	Japanese [61]
1–4	101	72–96	Brazilian [12]
2–5	95	84	US whites [20]
2–5	129	81	US Hispanic [20]
2–5	252	43	White (Netherlands) [8]
3–4	2728	74	South African black [28]
3–4	486	95	Chinese [62]
3–4	140	71	Latvian [63]
3–5	76	42	Uruguayan [64]
5	144	45	White (Finland) [65]
5	50	84	White (West Iceland) [11]

saliva and swab samples, and stimulated saliva samples had a better recovery than did nonstimulated samples. Others [23] have shown that different culture media can account for approximately 10% of the variation in the levels of MS. Because all cultivating techniques and selective media have limitations, and only a fraction of the microbiota grows on any artificial medium, cautious interpretation of various epidemiologic results across populations are needed.

Prevalence and distribution of MS in African American children

Information on MS prevalence and distribution in young African American children is lacking compared with that in other ethnic groups. Thibodeau et al [20] reported in 1993 that the MS prevalence for 2- to 5-year-old Head Start Program children was similar to that in African American (83%), white (81%), and Hispanic children (84%). The severity of infection, however, varied among the groups. African American children demonstrated higher MS levels (40%) compared with white children (28%) and Hispanic children (33%). A significant difference was also observed in caries activity measured by total decayed, missing, and filled tooth surfaces (DMFS) among the different ethnic groups. The mean dmfs was 3.33 ± 7.7 for African American children, 2.0 ± 3.4 for Hispanic children, and 2.5 ± 8.2 for 2- to 5-year-old white children. The reported variations in MS and caries prevalence among different ethnic groups were the focus of investigations that studied differences as a result of socioeconomic factors [24–27]. None of the studies, however, examined the correlation between ethnic groups and microbial risk factors in young children in relation to caries outcome.

Findings from a cross-sectional study

A cross-sectional epidemiologic study [66] of a group of low-income African American children was carried out in an inner-city elementary school in

Birmingham, Alabama in 1992. A total of 466 children in grades K through 5 were included. Plaque and saliva samples were collected from each subject and oral examinations were performed. A take-home self-administered questionnaire that included items on basic demographic factors, child's use of antibiotics, breast-feeding habits, caregiver history, sweet consumption, and tooth emergence was also completed by each mother. The main findings of the study are listed in Tables 2 and 3. The results can be summarized as: (1) MS prevalence was 92% in this African American child population. This prevalence (92%) was higher compared with the prevalence found either in American white children (84%) or in South African-Black children (74%) [28]; (2) MS prevalence was associated with the number of teeth in the oral cavity, age, mother as the primary caregiver, increase in frequency of sweet consumption, increase in number of people in the same household, and child's use of antibiotics; and (3) higher MS levels were associated with a higher number of decayed teeth. In most cases, the average age of the children was over 5-years-old when the initial infection with MS occurred. The study suggests that the percentage of children with MS positive was the true prevalence of MS colonization rather than the percentage of initial infection. Thus, these findings presented some similar trends compared to what have been reported for American-Caucasian children and provided consequential insights into the unique characteristics of MS prevalence associated with dental caries in an African-American child population.

Findings from longitudinal studies

The first longitudinal study of MS transmission in a Birmingham mother–child cohort was performed in 1985 to 1990. More than 1200 pregnant women attending the Public Health Clinics of Jefferson County of Alabama were screened at one of their prenatal visits [29–31]. Because African Americans comprise the majority of individuals residing in Jefferson County,

Table 2
Prevalence of mutans streptococci by age and gender among African American children

Variable	n	Prevalence (%)	95% confidence interval
Gender[a]			
Male	194	92	89.0–96.0
Female	159	91	87.0–96.0
Age group (years)[b]			
5–6	30	77	62.0–92.0
6–7	65	91	84.0–98.0
7–8	74	97	97.0–97.4
8+	184	92	88.0–96.0
Total	353	92	89.0–95.0

[a] Fisher exact test, $P = 0.85$.

[b] χ^2 for trend $= 4.3$, $P = 0.04$.

Data from Dasanayake AP, Roseman JM, Caufield PW, Butts JT. Distribution and determinants of mutans streptococci among African-American children and association with selected variables. Pediatr Dent 1995;17:192–8.

Table 3
Factors associated with mutans streptococci prevalence in African American children

Variable	Odds ratio	95% confidence interval
Ever use antibiotics	8.3	2.0–35.0
Antibiotic use at year 2	∞	1.1–∞[a]
Sweet consumption (frequency per day)[b]		
At least 3 times	4.9	1.3–18.0
More than 3 times	6.8	1.3–36.0
Number of people who lived with child (0–2 years)	1.5[c]	1.03–2.2
Total teeth present	1.4[c]	1.1–1.6
Total decayed teeth	1.3[c]	1.1–1.9

[a] Exact confidence interval.
[b] Chi-square test for trend = 5.1, $P = 0.02$.
[c] Unit odds ratio.
Data from Dasanayake AP, Roseman JM, Caufield PW, Butts JT. Distribution and determinants of mutans streptococci among African-American children and association with selected variables. Pediatr Dent 1995;17:192–8.

Alabama and they go to the Jefferson County Public Health Department (JCPHD) for their health care services, Birmingham-JCPHD was an ideal study site for recruiting an African American mother–child population to participate in a longitudinal study.

Thirty-nine mother–child pairs completed this 4-year study; 53% were African American and 47% were white (controls). Oral bacterial samples were obtained from the mothers and children at 3-month intervals from the third trimester of pregnancy until the children were 3 years old. A child was defined as infected by MS if any two consecutive saliva samples were positive for MS [30]. The major finding of the study was that infants acquired MS during a discrete time period, which was designated as the ''window of infectivity'' [31]. MS was detected in 25% of these 38 infants by 19 months of age and in 75% by 31 months of age. Time from emergence of first tooth was highly correlated with MS colonization in the children, which implied that the discrete nature of initial MS acquisition is directly related to the presence of newly emerged teeth, particularly to the availability of fissure surfaces. African American children exhibited a significantly lower percentage of initial MS acquisition than did the white children by the end of study. Several potential factors, including the mother's MS levels, the child's immune system, mother's use of antibiotics at the delivery, child's use of antibiotics during early childhood development, breast-feeding behaviors, and infants undergoing a change in their diet were hypothesized for further investigation. The finding that African American children experienced a higher incidence of dental caries than did white children, but a delayed MS initial acquisition, led us to focus further on African American families.

A second longitudinal study on MS transmission in an African American mother–child cohort was carried out in 1995 to 2000 [52–56]. Two hundred and ninety-six first-time mothers who attended a County Department of

Health obstetrics and gynecology clinic were recruited for the study during the second trimester of their pregnancy. This was a high-risk, low-income, and genetically homogeneous Birmingham cohort. The estimated bacterial level per mL of saliva for MS was obtained for all 296 mother–child pairs at 3-month intervals. The total count was transferred into a $\log_{10}$ value for statistical analyses. The time of acquiring MS for an infant was based on colony morphology identification on mitis-salivarius-bacitracin (MSB) medium. The results showed that infants acquired *S mutans* as early as 9 months of age and 75.6% of the children were infected with *S mutans* at 36 months of age, whereas 24.4% demonstrated undetectable levels of *S mutans* at the end of the study. The average age at which this group of children acquired *S mutans* was 24.4±8.0 months. This finding was in agreement with previously reported windows of infectivity for *S mutans* [31]. Using correlation analysis, a significant positive relationship was also found between tooth emergence and the initial acquisition of *S mutans* ($\log_{10}$ *S mutans* $= 1.99 + 0.14\times$ tooth, $r^2 = 0.35$, $P = 0.013$). The colonization of *S sanguinis*, however, was independent from the age and tooth emergence ($\log_{10}$ *S sanguinis* $= 5.00 + 0.02\times$ tooth, $r^2 = 0.04$, $P = 0.326$). These findings supported the idea that the availability of newly erupted primary tooth surfaces in the oral cavity fostered colonization by specific bacterial species among the infants [32–36]. The study further revealed that there were no significant differences in the initial acquisition of *S mutans* in the children with respect to gender, breast-feeding experience, length of breast feeding, mothers' *S mutans* level, and mothers' caries status.

In previous publications, we indicated several factors that might influence the results when studying MS acquisition in child populations [22,31]. For example, the ability to identify initial MS colonization by using conventional cultivation methods may not be sensitive enough to detect very low levels of MS; and the selective MSB medium might suppress MS growth—in particular, reducing *S sobrinus* growth. Evidence from other studies [10, 37–40] has shown an earlier time to infection by using various study methodologies. The application of new molecular techniques (eg, MS species-specific DNA probes) as they become available will help to determine the occurrence of certain microbes associated with caries progression and to ascertain the risk potential to children prior to the development of the disease.

Mutans streptococci transmission

The notion that mothers are the primary source of oral microbe transmission to their offspring was first suggested in the classic works of Keyes [41] with laboratory animals in the 1960s. Studies using bacteriocin profiles, serotyping, and genotyping [18,35,42–47] demonstrated that mothers are the primary source of MS for their infants, which we describe as "fidelity of transmission" [18]. Numerous studies [48–51] demonstrated a positive

correlation between the levels of *S mutans* in mothers and their children—that is, mothers with high levels of *S mutans* tend to have children who have high levels of *S mutans*. During the past 10 years, our laboratory has further developed a system of combining conventional microbiological cultivation, biochemical testing methods, chromosomal DNA fingerprinting, and other genetic profiling techniques for MS transmission study. We have been able to confirm the correlation between mother–child pairs but also more precisely identify the source of MS transmission from the mother to her child.

Fidelity of maternal MS transmission

The evidence of transmission from mother to child can be shown by comparing a particular genotype of MS of the mother with that of the child at initial MS acquisition. If the child's newly acquired MS stains are genotypically identical to those of the mother, the direction of transmission can be determined. In our previous publication [18], the degree of similarity between the genotype of the mother and child was labeled the fidelity of transmission. If the genotypes between a mother and her child were identical, we designated that as 100% fidelity of transmission. If the mother's and child's strains did not match, we designated that as 0% fidelity. For the purposes of statistical analysis, the outcome variables were dichotomized as "yes" for match and "no" for no match.

Our first fidelity study was completed in 1995 by using a chromosomal DNA fingerprinting technique [18]. As shown in Table 4, among the total 34 mother–child pairs, 71% of the children had MS strains that were identical to those of their mothers at the time of the initial MS acquisition. The MS genotypes of the remaining 29% of the children did not match the strains isolated from their mothers. The study further revealed that mothers

Table 4
Association between fidelity of mutans streptococci transmission within mother–infant pairs and demographic attributes

	Genotypic match				
	Yes		No		
Category	n	%	n	%	Total N
Mother–infant pair	24	70.6	10	29.4	34
Gender[a]					
Female	15	88.2	2	11.8	17
Male	9	52.9	8	47.1	17
Race[b]					
Black	14	87.5	2	12.5	16
White	10	55.6	8	44.4	18

[a] Chi-square test, $P = 0.02$.
[b] Fisher exact test, $P = 0.06$.

Data from Li Y, Caufield PW. The fidelity of initial acquisition of mutans streptococci by infants from their mothers. J Dent Res 1995;74:681–5; with permission.

transmitted the MS strain to female offspring with significantly greater fidelity (88%) than to male offspring (53%). The genotypes of MS from female children were six times more likely to match their mothers' strains than were those from male children (odds ratio = 6.7, 95% confidence ratio = 1.2–38.6, $P = 0.02$). More interestingly, African American mothers tended to transmit MS to their children with a higher fidelity rate than did white mothers. Findings of this study indicated that the conservation of MS within mother–child pairs is gender and race specific. The study did not examine the actual genetic or cultural determinants that may influence fidelity of transmission. Tooth emergence, however, was observed to be, on average, earlier in females (6.8 months) than in males (7.2 months), and a significant correlation between early tooth emergence and time to infectivity was also noted. These findings could at least partially explain the difference in the fidelity between genders. There are other developmental differences—including the development of the immune system between males and females—that may also induce a variation in MS transmission. Examination of the caretaker histories revealed that between 1 and 2 years of age, a greater proportion of the children without MS had primary caretakers other than their biological mothers, compared with the children with MS in which the primary caretaker was exclusively the mother. African American mothers tend to spend more time with their children than do white mothers and present a different nurturing practice. Thus, we hypothesized that genetic, environmental, and behavioral differences between African American families and white families could be contributing factors to the variation in MS transmission.

Further evidence that the mother is the major source of MS transmission to their child was provided by another prospective study that took place from 1995 to 1999 as part of the Specialized Caries Research Center (SCRC) project. Genetic relatedness of *S mutans* for African American families was analyzed using chromosomal DNA fingerprinting and arbitrarily primed polymerase chain reaction (AP-PCR) [46,54]. Fifty African American mother–child pairs were selected as a natural history cohort of the SCRC population core. At the end of the study, 26 of the 50 children (52.0%) were *S mutans* positive, and 21 out of 26 children (80.8%) harbored *S. mutans* strains identical to their mothers' at the time of initial acquisition. When studying genetic relatedness of bacterial profiles using AP-PCR, one critical factor is to compare DNA fingerprint profiles with an objective analytical method. In the current literature, unweighted pair group method with arithmetic averages (UPGMA) cluster analysis is the most commonly used method. Unlike the study of genomic structure by nucleotide sequence analysis, which is based on accurate oligonucleotide sequence information; DNA fingerprint profile analysis is based on pattern recognition. Similarity matrices are generated for statistical analyses. In our study, the fingerprint patterns of MS strains among different individuals was compared using the UPGMA cluster analysis generated by the DNA Diversity Database (BioRad, Hercules, CA). The results demonstrated that it is an informative approach in assessing

similarities of *S mutan* strains among different individuals [46]. Currently, a number of computer software programs are commercially available for conducting these types of analyses.

Maternal attributes to MS transmission

Effect of pregnancy on the maternal MS levels

Oral health and pregnancy have been linked in various ways. The effect of pregnancy on oral microflora of African American woman—has not been previously reported. In our Birmingham study of natural history of oral biota acquisition [52,53], we examined the oral microbial flora of a group of African American women during and after pregnancy and correlated these findings with pregnancy outcomes. The levels of specific oral organisms—that is, *S mutans*, *S sobrinus*, *S sanguinis*, *Lactobacillus acidophilus*, *L casei*, *Actinomyces naeslundii* genospecies-1, *A naeslundii* genospecies-2, total streptococci, and total cultivable microflora—were enumerated for 176 pregnant women. A total of 913 stimulated saliva samples were collected at the third trimester, at delivery, and at the second and fourth months after delivery, and were analyzed using correlation analyses, paired *t* tests, and multivariable analyses. The study found that the salivary levels of *L acidophilus*, *L casei*, *A naeslundii* genospecies-1, and *A naeslundii* genospecies-2 before delivery were significantly higher than the postpregnancy levels [52]. The level of MS decreased significantly after delivery [53]. A similar decrease was also observed for total streptococci, *S sanguinis*, *L casei*, *L acidophilus*, and *A naeslundii* genospecies-2. This decrease, however, was transient because the levels returned to predelivery levels within 2 to 8 weeks. The total cultivable bacterial counts remained unchanged. These results demonstrated a variation in prevalence and level of oral microbiota during and after pregnancy, and indicated that a selective depression of Gram-positive oral bacteria occurred shortly after delivery. Whether the depression affects oral biota transmission and acquisition is questionable. Our hypothesis is that oral bacterial biota, including the cariogenic flora, is established early in the lifetime of the infant and can influence lifelong susceptibility to caries. If the mother is the main source of early bacterial infection in her children, then the concentration, composition, and the dynamic changes of maternal oral bacterial during the pregnancy and childhood development warrant better understanding.

Effect of delivery on MS transmission

Although the mother was determined by genotyping to be the source of MS transmission, the same has not been observed with regard to fathers. We hypothesized that specific maternal attributes at delivery may contribute to an infant's susceptibility for selecting different indigenous biota that become colonized in the oral cavity. As a newborn acquires the maternal immunoglobulins through the placenta during prenatal development, and the

immune defense system is reinforced by extensive exposure to maternal microorganisms through the birth canal at birth, it is reasonable to speculate that the close similarity in immunological components between the mother and the infant would promote a greater degree of fidelity for oral indigenous biota transferring from the mother to her infant. Thus, it was hypothesized that birth by cesarean section would have an impact on an infant's oral biota development.

To test this hypothesis, as a part of the SCRC study, we followed 41 African American mother–child pairs from the third trimester of pregnancy until the children were 3 years old [54]. During this period, information concerning the infants was collected, including gender, birth status and birth weight, medical history of infectious diseases, and antibiotic use. Information on pregnancy, breast-feeding experience, MS level, antibiotic use, and caries status was also collected on the mothers. Oral bacterial samples were obtained by the same method as previously described. One interesting observation was that children born by cesarean section ($n = 6$) acquired MS significantly earlier and had higher levels than did vaginally delivered children ($n = 35$) [54]. The study also found that treatment with antibiotics for mothers at the delivery, or for children during their first year of life, significantly delayed the initial acquisition of MS by the children. The result suggests that maternal influence on MS acquisition and transmission begins at birth and may play a significant role in early establishment of oral biota in the African American children.

Effect of chlorhexidine treatment on MS transmission

Because dental caries is an infectious and transmissible disease, several clinical studies have been conducted to investigate whether antimicrobial treatment approaches could reduce levels of MS in mothers and thus moderate MS colonization in their children [6,30,50,56,57]. The objectives of these studies were to explore the possibility of breaking the infectious chain from mothers to children and, as a result, prevent dental caries. Chlorhexidine, fluoride, povidone iodine, and xylitol are among the chemotherapeutic agents that have been studied. Interestingly, most of the studies showed reductions in MS levels, but the effect on caries in children was marginal (0% to 40%) [55].

In a randomized clinical trial [56], we evaluated the effect of 10% chlorhexidine varnish (Chlorzoin) on maternal MS transmission, and subsequent caries experience of the children. Seventy-five African American mothers were randomly selected and assigned to either a treatment group ($n = 38$) or a control group ($n = 37$). Chlorhexidine or a placebo varnish was applied to the dentition of the mothers by a trained dental hygienist at a time when their first babies were 6 months old—the approximate time when the babies' first molars were emerging. Three more chlorhexidine applications were given to each subject at weekly intervals. Subsequently, a single application was given every 6 months after the first group of treatments

at 12, 18, 24, 30, and 36 months after delivery. The MS levels of both mothers and children were monitored at 3-month intervals. Repeated-measures analysis of variance and survival analyses have been used to compare the MS levels at each visit. As previously reported, the treatment group exhibited significant reduction in MS levels compared with the control group [30,56], and the MS levels in the treatment group remained significantly lower over the study period. This intervention, however, did not have a significant effect on MS colonization in the children or caries increment in either the mothers or the children. This finding was consistent with the results of a previous double-blind, placebo-controlled, randomized clinical trial of an iodine-NaF solution in an African American population [30] and with a clinical trial [57] that used 1% chlorhexidine/0.2% NaF in other populations. Several explanations can be offered for this lack of efficacy, including the time, frequency, and duration of applications and the type of chemotherapeutic agent. One study studies [58] speculated that antimicrobial treatments may be less effective in eliminating oral bacterial because of their inability to penetrate into the depths of subclinical lesions or pits and fissures, or because the children might have acquired MS before the intervention began, but at an undetectable level. In our Birmingham African American maternal cohort [52,54,56] many of the mothers had received high dosages of antibiotics during pregnancy for treatment of STD infection, which may have undermined the oral ecosystem via blood stream effect before the clinical trial. Therefore, antimicrobial treatment approaches to prevent MS transmission and caries should not just focus on a specific group of microorganisms. It is more important to understand the plaque microcommunity and the attributes that control oral ecological balance shifting from a caries-prone to a caries-free status. Simplified suppression of the entire biota, without acknowledging the overall effects on ecology, is unlikely to succeed.

Summary

In the current literature database, information on microbiological attributes to caries outcomes in African American populations is limited and scattered. Few reports have discussed MS infection and transmission from African American mothers to their children. During the past few years, the research group at the University of Alabama at Birmingham and the Jefferson County Public Health Department have done a series of extensive studies to systematically investigate the prevalence of MS colonization, the time of initial MS infection as defined as ''the window of infectivity,'' the source of MS transmission as defined as ''the fidelity of transmission,'' and the chemotherapeutic management of MS transmission and caries prevention. The objective of this report was to summarize the main significant findings generated during a period of 15 years of study. One limitation of the studies outlined in this article is that the research populations were predominately

African American families. The inclusion of white and other minorities would make the conclusions more generalizable to the US population as a whole. Nevertheless, the information presented in this report can serve as baseline knowledge for future studies of caries etiology in African American and other ethnic populations.

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