G Scientific Article

### Distribution and determinants of mutans streptococci among African-American children and association with selected variables

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### Abstract

Prevalence of mutans streptococci (MS) and the effect of selected variables, including the early childhood use of antibiotics on the oral colonization of MS, were studied among 353, 5- to 12-year-old African-American children using a cross-sectional study. MS prevalence was estimated using pooled plaque and stimulated saliva samples (spatula). Data on antibiotic use and other potential determinants of oral colonization of MS were obtained using a self-administered questionnaire. Antibiotic data were validated using health records of subjects. MS prevalence (92%, 95% Confidence Interval [CI] = 89-95%) was associated with dental caries (Odds Ratio [OR] = 2.5,95% CI = 1.3–6.2), age (Chi for trend = 4.3, P = 0.04), increased frequency of sweet consumption (Chi for trend = 5.1, P = 0.02), and increased number of teeth in the mouth (unit OR = 1.3, 95% CI = 1.1–1.6). Higher MS levels were associated with higher number of decayed teeth (P < 0.0001), and also, with having the mother as the primary caregiver during the second year of life (P = 0.02). Furthermore, children who took antibiotics during early childhood and those who lived in the same household with many others during the second year of life had a higher MS prevalence than those who did not (OR = 8.3, 95% CI = 2.0–35.0; unit OR = 1.5, 95% CI = 1.03-2.2, respectively). It is unclear why those exposed to antibiotics during the "window of infectivity" of MS exhibit a higher MS prevalence. Antibiotic-related oral ecological changes (i.e., lower levels of S. sanguis) and environmental changes (i.e., frequent exposure to sugar through most pediatric antibiotic preparations) may make the oral cavity more favorable for initial MS colonization. (Pediatr Dent 17:192-98, 1995)

ational as well as regional data indicate that the distribution of treated and untreated caries is not homogenous among population subgroups. African-Americans appear to be either at high risk for caries<sup>1</sup> or suffer the consequences of the disease most.<sup>1-3</sup> Therefore, we studied the epidemiology of mutans streptococci (MS), a group of organisms implicated in the etiology of caries, among African-American children.

African (black) children reportedly have a higher prevalence of MS<sup>4-7</sup> than U.S. Caucasian children of comparable ages.<sup>8</sup> Compared with Caucasian children, statistically significantly fewer African-American children acquire MS by age 3 years,<sup>9-10</sup> perhaps indicating that the age specific MS prevalence in African-American children is lower than in Caucasian children. One objective of the present study therefore, was to estimate the prevalence of detectable levels of MS among African-American children.

The other objective was to study the factors potentially associated with the oral colonization of MS. Antibiotic use during early childhood is a factor that may potentially be associated with oral MS colonization and MS levels. MS is susceptible to a number of antibiotics<sup>11, 12</sup> and the medical use of antibiotics can lower the levels of MS in the oral cavity.<sup>13, 14</sup> Furthermore, the long-term as well as sporadic use of antibiotics also reduce dental caries both in animals<sup>15–18</sup> and humans.<sup>19–</sup> <sup>26</sup> Since the initial oral colonization of MS has been shown to occur during a discrete "window of infectivity,"<sup>10</sup> we hypothesized a lower prevalence of MS among those who were given MS-susceptible antibiotics frequently within this window.

### Methods and materials

A cross-sectional epidemiological study of a group of low-income African-American children in an innercity elementary school in Birmingham, Alabama, was conducted during April and May of 1992. The procedures, possible discomforts or risks, as well as possible benefits were explained fully to the human subjects involved, and their informed consent was obtained according to the guidelines of the Institutional Review Board for Human Use at the University of Alabama at Birmingham and the Jefferson County Department of Health (JCDH) prior to the investigation.

Plague and saliva samples were collected from each subject and oral examinations were performed at the school with portable dental equipment. Plaque samples collected as described earlier<sup>27</sup> were plated directly on to one quadrant of a quarter plate, which was overfilled with mitis-salivarius-bacitracin agar (MSB).28 Samples taken from the two sides of the mouth were plated on two different quadrants of the same plate. Immediately after taking the plaque sample, a stimulated saliva sample was taken using the spatula method of Kohler and Bratthall.<sup>29</sup> Each side of the spatula was pressed against the agar surface of the other two quadrants of the same MSB plate. Agar plates were transferred to the lab within 2 hr of collection and processed as described before.<sup>10</sup> Identification of MS in each plate was confirmed using Gram staining and biochemical characteristics.<sup>30</sup> Semi-quantitation of MS was done using saliva samples obtained with the spatula according to the original procedure described by Kohler and Bratthall.<sup>29</sup> The reliability of MS enumeration was tested using 29 randomly selected plates read on two consecutive days.

Subsequent to bacteriological sampling, oral examination of each subject was performed by one of two experienced investigators from the Dental Bureau of the JCDH, using NIDR criteria. A pilot-tested, selfadministered questionnaire was sent home with each child for the mothers to complete and return to the teacher. This questionnaire asked about the child's use of antibiotics since birth. It also included questions on basic demographic factors, the number of family members, breast and night feeding habits, care-giver his-

# TABLE 1. COMPARISON OF AGE (YRS) AND GENDER BETWEEN PARTICIPANTS AND NONPARTICIPANTS AT DIFFERENT STAGES OF THE STUDY

Stage	Participants	Nonparticipants	P•
Bacteriological data			
N	353	113	
Age: mean (SD)	8.2 (1.6)	8.3 (1.7)	0.72
Gender: F/M	159/194	49/64	0.83
Dental data			
N	295	171	
Age: mean (SD)	8.2 (1.6)	8.2 (1.6)	0.94
Gender: F/M	139/156	69/102	0.18
<b>Ouestionnaire</b> data			
N	236	230	
Age: mean (SD)	8.0 (1.5)	8.4 (1.7)	0.005
Gender: F/M	110/126	98/132	0.40
Medical record data	a		
N	126	340	
Age: mean (SD)	8.2 (1.5)	8.2 (1.6)	0.91
Gender: F/M	60/66	148/192	0.50
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• For age - Student's t-test, For gender - Fisher's exact test.

tory, sweet consumption patterns, source of drinking water, approximate age at first tooth emergence, dental care, and general health.

Medical, dental, and emergency records of children of consenting mothers were reviewed by one investigator (APD), and detailed antibiotic prescription information was abstracted for each year of life. The validity of the mother's recall of her child's antibiotic exposure was tested against the prescription data obtained from health records. Since antibiotic information based on maternal recall was found to be considerably underestimated, data obtained from the health records were used in the analysis.

#### Statistical analysis

The reliability of detection of MS between examiners was tested using concordance rates and the Kappa statistic,<sup>31</sup> and within examiners, using the correlation coefficients. Both univariate and multivariable logistic regression analysis techniques were used to evaluate the association of the early childhood use of antibiotics and other factors with the presence and levels of MS and caries. Goodness of fit of the models was tested using the methods described by Hosmer and Lemeshow.<sup>32</sup> For all statistical tests, two-sided type I error probability  $\leq$ 5% was considered the level of significance.

### Results

The number of participants and nonparticipants (from the target population of 466 children in grades K–5) at each stage of the study and their age and gender distribution are given in Table 1. Although the subjects who provided questionnaire data were younger, subjects who participated in bacteriological and dental examinations and whose health records were reviewed did not differ from the nonparticipants with respect to age and gender.

MS detection between two examiners who initially read plates yielded 100% agreement. There was a high correlation (r = 0.98, P < 0.0001) between the first (mean = 17.5, SD = 23.8) and the second MS count (mean = 17.3, SD = 24.2) obtained from the same plate by one investigator on two consecutive days indicating a high reproducibility of MS counts.

### Prevalence of MS

The observed prevalence of detectable MS levels was 92% (95% CI = 89–95%). Among those with detectable MS levels, 19% had salivary MS levels corresponding with more than 10<sup>6</sup> CFU per ml saliva (> 100 CFU per 1.5 cm<sup>2</sup> of spatula impression); 33% had levels corresponding with 10<sup>5</sup>–10<sup>6</sup> CFU; and 48%, 1–10<sup>5</sup> CFU. MS prevalence was not statistically significantly different between males (92%) and females (91%). Except for the 5- to 6-year-old children who had a prevalence of 77%, all the older age groups showed a prevalence of more than 90%. This positive trend between MS prevalence and age was statistically significant (P = 0.04; Table 2).

## TABLE 2. PREVALENCE OF MUTANS STREPTOCOCCI BY AGE AND GENDER AMONG AFRICAN-AMERICAN CHILDREN

Variable	N	Prevalence (%)	95% Confidence Interval (%)
Gender•			
Male	194	92	89.0–96.0
Female	159	<del>9</del> 1	87.0-96.0
Age group (yrs) <sup>†</sup>			
56	30	77	62.0-92.0
6–7	65	91	84.0-98.0
7–8	74	97	97.2-97.4
8+	184	92	88.0-96.0
Total	353	92	89.0–95.0

• Fisher's exact test, P = 0.85.

<sup>+</sup> Chi<sub>(1)</sub> for trend = 4.3, P = 0.04.

TABLE 3. FACTORS ASSOCIATED WITH MUTANS STREPT-OCOCCI PREVALENCE IN AFRICAN-AMERICAN CHILDREN Odds 95% Confidence Variable Ratio Interval Ever use of antibiotics 8.3 2.0-35.0 Antibiotic use at year 2 1.1-∞• ~ Sweet consumption frequency/day<sup>‡</sup> At least 3 times 4.9 1.3-18.0 More than 3 times 6.8 1.3-36.0 Number of people who  $1.5^{+}$ 1.03-2.2 lived with child (0–2 yrs) Total teeth present  $1.4^{+}$ 1.1 - 1.6Total decayed teeth 1.3<sup>†</sup> 1.1 - 1.9

\* Exact Confidence Interval.

<sup>+</sup> Unit Odds Ratio.

\* Chi for trend = 5.1, P = 0.02.

### Association of antibiotic use with MS and caries

The review of medical records indicated that only 70% of the subjects had ever been prescribed antibiotics. The number of prescriptions given since birth ranged from 1 to 16 with a mean of 3.3 (SD = 3.3) and a median of 2. The most frequent illnesses for which antibiotics were prescribed were either unilateral or bilateral otitis media (58%). Antibiotic prescriptions also were given for upper respiratory track infections (24%), impetigo (10%), infected wounds and abscesses (2%), and less frequent conditions such as shigellosis, urinary tract infections, oral ulcers, burns, and antibiotic prophylaxis. The type of antibiotic prescribed also showed a wide variation (48% amoxyl, 12% bactrim, 12% penicillin, 9% erythromycin, etc.). An increased prevalence of MS was observed among those who had been given antibiotics compared with the group who had never been prescribed antibiotics (OR = 8.3, 95% CI = 2.0–35.0). Further analysis indicated that this association was actually confined to the antibiotic prescriptions given during the second year of life (Table 3). The number of prescriptions given over a lifetime was also significantly associated with MS prevalence (P = 0.002). The children who received antibiotics had more decayed teeth than those who did not, even though this association did not reach statistical significance (P = 0.07).

### Association between MS and dental caries

There was a statistically significant positive association between MS prevalence and caries in the primary dentition (OR = 2.8, 95% CI = 1.3-6.2) as well as caries in either dentition (OR = 2.8, 95% CI = 1.3-6.1). However, the positive association between caries in the permanent teeth and MS prevalence was not statistically significant (OR = 1.4, 95% CI = 0.5-3.9). Increasing caries levels in each dentition generally was associated with increasing salivary MS levels (Table 4). When this association was evaluated within each component of the caries experience, we observed the following results. The positive trend between the salivary MS levels and the number of untreated carious teeth per subject was significant (Chi = 31.6, P < 0.0001), but there was no statistically significant association between the number or the percentage of filled teeth and the salivary MS levels.

### Other factors associated with MS

Frequency of child's sweet consumption as reported by the mother also was positively associated with MS prevalence (Table 3). Compared with 5% of the children who did not eat sweets frequently, prevalence odds ratio among those who eat sweets at least three times a day was five times significantly higher, and in those who eat sweets more than three times a day, seven times significantly higher. The MS levels in saliva also showed a significant positive trend with frequency of sweet consumption (Chi for trend = 7.2, P = 0.007).

TABLE 4. ASSOCIATION BETWEEN SALIVARY LEVELS         OF MUTANS STREPTOCOCCI AND DENTAL CARIES         IN AFRICAN-AMERICAN CHILDREN <sup>48</sup>							
Salivary MS Levels (cfu/1.5cm²)	DMFT (mean)•	DMFS (mean) <sup>†</sup>	dft (mean)‡	dfs (mean) <sup>§</sup>			
0	0.19	0.19	0.89	1.63			
1–20	0.30	0.41	1.36	2.29			
21-100	0.62	0.80	2.63	4.80			
100+	0.81	1.11	2.60	4.65			

• P = 0.006.

 $^{+}$  P = 0.01.

P = 0.0001.

§ P = 0.0002 (ANOVA).

The number of people who lived in the same household with the child during the first two years of life and the total number of teeth and decayed teeth in the child's mouth at the time of examination also were significantly associated with MS prevalence (Table 3). There was a statistically significant positive association between the percentage of children whose mothers were the primary caregiver during the first 2 years of life and the current salivary MS levels of children (P = 0.02).

Antibiotic use during the second year of the life (OR  $= \infty$ ) and the number of people who lived with the child during the first two years (OR = 2.03,95% CI = 1.02-4.0) remained significant in the multivariable logistic regression model, which contained the significant variables that emerged from the univariate analysis.

### Discussion

Streptococcus mutans has been the subject of a large number of studies resulting in the publication of more than 2,000 MS-related articles between 1966–94. About 40% of these studies are related to human subjects. While most of these studies are on the microbiological aspects of MS, properly designed, conducted, and appropriately analyzed epidemiological studies among African-American populations are lacking in the literature. Here we present results from one such study.

In our cross-sectional epidemiological investigation of MS we obtained data on potential determinants of initial oral colonization of MS and restricted this information to time periods corresponding with the initial oral colonization of MS using relatively valid methods such as health records. While observing known associations for MS prevalence, we also observed two other associations that are inconsistent with the literature, namely the increased prevalence of MS among those who were given antibiotics and those who lived with many other household members during the second year of life (compared with those who did not). Since the above associations are based on only a subsample of the target population, we need to assess the possibility of selection bias. Selection bias occurs whenever the study participation is influenced by some individual characteristics related to the outcome of the study<sup>33</sup> (i.e., nonparticipants are all sick children). One way of assessing selection bias is to examine the comparability between participants and nonparticipants. In our study these two groups did not differ in gender or age except for the fact that children of the questionnaire respondents were slightly younger. This does not necessarily mean that the mothers of older children refused to provide questionnaire data, but may show a lower compliance by older children in taking the questionnaire home and returning it completed.

The two groups also were comparable with respect to socioeconomic status, since 99% of all children participated in the school free lunch program, which was based on low family income.

Information bias involving data on antibiotic use and the number of household members also can be ruled out as possible explanations for our results. At the time the questionnaire was given, neither the investigator who gathered antibiotic information from health records nor the mother was aware of the MS status of the subject. Therefore, differential misclassification of the above information (i.e., being more careful in reporting antibiotic and other information for those with MS) is unlikely. If there were errors in classifying this information, it should be nondifferential (i.e., errors in antibiotic and other information is equal for those with and without MS), a situation that would bias the odds ratios toward the null value of one. If that would have been the case, the real associations of the antibiotic use and the number of household members with MS prevalence should be even greater than observed here. Furthermore, if our data gathering procedures were subjected to considerable biases, our data would not reveal the known associations between MS and other factors.

In our study, based on either plaque or saliva samples, the prevalence of detectable MS levels among 5- to 12-year-old African-American children was estimated to be 92%. Using unstimulated saliva samples obtained from a group of U.S. Caucasian school children (10-15 years) and grown on MSB, Kingman et al.,8 reported an MS prevalence of 64%. For comparison purposes, if we based our MS detection only on saliva samples (even though we used stimulated saliva) our prevalence estimate still would be 92%. Kingman's estimate of 64% may be an underestimate since we already have shown that unstimulated saliva underestimates MS.9 In Kingman et al., study, 822% of the subjects had high levels of MS (>10<sup>5</sup> CFU/mL) as opposed to 52% in our study. These differences also may be partly due to methodological differences since stimulated saliva samples also yield higher levels of MS.9

Using the spatula method<sup>29</sup> and MSB, Carlsson et al.,<sup>4-5</sup> and Tayeb et al.,<sup>7</sup> reported a high prevalence of MS (96-98%) for children ages 10–14 years in a few African countries. According to these investigators, 40–45% of all children had MS levels > 100 CFU per 1.5 cm<sup>2</sup> of the spatula impression. The corresponding figure (based on 353 children) in our study was only 17%. These differences could be partly due to the younger age of our study subjects.

Age was associated significantly with MS prevalence in our study children between 5–8 years of age. The fact that the MS prevalence did not increase significantly with age after 8 years is consistent with the notion of "window of infectivity". Our earlier observations<sup>10</sup> and data from both cross-sectional and longitudinal studies of other investigators<sup>34–36</sup> indicate a rapid rise of MS prevalence with age among younger children, yielding an "S-shaped" curve for the plot between MS prevalence and age. We observe this distribution in our data.

We did not see an association between MS preva-

lence and gender, which also was consistent with the literature.<sup>37-38</sup> Sugar consumption and the number of teeth have earlier been shown to be significantly correlated with MS colonization,<sup>39</sup> and we also saw these associations in our data. The association we observed between the MS colonization in children and the mother being the primary caregiver is consistent with an earlier observation we made in a longitudinal study conducted in another predominantly African-American population.<sup>10</sup> The association between presence of MS and dental caries in our study was positive and significant (OR = 2.8, 95% CI = 1.3-6.2) and consistent with the observations made by the others. Brunelle et al.,<sup>40</sup> studied the MS prevalence in a larger sample of more than 1,000 children aged 5-17 years and reported similar results (OR = 2.2, 95% CI = 1.3–2.9). Perhaps due to the younger age of our study subjects, the positive association between the caries in permanent teeth and MS prevalence was not statistically significant in our study. Yet our data suggest a significant positive trend between MS levels and the number of untreated carious teeth. These findings also are consistent with the literature.41-42 The fact that we did not fail to observe these known associations in similar magnitudes and directions as reported earlier further illustrates the validity of our data gathering procedures.

Having examined the validity of our data, we need to provide probable explanations for our major findings. Loesche et al.,13 demonstrated that the fissure plaque of newly erupted first molars of children 6-7 years old who never received antibiotics had significantly higher proportions of MS (20% of the cultivable flora) compared with subjects who received antibiotics (13-14% of the cultivable counts). On the contrary, in our study we observed a higher prevalence of MS among those who were given antibiotics. By quantifying MS as a percentage of cultivable counts, Loesche et al.,<sup>13</sup> make direct inferences of their results difficult, because changes in these percentages may reflect either the changes in the denominator, the numerator, or both. Nevertheless, they did not observe an obvious effect of frequency of antibiotic usage upon the percentage of MS, while we observed a trend of higher MS prevalence with increasing number of antibiotic prescriptions (given during the second year of life as well as over a lifetime). Loesche et al.,<sup>13</sup> also failed to observe antibiotic-related changes in the proportions of the other organisms monitored, even though the same antibiotics that lower MS levels are shown to lower the levels of some other organisms they measured, such as lactobacilli.43 We could not evaluate this phenomenon in our study since we did not monitor the other oral bacteria. Failure to observe a dose-response effect of antibiotics on the percentage of MS and any effect on the other susceptible organisms alone would undermine the credibility of the associations observed by Loesche et al.<sup>13</sup> One possible explanation for this would be the poor quality of the antibiotic information in their study which was based on parental recall. This apparent limitation has been acknowledged by the authors. We have shown that the mother's recall of her child's antibiotic use during early childhood often is considerably underestimated.<sup>9</sup> In our study we used information obtained from health records, even though it's possible that the antibiotic information recorded on health records does not reflect the actual use due to noncompliance in purchasing or completing the prescriptions. Since more than 90% of our study subjects attended county health department clinics where most patients were covered by Medicaid, the proportion of unfilled prescriptions can be reasonably assumed to be very low.

Our observation of increased MS prevalence in those with increased exposure to antimicrobials was not without some precedent. In an intervention trial in which we treated the mothers' dentitions with an NaF-Iodine solution hoping to reduce MS transmission from mother to child,<sup>44</sup> treatment-group children experienced earlier colonization and higher incidence of MS and had more caries than the children in the control group, though these differences were not statistically significant, perhaps due to small study size. In addition, a group of children who received topical solutions of kanamycin also has been reported to show a shortterm increase in caries.<sup>45</sup>

We offer the following explanations for the association between antibiotic use during early childhood and higher MS prevalence. Organisms such as *S. sanguis* are considered to compete with MS for the same ecological niches. In a previous study, we observed that the initial oral colonization of *S. sanguis* occurs at a discrete time period (around 12 months after birth) prior to initial oral colonization of MS.<sup>10</sup> Perhaps suppression of the levels of those competing organisms by the antibiotics given during the second year of life make the oral cavity more favorable for the initial colonization of MS.

Another possible explanation is an underlying association between sugar and MS colonization. Most pediatric suspensions contain sugars<sup>46</sup> which come in direct contact with teeth frequently as children repeatedly take antibiotic preparations for illnesses such as recurrent otitis media. Due to the tenacity of these preparations, these sugars can remain on teeth for prolonged time periods. The majority of our subjects were repeatedly given amoxil or a derivative in suspension form as a treatment for bilateral or unilateral otitis media for periods up to a month at a time. The drug literature indicates that these preparations contain sucrose as a preservative. Roberts and Roberts<sup>47</sup> showed that children who received sucrose-based medicines had significantly more caries and gingivitis. In our study, we observed that the antibiotic use was positively associated with decayed component of the caries experience, even though it did not reach statistical significance (P = 0.07). In contrast, statistically significantly lower caries levels at ages 3 and 4 years have been reported in a

group of adenoidectomized children who received twice the amount of sucrose containing syrup medications and twice the amount of antimicrobial syrup medications compared with a control group.<sup>26</sup> However, after 4 years of age, the lower caries levels in the treatment-group children were followed by an accelerated formation of new caries lesions. Based on all these, it can be argued that the increased MS prevalence among those who are exposed to antibiotics at an earlier age may be due to sugar in the antibiotic preparations. However, this argument is somewhat weakened due to the fact that we did not have data on actual "sugar hit" from medications. Our inability to control for the amount of sugar in these suspensions in evaluating the MS-antibiotic association is a limitation of our study that should be overcome in future studies.

The children who became sick more often and hence had higher frequency of antibiotic use also may have had poorer dental and general health practices. However, we did not see an association between the frequency of brushing or the use of mouth washes and the number of antibiotic prescriptions given, thus minimizing this possibility. Another explanation would be that the children who had frequent antibiotics represent a group of sicker children who may be immunologically different from the others. We did not have enough data to either support or refute this notion.

Our data also support the notion that the increased exposure within the family unit increases MS prevalence. In a similar cross-sectional study conducted among younger children (3-24 months), Stiles et al.48 failed to observe an association between the increased number of the older children in the household and MS prevalence in the mouth and feces of children. However, reanalysis of their data presented in Table 4 indicates an association in the same direction as observed in our study, though it does not reach statistical significance (OR = 1.3, 95% CI = 0.6–2.8). One explanation for the disparity of results between our study and Stiles et al., study48 is the difference in ages of the study subjects. MS prevalence in children younger than 24 months of age is very low (21%) compared with the prevalence (92%) we observed in our study. Another difference is that Stiles et al.48 restricted the household number only to older siblings. Since the possibility exists that family members other than siblings also may transmit MS, we obtained information on all household members and restricted this information to an etiologically meaningful time period (12-24 months).

### Conclusions

- 1. In our study of the epidemiology of MS in a group of inner-city, low-income African-American children, we observed a high prevalence of detectable MS levels.
- 2. Prevalence as well as the MS levels appear to be age related but not gender related.
- 3. Children who took antibiotics and lived with

many household members during a time period when the child was at high risk for initial MS colonization exhibited a higher prevalence of detectable MS levels.

4. Among the other factors significantly associated with the presence of detectable levels of MS in the mouth of children were the frequency of sweet consumption, the number of teeth present in the mouth, amount of caries, and having the mother as the primary caregiver during the second year of life.

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- 1. Davis AC, Lockwood SA: Dental disease prevalence in Alabama school aged children. J Alabama Dent Assoc 75:18–30, 1991.
- 2. US Department of Health and Human Services, National Institute of Dental Research: Oral health of United States children. The national survey of dental caries in school children 1986–1987. DHHS Pub NO (PHS) 85–2247, Bethesda, MD, 1989.
- 3. US Department of Health and Human Services, National Health and Nutrition Examination Survey, National Center for Health Statistics, Hyattsville, MD, 1987.
- Carlsson P, Olsson B, Bratthall D: The relationship between the bacterium Streptococcus mutans in saliva and dental caries in children in Mozambique. Arch Oral Biol 30:265– 68, 1985.
- Carlsson P, Gandour IA, Olsson B, Rickardsson B, Abbas K: High prevalence of mutans streptococci in a population with extremely low prevalence of dental caries. Oral Microbiol Immunol 2:121–24, 1987.
- Carlsson P: Distribution of mutans streptococci in populations with different levels of sugar consumption. Scand J Dent Res 97:120–25, 1989.
- 7. El Tayeb IY, Bratthall D, Carlsson P: Dental caries and Streptococcus mutans in Sudanese children. Odontostomatol Trop 8:77–80, 1985.
- Kingman A, Little W, Gomez I, Heifetz SB, Driscoll WS, Sheats R, Supan P: Salivary levels of Streptococcus mutans and lactobacilli and dental caries experience in a US adolescent population. Community Dent Oral Epidemiol 16:98– 103, 1988.
- 9. Dasanayake AP: Epidemiology of mutans streptococci: validity of detection, prevalence, and its association with the use of antibiotics among African-American children, and the maternal transmission (dissertation). Birmingham (AL), University of Alabama at Birmingham, 1993.
- 10. Caufield PW, Cutter GR, Dasanayake AP: Initial acquisition of mutans streptococci by infants: evidence for a discrete window of infectivity. J Dent Res 72:37–45, 1993.
- Baker CN, Thornsberry C: Antimicrobial susceptibility of Streptococcus mutans isolated from patients with endocarditis. Antimicrob Agents Chemother 5:268–71, 1974.
- 12. Ferretti JJ, Ward M: Susceptibility of Streptococcus mutans

to antimicrobial agents. Antimicrob Agents Chemother 10:274-76, 1976.

- Loesche WJ, Eklund SA, Mehlisch DF, Burt B: Possible effect of medically administered antibiotics on the mutans streptococci: implications for reduction in decay. Oral Microbiol Immunol 4:77–81, 1989.
- Staves E, Tinanoff N: Decline in salivary S. mutans levels in children who received short-term antibiotic therapy. Pediatr Dent 13:176–78, 1991.
- McClure FJ, Hewitt WL: Relation of penicillin to induced rat caries and oral L. acidophilus. J Dent Res 52:441–43, 1946.
- Zander HA, Bibby BG: Laboratory and animal studies on the effect of penicillin on caries activity. J Dent Res 26:454– 55, 1947.
- Stephan RM, Fitzgerald RJ, McClure FT, Harris MR, Jordan H: Comparative effects of penicillin, bacitracin, chloromycin, aureomycin and streptomycin on experimental dental caries and on certain oral bacteria in the rat. J Dent Res 31:421– 27, 1952.
- Rosen S, Ragheb HS, Hoppert CA, Hunt HR: Effects of penicillin and Terramycin on dental caries and certain oral microflora in Hunt-Hoppert caries susceptible rats. J Dent Res 35:399-403, 1955.
- 19. Hill JT, Kniesner AH: Penicillin dentifrice and dental caries experience in children. J Dent Res 28:263–66, 1949.
- Zander HA: Effect of penicillin dentifrice on caries incidence in school children. J Am Dent Assoc 40:569–74, 1950.
- Littleton NW, White CL: Dental findings from a preliminary study of children receiving extended antibiotic therapy. J Am Dent Assoc 68:520–25, 1964.
- 22. Handelman SL, Mills JR, Hawes RR: Caries incidence in subjects receiving long term antibiotic therapy. J Oral Ther 2:338–45, 1966.
- 23. Brearley LJ, Porteous JR: Characteristics and caries experience of tetracycline affected dentition. J Dent Res 52:508– 16,1973.
- 24. Weld HG, Sandham HJ: Effect of long term antibiotic therapy with penicillin and sulfadiazine on Streptococcus mutans. Antimicrob Agents Chemother 10:200–4, 1974.
- 25. Robertson PB, Wright TE 3d, Mackler BF, Lenertz DM, Levy BM: Periodontal status of patients with abnormalities of the immune system. J Periodontal Res 13:37–45, 1978.
- Karjalainen S, Rekola M, Ståhlberg MR: Long-term effects of syrup medications for recurrent otitis media on the dental health of 6- to 8-year-old children. Caries Res 26:310–14, 1992.
- Kristoffersson K, Bratthall D: Transient reduction of Streptococcus mutans interdentally by chlorhexidine gel. Scand J Dent Res 90:417–22, 1982.
- Gold OG, Jordon HV, Van Houte JV: A selective medium for Streptococcus mutans. Arch Oral Biol 18:1357–64, 1973.
- 29. Köhler B, Bratthall D: Intrafamilial levels of Streptococcus mutans and some aspects of the bacterial transmission. Scand J Dent Res 86:35–42, 1978.
- 30. Shklair IL, Keene HJ: A biochemical scheme for the separation of the five varieties of Streptococcus mutans. Arch Oral Biol 19:1079–81, 1974.

- Cohen J: A coefficient of agreement for nominal scales. Educ Psychol Measure 20:37–46, 1960.
- 32. Hosmer DW, Taber S, Lemeshow S: The importance of assessing the fit of logistic regression models: a case study. Am J Publ Health 81:1630–35, 1991.
- Greenland S: Response and follow-up bias in cohort studies. Am J Epidemiol 106:184–87, 1977.
- Carlsson J, Grahnén H, Jonsson G: Lactobacilli and streptococci in the mouth of children. Caries Res 9:333–39,1975.
- 35. Köhler B, Andréen I, Jonsson B: The effect of caries preventive measures in mothers on dental caries and oral presence of the bacteria Streptococcus mutans and lactobacilli in their children. Arch Oral Biol 29:879–83, 1984.
- 36. Masuda N, Sobue S, Hamada S: Longitudinal survey of the distribution of various serotypes of Streptococcus mutans in infants. J Clin Microbiol 10:497–502, 1979.
- 37. Klock B, Krasse B: Microbial and salivary conditions of 9- to 12-year-old children. Scand J Dent Res 85:56–63, 1977.
- Kohler B, Bjarnason S: Mutans streptococci, lactobacilli, and caries prevalence in 11- and 12-year-old Icelandic children. Community Dent Oral Epidemiol 15:332–35, 1987.
- Dähllof G, Grinderfjord M, Ekstrom G, Höjer B, Modéer T: Prevalence of mutans streptococci in 2.5 year old children. Swed Dent J 15:44, 1991.
- Brunelle JA, Little WA, Stiles HM: Comparison of mutans counts from three media and dental caries susceptibility. J Dent Res 71:129, 1992.
- Granath L, Cleaton–Jones P, Fatti LP, Grossman ES: Prevalence of dental caries in 4- to 5-year-old children partly explained by presence of salivary mutans streptococci. J Clin Microbiol 31:66–70, 1993.
- 42. Shi Y, Barmes D, Bratthall D, Leclercq MH: WHO pathfinder caries survey in Beijing extended with data for prevalence of mutans streptococci. Int Dent J 42:31–36, 1992.
- White BJ, Kniesner AH, Hill JT: Effect of small amounts of penicillin on the oral bacterial flora. J Dent Res 48:267–71, 1949.
- 44. Dasanayake AP, Caufield PW, Cutter GR, Stiles HM: Transmission of mutans streptococci to infants following short term application of iodine–NaF solution to mothers' dentition. Community Dent Oral Epidemiol 21:136–42, 1993.
- 45. Loesche WJ, Bradbury DR, Woolfolk MP: Reduction of dental decay in rampant caries individuals following short term kanamycin treatment. J Dent Res 56:254–65, 1977.
- Johnston DI: Sugar free medicines Are you using them? Arch Dis Child 61:216–17, 1986.
- Roberts IF, Roberts GJ: Relation between medicines sweetened with sucrose and dental disease. Br Med J 2:14–16, 1979.
- 48. Stiles HM, Meyers R, Brunelle JA, Wittig AB: Occurrence of Streptococci mutans and Streptococci sanguis in the oral cavity and feces of young children. In: Proceedings, Microbial Aspects of Dental Caries. Stiles HM, Loesche WJ, O'Brien TC, eds. Vol. 1. Sp. Supp., Microbiology Abstracts. New York: Information Retrieval Inc., 187–99, 1976.