Caries experience and cariogenic markers in HIV-positive children and their siblings

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Abstract

The purpose of this cross-sectional, masked study was to compare the oral status of perinatally HIV-infected children with their uninfected siblings living in the same environment. A secondary purpose was to compare HIV-positive children for differences in oral health with respect to disease advancement. One hundred forty-seven children were examined in their homes and meeting places, using NIH criteria for caries diagnosis. Significant differences were found in the number of caries-free children ($P < 0.05$), past caries experience ($P < 0.003$), subsurface demineralizations ($P < 0.0001$), and caries-related bacteria ($P < 0.05$). However, differences in caries prevalence were not found in the 3- to 6-year-old subgroup. Caries prevalence ($P < 0.001$) and levels of caries-related flora in saliva were correlated to years since diagnosis (mutans streptococci $P < 0.008$, lactobacilli $P < 0.02$). Children with a more advanced disease stage had significantly more caries ($P < 0.02$). Among the HIV-infected children, the frequency of carbohydrate intake was clearly correlated to caries ($P < 0.003$) and to lactobacilli levels ($P < 0.0001$). It is concluded that children with perinatally acquired HIV are at greater risk for caries than their siblings, more so with advancing disease. (Pediatr Dent 18:129–36,1996)

Reports concerning the oral pathoses observed in pediatric HIV infection are steadily accumulating in the literature.[1–12] Caries has been reported by several investigators.[2, 3, 6–8, 11, 12] Schiødt[2] reported rampant caries associated with salivary gland disease. Leggott et al.[3] presented case reports that suggested caries prevalence may be elevated in HIV-infected children. Both Howell[6] and Valdez et al.[11] reported caries as the most common oral disease. In each of these two studies, children of all ages, routes of transmission, age at transmission, and disease stage were included in the study. All children were HIV-positive, and these earlier studies have not been masked. Until now, the oral disease observed has not been compared to uninfected children born into the same environment where disease rates may be higher in general. The majority of children born to HIV-positive mothers in the greater New York area are born into extreme poverty.[13–16] With this poverty follow many factors, such as malnutrition and stress, which may predispose even uninfected children to poor health, including poor oral health and poor immune function.[17] In this masked, cross-sectional study, we examined 147 children in households where children with perinatally acquired HIV were being raised. The oral health of the HIV-positive children along with their eating and medication patterns were then compared with the HIV-negative siblings. The differences in oral findings in age subgroups were examined as well as the differences within the group of HIV-positive children at different stages of disease advancement. In this paper, findings of caries, white spot lesions, lactobacilli and mutans streptococci levels in saliva, and the frequency of carbohydrate consumption are reported.

Methods and materials

Population

The population comprised 147 children recruited through local support and network organizations for women and families affected by HIV. Only children with perinatally acquired HIV and their siblings were included for study. Ninety of the examined children were HIV positive, 57 were HIV negative. Sample size for populations with a high prevalence of oral disease should be 40–50 subjects.[18] Additional HIV-positive subjects were examined to allow for intragroup nonparametric comparisons.[19] This sample size allows for a level of significance of 5% and 90% power.

All children had been born and raised in Newark area communities, which are unfluoridated with low natural fluoride levels in the water supplies. Children whose HIV status was indeterminate or undetermined were excluded from this study. This study was approved by an internal review board at the University of Medicine and Dentistry of New Jersey-New Jersey Dental School.

Examination

The children were examined in their homes or at meetings and social gatherings on mornings during summer vacation, at least 1 hour after breakfast or morning snack. The study was carefully explained to the caretakers. Guardians were also requested to give written permission for the examiner to access medical information about the child. The examiner did not inquire about the health status of the child until after the examination was completed; however, the study could not be completely masked because the health status of the child was occasionally obvious or information was incidentally volunteered. One trained and calibrated examiner (A.M.) conducted all examinations. Inter-rater reliability was determined prior to beginning the study by comparison with another experienced and previously calibrated examiner (P.A.M.). Training included several didactic sessions reviewing standard diagnostic criteria, and clinical examinations of 10 children. Intrarater reliability was determined by re-examination of nine patients without the examiner's prior knowledge during the study.

The samples for cultivation of mutans streptococci were collected using the method described by Jensen and Bratthall.[20–22] Sterile paraffin is masticated to displace plaque into saliva. Residual pooled saliva in the mouth is then used to inoculate a smooth plastic strip, which is suspended in selective media (Dentocult SM Strip™, Orion Diagnostica, Espoo, Finland). Mycotic contamination, which was assumed to be a potential problem,[23] cannot adhere to the smooth vertical surface.[24, 25]

The cultures for oral lactobacilli were collected as described by Larmas.[26] Expectorated saliva was used to inoculate dip slides coated with selective media (Dentocult-LB™, Orion Diagnostica, Espoo, Finland). The coded samples were transported to the laboratory at the University of Medicine and Dentistry of New Jersey for immediate incubation in air at 37° C. Mutans cultures were evaluated after 48 hr and lactobacilli cultures were evaluated after 4 days by a laboratory technician who had no knowledge of the children's health or HIV status.

The children were examined for caries by placing the child's head on a patient bib on the examiner's lap, using a number 23 explorer and mouth mirror and high intensity light (Mag Instrument™, Ontario, Canada). Small children lay down and larger children sat up, as described by Westphal.[27, 28] The maxillary arch was examined, the child was asked to swallow, and then the mandibular arch was examined. When a drier field was needed, sterile cotton rolls were available, although not routinely used. The diagnostic criteria for manifest caries were those of the U.S. National Institutes for Health (NIH).[29]

In addition to the diagnosis of manifest — cavitated carious lesions — a separate record was also kept of white spot lesions of those smooth surfaces that could be seen. Because of diagnostic difficulties, incipient lesions of fissures were not registered. The following criteria were used to diagnose smooth surface white spot lesions (subsurface demineralizations): 1) rough enamel surface, 2) enamel that had lost its translucency and/or was judged by the examiner to be an active lesion as indicated by its chalky color, and 3) the lesion's location, (i.e. the lesion was still in a plaque retention area, and the lesion did not fulfill NIH criteria for manifest caries).[30, 31] White spot lesions were recorded as a separate subcategory of "normal", and not reported in either the DMFT/deft or DMFS/defs analyses. This was to enable comparison to other reports on caries that use NIH diagnostic criteria.

Caretaker interview

After the child was examined, the caretakers were interviewed. The child's health status, health history, including prescribed medication regimens, and a 24-hr diet and medication recall were recorded. The examination and interview forms and microbial samples were coded for data entry and analysis without use of names or other patient identification. Each child received a treatment urgency rating similar to that used in the Arizona Pathfinder Survey,[32] which was explained to the caretaker. Each caretaker was provided oral and written information about available oral health care facilities for their children. All were encouraged to seek regular preventive services.

Data analysis

Ages of the participants in all subgroups were compared using a *t*-test procedure. Chi-square analysis and Fisher's exact test were used to examine race and sex differences. The microbial colony forming units (CFU) counts were considered to be ranks rather than a numerical value. Differences in CFU distributions of HIV-positive versus HIV-negative groups were examined with the Wilcoxon rank sum test. Caries findings did not conform to a normal distribution and displayed large standard deviations. Caries distributions were therefore analyzed nonparametrically, using a Wilcoxon rank sum test. For correlation analysis, the Kendall *tau b* coefficient was used. Differences in the proportions of caries-free children were evaluated using Fisher's exact test.

Results

Rater-reliability

Inter-rater reliability was calculated to be 95%. Intrarater reliability for caries was 91%.

Demographic data

Ninety HIV-positive children participated in this study. In this group there were 45 males and 45 females; mean age was 5.1 years with a range of 0.3–14 years. Racial breakdown was 77 black, one white, and 12 Hispanic children. In the HIV-negative group there were 27 males and 30 females with a mean age of 6.2 years

(range 1–14 years). Racial breakdown was 53 black, no white, and four Hispanic children. There were no significant differences in the makeup of the HIV-positive or -negative groups as to age, race or sex, even when analyzed by age subgroups.

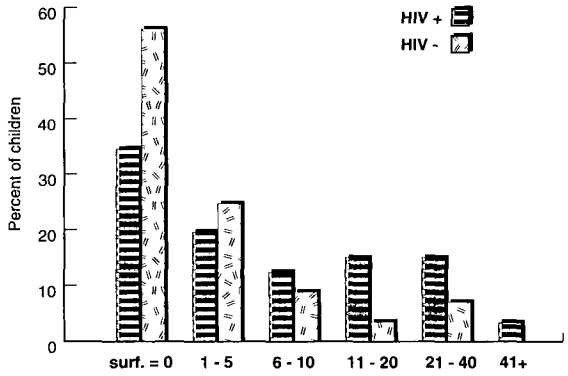

Figure. The distribution of DMFS/defs in HIV-positive (N = 89) and HIV-negative (N = 57) children. A significant difference existed.

Caries

The Figure shows the caries distribution in the HIV-positive and -negative groups. The HIV-positive children had a significantly different caries distribution with fewer caries-free surfaces and significantly greater number of carious surfaces (Wilcoxon rank sum test, $P = 0.003$). Table 1 compares caries in all dentate children, and in children older than 6, 3 to 6 years of age, and younger than 3. In general, there was a significantly higher prevalence of caries in the HIV-positive group. The higher caries prevalence was found in the primary dentitions of the older children (> 6 years). The preschool children (age 3–6 years) did not have significant differences in caries prevalence. Twenty-three dentate children who were younger than 3 (15 HIV-positive and eight HIV-negative) were examined for differences in caries prevalence using Fisher's exact test. None were found (Table 1).

There were also significant differences in the numbers of caries-free children as seen in Table 2. Again differences were due to the HIV-positive children in the older than 6 group. Only 6.7% of the HIV-positive children older than 6 were caries-free compared with 33% in the HIV-negative cohort.

Also notable is the significant difference in the primary dentition when the HIV-positive and HIV-negative groups were compared. The percentage of HIV-positive children in the older than 6 group who had a caries-free primary dentition was significantly lower in the HIV-negative group (11% versus 62.5%, $P < 0.05$).

In addition to examining the number of caries-free children and the caries prevalence, a separate analysis was performed to evaluate for nursing bottle caries patterns. All children with primary maxillary central incisors were included, and the prevalence of caries on the palatal surface of these teeth was calculated. Of 64 HIV-positive children, 19 (29.7%) had palatal caries. Of 29 HIV-negative children, only one (3.4%) had palatal caries of maxillary central incisors. This is a significant difference as determined using a Fisher's exact test ($P < 0.03$).

TABLE 1. CARIES IN ALL DENTATE CHILDREN, CHILDREN > 6 YEARS, AND CHILDREN 3–6 YEARS

	Status	*N*	*Mean*	*SD*	*Range*	*P*
DMFS/defs						
All children	HIV+	87	10.4	15.2	0–86	0.003*
	HIV-	57	4.4	8.9	0–37	
> 6 yrs	HIV+	30	15.7	14.0	0–60	0.004*
	HIV-	21	5.9	9.3	0–37	
3–6 yrs	HIV+	39	8.2	16.9	0–86	0.3
	HIV-	26	4.8	9.9	0–37	
DMFT/deft						
All children	HIV+	87	4.1	4.8	0–20	0.002*
	HIV-	57	2.0	3.4	0–12	
> 6 yrs	HIV+	30	6.4	4.3	0–16	0.003*
	HIV-	21	2.9	3.5	0–11	
3–6 yrs	HIV+	39	3.2	4.9	0–20	0.29
	HIV-	26	2.7	3.5	0–12	
DMFT						
All children	HIV+	40	0.98	1.4	0–6	0.7
	HIV-	33	1.0	1.6	0–5	
> 6 yrs	HIV+	30	1.3	1.5	0–6	0.5
	HIV-	21	1.6	1.7	0–5	
3–6 yrs	HIV+	9	0	0	0	0.16
	HIV-	12	1	3.9	0–1	
deft						
All children	HIV+	84	3.8	4.5	0–20	0.0005*
	HIV-	52	1.5	3.0	0–12	
> 6 yrs	HIV+	27	5.7	3.7	0–16	0.003*
	HIV-	16	1.7	2.8	0–8	
3–6 yrs	HIV+	39	3.2	4.9	0–20	0.25
	HIV-	26	2.0	3.6	0–12	

* Significant difference in mean lesions (*t*-test), significant difference in caries distribution (Wilcoxon rank sum test; $P < 0.05$).

Table 2. Number and percentage of caries-free children in the different dentitions and age groups

	< 3 Years			3 to 6 Years			> 6 Years			All Dentate Children		
	N	Caries-Free	%	N	Caries-Free	%	N	Caries-Free	%	N	Caries-Free	%
Entire dentition												
HIV+				39	18	46.2	30	2	6.7*	87	30	34.5*
HIV-				26	15	57.7	21	7	33.0	57	32	56.1
Permanent dentition												
HIV+				9	9	100.0	30	11	36.7	40	20	50.0
HIV-				12	10	83.3	21	9	42.9	33	19	56.7
Primary dentition												
HIV+	15	11	73.3	39	18	46.2	27	3	11.0*	84	31	36.9*
HIV-	8	8	100.0	26	15	57.7	16	10	62.5	52	35	67.3

* Significant difference. Fisher's exact test $P < 0.05$.

Table 3. Distribution of mutans streptococci levels in HIV-positive and HIV-negative children

			Children With (%)		
Group	Status	N	$< 10^5$ CFU*	10^5–10^6 CFU*	$> 10^6$ CFU*
All children	HIV+	88	29.5	28.4	42.0†
	HIV-	55	50.9	36.4	12.7
> 6 yrs	HIV+	25	18.5	25.9	55.6†
	HIV-	16	25.0	56.3	18.8
3–6 yrs	HIV+	38	28.9	42.1	28.9
	HIV-	24	50.0	33.3	16.7

* CFU/ml of saliva; † Significantly different $P < 0.05$; chi square analysis.

Table 4. Distribution of lactobacilli in HIV-positive and HIV-negative children

			Children With (%)		
Group	Status	N	$< 10^3$ CFU*	10^3–10^4 CFU*	$> 10^5$ CFU*
All children	HIV+	87	16.1	33.3	50.6†
	HIV-	55	25.5	49.1	25.5
> 6 yrs	HIV+	30	6.7	23.3	70.0†
	HIV-	21	19.0	57.1	23.8
3–6 yrs	HIV+	37	27.0	27.0	45.9
	HIV-	24	33.3	33.3	33.3

* CFU/ml of saliva; † Significantly different $P < 0.05$; chi square analysis.

Subsurface demineralization of smooth surfaces

Smooth surface demineralizations were recorded during the caries examination but were not reported in the DMF/def scores. The mean number of white spot lesions on smooth surfaces was significantly higher in the HIV-positive group when compared to the HIV-negative group (2.2 ± 6.0 versus 0.1 ± 0.5; Wilcoxon rank sum test, $P < 0.0001$). Also, the percent children without any white spot lesions was significantly lower in the HIV-positive group (34.5 % versus 56%; Fisher's exact test, $P < 0.02$).

Assessment of mutans streptococci and lactobacilli

The HIV-positive children had significantly higher mutans streptococci scores (Wilcoxon rank sum, $P < 0.001$; Table 3). The percent of children harboring more than one million CFU mutans streptococci /ml saliva was significantly higher in the HIV-positive group. Forty-two percent of HIV-positive children harbored greater than 10^6 CFU/ml compared with only 12.7% of the HIV-negative group, while 29.5% of the HIV-positive group harbored less than 10^5 CFU compared to 50.9% in the HIV-negative group. These proportions are significantly different (chi-square, $P < 0.001$; Table 3).

The greatest differences in mutans streptococci levels were seen among the older children.

The difference in levels of lactobacilli between HIV-positive and HIV-negative groups was significant (Wilcoxon rank sum test; $P < 0.006$). The mean rank lactobacilli score was 2.3 ± 1.5 for the HIV-positive group compared with 1.6 ± 1.4 for the HIV-negative group. The percentage of children who were heavily colonized (> 100,000 CFU/ml saliva) was also significantly higher in the HIV-positive group for all children and for children older than 6 years, as seen in Table 4.

Correlation of mutans streptococci and lactobacilli to caries

A correlation analysis was done between levels of mutans streptococci and caries and lactobacilli and car-

ies. For both the HIV-positive group and the HIV-negative group, clear correlations were demonstrated both for mutans streptococci (HIV-positive: r = 0.3, $P = 0.0008$; HIV-negative: r = 0.5, $P = 0.0001$) and lactobacilli (HIV-positive: r = 0.3, $P = 0.0001$; HIV-negative: r = 0.3, $P = 0.006$) to the DMF / def.

The use of antibiotics as a confounder on microbial data was evaluated by excluding children who took antibiotics within the last month. No confounding effect was found.

Table 5. Frequency of carbohydrate intake and correlation to levels of microbes and caries, Kendall *tau b* correlation coefficients

	Frequency of Carb. Intake			*DMFT/deft*		*Mutans*		*Lactobacillus*	
Group	*N*	*Mean*	*(± SD)*	*r*	*P*	*r*	*P*	*r*	*P*
HIV+	88	6.8*	(± 2.3)	0.24	0.03†	0.14	0.1	0.31	0.0004§
HIV-	57	6.1	(± 2.1)	0.17	0.1	0.18	0.1	0.26	0.02§
Entire N	142	6.5	(± 2.2)	0.25	0.0001†	0.19	0.005‡	0.32	0.001§

* No difference in the means or distribution. Wilcoxon rank sum $P > 0.05$.

† A positive correlation was found between intake frequency and caries for the HIV+ group and the entire N.

‡ Significant correlation between mutans streptococci and intake frequency for the entire group (N).

§ Significant correlation between intake frequency and lactobacilli levels in all groups.

Frequency of carbohydrate consumption

Medication regimens and the 24-hr diet recall were evaluated. Carbohydrate-containing medications that were taken between meals were counted as a carbohydrate intake. No differences in the frequency of carbohydrate consumption were seen in the two groups (HIV-positive mean = 6.8 ± 2.3; HIV-negative mean = 6.1 ± 2.1). In the HIV-positive group, the frequency of carbohydrate intake correlated with caries experience (Kendall *tau b*, r = 0.24, $P = 0.003$) and with levels of lactobacilli (r = 0.31, $P = 0.0004$), but not with the levels of mutans streptococci. These data are shown in Table 5.

In the HIV-negative group frequency of carbohydrate intake correlated with the level of the lactobacilli only (r = 0.26, $P = 0.02$).

Associations of findings with advancement of HIV disease

The data were examined for differences in caries experience, levels of microbes, and frequency of carbohydrate intake between HIV-positive children who had a CDC classification of P1 or P2, using a Wilcoxon rank sum test. The only significant difference found between children in the two disease stages was in caries experience. Children with a more advanced disease stage had a higher caries prevalence. Again, this was due to differences in caries of the primary dentition. The mean DMFT / deft for the P1 group was 2.2 ± 3.2, and for the P2 group the mean DMFT / deft was 4.9 ± 5.0 ($P < 0.02$). The mean deft for the P1 group was 2.0 ± 2.8, and for the P2 group the mean deft was 4.4 ± 4.8 ($P < 0.02$).

The number of years since the child had been diagnosed as HIV positive was examined and found to correlate to caries experience, mutans streptococci levels, lactobacilli levels, and to frequency of carbohydrate intake. However, the DMFT did not correlate with years since diagnosis. These data are presented in Table 6.

Table 6. Correlation of caries, microbial burden, carbohydrate intake to years of illness

N	*Variables*	*Years Since Diagnosis*	
82	Lactobacilli levels	r = 0.20	$P = 0.02$*
83	Mutans streptococci	r = 0.23	$P = 0.008$*
84	Carb. intake frequency	r = 0.22	$P = 0.008$*
82	DMFT / deft	r = 0.28	$P = 0.0009$*
38	DMFT	r = 0.12	$P = 0.39$
79	deft	r = 0.28	$P = 0.001$*

* Correlation is positive and significant. Kendall tau b correlation coefficients.

Discussion

Children with perinatally acquired HIV conform to two age distributions: the short-term survivors who often die in infancy, and the long-term survivors who survive infancy with few manifestations of the infection.[33] The long-term survivor group has a long tail of survivors and peaks at around age 4. Long-term survivors were the primary participants in our present study, since 95% of subjects were between the ages of infancy and 11, with a normal distribution. Consequently, many of the subjects in this study had been relatively healthy in early childhood, which needs to be considered when comparing studies. In general, it was found that perinatally infected HIV-positive children have an increased prevalence of caries and more markers of current caries activity than their HIV-negative siblings. The higher level of past caries experience was most significant in the older children in teeth that had been in the oral cavity for some time, that is, in the primary dentition. Furthermore, in this study no differences in caries prevalence were found in the preschool children. In contrast, Valdez et al.[11] reported that caries prevalence was high in their study population. However, all of the children in the Valdez study were classified as having an advanced disease stage. These findings may be consistent with our study which also reports that sicker children have a higher caries prevalence.

Howell[6] also found a higher caries prevalence than in the general population. It was suggested that nutritional therapy in infancy and sugary medications could be the cause.[7] Despite the higher caries prevalence among HIV-positive children in general, it could not be demonstrated that the HIV-positive children were exposed to carbohydrates more frequently than their siblings even when medication regimens were considered. Moreover, in our study population many of the children had been relatively healthy infants and were still in fairly good health and did not receive nutritional supplements. However, the children with more advanced disease who were likely to be receiving hypernutrition, had a significantly higher caries prevalence.

Although the younger children were not found to have more caries than their siblings, more caries consistent with a nursing bottle decay pattern were found among the HIV-positive children, as well as more white spot lesions. Leggott et al.[3] and Schiøt[2] have reported cases of salivary gland disease and smooth surface decay. It is tempting to speculate that HIV-positive children display a different decay pattern, which might be related to decreased salivary flow[34] and the use of viscous, sugary nutrient supplements. This hypothesis would be consistent with the findings reported in our study.

Studies of caries and the dentate oral flora in young children require a large sample size[18, 28, 35, 36] because of the very skewed distribution of caries in young children and the wide variations in ages at which children contract oral flora components.[35–40] In this study, there were only 23 children younger than age 3, so this analysis is less powerful than most in this study. It may be that a larger sample would have shown differences in caries in this group, as suggested by the finding of more nursing bottle-type caries in the HIV-positive group as a whole. The findings of this study indicate that caries studies in infants perinatally infected with HIV should take into account the health status of the child and the need for a large sample size.

In this evaluation of mutans streptococci and lactobacilli as potential markers of current caries risk, a significantly greater number of HIV-positive children harbored high levels of both when compared to their HIV-negative siblings. A possible explanation for this finding of increased salivary bacteria is that dehydration or salivary gland disease may directly decrease salivary function[2, 34] and promote cariogenic factors. It has been demonstrated recently[41] that infants frequently exposed to the mother's saliva in the predentate period have lower oral burdens of mutans streptococci in the dentate period. It is thought that early inoculation initiates and elevates the child's immune response to mutans streptococci, thereby conferring a defense by the time the child is colonized with dentate flora. Thus, a lack of early salivary exchange between HIV-positive children and their caretakers may have contributed to the higher mutans levels detected in HIV-infected children. It is also conceivable that the children lose their immune response to caries-associated flora as the disease progresses, which would explain why higher levels of mutans streptococci were found among the older HIV-positive children in particular.

Changes in the DMFS/defs scores over time could not be calculated because of the cross-sectional nature of our study. Thus, the current caries activity of the children[42, 43] could not be determined. Because this was a population with a progressing disease and multiple, changing medications, past caries experience may not be indicative of the current caries activity in the HIV-positive population.[44] Consequently, other factors often associated with ongoing caries activity, such as frequent snacking,[45] the oral burden of caries-associated microbes, and the presence of active-appearing white spot lesions[46] were investigated.

Children with HIV infection intermittently receive antibiotics and frequently take prolonged courses of antibiotics, often in low doses. It was anticipated that these children would therefore have a weak correlation between past caries experience and current levels of oral microbes. Any effect the medications may have had was not great enough to disrupt the correlation between the measured bacteria and the DMFS/defs of the HIV-positive group. It may be that the systemic antibiotics were not reaching the intraoral niches due to poor salivary function.

Among the HIV-positive children, there was a clear correlation between the frequency of carbohydrate intake and caries experience, even though they had no more carbohydrate exposure than their siblings. This may indicate that these children are especially susceptible to the effects of frequent exposure to carbohydrates. Mutans streptococci levels were not correlated to the frequency of carbohydrate exposure, but were correlated to caries experience. Again, these findings would be consistent with decreased salivary flow, or some other predisposing factor.

It has been suggested[7, 11] that increased caries in HIV-positive children may be due to the hypernutrition therapy. Indeed, hypernutrition therapy is common in HIV-positive children, particularly as the disease progresses. This may coincide with declining caries defenses.[47] It seems likely that the use of nutritional supplements in small portions many times a day may influence the development and progression of caries.[45] However, we did not find that the HIV-infected children were consuming fermentable carbohydrates more frequently than their siblings, since almost all the subjects were frequent snackers.

Because caries is a multifactorial disease, no single test or examination can be performed that is highly sensitive or selective at the individual level for diagnosing ongoing caries activity. It has been shown, however, that by the combined measure of several causative and predictive factors, a group's relative risk for car-

ies activity can be estimated.[48-58] Although caries activity in this cross-sectional study could not be measured, several different indicators demonstrate that these children are at risk for caries activity, increasingly so with age. This is likely related to the increased prevalence of HIV-related illness with increasing age in long-term survivors.

Conclusions

1. Children with perinatally acquired HIV are more likely to have heavier oral burdens of lactobacilli and mutans streptococci than uninfected children in the same household.
2. Fewer children with perinatally acquired HIV are caries free than are uninfected siblings in the same households.
3. Caries prevalence is higher among HIV-positive children than among their uninfected siblings.
4. In perinatally infected HIV-positive children, caries is more prevalent in children with more advanced disease.

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1. Schiødt M, Greenspan D, Levy JA, Nelson JA, Chernoff D, Hollander H, Greenspan JS: Does HIV cause salivary gland disease? AIDS 3:819–22, 1989.
2. Schiødt M, Greenspan D, Danieli TE, Nelson J, Leggott PJ, Wara DW, Greenspan JS: Parotid gland enlargement and xerostomia associated with labial sialadentis in HIV-infected patients. J Autoimmun 2:415–25, 1989.
3. Leggott PJ, Robertson P, Greenspan D, Wara D, Greenspan J: Oral manifestation of primary and acquired immunodeficiency diseases in children. Pediatr Dent 9:98–104, 1987.
4. Goddart D, Francois A, Ninane J, Vermylen CH, Cornu G, Clapuyt PH, Claus D: Parotid gland abnormality found in children seropositive for the human immunodeficiency virus (HIV). Pediat Radiol 20:355–57, 1990.
5. Ketchem L, Berkowitz R, McIlveen L, Forrester D, Rakusan T: Oral findings in HIV seropositive children. Pediatr Dent 12:143–46, 1990.
6. Howell R: Oral manifestations and laboratory values in children infected with the Human Immunodeficiency Virus Type I, Masters Thesis UMDNJ, 1991.
7. Howell R, Jandinski J, Palumbo P, Shey Z, Houpt M: Dental caries in HIV-infected children. Pediatr Dent 14:370–71, 1992.
8. Leggott PJ: Oral manifestations of HIV infection in children. Oral Surg Oral Med Oral Pathol 73:187–92, 1992.
9. Katz MH, Mastrucci M, Leggott P, Westenhouse J, Greenspan, Scott G: Prognostic significance of oral lesions in children with perinatally Acquired Human Immunodeficiency Virus infection. Am J Dis Child 147:45–48, 1993.
10. Moniaci D, Cavallari M, Greco D, Bruatto M, Raiteri R, Palombo E, Tovo PA, Sinicco A: Oral lesions in children born to HIV-1 positive women. J Oral Pathol Med 22:8–11, 1993.
11. Valdez IH, Pizzo PA, Atkinson J: Oral health of pediatric AIDS patients: a hospital-based study. ASDC J Dent Child 61:114–18, 1994.
12. Ramos-Gomez F, Greenspan D, Greenspan JS: Orofacial manifestations and management of HIV-infected children. Oral Maxillofacial Surg Clinics NA 6:37–47, 1994.
13. U.S. Department of Health, Education and Welfare, Public Health Service, Centers for Disease Control: AIDS in Women: United States, 39:845–46, Atlanta, 1990.
14. U.S. Department of Health, Education and Welfare, Public Health Service, Centers for Disease Control: HIV/AIDS May surveillance report, Atlanta, 1993.
15. U.S. Department of Health, Education and Welfare, Public Health Service, Centers for Disease Control: Estimates of HIV prevalence and projected AIDS cases. Morbidity and mortality weekly report 39:110–119, Atlanta, 1990.
16. Yogev R, Connor E: Management of HIV-infection in infants and Children, 1st Ed. Philadelphia: CV Mosby Co, 1991.
17. Abbas AK, Lichtman AH, Pober JS: Congenital and acquired immunodeficiencies. In: Cellular and Molecular Immunology, 2nd Ed. MJ Wonsiewicz, Ed. Philadelphia: WB Saunders Co, 1994, pp 378–96.
18. World Health Organization: Guide to epidemiology and diagnosis of oral mucosal diseases and conditions. Comm Dent and Oral Epidemiol 8:1–26, 1980.
19. Cohen J: Statistical Power Analysis for the Behavioral Sciences, revised Ed. New York: Academic Press, 1977.
20. Jensen B, Bratthall D: A new method for the estimation of mutans streptococci in human saliva. J Dent Res 68:468–71, 1989.
21. Newbrun E, Matsukubo T, Hoover C, Graves R, Brown A, Disney J, Bohannan H: Comparison of two screening tests for *Streptococcus mutans* and evaluation of their suitability for mass screenings and private practice. Comm Dent Oral Epidemiol 12:325–31, 1984.
22. Denepitiya L, Kleinberg I: A comparison of the microbial compositions of pooled human dental plaque and salivary sediment. Arch Oral Biol 27:739–45, 1982.
23. Berkowitz R, Obeid G, McIlveen L, Getson P, Mohla C, Campos J: Comparison of two sampling techniques for quantitation of oral yeast levels. Pediatr Dent 16:62–63, 1994.
24. Beighton D: A simplified procedure for estimating the level of *Streptococcus mutans* in the mouth. Br Dent J 160:329–30, 1986.
25. Matsukubo T, Ohta K, Macki Y, Takeuchi M, Takazoe I: A semi-quantitative determination of *Streptococcus mutans* using its adherent ability in a selective medium. Caries Res 15:40–45, 1981.
26. Larmas M: A new dip-slide method for the counting of salivary lactobacilli. Proc Finn Dent Soc 71:31–35, 1975.
27. Westphal P, Westphal I, Narayana AS: Tandhalsovårdsprojekt i Andhra Pradesh — Indien. Tandläkartidningen 83:428–36, 1991.
28. World Health Organization: Oral Health Surveys—Basic Methods. Third Ed. Geneva 1987.

29. NIDR, U.S. Department of Health and Human Service, Oral Health of United States Children. The National Survey of Dental Caries in US School Children 1986–1987. NIH Pub No. 89–2247, 1989.
30. Kolmakow S, Nyyssonen V, Honkala E: Determining the caries at-risk child: Focal demineralization in children's teeth. J Pedod 9:58–66, 1984.
31. Pienihäkkinen K: Caries prediction through the combined use of incipient caries lesions, salivary buffering capacity, lactobacilli and yeasts in Finland. Comm Dent Oral Epidemiol 15:325–28, 1987.
32. Arizona Department of Health Services: Arizona Pathfinder health survey, 1990.
33. Auger I, Thomas P, DeGruttola V, Morse D, Moore D, Williams R, Truman B, Lawrence CE: Incubation periods for pediatric AIDS patients. Nature 336:575–77, 1988.
34. Courderc L-J, D'Agay M-F, Danon F, Harzic M, Brocheriou C, Clauvel J-P: Sicca complex and infection with HIV. Arch Intern Med 147:898–901, 1987.
35. Catalanotto FA, Shklair IL, Keene HJ: Prevalence and localization of Streptococcus mutans in infants and children. J Am Dent Assoc 91:606–609, 1975.
36. Berkowitz RJ, Jordan HV, White G: The early establishment of Streptococcus mutans in the mouths of infants. Arch Oral Biol 20:171–74, 1975.
37. Köhler B and Bratthall D: Intrafamilial levels of Streptococcus mutans and some aspects of the bacterial transmission. Scand J Dent Res 86:35–42, 1978.
38. Kristoffersson K, Gröndahl H-G, Bratthall D: The more Streptococcus mutans, the more caries on approximal surfaces. J Dent Res 64:58–61, 1985.
39. Lang NP, Holtz PR, Gusberti FA, Joss A: A longitudinal clinical and microbiological study of the relationship between infection with Streptococcus mutans and the development of caries in humans. Oral Microbiol Immun 2:39–47, 1987.
40. Köhler B, Andreen I, Jonsson B: The earlier the colonization by mutans streptococci the higher the caries prevalence at 4 years of age. Oral Microbiol Immunol 3:14–17, 1988.
41. Aaltonen A, Tenovuo J: Association between mother-infant salivary contact and caries resistance in children a cohort study. Pediatr Dent 16:110–16, 1994.
42. Krasse B: Caries Risk. Chicago: Quintessence Publ Co, 1985.
43. Ericsson Y, Hardwick L: Individual diagnosis, prognosis and counselling for caries prevention. Caries Res 12:94–102, 1978. (Suppl 1)
44. Alaluusua S, Kleemola-Kujala E, Nyström M, Evälahti M, Grönroos L: Caries in the primary teeth and salivary Streptococcus mutans and lactobacillus levels as indicators of caries in the permanent teeth. Pediatr Dent 9:126–30, 1987.
45. Gustavsson BE, Quesnel CE, Lanke LS, Lundquist C, Grahnen H, Bonow BE, Krasse B: The Vipeholm dental caries study. The effect of different levels of carbohydrate intake on caries activity in 436 individuals observed for five years (Sweden). Acta Odont Scand 11:232–364, 1954.
46. Seppä L, Hausen H: Frequency of initial caries lesions as predictor of future caries increment in children. Scand J Dent Res 96:9–13, 1988.
47. Itescu S, Brancato LJ, Winchester R: A sicca syndrome in HIV infection: Association with HLA-DR5 and CD8 lymphocytosis. Lancet No. 8661:466–68, 1989.
48. Kidd EAM: Role of Chlorhexidine in the management of dental caries. Int Dent J 41:279–86, 1991.
49. Edwardsson S, Koch G, Obrink M: Strep sanguis, Strep mutans, Strep salivarius in saliva. Prevalence and relation to caries increment and prophylactic measures. Odontol Revy 23:279–96, 1972.
50. Crossner CG: Salivary lactobacillus counts in the prediction of caries activity. Community Dent Oral Epidimiol 9:182–90, 1981
51. Köhler B, Petterson B-M, Bratthall D: Streptococcus mutans in plaque and saliva and the development of caries. Scand J Dent Res 89:19–25, 1981.
52. Zickert I, Emilson CG, Krasse, B: Correlation of level and duration of Streptococcus mutans infection with the incidence of caries. Infect Immun 39:982–85, 1983.
53. Alaluusua S, Renkonen OV: Streptococcus mutans establishment and dental caries establishment in children from 2 to 4 years old. Scand J Dent Res 93:453–57, 1983.
54. Stecksén-Blicks C: Salivary counts of lactobacilli and Streptococcus mutans in caries prediction. Scand J Dent Res 93:204–12, 1985.
55. Klock B, Krasse B: A comparison between different methods for the prediction of caries activity. Scand J Dent Res 87:129–39, 1979.
56. Krasse B: Biological factors as indicators of future caries. Int Dent J 38:219–25, 1988.
57. Crossner CG, Claesson R, Johansson T: Presence of mutans streptococci and various types of lactobacilli in interdental spaces related to development of proximal carious lesions. Scand Dent Res 97:307–15, 1989.
58. Bowden GHW: Which bacteria are cariogenic in man? In: Dental Caries: Markers of High and Low Risk Groups, 1st Ed. NW Johnson Ed. Cambridge: Cambridge University Press, 1991, pp 266–86.