Preschool Caries as an Indicator of Future Caries: a Longitudinal Study

Benjamin Peretz, DMD Diana Ram, Dr. Odont Elinor Azo, DMD Yaakov Efrat, DMD

Dr. Peretz is clinical associate professor, Dr. Ram is clinical lecturer, and Dr. Efrat is a staff member, Department of Pediatric Dentistry, The Hebrew University Hadassah School of Dental Medicine, Jerusalem, Israel; Dr. Azo is in private practice, Tel-Aviv, Israel. Correspond with Dr. Peretz at benny@cc.huji.ac.il

Abstract

Purpose: This study was performed to compare the increment of carious surfaces per year in preschool-age children with early childhood caries (ECC), children with posterior caries only, and caries-free children after 7 to 10 years.

Methods: One hundred and fifty files of children were included in the study. The first examination referred to the ages of 3 to 5 years (T1), and follow-up visits took place after at least 7 years (T2). The number of carious surfaces was recorded. The study population was divided into 3 groups: (1) caries free children (CF), (2) children with ECC, and (3) children with posterior caries only (PC). There were 50 patients in each group.

Results: Children with ECC had 1.15±0.97 new affected surfaces per year, while caries-free children had an increment per year of 0.41±0.60, and children with posterior caries only showed an increment per year of 0.74±0.64. A statistically significant difference was found between the ECC and caries-free groups, and between the ECC and posterior caries groups. The high increment in the ECC group is influenced by the high number of affected surfaces in the primary teeth.

Conclusions: Children with ECC may have a high risk to develop future carious lesions compared with caries-free children. Children with posterior caries demonstrate less carious lesions by the age of 12 years, however, they resemble ECC children when they reach their mid-teens. (*Pediatr Dent*. 2003;25:114-118)

KEYWORDS: EARLY CHILDHOOD CARIES, CARIES PREDICTION

Received March 6, 2002 Revision Accepted July 29, 2002

Early childhood caries (ECC) is a unique form of rampant caries that develops in the primary dentition soon after the eruption of the first teeth. A variety of factors have been suggested to be associated with ECC. Biological factors such as the presence of high counts of Mutans streptococci (MS), as well as social/demographic/behavioral factors:

1. lack of access to dental care;
2. lack of dental insurance for the children;
3. low family income and the educational level of the mother of the child;
4. poor hygiene and dietary habits.[1-5]

The biology of the mouth may be modified by several factors unique to young children related to the immaturity of the host defense system, as well as behavioral patterns associated with feeding and oral hygiene in early childhood.[6] It is believed that the condition is progressed and exacerbated by prolonged use of sweet drinks in a nursing bottle, particularly night feeding or during day naps.[1]

Clinically, the decay is first found in the maxillary primary incisors; later it spreads to the maxillary molars, mandibular molars, and, rarely, the mandibular incisors.

Despite extensive use of the bottle, many children do not suffer from caries at all. This finding may point towards a different susceptibility of these caries-free children to caries. Predisposing factors such as the mothers' course of pregnancy or instrumental delivery have been suggested to affect the developing teeth and cause some form of hypoplasia, which, in turn, make the teeth more vulnerable to caries.[7] ECC may not be the only form of caries observed in young children. Often, caries may appear on molar teeth only, without involving the front teeth.

A question is often asked regarding the risk of children with ECC to future caries in comparison to other children.

The dental literature suggests some evidence that children who experience ECC continue to be at high risk for new lesions as they get older, both in the primary and permanent dentitions (in the first permanent molars).[8-13] Furthermore, it has been found that children who participated in a prevention program that included topical application of fluoride, oral hygiene, use of fluoride toothpaste, diet advice and regular follow-up, showed more new lesions in the ECC group than in the control group. Their control group included children with caries who did not have ECC but were not caries free.[14]

Another study has determined that the possibility of developing caries in the ECC group is 2 times more than children without ECC.[15] In a more recent study, Almeida et al,[16] concluded that children with ECC who were treated under general anesthesia developed about 4 times more carious lesions after 2 years of follow-up than a control group. This future higher susceptibility to dental caries in children who had ECC has been associated with higher counts of MS in these children.[17]

While most studies regarding future occurrence of caries have been conducted comparing children with ECC with caries-free children, this manner may not reflect reality, where posterior caries may be present without involving the front teeth.[14,18]

There is a paucity of data regarding whether future caries susceptibility in children with posterior caries only is different from children with ECC.

Therefore, the purpose of the present study was to compare the increment of carious surfaces per year in children who had ECC with children who had posterior caries only, and caries-free children, after 7 to 10 years.

Methods

Data was obtained from 150 files of children who were treated in 2 private pediatric dental clinics (Jerusalem and Petah Tikva) according to the following criteria:

1. The first examination was between the age of 3 and 5 years (T1).
2. Children were treated, if needed, and presented for follow-up visits at least 7 years since the first visit (T2).
3. Both clinical and radiographic examinations (2 bitewing radiographs) were available at T1 and T2.
4. All subjects were healthy children.
5. Children did not have orthodontic treatment during the follow-up period.
6. Children had no trauma of the maxillary incisors during the follow-up period.
7. In each clinic, the same experienced pediatric dentist examined the children at T1 and T2 (DR in Jerusalem and YE in Petah Tikva).

The study population was divided into 3 groups, with 50 patients in each group (25 from each clinic) in the following manner:

1. group 1—caries free children (CF);
2. group 2—children with ECC (caries in at least 2 upper incisors);
3. group 3—children with caries in the posterior teeth (PC): caries in at least 1 primary molar, while the incisors and canine were caries free.

The number of patients in each group (50 patients, 25 in each clinic) was the minimum required for statistical analysis. Files were reviewed in alphabetical order until 25 patients in each clinic for each group were included.

The group of posterior teeth only was included as a control group, according to Sclavos et al,[14] and Johnsen et al,[18] who established that a caries-free group cannot be used as a control because these children may have a special or different sensitivity to caries. The number of carious tooth surfaces at T1 (primary teeth) and T2 (primary and permanent teeth) was recorded for each patient.

Missing teeth at follow-up were not calculated. They were assumed exfoliated. Stainless steel crowns were considered as 5 surfaces affected.

ANOVA with Scheffe's test was done to compare the mean number of affected surfaces at T1 and at T2, as well as the increment of affected surfaces in the 3 groups.

Table 1. Mean Number of Affected Surfaces at T1 and T2,Increment and Increment per Year

Group	Affected surfaces at T1	Affected surfaces at T2 (primary)	Affected surfaces at T2 (permanent)	Affected surfaces at T2 (total)	Increment	Increment per year
1=CF N=50	0	2.0±4.30	1.1±1.48	3.10±4.64	3.10±4.64	0.41±0.60
2=ECC N=50	16.25±8.35	8.62±9.57	3.06±6.50	11.68±9.58	8.86±7.64	1.15±0.97
3=PC N=50	8.92±4.48	2.52±4.11	4.72±6.19	7.24±5.89	6.04±5.34	0.74±0.64
Total (groups 1+2+3)N=150	8.39±8.60	4.38±7.13	2.96±4.74	7.34±7.81	6.00±6.42	0.77±0.81
Groups significantly different *	1 vs 2: *P*<.01 1 vs 3: *P*<.01 2 vs 3: *P*=.03	1 vs 2: *P*<.01 2 vs 3: *P*<.01	1 vs 2: *P*=.03 1 vs 3: *P*=.01	1 vs 2: *P*<.01 2 vs 3: *P*<.01	1 vs 2: *P*<.01 1 vs 3: *P*<.01	1 vs 2: *P*<.01 2 vs 3: *P*<.01

*ANOVA with Scheffe for multiple comparisons.

Table 2. Mean Number of Affected Surfaces at T1 and T2, and Increment per Year by Age Groups

Group	Age groups	Affected surfaces at T1	Affected surfaces at T2 (primary)	Affected surfaces at T2 (permanent)	Affected surfaces at T2 (total)	Increment per year
1=CF	10-12 y (N=23)	0	3.60±5.76	0.92±0.44	4.52±6.20	0.60±0.79
	13-15 y (N=27)	0	0.62±1.62	1.26±1.58	1.88±2.20	0.25±0.29
2=ECC	10-12 y (N=32)	17.46±8.85	12.31±10.01	2.03±1.11	14.34±9.90	1.37±1.01
	13-15 y (N=18)	14.11±7.11	2.05±3.15	4.89±3.83	6.94±6.98	0.76±0.77
3=PC	10-12 y (N=17)	10.94±4.60	4.76±5.46	4.12±2.52	8.88±5.98	0.88±0.66
	13-15 y (N=33)	7.87±4.10	1.36±2.64	5.03±3.1	6.39±5.74	0.67±0.63
Total	10-12 y (N=72)	10.34±9.83	7.75±8.82	2.16±2.19	9.91±9.01	1.01±0.92
	13-15 y (N=78)	6.58±6.88	1.26±2.50	3.70±3.09	4.96±5.59	0.54±0.61
Significance for age groups*		*P*<.01	*P*<.01	NS	*P*<.01	*P*<.01

*Two-way ANOVA with group and age groups as factors.

Results

Table 1 shows the mean number of surfaces affected with caries for all the groups (caries free, ECC, and posterior caries), at T1 (when they presented themselves for clinical examination), and at T2 (after the follow-up period), as well as the increment and the increment per year. The mean number of affected surfaces was highest among the ECC group at T1 and at T2. For the primary teeth, the highest mean number of affected surfaces was found among the ECC group (statistically significant in comparison with CF and PC groups, *P*=.000 and *P*=.030, respectively). For the permanent teeth, the highest mean number of affected surfaces was found among the PC group (statistically significant difference in comparison with CF group, *P*=.000, but not with ECC group) .

Since the follow-up time was different for each patient, (60 patients were followed up after 7 years, 50 after 8 years, 22 after 9 years, and 18 after 10 years), the increment per year was a better way to describe the new carious surfaces (treated or untreated) that were added. Comparison between the groups was done using 2-way ANOVA.

When increments per year among the 3 groups of the study were compared, children with ECC had 1.15±0.97 new affected surfaces per year, caries-free children had an increment per year of 0.41±0.60, and children with posterior caries only showed an increment per year of 0.74±0.64. A statistically significant difference was found between the ECC and caries-free groups, and between ECC and posterior caries groups.

Table 2 shows the mean number of affected surfaces at T1 and at T2, and the increment per year by age groups. At T1, the younger children (10-12 years old) showed a higher number of affected surfaces than the older children (13-15 years old) in 2 groups (ECC and PC). At T2, the younger children showed a higher number of affected surfaces in the primary teeth and in the total number of affected surfaces. The highest number of affected surfaces at T2 was found in the younger children of the ECC group (12.31±10.01). With respect to the permanent teeth, the older children demonstrated higher scores in all 3 groups. The highest number of affected surfaces was among the older children of the PC group (5.03±3.1). Also, in the permanent teeth in the ECC group, the older children had over 2 times more affected surfaces than the younger children (4.89±3.83 and 2.03±1.11, respectively). In the PC group, the older children had nearly 25% more affected surfaces than the younger children (5.01±3.1 and 4.12±2.52 respectively).

As for the increment per year, the youngest children showed higher scores when compared with the eldest children (13-15 years old): 0.6 and 0.25, respectively, for the caries-free children; 1.37 and 0.76, respectively, among the ECC children; and 0.88 and 0.67, respectively, among the PC children.

When the increments per year in the different groups were compared, in the younger age (10-12 years old) the ECC group showed 2.3 times more new lesions than the caries-free group and 1.6 times more than the posterior caries only group (ECC=1.37, CF=0.6, PC=0.88). The difference was statistically significant.

When the increments per year in the older age group (13-15 years old) were compared, children in the ECC group showed 3 times more lesions than caries-free children and 1.2 times more than the posterior caries group (ECC=0.76, CF=0.25, PC=0.67). The difference was statistically significant.

Table 3 shows the percentage of affected surfaces by location on the tooth at T2 in the permanent molar teeth in the CF, ECC, and PC groups. In all groups, the occlusal

Table 3. Percentage of Affected Surfaces in the Permanent Molar Teeth at T2

	Occlusal	Proximal	Buccal/ lingual	
CF	58%	25%	17%	100%
ECC	43%	30%	18%	100%
PC	53%	28%	29%	100%
Total mean	51% (SD 7.6)	28% (SD 2.5)	21% (SD 6.7)	*P*=.002*

*ANOVA.

surface was the most affected surface (58%, 43%, and 53% respectively). The second most affected surface was the proximal surface in the CF, ECC, and total mean surfaces, and the buccal/lingual area in the PC group.

No difference was found in the increment per year between both clinics.

Discussion

The findings of this study show that after 7 to 10 years, children from the ECC group demonstrated the highest increment per year of affected tooth surfaces compared to children with caries in their posterior teeth and to children who were caries free. This finding is in agreement with previous studies, which determined ECC to be a high-risk indicator for future caries development.[14-16,18]

A few differences between this study and the others are:

1. The time of follow-up (7-10 years in this study) is much longer than in previous studies (from a few months to 2 years).
2. A large study group—150 patients in this study.
3. The ECC group in this study was compared with 2 different groups: caries-free and posterior caries to avoid the problem of comparison with caries-free children only.

The findings of this study show that in primary teeth, the ECC group demonstrated significantly more affected surfaces at T2 than children with PC or CF children. However, in the permanent teeth, the PC group demonstrated the highest number of affected surfaces, although the difference was not statistically significant.

Looking at this study's findings by age groups reveals that the total number of affected surfaces in primary teeth at T2 among the ECC children was the highest in the younger age (12.31±10.01), as was the total mean number of affected surfaces (14.34±9.90), and the increment per year (1.37±1.01). Regarding the permanent teeth, the highest number of affected surfaces was observed among the older children of the PC group.

The findings of this study also show that, in the ECC group, the older children had over 2 times more affected surfaces in the permanent teeth than the younger children. In the PC group, the older children had nearly 25% more affected surfaces of the permanent teeth than the younger children. This may indicate a higher number of affected surfaces in the younger children in this group. Furthermore, it may be that the younger children have not had enough time to exfoliate more teeth.

Based on the pattern of the appearance of the increment per year of affected surfaces in the primary teeth and the finding that the increment per year was highest among the ECC children, it seems that these results were probably mainly influenced by the high number of affected surfaces in the primary teeth.

The results may point to the fact that, until 12 years of age, ECC children may be considered a unique group. However, when ECC and PC children approach the mid-teenage years, they demonstrate a similar number of affected surfaces. Thus, both ECC and PC children constitute a particular group of children in early childhood with caries on anterior or posterior teeth who are more susceptible to future caries in their adolescence than caries-free children. The difference may be explained by the existence of social/demographic factors in these children as well as biological factors such as a different ecology in the oral cavity.[6]

The high counts of MS in children with ECC has been suggested as a primary cause for the disease.[2] A recent study which used molecular identification methods found, besides MS, a number of other species not previously associated with dental caries in children with ECC compared to caries-free children: *S sanguinis* was associated with health, and, in order of decreasing cell numbers, *Actinomyces gerencseriae*, *Bifidobacterium*, MS*, Veillonella*, *S salivarius*, *S constellatus*, *S parasanguinis*, and *Lactobacillus fermentum* were associated with caries.[9] The biology of the mouth may be modified by behavioral patterns associated with feeding and oral hygiene in early childhood and lack of access to dental care.[5,6]

The similarity among children in their mid-teenage years regarding the number of affected surfaces in permanent teeth between ECC and PC groups may be supported by the findings of Ramos-Gomez et al,[6] who found salivary MS levels among children with ECC to be higher than would be expected in a dentally healthy population, but lower than levels reported among older children at high risk for caries.

Finally, the surface on the molars that was mostly affected was the occlusal surface. This is not surprising, since the first molar erupts usually at 6 years of age with unmatured enamel containing pits and fissures on its occlusal area thus being most susceptible to caries.

This study faces some limitations, which mostly concern methodological aspects:

1. A possibility exists that, over a 10-year period, the practioner's criteria for caries identification and diagnostic methods may change.
2. Significant selection bias likely occurs when some patients who started at ages 3 to 5 (T1) are lost to the practice by T2.
3. Continuous dental care is itself a significant predictor of caries increment because lesions get identified early in their development and result in restorations that become caries markers.
4. The design of this study missed any lesion that developed in primary teeth that subsequently exfoliated between T1 and T2. Thus, averaging the number of carious surface increments at T2 over the number of years between T1 and T2 may underreport caries incidence.

Further research is needed to isolate factors influencing the appearance of caries in childhood.

Conclusions

1. Children with ECC may have a high risk to develop future carious lesions compared with caries-free children and children with posterior caries until the age of 12 years.
2. Children with posterior caries resemble ECC children when they reach their mid-teenage years.

References

1. O'Sullivan DM, Tinanoff N. Social and biological factors contributing to caries of the maxillary anterior teeth. *Pediatr Dent*. 1993;15:41-44.
2. Tinanoff N, O'Sullivan DM. Early childhood caries: overview and recent findings. *Pediatr Dent.* 1997; 19:12-16.
3. Mohan A, Morse DE, O'Sullivan DM, Tinanoff N. The relationship between bottle usage/content, age, and number of teeth with mutans streptococci colonization in 6-24-month-old children. *Community Dent Oral Epidemiol.* 1998;26:12-20.
4. Douglass JM, Tinanoff N, Tang JM, Altman DS. Dental caries patterns and oral health behaviors in Arizona infants and toddlers. *Community Dent Oral Epidemiol*. 2001;29:14-22.
5. Ramos-Gomez FJ, Weintraub JA, Gansky SA, Hoover CI, Featherston JD. Bacterial, behavioral and environmental factors associated with early childhood caries *J Clin Pediatr Dent*. 2002;26:165-173.
6. Seow WK. Biological mechanisms of early childhood caries. *Community Dent Oral Epidemiol*. 1998; 26(suppl):8-27.
7. Peretz B, Kafka I. Baby bottle tooth decay and complications during pregnancy and delivery. *Pediatr Dent*. 1997;19:34-36.
8. Johnsen DC, Schechter TG, Gerstenmaier JH. Proportional changes in caries patterns from early to late primary dentition. *J Public Health Dent.* 1987;47:5-9.
9. Kaste LM, Marianos D, Chang R et al. The assessment of nursing caries and its relationship to high caries in the permanent dentition. *J Public Health Dent*. 1992;52:64-68.
10. O'Sullivan DM, Tinanoff N. Maxillary anterior caries associated with increased caries risk in other primary teeth. *J Dent Res*. 1993;72:1577-1580.
11. O'Sullivan DM, Tinanoff N. The association of early dental caries patterns in preschool children with caries incidence. *J Public Health Dent*. 1996;56:81-83.
12. Litt M, Reisine S, Tinanoff, N. Multidimensional causal model of dental caries development in low-income preschool children. *Public Health Reports.* 1995;110:607-617.
13. Berkowitz RJ, Moss M, Billings RJ et al. Clinical outcomes for nursing caries treated using general anesthesia. *J Dent Child*. 1997;64:210-211.
14. Sclavos S, Porter S, Seow WK. Future caries development in children with nursing bottle caries. *J Pedod*. 1988;13:1-10.
15. Al-Shalan TA, Erickson PR, Hardie NA. Primary incisor decay before age 4 as a risk factor for future dental caries. *Pediatr Dent*. 1997;19:37-41.
16. Almedia AG, Roseman M, Sheff M, Huntington N, Hughes C. Future caries susceptibility in children with early childhood caries following treatment under general anesthesia. *Pediatr Dent.* 2000;22:302-306.
17. Burt BA, Loesche WJ, Eklund SA, Earnest RW. Stability of Streptococcus mutans and its relationship to caries in a child population over 2 years. *Caries Res*. 1983;17:532-542.
18. Johnsen DC, Gerstenmaier JH, Disantis TA, Berkowitz R. Susceptibility of nursing-caries children to future proximal molar decay. *Pediatr Dent*. 1986;8:168-170.
19. Becker MR, Paster BJ, Leys EJ, Moeschberger ML, Kenyon SG, Galvin JL, et al. Molecular analysis of bacterial species associated with childhood caries. *J Clin Microbiol.* 2002;40:1001-1009.