



Prevalence of Dental Caries and Enamel Defects in Connecticut Head Start Children

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Abstract

Purpose: The aim of this study was to determine the prevalence of dental caries and enamel defects in a sample of predominantly African American and Hispanic children from an inner city Head Start program located in Connecticut.

Methods: A total of 517 children were examined for dental caries and enamel defects. Children's caries experience was described using the dmfs/t indices, and dental defects were described using a modified developmental defects of enamel index.

Results: The mean dmfs was 3.0, and 38% of the children had caries. The prevalence of enamel defects was 49%. When analyzed by race/ethnicity no significant differences in dmfs scores or the prevalence of defects were observed. The majority of defects were located on anterior teeth, and the type of defect varied with the location. On the buccal surface of canines, hypoplasia accounted for 70% of the lesions. On maxillary anterior teeth, linear opacities accounted for 50% of the lesions. A positive association between enamel defects and caries was observed.

Conclusions: The prevalence of caries and defects in these Head Start children was high, with most defects located on anterior teeth. Enamel defects were associated with an increased caries incidence. (*Pediatr Dent.* 2003;25:235-239)

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The most recent US national survey on dental caries, carried out from 1988-1991, found a mean dfs score of 1.2 in 2- to 4-year-old children.¹ However, certain populations experience a greater level of caries. The prevalence of caries in low-income United States Head Start preschool children (aged 3 to 4 years) ranges from 24% to 59%, and mean caries scores range from a mean dmft of 1.3 to a mean dfs of 11.²⁻⁵ However, most reports are more than 10 years old.

Caries has been suggested to be higher in low-income children due to a multiplicity of factors including lack of access to care, poor nutrition, and more fatalistic health beliefs.⁶ A more tangible cause may be a higher incidence of dental defects, which has been suggested to predispose a tooth to increased caries risk and has been associated with increased levels of dental caries.⁷ Enamel defects may result from systemic, genetic, or environmental factors such as birth prematurity, low birth weight, infections, malnu-

trition, or metabolic disorders—many of which have a higher incidence in low socioeconomic families.⁸ Also, it has been hypothesized that a low serum calcium concentration during enamel formation may be a factor.⁹

In certain anatomic locations, such as canines, it has been suggested that hypoplasia could be caused by minor physical trauma to the face approximately 6 months after birth due to insufficient cortical bone protecting the canine crypt.¹⁰ Similarly, localized trauma from intubation of premature infants has been suggested to be the cause of some lesions on maxillary incisors.¹¹ In addition, studies in twins are suggestive of a possible genetic predisposition in the formation of the lesion.¹² The prevalence rates for enamel defects range from 4% to 60%, depending on the population studied, teeth examined, and diagnostic criteria used.⁷

The aim of this paper is to ascertain the prevalence of caries and enamel defects in an inner city Head Start program

Table 1. Caries Experience and Treatment Levels by Age

Age	N	Caries prevalence	Mean dmfs	Mean dmfs among caries positive children	d/dmfs
<3 y	27	19%	0.3	1.8	100%
3 y	221	33%	2.8	8.4	39%
≥4 y	269	45%	3.6	7.9	45%
Total	517	38%	3.1	7.9	43%

located in Hartford, Conn and determine if an association between caries and enamel defects exists.

Methods

In October 1999, 517 3- to 5-year-old children were examined for dental caries and enamel defects as part of their annual oral health screening. The children were enrolled in the Head Start program located in Hartford, Conn. Head Start is a federally funded preschool program designed for children from families whose household incomes generally fall below US government poverty guidelines. All children present at the Head Start centers on the day of the examinations with a signed consent form were examined. Excluded from the study were children that refused the dental examination. The study was approved by the University of Connecticut Health Center's Institutional Review Board.

Dental examinations were conducted onsite using no. 23 sickle-shaped explorers, nonmagnifying front surface mirrors, and focusable flashlights. Visible plaque was removed with gauze. Diagnostic criteria for the coronal caries examination were modified from those developed by Radike.¹³ Fissure surfaces were diagnosed carious if an explorer resisted removal after insertion into a fissure with moderate pressure and/or loss of translucency of enamel adjacent to the fissure. Buccal or lingual surfaces were diagnosed as carious if penetrated by an explorer or if enamel could be scraped away by the explorer. Proximal surfaces were diagnosed as carious if the marginal ridge showed opacity or if the explorer recorded discontinuity along with other signs such as an opacity. No radiographs were taken.

A modified developmental defects of enamel (DDE) index was used for charting enamel defects. An opacity was recorded if there was a change in the translucency of the enamel but only if the enamel was of normal thickness with a smooth surface that could not be scraped or penetrated by an explorer. Opacities were white, yellow, cream, or brown in color. They were classified as:

1. linear, if the defect followed the lines of development of the teeth;
2. demarcated, if the defect had a distinct and clear boundary with the adjacent normal enamel; and
3. diffuse, if the defect had no clear boundary with the adjacent normal enamel.

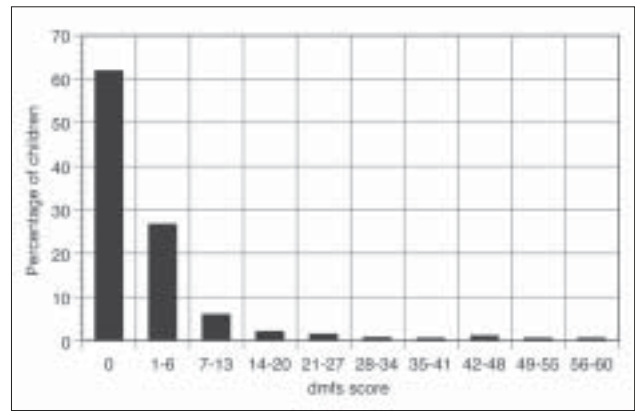


Figure 1. Distribution of dmfs scores.

Enamel hypoplasia was recorded if there was a quantitative loss of enamel or break in either the enamel surface or in the form of pits, grooves, or other malformations.¹⁴

Two dentists performed the examinations, and 2 separate dentists recorded all data. Calibration of the 2 dental examiners consisted of reviewing the caries and enamel defects diagnostic criteria 2 weeks prior to the examinations. Examiners had 2 practice sessions on 10 subjects each with postexamination discussion and resolution of differences. Field examinations were made on a random sample of 10 subjects (not included in the main data set), each of whom were examined independently by the 2 dentists. Using individual tooth surfaces as the level of agreement, kappa scores of 0.82 and 0.70 for dental caries and enamel defects, respectively, were obtained.

Caries and defects were charted on a form that specified each primary tooth surface as being missing, sound, or having caries, restorations, sealants, hypoplasia, or opacities (linear, demarcated, or diffuse). Multiple codes per surface were recorded as necessary. Teeth were determined to be lost due to trauma or exfoliation if adjacent incisors had no caries. These teeth were not included in the analysis. Maxillary incisors not present that had adjacent carious incisors were considered to have been carious and extracted. This assessment of missing teeth is similar to the methods used in British surveys of children's dental health.¹⁵ Data regarding the subject's date of birth and race/ethnicity were also collected.

Children's caries experience was analyzed using traditional dmfs/t scores. Enamel defects were categorized by location. Maxillary anterior defects included all lesions affecting the maxillary incisors. Canine defects included only the buccal surfaces of all canines. Statistical differences between dmfs/t scores were evaluated using the Mann-Whitney test and differences in prevalence of caries and defects was analyzed by the chi-square test.

Results

A total of 517 eligible (250 female and 267 male) children with ages ranging from 2.7 to 4.9 years old were examined for dental caries and enamel defects. The majority of the

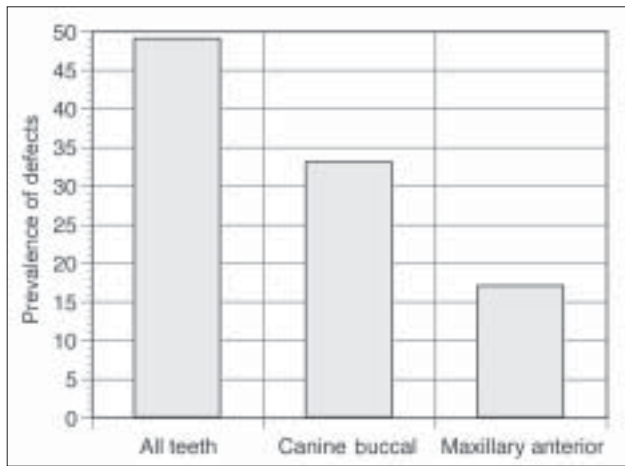


Figure 2. Prevalence of enamel defects by location.

children were either African American (59%) or Hispanic (39%). The mean dmfs score of the children was 3.1, ranging from 0.3 in children less than 3 years old to 3.6 in children 4 years old. Among the caries-positive children, the mean dmfs was more than twice as high, at 7.9 (Table 1). The frequency distribution of dmfs scores was skewed, with 62% of children having a dmfs score of 0 and 13% of the children having dmfs scores equal or greater than 7, accounting for 78% of the disease in the population (Figure 1). When analyzed by race/ethnicity, no differences in the dmfs scores were observed. Treatment levels were low and no treatment had been rendered to children less than 3 years old (Table 1).

The prevalence of enamel defects among all subjects was 49%, with the prevalence of defects on canine buccal surfaces being 33% and on maxillary anteriors being 17% (Figure 2). There was a mean number of 1.4 defects per child, with a mean number of 0.5 canine buccal defects per child and a mean number of 0.4 maxillary anterior defects per child. The types of defects varied by location. On the canine buccal surfaces, hypoplasia accounted for 70% of the lesions. On the maxillary anterior teeth, linear opacities accounted for 50% of the lesions, while hypoplasia accounted for only 14% of the lesions (Figure 3). There was no significant difference in the prevalence of defects between different racial/ethnic groups.

A significant association between enamel defects and caries was observed in this study. Children with enamel defects had a mean dmfs score and caries prevalence twice that of those without defects. When analyzed by defect location, those with maxillary anterior defects had a higher mean dmfs score of 5.1 compared to 4.28 in children with any type of defect (Table 2).

Discussion

The prevalence of caries in this population is lower than the majority of previous reports about children in Head Start programs that have examined a variety of racial/ethnic populations.¹⁶

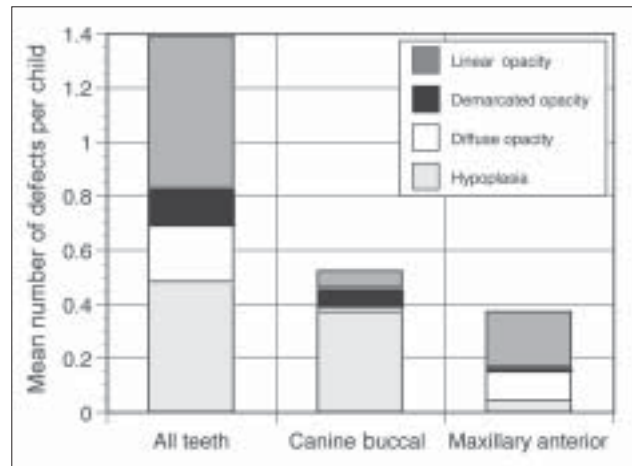


Figure 3. Mean number of enamel defects per child by location and type.

Table 2. Mean dmfs Scores and Caries Prevalence by Presence of Enamel Defects

	Mean dmfs*	Caries prevalence†
Children with any type of enamel defects	4.28	50%
Children without any type of enamel defects	1.86	26%
Children with maxillary anterior enamel defects	5.12	67%
Children without maxillary anterior enamel defects	2.56	31%

*Statistically significant differences ($P < .01$) by Mann-Whitney test between those with and without defects.

†Statistically significant differences ($P < .01$) by chi-square test between those with and without defects.

However, there remains a subset of children with high caries levels, as 10% of the children accounted for 73% of the disease. Among those with caries, the mean dmfs was double that of the mean for the whole population, a finding similar to other studies.² These results emphasize the need to identify and target additional preventive services for those children at high risk of developing caries.

It has been suggested that racial/ethnic minorities show an increased risk of caries.¹ However, it has been difficult to separate the cultural influences of ethnicity from the effects of low socioeconomic status or poverty status on the prevalence of dental caries.⁶ In the present study, where no differences in the prevalence of dental caries between Hispanic and African American children were found, the children were homogeneous in respect to geographic location and socioeconomic status, as measured by income. This finding is similar to that of Tang et al.² These conflicting reports suggest that, at the present time, race/ethnicity may not be a good indicator of caries risk.

The prevalence of enamel defects in this study was 49%. Studies carried out in developed countries not targeting high-risk groups such as premature infants are limited in

number. Among United States studies, one study of mainly white children located in Iowa found the prevalence of enamel hypoplasia to be 6% and the prevalence of isolated opacities to be 27%.¹⁷ Another study located in California examining predominantly white and Hispanic children found an overall prevalence of enamel defects to be 33%, with the prevalence of enamel hypoplasia and opacities being 21% and 12%, respectively.¹⁸ The higher prevalence of enamel defects in the present study population may be due to the low socioeconomic status which has been shown to be associated with a high prevalence of enamel defects.⁸

Primary canines have long been known to be prone to enamel defects. The prevalence of enamel defects on the buccal surface of canines in the present study was 33%. Previous clinical studies in United States populations that included African American and white children have shown the prevalence of such defects to be 9% to 45%.¹⁹⁻²²

Some of these studies have shown racial/ethnic differences between African American and white children with respective prevalences of 33% and 17% in one study²⁰ and 10% and 7% in another.²¹ In the present study, no variations between African Americans and Hispanics were found.

In the present study, although no enamel defects were found on the mandibular incisors, the prevalence of enamel defects on maxillary anterior teeth was 30%, and the majority were linear opacities suggesting a time-dependent environmental or systemic cause. These findings suggest that site-specific factors account for these defects. However, it is also possible that environmental factors may play a role and lesions on the other teeth may be at the microscopic rather than macroscopic level.

In the present study, children with enamel defects had more than twice the level of caries compared to those without enamel defects. Many other studies have also found strong associations between enamel defects and caries.^{7,23} Children with defects may be more susceptible to caries due to decreased caries resistance from the loss of the enamel or due to imperfect enamel.^{7,8}

Conclusions

1. Although caries was prevalent in Connecticut Head Start children, a mere 10% of the children accounted for more than 70% of the disease.
2. No differences were found between the prevalence of caries in African American or Hispanic children.
3. Enamel defects were highly prevalent, with most defects located on the maxillary incisor teeth or the buccal surfaces of the canines.
4. The presence of enamel defects was associated with increased caries experience.

References

1. Kaste LM, Selwitz RH, Oldakowski RJ, Brunelle JA, Winn DM, Brown LJ. Coronal caries in the primary

- and permanent dentition of children and adolescents 1-17 years of age: United States, 1988-1991. *J Dent Res.* 1996;75(special issue):631-641.
2. Tang JMW, Altman DS, Robertson DC, O'Sullivan DM, Douglass JM, Tinanoff N. Dental caries prevalence and treatment levels in Arizona preschool children. *Public Health Rep.* 1997;112:319-329.
3. Barnes GP, Parker WA, Lyon TC, Drum MA, Coleman GC. Ethnicity, location, age, and fluoridation factors in baby bottle tooth decay and caries prevalence of Head Start Children. *Public Health Rep.* 1992;107:167-173.
4. Trubman A, Silberman SL, Meydrech EF. Dental caries assessment of Mississippi Head Start children. *J Pub Health Dent.* 1989;49:167-169.
5. Jones DB, Schlife CM, Phipps KR. An oral health survey of Head Start children in Alaska: oral status, treatment needs, and cost of treatment. *J Pub Health Dent.* 1992;52:86-93.
6. Reisine S, Douglass JM. Psychosocial and behavioral issues in early childhood caries. *Community Dent Oral Epidemiol.* 1998;26(suppl 1):32-44.
7. Seow WK. Biologic mechanisms of early childhood caries. *Community Dent Oral Epidemiol.* 1998;26(suppl 1):8-27.
8. Seow WK. Enamel hypoplasia in the primary dentition: a review. *J Dent Child.* 1991;58:441-449.
9. Nikiforuk G, Fraser D. The etiology of enamel hypoplasia: a unifying concept. *J Pediatr.* 1981;98:888-893.
10. Skinner MF, Hung JT. An enigmatic hypoplastic defect of the deciduous canine. *Am J Phys Anthropol.* 1989;79:159-175.
11. Fadavi S, Adeni S, Dziedzic K, Punwani I, Vidyasagar D. The oral effects of oro-traqueal intubation in prematurely born preschoolers. *J Dent Child.* 1992;59:420-424.
12. Taji S, Hughes T, Rogers J, Townssend G. Localised enamel hypoplasia of human deciduous canines: genotype or environment. *Aust Dent J.* 2000;45:83-90.
13. Radike AW. Criteria for diagnosis of dental caries. In: *Proceedings Clinical Testing of Cariostatic Agents.* Chicago, Ill;1972.
14. Federation Dentaire Internationale Commission on Oral Health Research and Epidemiology: A review of Developmental Defects of Enamel (DDE Index). *Int Dent J.* 1992;42:411-426.
15. O'Brien M. Children's dental health in the United Kingdom 1993. London, England: Office of Population Censuses and Surveys; 1994.
16. Edelstein BL, Douglass CW. Dispelling the myth that 50 percent of US schoolchildren have never had a cavity. *Public Health Rep.* 1995;110:522-530.
17. Slayton RL, Warren JJ, Kanellis MJ, Levy SM, Islam M. Prevalence of enamel hypoplasia and isolated opacities in the primary dentition. *Pediatr Dent.* 2001;23:32-36.

18. Nation WA, Matson L, Peterson JE. Developmental enamel defects of the primary dentition in a group of Californian children. *J Dent Child.* 1987;54:330-334.
19. Badger GR. Incidence of enamel hypoplasia in primary canines. *J Dent Child.* 1985;52:57-58.
20. Silberman SL, Trubman A, Duncan WK, Meydrech EF. Prevalence of primary canine hypoplasia of the mandibular teeth. *Pediatr Dent.* 1991;13:356-360.
21. Duncan WK, Silberman SL, Trubman A, Meydrech EF. Prevalence and racial distribution of primary canine hypoplasia of the maxillary canine. *Pediatr Dent.* 1994;16:365-367.
22. Silberman SL, Duncan WK, Trubman A, Meydrech EF. Primary canine hypoplasia in Head Start children. *J Public Health Dent.* 1989;49:15-18.
23. Li Y, Navia JM, Bian JY. Caries experience in deciduous dentition of rural Chinese children 3-5 years old in relation to the presence or absence of enamel hypoplasia. *Caries Res.* 1996;30:8-15.

ABSTRACT OF THE SCIENTIFIC LITERATURE



EFFECTS OF DAILY FLUORIDE ON GLASS IONOMER RESTORATIVES

It is known that glass ionomer cements absorb and release fluoride following single fluoride exposure. However, limited information exists as to whether daily tooth-brushing with a fluoride dentifrice will optimally charge these restorations or if the addition of supplementary fluoride sources will augment fluoride uptake and release. With this in mind, the purpose of this study was 2-fold: (1) to compare the methods for recharging glass ionomer, resin-modified glass ionomer and polyacid-modified composite resin restorations; and (2) to determine whether saturation levels exist for these materials. For this, 96 specimens (32 of each material: Ketac-Fil, Photac-Fil, and Dyract AP) were prepared in cylindrical Delrin molds with dental floss incorporated for suspension into the test medium. Samples were placed in a pH cycling system consisting of a demineralizing solution and a remineralizing solution to mimic a high-caries risk situation. Specimens were subdivided into 4 groups receiving the following fluoride exposures: (1) control; (2) tooth brushing 1 time/day; (3) tooth-brushing 2 times/day; and (4) tooth-brushing 2 times/day+a fluoride rinse every day for 7 days. Fluoride content was measured using an expandable ion analyzer. The results showed a significant decrease in fluoride release for all materials from days 1 to 3. All specimens released more fluoride when immersed in the demineralizing solution than in the mineralizing solution. By day 7, Photac-Fil (resin-modified glass ionomer) demonstrated both the greatest total fluoride release and the greatest rechargeability. This was followed by Ketac-Fil (glass ionomer) and Dyract (compomer). Group 4 produced the greatest amount of "recharging." In conclusion, the authors state that home care fluoride regimens provide adequate fluoride exposure for recharging glass ionomer materials and that both the resin-modified glass ionomer and the glass ionomer showed the greatest amount of rechargeability.

Comments: This study highlights the fact that a compomer, in high-risk children, is not the material of choice. In this, as in other studies, it produced the least amount of fluoride release. It would have been better if the authors used a traditional composite material as a control for comparison among the different materials. KV

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Freedman R, Diefenderfer KE. Effects of daily fluoride exposures on fluoride release by glass ionomer-based restoratives. *Oper Dent.* 2003;28:178-185.

34 references