

Prevalence of enamel hypoplasia and isolated opacities in the primary dentition

Rebecca L. Slayton, DDS, PhD John J. Warren, DDS, MS Michael J. Kanellis, DDS, MS Steven M. Levy, DDS, MPH Mahbubul Islam, BA

Dr. Slayton is assistant professor and Dr. Kanellis associate professor, Department of Pediatric Dentistry; Dr. Warren is assistant professor and Dr. Levy is professor, Department of Preventive & Community Dentistry; and Dr. Islam is a statistician, they are all at The University of Iowa, College of Dentistry. Correspond with Dr. Slayton at rebecca-slayton@uiowa.edu.

Abstract

Purpose: Enamel hypoplasia is of interest to both the clinician and the basic scientist because it may indicate an increased risk for caries and can contribute to the understanding of enamel development. The purpose of this paper is to report the prevalence of enamel hypoplasia and isolated enamel opacities in a cohort of healthy, well-nourished children in Iowa.

Methods: The study sample consisted of 698 children examined at 4-5 years of age. Individual tooth surfaces were scored for the presence of enamel hypoplasia (EH) and isolated enamel opacities. Prevalence of EH and isolated opacities were determined by tooth type and by gender.

Results: Six percent of the children examined had at least one tooth with EH; 27% had at least one tooth with isolated enamel opacities. There was no difference in the prevalence of EH between boys and girls, but significantly more boys than girls had enamel opacities.

Conclusions: The prevalence of enamel defects in this study group is comparable to that seen in other studies of normally developed children except that in this study, the primary tooth types most commonly affected with enamel hypoplasia or isolated opacities were mandibular second molars and maxillary second molars, respectively. (Pediatr Dent 23:32-36, 2001)

isturbances during tooth development can be manifested as enamel hypoplasias, diffuse or demarcated enamel opacities or enamel hypomineralization. These defects can be the result of hereditary factors (as in amelogenesis imperfecta and trichodentoosseous syndrome)^{1.2} or environmental factors (both pre- and post-natally).³

Enamel hypoplasia (EH) is defined as a deficiency of enamel formation. This is seen clinically as pits, grooves, or generalized lack of surface enamel. Enamel hypoplasia is important clinically because it can result in increased caries susceptibility, increased wear, tooth sensitivity and poor esthetics. This type of enamel defect may also provide valuable clues about the child's early environment and may be predictive of similar disturbances in the permanent dentition.³

The published prevalence of EH in the primary dentition varies from 2-99% of children examined depending on the racial, ethnic, nutritional, or socioeconomic status of the child, birth weight, the type of classification system used, and the method of examination.⁴⁻⁹ There is a greater prevalence of EH in children from developing countries,^{10,12} children with

chronic or acute malnutrition,^{10,12} and children with very low birth weight.⁴ Other factors that are significantly associated with EH include a history of maternal smoking, lack of prenatal care during the first trimester, elevated blood lead levels, postnatal measles infection and perinatal intubation.^{4,13-15} Interestingly, enamel hypoplasia of primary teeth is more common in children with cerebral palsy, mental retardation, or hearing defects.¹⁶ This suggests that the systemic disturbances that interfere with neurological development may also interfere with tooth development.

Distinct from EH, enamel opacities may be white, yellow, or brown in color with an intact surface, are usually well demarcated, and are round to oval in shape.¹⁷ Enamel opacities represent a mild disruption of enamel formation compared to enamel hypoplasias. They may occur as a result of a developmental disturbance during amelogenesis or because of mechanical trauma during the maturation phase of enamel formation.⁷ Isolated enamel opacities do not usually lead to an increased risk for caries but can cause significant esthetic challenges.

The prevalence of non-fluoride opacities in primary teeth ranges from 1-98% of children studied.^{5,7,10,18-21} The prevalence of isolated opacities is lower among Asian children than among Caucasian or Hispanic children and is more prevalent in malnourished children.

Direct comparisons of the prevalence of EH and isolated opacities from previous studies are virtually impossible because of differences in classification methods, examination techniques, epidemiological factors and the manner of reporting findings. In Table 1, findings from previous studies are summarized and differences in classification systems are noted.

Relatively few studies have examined the prevalence of enamel defects in the primary dentition of healthy, well-nourished children in the United States.¹⁸ The purpose of this paper is to report the prevalence of isolated enamel opacities and enamel hypoplasia in the primary dentition of a cohort of children in Iowa who have been followed since birth.

Methods

Children included in this study were part of The Iowa Fluoride Study cohort,²²⁻²⁶ which has been followed prospectively since birth. The mean age at examination for the children was 4.6 years. A total of 698 children (359 females and 339 males)



Fig 1. Maxillary primary dentition with enamel hypoplasia evident on both second molars.



Fig 2. Creamy white enamel opacities are present on maxillary and mandibular primary canines.

were examined for enamel hypoplasias of any type and for isolated opacities at the same time that dental fluorosis and caries were assessed. Characteristics and prevalence of primary tooth fluorosis^{27,28} and cavitated vs. non-cavitated lesions²⁹ in these subjects have been reported separately.²⁹ Fluorosis and nonfluoride (isolated) opacities were differentiated using Russell's criteria¹⁷ and features of the developmental defects of enamel index (DDE).³⁰ These techniques differentiate between the two types of lesions based on the shape and demarcation of the lesion, the color, area, and teeth affected. Non-fluoride opacities are most commonly creamy-yellow to brown in color, well demarcated, and on the smooth surface of the tooth. In contrast, fluorosis is more symmetrical and more diffuse, with white lines or patches that lack well-defined margins.

Examinations were conducted using a portable chair and exam light by one of two trained and calibrated examiners. Teeth were evaluated for presence of enamel hypoplasia and isolated opacities using a mouth mirror and exam light, but without drying the teeth. Presence or absence of enamel hypoplasia and isolated opacities was recorded separately for each tooth, but the specific locations on the tooth were not noted. Deficiencies in enamel formation such as pits and linear grooves were recorded as enamel hypoplasia (Fig 1). Localized opacities that were white, creamy, yellow, or brownish in color were scored as isolated opacities (Fig 2).

Interexaminer reliability for hypoplasia and isolated opacities was assessed by examination of about 10% of subjects by both examiners throughout data collection, between August 1997 and March 2000. Percent agreement and kappa statistics were computed at the subject and tooth levels. At the person level, there was 100% agreement; at the tooth level, percent agreement was 92% and kappa was 0.69.

Data were entered using SPSS[®] Data Entry software and descriptive statistics were generated and statistical tests conducted using SPSS^{®31} and SAS.^{®32} P values less than 0.05 were considered to be statistically significant.

Results

Study group characteristics

The children in this study were relatively homogeneous in socioeconomic status (SES) and in race, with almost all (98%) being Caucasian. At the time of recruitment (1992 to 1994), 50% of families had incomes greater than \$40,000 per year (US dollars), 38% had family incomes between \$20,000-\$40,000 per year, and 12% had incomes less than \$20,000 per year. There were no reports of malnourishment and those children with birth weights less than 2500g were designated low birth weight. There were 25 children (4%) who were less than 2500g at birth. None of the children in the study met the criteria for very low birth weight (1000g-1500g) and none were classified as extremely low birth weight (less than 1000g). No attempt was made to keep a detailed record of each child's medical history from birth until the time of examination.

Enamel hypoplasia

Of the 698 children examined, 44 (6%) had enamel hypoplasia on at least one primary tooth, 3% had one tooth affected, 2% had two teeth affected, <1% had three teeth affected, <1%had 4 teeth affected and only one individual had more than four affected teeth. The appearance of this individual's teeth was consistent with a diagnosis of hypoplastic-type amelogenesis imperfecta. The mean number of teeth with enamel hypoplasia in the entire sample was 0.13. Among those individuals with any hypoplasia, the mean number was 2.02. There was no statistically significant difference in prevalence between males (7%) and females (6%). Although it would be expected that enamel hypoplasias due to environmental stress would occur bilaterally, in just over half of the cases (24 out of 44), only one tooth was affected. Hypoplastic lesions were just as likely to occur on the left side as they were on the right side of the mouth.

Figure 3 shows the percentages of affected teeth by individual tooth among those children with at least one hypoplastic tooth. The primary second molars were the most commonly affected teeth. EH was found most frequently on the mandibular primary second molars (teeth 'K' and 'T'), with 30% and 27% of all affected teeth, respectively. The maxillary primary second molars (teeth 'A' and 'J') had 18% and 16% affected teeth, respectively. Primary canines on the left side of the mouth (teeth 'H' and 'M') were more likely to have EH than primary canines on the right side of the mouth (teeth 'C' and 'R'). Sixteen percent of each of the left primary canines was affected while 7% of each of the right primary canines had EH. The mandibular primary incisors were the least likely to be affected (2% for each tooth).

Isolated opacities

Isolated opacities occurred in 188 (27%) of the subjects. The majority of these (16%) had only one tooth affected, 5% had 2, 4% had 3, 1% had 4, and 1% had 5 or more affected teeth. The mean number of teeth with opacities for the entire sample

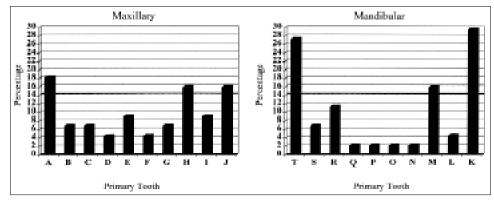


Fig 3. Graphic representation of the percentage of primary teeth with enamel hypoplasia among those children with at least one hypoplastic tooth.

was 0.50. Among those individuals with at least one opacity, the mean number of affected teeth was 1.85. Opacities were significantly more prevalent in males (31%) than in females (23%) (P=0.018). Among the subjects with at least one opacity, the tooth type most commonly affected was the maxillary second primary molar (26% and 20% teeth affected). The next most frequently affected teeth were the mandibular canines (17% and 15%). There was a significant difference overall in the prevalence of any isolated opacities in the maxilla (3%) and the mandible (2%) (P=0.018).

Figure 4 shows the percentages of teeth with isolated opacities by individual tooth among the subjects with at least one opacity. As mentioned previously, the primary second molars were the most commonly affected teeth, with the most prevalent opacity found on the right maxillary primary second molar (tooth 'A'). Among children with at least one opacity, 26% had this tooth affected. Each canine was affected among 12% to 17% of the subjects, each first molar among 3% to 7% of subjects, and each lower incisor in less than 1%. The prevalence of opacities on each primary maxillary incisor ranged from 6% to 7% among those with at least one opacity.

Family income, maternal education, maternal age

There was no significant difference in the prevalence of either enamel hypoplasia or isolated enamel opacities by family income or by maternal education. Subjects were divided into five different categories based on their mothers' ages at the time of

Table 1. Prevalence of Enamel Defects in other Studies							
Study Group	Age (YR)	Enamel Defects (%)	Enamel Hypoplasia (%)	Enamel Opacities (%)	Ν	Index used	Reference
Japan	3		2%	>1%	2,733	$\mathbf{NR}^{\dagger\dagger}$	Yonezu et al, 1997 ⁵
Thailand	1 – 4•		23%	9%	344	$\mathbf{NR}^{\dagger\dagger}$	Kanchanakamol et al, 1996 ¹⁰
China	3 – 5	24%	22%	2%	1,344	DDE"	Li et al, 1996 ¹¹ Li et al, 1995 ²⁰
USA (Mississippi)	4 - 8		27% (canines)		2,686	SHI^\dagger	Silberman et al, 1991 ³⁴
Pakistan	5 - 8		35% (canines)		113	$\mathbf{NR}^{\dagger\dagger}$	Lukacs, 1991 ¹²
Australia	Child	9% (normal) 57% (premature)			8,411 ^{‡‡}	mDDE [‡]	Hall, 1989 ³⁷
Mexico	5 - 15		6%		300	DDE"	Goodman et al, 1987 ³³
USA	1 – 11		45% (canines)		220	$NR^{\dagger\dagger}$	Badger, 1985 ³⁸
USA (California)	3 - 6	33%	21%	12%	300	mDDE [‡]	Nation et al, 1987 ¹
Saudi Arabia	2 - 6		15%	12%	390	mDDE [‡]	Rugg-Gunn et al, 1998 ¹⁹
Australia (Aborig-inal)	4.4 +/- 0.8	98%	99%	98%	68	DDE 	Seow et al, 1996 ⁶ Pascoe & Seow, 1994 ³⁵
Great Britain	6		4%	33%	303	Al-Alousi ^{‡‡‡}	Murray & Shaw, 1979 ²¹

¹Malnourished, "Developmental Defects of Enamel Index⁹, [†]Simplified Hypoplasia Index⁸, ^{††}Not Reported, [‡]Modified Developmental Defects of Enamel Index³⁰, ^{‡‡}Based on chart review, ^{‡‡‡}(Al-Alousi et al, 1975)³⁹

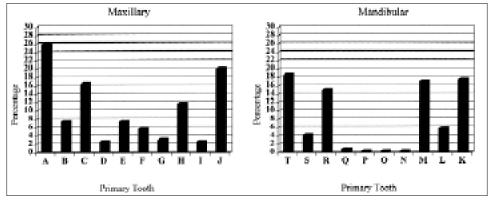


Fig 4. Graphic representation of the percentage of primary teeth with isolated enamel opacities among children with at least one opacity.

their birth. Analysis of enamel hypoplasias by maternal age showed no significant differences among the groups.

Analysis of isolated enamel opacities by maternal age showed a statistically significant difference among the groups (P=0.043, chi-square), with the highest occurrence among the children with the youngest mothers (< 20 years) at 58% vs. 27% for Group II (20-24 years), 32% for Group III (25-29 years), 23% for Group IV (30-34 years), and 26% for Group V (>35 years).

Discussion

The data for this paper were obtained as part of a longitudinal study of children in the Iowa Fluoride Study. The primary objectives of the study have been to collect information relative to fluoride exposures and ingestion and to relate these to the presence and severity of dental fluorosis and caries. Documentation of enamel hypoplasias and enamel opacities was done to provide a more complete picture of the developmental enamel defects that occurred in this study group. The sample is not fully representative of any particular group of individuals. It primarily represents children whose parents have chosen to keep them in the study, and are of relatively high SES.²³ Comparisons of findings in this study with those in other similar studies must be done with caution because of the differences in populations, environmental influences, and methods of reporting. The purpose of this part of the study was to report the prevalence of enamel hypoplasia and isolated (non-fluoride) opacities in a cohort of healthy Iowa children.

In the current study, we found the prevalence of enamel hypoplasia in primary teeth to be 6%. This falls within the lower end of the range seen in other studies. In previous studies, the populations with the lowest prevalence of enamel hypoplasia were from Japan (2%)⁵ and Mexico (6%).³³ In general, a higher prevalence of EH was reported among African-American children,^{18,34} aboriginal children,^{6,35} and children with chronic malnutrition.^{10,11} The Iowa study sample had fewer than 3% non-Caucasians and the children were healthy and well nourished. The relatively low prevalence of EH in the Iowa sample may be partially explained by the exclusion of teeth with stainless steel crowns. The crowns could have been placed to treat either caries or enamel hypoplasia. Treatment effects such as this would result in an overall reduction in the reported prevalence of hypoplasia.

The percentage of children in this study with enamel opacity on at least one tooth was 27%. In other studies, the prevalence ranged from less than 1% to 98% of children with at least one affected tooth. The lowest prevalence was seen in Asian children^{5,11}and the highest prevalence was in Australian Aboriginal children.³⁵ The prevalence of enamel opacities in the Iowa study sample was similar to that seen in a study of children in Great Britain (33%).²¹

Of interest is the difference in prevalence of EH and isolated opacities within the same study sample. In most other studies, EH was more prevalent than isolated opacities. In the current study, that trend was reversed. One potential explanation is that the examination technique resulted in the underdetection of teeth with hypoplastic lesions due to prior treatment with stainless steel crowns (as mentioned above). Another possibility is that fluoride-related opacities were incorrectly scored as isolated (non-fluoride) opacities. The potential for this type of diagnostic error was minimized by using Russell's criteria and criteria similar to the DDE index (described above) to differentiate isolated opacities from fluorosis. In addition, the designer of the Tooth Surface Index of Fluorosis (TSIF) index provided extensive training and calibration of the examiners in the diagnosis of fluorosis.³⁶

Women who gave birth when they were less than 20 years old were more likely to have children with enamel opacities but not with enamel hypoplasias. In some cases, young mothers are more likely to have babies born prematurely or with low birth weight. There is clear evidence to show that low birth weight babies (<2500g) are at a greater risk for developing enamel defects in the primary dentition.⁴

Defects such as enamel hypoplasia and isolated enamel opacities occur as a result of disruptions in enamel development. A variety of environmental and genetic factors have been shown to contribute to the formation of these defects, including malnourishment, mechanical trauma, racial or ethnic background, and lack of prenatal care. With continued investigation of these defects, a better understanding of the process of enamel development and the factors that interfere with it will be reached.

Conclusions

- 1. Among a convenience sample of 698 primarily caucasian children in Iowa, the prevalence rate for enamel hypoplasia was 6% and for isolated enamel opacities was 27%.
- 2. The primary tooth types most commonly affected with enamel hypoplasia or isolated opacities were mandibular second molars (28%) and maxillary second molars (25%), respectively.
- 3. Enamel hypoplasia was more common on mandibular canines (27%) than on maxillary canines (23%); a similar pattern was present for isolated opacities where mandibu-

lar canines (32%) were more frequently affected than maxillary canines (28%).

4. Isolated opacities were more common in males than females.

The study was supported by NIH grants 2RO1-DE09551, 2P30-DE10126 and CRC-RR00059

References

- 1. Witkop CJ, Jr.: Amelogenesis imperfecta, dentinogenesis imperfecta and dentin dysplasia revisited: problems in classification. J Oral Pathol 17:547-53, 1988.
- 2. Wright JT, Roberts MW, Wilson AR, Kudhail R: Trichodentoosseous syndrome. Features of the hair and teeth. Oral Surg Oral Med Oral Pathol 77:487-93, 1994.
- 3. Seow WK: Enamel hypoplasia in the primary dentition: a review. ASDC J Dent Child 58:441-52, 1991.
- 4. Seow WK: Effects of preterm birth on oral growth and development. Aust Dent J 42:85-91, 1997.
- Yonezu T, Hayashi Y, Sasaki J, Machida Y: Prevalence of congenital dental anomalies of the deciduous dentition in Japanese children. Bull Tokyo Dent Coll 38:27-32, 1997.
- 6. Seow WK, Amaratunge A, Bennett R, Bronsch D, Lai PY: Dental health of aboriginal pre-school children in Brisbane, Australia. Community Dent Oral Epidemiol 24:187-190, 1996.
- 7. Needleman HL, Leviton A, Allred E: Macroscopic enamel defects of primary anterior teeth—types, prevalence, and distribution. Pediatr Dent 13:208-16, 1991.
- 8. Silberman SL, Trubman A, Duncan WK, Meydrech EF: A simplified hypoplasia index. J Public Health Dent 50:282-84, 1990.
- 9. Ainamo J: An Epidemiological Index of Developmental Defects of Dental Enamel (DDE Index), FDI Technical Report No. 15. Int Dent J 32:159-66, 1982.
- Kanchanakamol U, Tuongratanaphan S, Tuongratanaphan S, Lertpoonvilaikul W, Chittaisong C, Pattanaporn K et al: Prevalence of developmental enamel defects and dental caries in rural pre-school Thai children. Community Dent Health 13:204-207, 1996.
- 11. 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 30:8-15, 1996.
- Lukacs JR: Localized enamel hypoplasia of human deciduous canine teeth: prevalence and pattern of expression in rural Pakistan. Hum Biol 63:513-22, 1991.
- Needleman HL, Allred E, Bellinger D, Leviton A, Rabinowitz M, Iverson K: Antecedents and correlates of hypoplastic enamel defects of primary incisors. Pediatr Dent 14:158-66, 1992.
- Brook AH, Fearne JM, Smith J: Environmental causes of enamel defects. Ciba Foundation Symposium 205:212-21, 1997.
- 15. Fadavi S, Adeni S, Dziedzic K, Punwani I, Vidyasagar D: The oral effects of orotracheal intubation in prematurely born preschoolers. J Dent for Children Nov-Dec:420-24, 1992.
- Bhat M, Nelson KB: Developmental enamel defects in primary teeth in children with cerebral palsy, mental retardation, or hearing defects: a review. Adv Dent Res 3:132-42, 1989.
- 17. Russell AL: The differential diagnosis of fluoride and nonfluoride enamel opacities. Journal of Public Health Dentistry 21:143-46, 1961.

- Nation WA, Matsson L, Peterson JE: Developmental enamel defects of the primary dentition in a group of Californian children. ASDC J Dent Child 54:330-34, 1987.
- 19. Rugg-Gunn AJ, Al Mohammadi SM, Butler TJ: Malnutrition and developmental defects of enamel in 2- to 6-year-old Saudi boys. Caries Res 32:181-92, 1998.
- Li Y, Navia JM, Bian JY: Prevalence and distribution of developmental enamel defects in primary dentition of Chinese children 3-5 years old. Community Dent Oral Epidemiol 23:72-79, 1995.
- 21. Murray JJ, Shaw L: Classification and prevalence of enamel opacities in the human deciduous and permanent dentitions. Arch Oral Biol 24:7-13, 1979.
- 22. Levy BT, Bergus GR, Levy SM, Slager SL, Kiritsy MC: Longitudinal feeding patterns of Iowa infants. Ambulatory Child Health 2:25-34, 1996.
- Levy SM, Kiritsy MC, Slager SL, Warren JJ, Kohout FJ: Patterns of fluoride dentifrice use among infants. Pediatr Dent 19:50-55, 1997.
- 24. Heilman JR, Kiritsy MC, Levy SM, Wefel JS: Fluoride content of infant foods and cereals. JADA 128:857-63, 1997.
- 25. Levy SM, Kiritsy MC, Slager SL, Warren JJ: Patterns of dietary fluoride supplement use during infancy. J Public Health Dent 58:228-33, 1998.
- 26. Warren JJ, Levy SM, Nowak AJ, Tang S: Non-nutritive sucking behaviors in pre-school children: A longitudinal study. Pediatr Dent 2000.
- Levy SM, Warren JJ, Kanellis MJ, Kirchner HL: Dental Fluorosis Prevalence in the Primary Dentition. J Dent Research 79:158, 2000.
- Warren JJ, Kanellis MJ, Levy SM: Fluorosis of the primary dentition: What does it mean for permanent teeth? JADA 130:347-56, 1999.
- Warren JJ, Kanellis MJ, Levy SM: Patterns of decay and fluorosis in the primary dentition. J Public Health Dent 59:103, 1999.
- Clarkson J: A review of the developmental defects of enamel index (DDE Index). International Dental Journal 42:411-26, 1992.
- 31. SPSS User's Guide 7.5. 1996.
- 32. SAS Procedures Guide. 1999.
- 33. Goodman AH, Allen LH, Hernandez GP, Amador A, Arriola LV, Chavez A et al: Prevalence and age at development of enamel hypoplasias in Mexican children. Am J Phys Anthropol 72:7-19, 1987.
- 34. Silberman SL, Trubman A, Duncan WK, Meydrech EF: Prevalence of primary canine hypoplasia of the mandibular teeth. Pediatr Dent 13:356-60, 1991.
- 35. Pascoe L, Seow WK: Enamel hypoplasia and dental caries in Australian aboriginal children: prevalence and correlation between the two diseases. Pediatr Dent 16:193-99, 1994.
- Horowitz HS, Driscoll WS, Meyers RJ, Heifetz SB, Kingman A: A new method for assigning the prevalence of dental fluorosis—the Tooth Surface Index of Fluorosis. JADA 109:37-41, 1984.
- Hall RK: Prevalence of developmental defects of tooth enamel (DDE) in a pediatric hospital department of dentistry population (1). Adv Dent Res 3:114-19, 1989.
- Badger GR: Incidence of enamel hypoplasia in primary canines. ASDC J Dent Child 52:57-58, 1985.
- Al-Alousi W, Jackson D, Compton G, Jenkins OC: Enamel mottling in a fluoride and in a non-fluoride community. Br Dent J 138:56-60, 1975.