Macroscopic enamel defects of primary anterior teeth — types, prevalence, and distribution

Howard L. Needleman, DMD Alan Leviton, MD Elizabeth Allred, MS

Abstract

A total of 40.7% of 509 exfoliated primary anterior teeth from children who were healthy products of uneventful pregnancies exhibited at least one macroscopic enamel defect. Twenty per cent of the teeth exhibited hypoplastic defects (HD), 12.4% exhibited white-cream opacities (WCO), and 9.8% exhibited yellow-brown opacities (YBO). Slightly more than a third (33.6%) of the teeth had defects we considered to be developmental enamel defects (DED). The occurrence of DED did not vary with gender, side of mouth, individual tooth types, or racial background. DED occurred with increased frequency on maxillary teeth, facial surfaces, and the middle third of affected surfaces. These locations have thicker enamel than other sites and may be more susceptible to insult if vulnerability is a function of metabolic demand of the rapidly secreting ameloblasts. Twenty-five per cent of the maxillary incisors and 10.1% of the mandibular incisors exhibited HD whose locations coincided with enamel forming at birth. A third (33.3%) of the canines exhibited HD, which occurred most commonly in the middle third of the facial surfaces. These defects are believed to occur approximately six months postnatally and may be primarily due to mechanical trauma. YBO most commonly occurred on the middle third of the facial surfaces, while WCO on the gingival third. Neither YBO nor WCO followed a chronologic pattern. (Pediatr Dent 13:208–16, 1991)

Introduction

The prevalence of macroscopic enamel defects in the primary dentition ranges from 0 to 85% of the children studied depending upon: 1) the types of defects evaluated; 2) the classification system used; 3) methods used in detection and recording and; 4) the genetic, racial, ethnic, medical, or socioeconomic status of the populations studied (Small and Murray 1978; Cutress and Suckling 1982; Pindborg 1982; Bhat and Nelson 1989). Macroscopic enamel defects of the primary dentition are reported to occur in 5.9 to 33.0% of normal children (Holm and Arvidsson 1974; Murray and Shaw 1979; Nation et al. 1987; Hargreaves et al. 1989) and in 0 to 73% of children from so-called underdeveloped countries or populations (Jelliffe et al. 1961; Sweeney and Guzmán 1966; Sweeney et al. 1969; Jelliffe and Jelliffe 1971; Sweeney et al. 1971; Enwonow 1973; Infante 1974; Infante and Gillespie 1974; Infante and Gillespie 1977; Goodman et al. 1987). Most of the defects described in these studies occur on the facial surfaces of the maxillary primary incisors and coincide with the so-called neonatal line.

Hypoplasia of the enamel on the facial surface of primary canines is reported to occur in 2 to 45% of children (Schroeder and Hammer 1984; Badger 1985; Brown and Smith 1986; Duncan et al. 1988; Silberman et al. 1989; Skinner and Hung 1989). This defect may be the result of a combination of a systemic disturbance (hypocalcemia) and local trauma (Skinner and Hung 1989).

In this paper we document the prevalence, location, and types of macroscopic developmental enamel defects found on exfoliated primary teeth from a sample of normal children, and offer a hypothesis that accounts for the pattern of defect distribution.

Materials and Methods

Six hundred and seventy exfoliated primary teeth were obtained from children who participated in a study of effects of prenatal and postnatal lead exposure on behavior and cognitive and perceptual function. These children, healthy products of uneventful pregnancies, were born between April 1, 1979, and March 31, 1980, at what was then the Lying-In Division of the Boston Hospital for Women and is now part of the Brigham and Women's Hospital, Boston, MA. Ninety-five per cent of the children had birth weights greater than 2500 g, and 95% had gestational ages of more than 36 weeks. Teeth were requested so that dentin lead content could be measured. Parents mailed the teeth along with a form that specified the tooth's location in the mouth and the date of exfoliation. If more than one tooth was mailed, only the first tooth received was included in the study. Before the teeth were destroyed to analyze dentin lead levels, they were prepared for visual examination by cleaning the outer surfaces with dental scalers and a standard dental prophylaxis cup on a slow-speed handpiece using flour of pumice. The pumice had been precleaned with warm EDTA which removed the lead content of the pumice, preventing contamination of the teeth for subsequent lead analyses. All teeth were dried and examined by the principal author (HLN) using a 4x magnifying loop under direct

illumination. Intraobserver reliability was tested by having the examiner randomly and blindly reevaluate 123 of the sample teeth at different sessions.

The type and location of each tooth was identified. Defects were recorded using the DDE Index, an epidemiological index of developmental defects of dental enamel developed by the Commission on Oral Health, Research and Epidemiology of the Federation Dentaire Internationale (1982). In this classification system defects are categorized as: 1) white-cream opacities (WCO); 2) yellow-brown opacities (YBO); 3) hypoplastic pits; 4) hypoplastic horizontal grooves; 5) hypoplastic vertical grooves; 6) missing enamel; or 7) discolored enamel. Hypoplasia is defined as a quantitative defect of enamel associated with a reduced thickness of enamel. An opacity is a qualitative defect characterized by an abnormality in the translucency of enamel. For this study, hypoplastic pits and grooves were combined into one category of defect, i.e., hypoplastic defect (HD). Teeth with missing or discolored enamel were excluded from the sample. Location of the defects was recorded by surface (facial, lingual, mesial, distal, or incisal/cuspal) and location on the affected surface (gingival third, middle third, or incisal third).

To determine if the distribution of HD reflected a developmental pattern, the six types of anterior teeth also were analyzed by pairing them into three chronological groupings. Each grouping had approximately the same amount of the enamel calcified at birth (Lunt and Law 1974). The maxillary central and lateral incisors were the first of the three groupings to begin calcification, with 5/6 and 2/3 of their crowns respectively, calcified at birth. The mandibular central and lateral incisors are the next grouping developmentally, with 3/5 of their crowns calcified at birth. The canines, both maxillary and mandibular, are the last of the three groupings to calcify, with only a third of their crowns calcified at birth.

The Fischer's exact test was used to calculate the probability that the observed distributions represented random phenomena.

Results

Of the 670 primary teeth received, 153 were not evaluated because of: 1) damage during mailing; 2) the presence of caries; 3) the presence of restorations; or 4) significant portions of missing enamel due to fracture of the desiccated tooth. Primary anterior teeth (central incisors, lateral incisors and canines) accounted for 509 of 517. The remaining 8 teeth were posterior primary teeth and were not used in the analyses. Therefore, the final data set consisted of 509 teeth.

Eighty-six per cent of the teeth were obtained from Caucasians, 7.2% from Blacks, 3.8% from Hispanics, and 3.0% from other minority groups, while 55.2% of the teeth were from females, and 44.8% from males. The percentages of teeth obtained from the right and left sides were 50.3% and 49.7%, respectively. Incisors accounted for 89.3% (50.2% maxillary, 39.1% mandibular) and canines, 10.7% (2.2% maxillary, 8.5% mandibular) of the sample.

One hundred and twenty-three teeth were evaluated twice without the examiner being informed. This allowed an assessment of intraobserver reliability. Kappa scores were 0.49 for overall agreement, 0.56 for recording of opacities and 0.66 for recording of HD. These values are within the ranges seen for the reading of radiographs, electrocardiograms and other diagnostic procedures (Koran 1975).

Tables 1 and 2 (see next page) present the prevalence of defects identified in the sample by arch, tooth type, and location (surface and location on the affected surface). At least one macroscopic enamel defect was found on 40.7% of the 509 teeth examined. Eight teeth exhibited more than one type of defect. HD were the most prevalent defect (20.0%), followed by WCO (12.4%), and YBO (9.8%). The maxillary central and maxillary lateral incisors exhibited the highest prevalence of defects (51.6% and 49.2%, respectively). Defects occurred more frequently (50.2%) on the maxillary primary anterior teeth than on their mandibular counterparts (30.2%). The facial surface was affected most frequently (46.4%), while the incisal/cuspal surface was least often affected (1.9%). The middle third of the affected surface was the most prevalent location of HD (50.0%) and YBO (60.0%), while WCO were observed most frequently on the gingival third (44.4%).

Hypoplastic Defects

HD were the most prevalent type of defect of the sample as a whole (20.0%) as well as of all individual tooth types with the exception of the mandibular lateral incisor, where they were equal in prevalence (10.7%) to YBO and WCO. Defects occurred on 33.3% of the canines (45.5% maxillary and 30.2% mandibular) and on 18.5% of the incisors (25.0% maxillary and 10.1% mandibular). HD also were more prevalent in the maxillary arch, on the facial surface, and on the middle third of the affected surface (Tables 1 and 2). HD were the most prevalent defect on all surfaces except the mesial surface (Fig 1, see page 211), and in all locations except the gingival third (Fig 2, see page 211).

HD were most prevalent on the incisal third of the mandibular incisors (50.0%), while the middle third was the most prevalent site for both the maxillary incisors (50.0%) and canines (72.2%, Table 3, see page 211).

Opacities

WCO occurred on 12.4% of teeth examined, while YBO occurred on 9.8% of the sample. Collectively, the

Table 1. Prevalence of enamel defects by tooth types (row %)

	N	Any Defect	HD	YBO	WCO
Total Sample	509	40.7	20.0	9.8	12.4
Maxillary Arch	267	50.2	25.8	10.5	15.4
Central Incisor	128	51.6	25.0	16.4	12.5
Lateral Incisor	128	49.2	25.0	5.5	19.5
Canine	11	45.5	45.4	0.0	0.0
Mandibular Arch	242	30.2	13.6	9.1	9.1
Central Incisor	96	24.0	9.4	9.4	7.3
Lateral Incisor	103	31.1	10.7	10.7	10.7
Canine	43	41.9	30.2	4.6	9.3

HD = Hypoplastic defect, YBO = Yellow-brown opacity, WCO = White-cream opacity

Table 2. Prevalence of enamel defects by surface and location on surface (column %)

	Any Defect	HD	YBO	WCO
Total Sample	102	50	63	11
Surface				
Facial	46.4	52.9	34.0	42.9
Mesial	23.2	13.7	30.0	33.3
Distal	16.9	13.7	26.0	15.9
Lingual	11.6	15.7	10.0	7.9
Incisal/Cuspal	1.9	3.9	0.0	0.0
Location on Affected Surface				
Middle Third	48.8	50.0	60.0	38.1
Incisal Third	26.1	31.4	28.0	17.5
Gingival Third	23.7	16.7	10.0	44.4
Whole Surface	1.4	2.0	2.0	0.0

HD = Hypoplastic defect, YBO = Yellow-brown opacity, WCO = White-cream opacity

opacities occurred more frequently on the maxillary primary anterior teeth than the mandibular, on the incisors than the canines, and on the facial surface than the other surfaces (Tables 1 and 2). WCO were the most prevalent defect on the mesial surfaces (42.0%, Fig 1) and on the gingival third of the affected surface (56.0%, Fig 2). In contrast, YBO were most prevalent on the distal surface (35.1%, Fig 1) and on the middle third of the affected surface (28.6%, Fig 2).

WCO occurred with approximately equal frequency on the gingival and middle third locations of the incisors and canines, while YBO occurred most frequently on the middle third location for these three developmental groups (Table 3). WCO were the least prevalent defect on all locations of the affected surface with the exception of the gingival third where they were the most prevalent (56.0%, Fig 2).

Developmental Enamel Defect Grouping

We classified as developmental enamel defects (DED) all HD, YBO, and selected WCO. The gingival third location of WCO accounted for 70.4% of the facial surfaces affected, compared to 40.0% of the lingual, 23.8% of the mesial, and 20.0% of the distal surfaces. In addition, the middle third location accounted for 70.0% of the distal surfaces and 57.1% of the mesial surfaces affected, compared to 20.0% of the lingual and 14.8% of the facial surfaces (Fig 3, see next page). These sites are in fact, common areas for enamel decalcification due to accumulations of plaque. Thirty-six teeth had only one defect; WCO located either on the gingival third of the facial surface or on the middle third of the mesial and distal surface. These were eliminated from the analysis because these sites were not considered to be developmental in origin. Therefore, WCO that were considered to be developmental in origin occurred on only 3.9% of the teeth examined. No such disparity occurred for YBO (Fig 3).

The prevalence of DED in the primary anterior teeth of children did not differ appreciably between groups classified by: 1) gender — males 34.9%, females 32.0% ($P = .51$), 2) location of the tooth — right side 34.8%, left side 32.4% ($P = .64$), 3) tooth types — central incisors 33.9%, lateral incisors 32.5%, canines 37.0% ($P = .81$) or 4) racial background — Caucasian 31.9%, Black 41.7%, Hispanic 31.6%, other 53.3% ($P = .23$). Significant differences were not seen when Caucasians were compared to all others ($P = .13$), or when Blacks were compared to all others ($P = .36$).

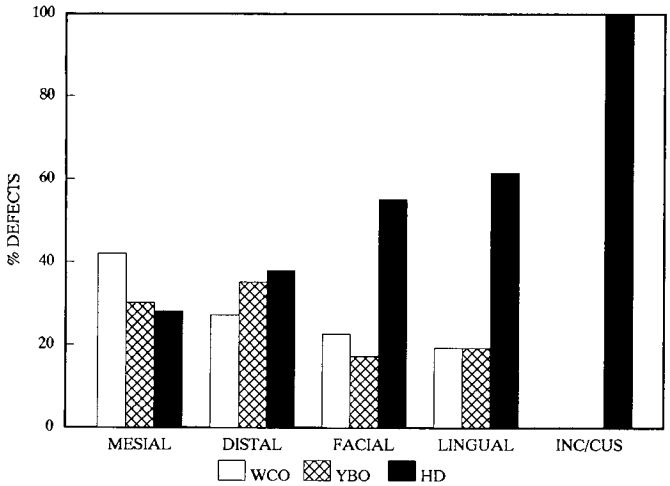

Fig 1. The prevalence of defects by tooth surface.

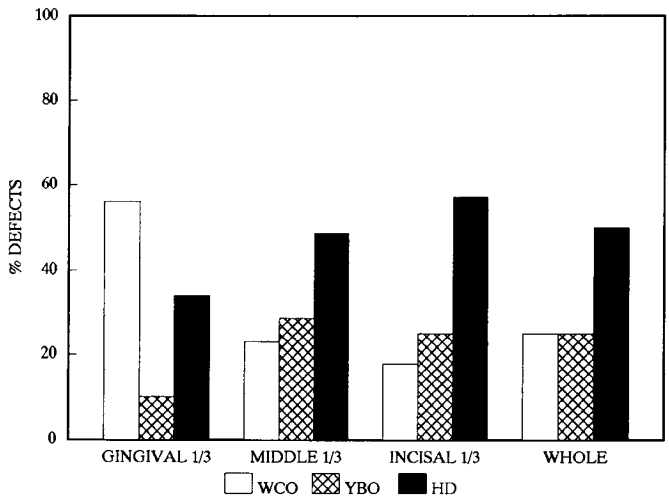

Fig 2. The prevalence of defects by location on affected surfaces.

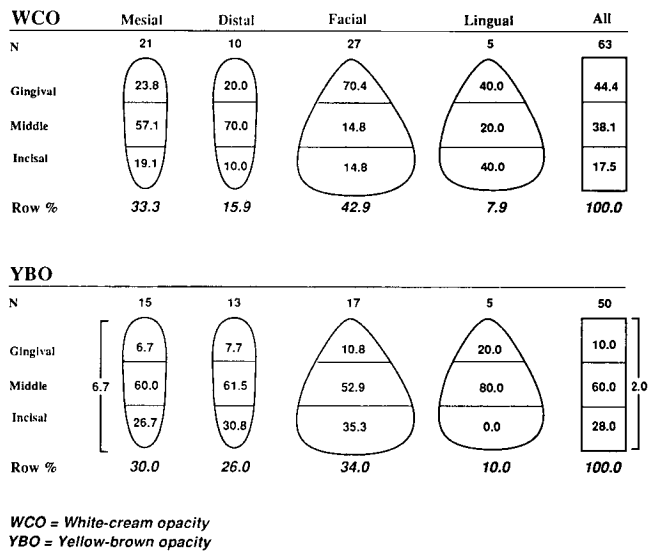

Fig 3. The prevalence of enamel defects by surface and location on surface (column %).

Statistically significant differences in the prevalence of DED in the primary anterior teeth of children were seen between groups of teeth classified by: 1) arch — maxillary 42.7%, mandibular 23.6% (P « .001); 2) surface — facial (45.0%) when compared to all other surfaces as a group (mesial 21.6%, distal 17.0%, lingual 14.0%, and incisal/cuspal 2.3%, P « .001); and 3) location on the affected surface — middle third (49.1%) when compared to all other locations as a group (incisal third 39.6%, gingival third 17.6%, and whole 1.7%, P « .001).

Discussion

Hypoplastic Defects of Primary Incisors

Prevalence

We found that 25.0% of the primary maxillary incisors of normal children in our sample had HD, considerably lower than prevalence rates of 31 to 73% among children from underdeveloped countries or populations (Jelliffe et al. 1961; Sweeney and Guzmán 1966; Sweeney et al. 1969; Jelliffe and Jelliffe 1971; Sweeney et al. 1971; Enwonow 1973; Infante 1974; Infante and Gillespie 1974; Infante and Gillespie 1977; Goodman et al. 1987) and among children with various medical problems where rates have been as high as 85% (Small and Murray 1978; Cutress and Suckling 1982; Pindborg 1982; Seow et al. 1984; Bhat and Nelson 1989). These

Table 3. Prevalence of enamel defects by location on surface for developmental pairings (column %)

	ManI	*MaxI*	*Canines*
HD	(N = 20)	(N = 64)	(N = 18)
Gingival Third	20.0	14.1	22.2
Middle Third	30.0	50.0	72.2
Incisal Third	50.0	32.8	5.6
Whole Surface	0.0	3.1	0.0
YBO	(N = 20)	(N = 28)	(N = 2)
Gingival Third	21.0	10.7	0.0
Middle Third	68.4	57.1	50.0
Incisal Third	5.3	32.1	50.0
Whole Surface	5.3	0.0	0.0
WCO	(N = 18)	(N = 41)	(N = 4)
Gingival Third	44.4	43.9	50.0
Middle Third	44.4	34.1	50.0
Incisal Third	11.1	22.0	0.0
Whole Surface	0.0	0.0	0.0

ManI = Mandibular incisors, MaxI = Maxillary incisors, HD = Hypoplastic defects, WCO = White-cream opacities, YBO = Yellow-brown opacities

observations suggest that significant systemic insults to a child with developing enamel increase the chance for macroscopic enamel defects of the primary maxillary incisors to develop.

Chronology

The HD of the primary maxillary incisors in our study generally appeared as small pits and were often found in a linear pattern or formed a groove that followed the incremental calcification pattern of these teeth. They occurred most frequently on the incisal third of the mandibular incisors and on the middle third of the maxillary incisors. Since maxillary incisors begin to calcify earlier than the mandibular incisors (Via and Churchill 1959; Nomata 1964; Kraus and Jordan 1965; Lunt and Law 1974; Levine et al. 1979), a single insult causing hypoplasia of developing enamel would result in HD occurring more gingivally on the maxillary incisors than on the mandibular incisors. This is consistent with the finding in our sample. Therefore, the insult(s) responsible for the HD of maxillary incisors probably occurred at approximately the same time as the HD of the mandibular incisors and may even be the very same or similar insult.

The most frequent location of the HD on the maxillary incisors in our study corresponds closely to the so-called neonatal line, which is a prominent incremental line in the enamel and dentin. This line is believed to emanate from a transient neonatal hypocalcemia, a nutritional disturbance which is regarded as a physiological event (Bergman 1974; Norén 1984). HD lesions are formed during the short secretory phase of amelogenesis (Suckling and Thurley 1984; Suckling 1989).

HD of the maxillary incisors in our sample occurred most frequently on the middle third of the crown, which is slightly more incisally placed than the location of the neonatal line as reported in the classical literature (Rushton 1933; Schour 1936; Schour and Kronfeld 1938; Kronfeld and Schour 1939; Massler et al. 1941; Sarnat and Schour 1941, 1942). Those studies reported the placement of the neonatal line on the facial surface of the MaxI at the point of enamel calcification occurring at birth, i.e., gingival 5/6 (Lunt and Law 1974).

The higher prevalence in our study of HD on the middle third of the maxillary incisors (50.0%) when compared to the gingival third (14.1%) may indicate that the insult occurred prenatally, the maxillary incisors in our sample have a slower developmental sequence than documented in the literature, or surface enamel defects resulting from a perinatal insult are found higher on the crown than previously believed. This third explanation is consistent with Mayer and Baume's (1966) report of the occurrence of the neonatal line on the middle third of the crown. It also is compatible with the hypothesis of Angelos et al. (1989) that any insult occurring at birth, when 5/6 of the crown has only partially calcified at the dentinoenamel junction, would result in a surface HD located more incisally on the crown because calcification of the enamel occurs in an oblique front (Fig 4). Thus, surface macroscopic neonatal lines should occur on the middle third of the facial surface because this is where enamel calcification is being completed on the tooth surface at birth. This hypothesis explains the higher prevalence of HD lesions in our sample occurring in the middle third of the maxillary incisor crowns as opposed to the classic description of the neonatal line occurring at the 5/6 level on the crown.

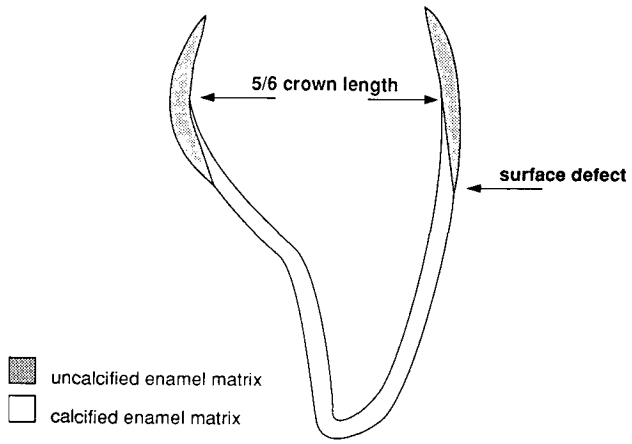

Fig. 4. Calcification status of primary maxillary incisor at birth.

Hypoplastic Defects of Primary Canines

Prevalence

Hypoplastic defects occurred in 33.3% of the 54 primary canines examined in this study. Because we examined only one primary canine per child, the prevalence of this defect might actually have been greater had we examined all the primary canines from each child. Our prevalence, however, is consistent with those reported in several other studies (Schroeder and Hammer 1984, Badger 1985, Brown and Smith 1986, Duncan et al. 1988, Silberman et al. 1989) and higher than the 2.4% reported by Skinner and Hung (1989, Table 4, see next page). These studies differ in methods and nomenclature. For example, in some studies all the primary canines of an individual were examined and results are reported in percentage of canines affected. In other studies, results are given in terms of percentage of children affected. HD were more prevalent in the maxillary canines (45.5%) than the mandibular (30.2%) in our study. In the only other study to make this comparison (Brown and Smith 1986), the reverse pattern was reported (18.2% maxillary, 31.8% mandibular). Both studies, however, have similar prevalences for the mandibular canines.

Table 4. Prevalence of primary canine hypoplasia

Study	*Sample*	*Percentage Affected*	*Procedure & Comments*
Schroeder & Hammer (1984)	196 canines US	28.0 canines	?
Badger (1985)	55 children 1.5–11.5 years old, US	45.0 children 22.0 canines	L,M & E
Brown & Smith (1986)	112 children 3–10 years old, Indiana	35.7 children	L, M & E, P mandibular > maxillary
Duncan et al. (1988)	334 children 3–5 years old, Mississippi	37.1 children	L, M & E
Skinner & Hung (1989)	1350 children 3–5 years old, Canada	2.4% all children 13.8% of Indo-Asian subset	normal oral screening mandibular > maxillary right > left
Silberman et al. (1989)	371 children 3–5 years old, Mississippi	34.5% children	L, M & E 334 of 371 Duncan et al.'s sample, black > caucasian
Needleman et al. (1991)	54 canines Boston	33.3% canines	L, M & E, D, P

L = light, M & E = mirror and explorer, D = dried, P = prophylaxis

Many clinicians might be surprised at our results indicating that HD occurred almost as frequently on the primary maxillary incisors (25.0%) as on the primary canines (33.3%). The HD of the incisors in our study tended to be smaller and less noticeable than those on the canines. Thus, perhaps our detailed examination with a magnifying loop of exfoliated rather than in situ teeth allowed us to identify defects not usually clinically detectable.

Chronology

The enamel of the middle third of primary canines calcifies at approximately six months postnatally — considerably later than the incisors (Skinner and Hung 1989). Because the HD of the canines we examined were located most commonly in the middle third as it was on the maxillary incisors, the insult responsible for HD of canines and maxillary incisors does not occur at the same time.

The character of the facial HD of the canines was typically a round or ovoid depression on the mesial or central portion of the middle third of the facial surface. This is in contrast to the linear pattern noted on the incisors, and supports the view that defects on canines are due to different insults than those causing defects on incisors. Skinner and Hung (1989) hypothesized that the HD of canines is caused by "minor physical trauma to the face... which damages the developing tooth crown through deficient cortical bone over the canine crypt." They suggested for example, that the mouthing of objects by infants may be responsible for such defects.

Opacities

Of the incisors examined, 22% had enamel opacities; however, only 13.5% were considered to be developmental in origin. This prevalence is consistent with those of similar studies (Table 5, see next page). It should be noted that these teeth were examined after prolonged desiccation which may have made opacities easier to detect or even artifactual. YBO occurred most often on the middle third and WCO on the gingival third of the affected surfaces on both mandibular and maxillary incisors, thus differing from the pattern seen with HD. Suckling (1989) has suggested that opacities occur during the secretory phase of amelogenesis as a result of less severe insults than those causing HD. Because opacities also may be the result of any disturbance during the longer maturation phase of enamel formation, their time of development is very difficult to assess accurately (Suckling 1989). Alternatively, the etiology of opacities may not be primarily developmental in origin as it is for HD.

Because YBO occur on the middle third of both the mandibular and maxillary incisors, which calcify at different times, the local trauma hypothesis of Skinner and Hung (1989) accounting for HD of canines also may explain the etiology of YBO. The high prevalence of WCO on the gingival third of facial surfaces and on the middle third of mesial and distal surfaces of all three tooth groupings i.e., maxillary incisors, mandibular incisors, and canines might be explained by plaque accumulation in this area with subsequent "white spot" decalcification. The etiology of WCO occurring on loca-

Table 5. The prevalence of developmental defects of primary dentition of "normal" populations

Study	Sample	Percentage of children affected			Methodology & Comments
		Total	HD	Opacities	
Holm & Arvidsson (1974)	208, 3 year olds Sweden	14.0	5.0	9.0	L, M & E, D
Murray & Shaw (1979)	303, 6 year olds England	32.7	4.3	28.4	L
Nation et al. (1987)	300, 3–6 year olds California	33.0	21.0	12.0	L, M & E, D, P
Hargreaves et al. (1989)	1491, preschool South Africa	5.9–16.3			? Methodology 4 different ethnic groups
Needleman et al. (1991)	509 anterior teeth Boston	33.6	20.0	13.5	L, M & E, D, P % teeth examined

L = light, M & E = mirror and explorer, D = dried, P = prophylaxis

tions other than the gingival third also may involve mechanical insult, as may be the case for all YBO and HD of the canines.

Developmental Enamel Defects Grouping

Prevalence

In this study, 40.7% of the primary anterior teeth examined were designated initially as having enamel defects according to the FDI classification system for developmental defects of dental enamel. This classification system, designed to allow for accurate recording of "developmental" defects, does not distinguish between white-cream opacities and white lesions that are probably not developmental in origin.

We found an unusually high prevalence of WCO on the gingival third of the facial surface and middle third of the mesial and distal surfaces. These sites on primary anterior teeth are where "white spot" enamel decalcifications often develop as a result of plaque accumulation. Since WCO at these locations probably were not developmental defects, we eliminated them from the analyses of developmental defects. By doing so, we hoped to obtain more accurate estimates of the prevalence and distribution of developmental enamel defects. Eliminating 36 teeth with such WCO reduced the prevalence of these defects to 3.9% (20/509), resulting in a 33.6% prevalence of developmental enamel defects (DED) in our sample. This rate is consistent with prevalences of DED found in primary dentitions of normal populations as reported by Murray and Shaw (1979) and Nation et al. (1987), and significantly higher than those reported by Holm and Arvidsson (1974) and Hargreaves et al. (1989, Table 4). HD accounted for 59.6% of the DED, which is similar to the 63.6% reported by Nation et al. (1987). HD occurred more frequently in our sample than opacities, even though HD are presumed to require a more severe developmental insult than is needed to produce opacities.

Our prevalences are based on the examination of a single primary anterior tooth from an individual child. In the studies of normal populations previously cited, prevalences were based on an examination of the entire dentition of each child. Therefore, our prevalence of 33.6% may be an underestimate because more defects might have been detected if all 20 primary teeth of each child had been examined.

Tooth examination procedures also differ among studies. In most studies, the dentitions were examined in a clinical setting and varied in illumination, equipment, preexamination prophylaxis, and drying (Table 4). Such examinations also precluded detection of defects in areas of tooth contact. In our study, individual teeth were critically examined in hand using a 4x magnifying loop with excellent illumination after careful cleaning and drying. If similar critical examinations could be made "in the field," actual prevalences for DED of the primary dentition in the previously reported studies also might have been greater.

HD were the most prevalent defects of all individual anterior primary teeth examined in this study. In addition, they were the most prevalent defect on four of the five surfaces examined and on all locations of the affected surface. They occurred most frequently on the facial surface (52.9%, $P \ll .001$), especially on the canines (84.4%).

The lack of differences in the prevalences of DED between gender and between right and left sides of the mouth are expected and are in agreement with most similar studies. Only Brown and Smith (1986) found a higher prevalence of HD in males. Skinner and Hung reported a higher prevalence of HD on the right side of the mouth. Their sample of Vancouver children however, had an extremely low prevalence of defects (2.4%).

Our finding of higher DED prevalences among Black children (41.7%) than among non-Black children (33.0%), did not achieve nominal statistical significance. Others, however, have found that Blacks had HD prevalences that are approximately twice as high as the rates in Caucasians (Nation et al. 1987; Hargraeves et al. 1989; and Skinner and Hung 1989; Silberman et al. 1989).

Distribution and Proposed Etiology

The location and distribution of developmental defects on enamel surfaces usually have been explained entirely in terms of the chronology of developing enamel (Goodman and Aramelagos 1985). The time of crown development is certainly a critical factor in determining the distribution of enamel hypoplasias. The prevalence of DED in our study was significantly higher on the maxillary teeth, on the facial surfaces (lowest on the incisal/cuspal) of affected teeth and on the middle third (lowest on the gingival third) of the affected surface. If DED reflected systemic factors, then all actively calcifying teeth would be affected equally with distribution of the defects on all surfaces of the crown.

Goodman and Armelagos (1985) reported that the distribution of enamel hypoplasias occurred most commonly on the middle third of all permanent tooth types examined. They suggested that biological gradients in susceptibility to ameloblastic disruption, as well as morphological factors, such as enamel prism length and direction might, in addition to chronological development, effect the distribution of enamel defects.

The thickness of enamel might explain our results that developmental defects were seen most commonly on maxillary teeth, facial surfaces, and the middle third of the crown. The primary maxillary anterior teeth have thicker enamel then their mandibular counterparts. Primary anterior teeth generally have thicker enamel on their facial surfaces and in the middle third of the crown. In addition, the incisal/cuspal third of exfoliated primary anterior teeth have the thinnest enamel and usually are worn significantly, precluding observation of enamel defects in many instances.

Kraus and Jordan (1965) explain that the varying thicknesses of enamel in the same tooth may be due to "different rates of enamel apposition in different parts of the same tooth... regardless of whether or not the ameloblastic life spans differ, or whether or not calcification ceases simultaneously throughout the crown..." This postulate is supported by our observations that the thickest surfaces and locations exhibited the highest prevalence of DED. If the secretion and maturation of enamel occurs most rapidly on these thicker teeth, surfaces, and locations, then the greater metabolic demand of the ameloblasts in these areas might make them especially vulnerable to any insult. A severe metabolic disturbance might affect all teeth and surfaces, while a milder perturbation might preferentially affect the most metabolically active ameloblasts or the most rapidly maturing enamel.

Susceptibility to local trauma, however, may be more a function of tooth location than of metabolic demand. For example, because of their position in the oral cavity and within the dental arch, the maxillary primary dentition and middle third of the facial surfaces may be most susceptible to local mechanical insults. These locations are especially prone to trauma because of the labial position of the maxilla and have little protection due to a thin cortical plate. Metabolic and mechanical insults also may act together. Seow et al. (1989) suggested that neonatal hypocalcemia may decrease the thickness of the cortical mass of bone, thereby increasing the chance of mechanical trauma to the middle third of the facial surfaces of the primary teeth.

In summary, hypoplastic defects of the primary maxillary incisors are probably due to a perinatal systemic insult to susceptible secreting ameloblasts. Hypoplastic defects of the primary canines and all opacities may be primarily the result of mechanical trauma. Their development also may be influenced by systemic insults, which further increase the susceptibility of the secreting or maturing ameloblasts to mechanical trauma.

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Dr. Needleman is associate dentist-in-chief, The Children's Hospital and associate clinical professor in pediatric dentistry, Harvard School of Dental Medicine, Boston, MA. Dr. Leviton is associate in neurology, The Children's Hospital and associate professor of neurology, Harvard Medical School. Ms. Allred is senior systems analyst, Neuroepidemiology Unit, The Children's Hospital, Boston, MA.

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