Prevalence and Age at Development of Enamel Hypoplasias in Mexican Children

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ABSTRACT   Enamel hypoplasias, deficiencies in enamel thickness resulting from disturbances during the secretory phase of enamel development, are generally believed to result from nonspecific metabolic and nutritional disruptions. However, data are scarce on the prevalence and chronological distribution of hypoplasias in populations experiencing mild to moderate malnutrition. The purpose of this article is to present baseline data on the prevalences and chronological distributions of enamel hypoplasias, by sex and for all deciduous and permanent anterior teeth, in 300 5 to 15-year-old rural Mexican children.

Identification of hypoplasias was aided by comparison to a published standard (Federation Dentaire Internationale: Int. Dent. J. 32(2):159–167, 1982). The location of defects, by transverse sixths of tooth crowns, was used to construct distributions of defects by age at development.

One or more hypoplasias were detected in 46.7% (95% CI= 40.9–52.5%) of children. Among the unworn and completely erupted teeth, the highest prevalence of defects was found on the permanent maxillary central incisors (44.4% with one or more hypoplasias), followed by the permanent maxillary canine (28.0%) and the remaining permanent teeth (26.2 to 22.2%) Only 6.1% of the completely erupted and unworn deciduous teeth were hypoplastic. The prevalence of enamel defects on the permanent teeth was up to tenfold greater than that found in studies of less marginal populations that used the FDI method.

The prevalence of defects in transverse zones suggests a peak frequency of hypoplasias during the second and third years for the permanent teeth, corresponding to the age at weaning in this group. In the deciduous teeth, a smaller peak occurs between 30 and 40 weeks post gestation. The frequency of defects after three years of age is slightly higher in females than males, suggesting a sex difference in access to critical resources.

Dental enamel hypoplasias have long been considered to result from periods of general physiological disruption (Kreshover, 1960) and to reflect the chronological pattern of stress during periods of enamel development (Sarnat and Schour, 1941). Laboratory research designed to induce defects in experimental animals has demonstrated that a wide variety of noxious stimuli, including fever (Kreshover and Clough, 1953), infectious agents (Kreshover et al., 1953, 1954), and under- and overnutrition (Becks and Furata, 1941; Mellanby, 1929; Wolbach and Howe, 1933), may cause enamel defects.

The applicability of these studies to humans is supported by the results of early

clinical studies on the association between enamel defects and diseases in utero and during infancy (Evans, 1947; Grahnen and Selander, 1954). In summarizing the results of these early studies, Kreshover (1960:166) states that

> ample clinical and experimental evidence exists to suggest that developmental tooth disturbances are generally nonspecific in nature and can be related to a wide variety of systemic disturbances, any of which, depending upon their degree of severity and degree of tissue response, might result in defective enamel....

This conclusion is supported by more recent reviews of the epidemiology of dental enamel defects (Cutress and Suckling, 1982; Pindborg, 1982). Cutress and Suckling (1982) note that nearly 100 factors have now been reported to be associated with enamel defects. Since a wide variety of insults may cause hypoplastic defects and their appearance seems to be unrelated to the type of causal agent (Suckling and Thurley, 1984), enamel hypoplasias are best considered to be nonspecific indicators of metabolic and nutritional disruption (Cutress and Suckling, 1982; Guita, 1984; Pindborg, 1982; Shafer et al., 1983; Yaeger, 1980).

Although enamel defects are generally agreed to reflect a period of metabolic and/or nutritional disruption, few studies have been designed to assess the utility of enamel defects as supplemental indicators of adverse living conditions. In the 1960s and early 1970s a series of investigators noted high frequencies of enamel hypoplasias and other enamel defects in populations from underdeveloped regions (Arkle, 1962; Baume and Meyer, 1966; Enwonwu, 1973; Infante, 1974; Infante and Gillespie, 1974; Moller et al., 1972; Sweeney and Guzman, 1966; Sweeney et al., 1969, 1971). For example, Sweeney et al. (1971) noted that 73% of Guatemalan children with third-degree malnutrition and 43% of children with second-degree malnutrition had hypoplasias in enamel formed prior to the diagnosis of malnutrition. Jelliffe and Jelliffe, in commenting on the studies of Sweeney and others and citing their own data on enamel hypoplasias and circular caries (hypoplastic defects with caries formation at the site of the defect) in the Caribbean (Jelliffe and Jelliffe, 1961; Jelliffe et al. 1954,

1961), conclude that "further studies of its etiology and public health consequences seem overdue" (1971:893). However, we are aware of only one study published after 1974 on enamel defects in a marginal population. This study (Sawyer and Kwoku, 1985) of 45 malnourished children supports the results of Sweeney et al. (1971).

While a handful of studies exist on the prevalence of enamel defects in marginal populations, it is rare to find studies of the age at development or chronology of enamel defects in any group. The first chronology of enamel defects was constructed by Sarnat and Schour (1941), based on 60 children from the greater Chicago area. They found that two thirds of defects developed during the first year and all but 2% of the remaining defects developed after 35 months. Since this time, analyses of enamel hypoplasias in permanent teeth of skeletal series have established peak frequencies of defects between 2 and 5 years of age (Goodman et al., 1984; Schulz and McHenry, 1975; Swardstedt, 1966), and Cook and Buikstra (1979) and Blakey and Armelagos (1985) have showed that enamel defects of deciduous teeth in prehistoric Amerindians tend to occur during the third trimester and perinatally. We are aware of only one description of the chronology of defects in contemporary populations. In a pilot study of Jordanian children from two villages and a nomadic group, Alcorn and Goodman (1985) found a peak period of hypoplastic defects between 1 and 3 years of age.

The decline in studies of enamel defects in marginal populations may be attributed to a series of methodological problems and an inadequate understanding of intratooth variations in susceptibility to defects. Fortunately, three issues that had not been addressed in the 1970s have now been resolved, at least to a degree: 1) establishment of a replicable typology of enamel defects, 2) establishment of a method for assessing the age of individuals at the time of development of defects, and 3) development of understanding of the degree of differential susceptibility of teeth to defects and the role of these differences in explaining previous research findings.

In 1977 a working group of the Federation Dentaire Internationale (FDI) proposed a system for the coding of dental defects suitable for epidemiological study (FDI, 1982). The key feature of this report is the inclusion of a series of high quality color prints of dental defects to be used as standards (FDI,

1982:162–163). The authors of this report note that the lack of a well-defined and accepted classification of defects has inhibited both the comparability and replicability of results. Two epidemiological studies of dental defects using FDI coding have included intraobserver reliability estimates. Murray et al. (1984) noted an 82% agreement in the recording of opacities, and Suckling and Pearce (1984) and noted a 95% agreement on the presence of a defect of any type.

Establishing the age of an individual at the time of defect development requires the use of a tooth development standard and a method for locating defects on tooth crowns. For most purposes, the calcification standard of Massler et al. (1941) has been used. The location of defects in skeletal samples has been achieved by measurement of the distance of a defect from a landmark such as the cementoenamel junction. Such a method, however, is impractical in epidemiological studies of living subjects.

In a pilot study with low-birthweight infants, it was noted that a trained observer could reliably locate defects by transverse sixths of tooth crowns without use of a measurement device (Goodman, unpublished observations). Division of crowns into sixths yields chronological zones of approximately 2 to 3 months duration in the deciduous anterior dentition and approximately 7 to 12 months duration in the permanent anterior dentition.

Finally, Goodman and Armelagos (1985a,b) have noted up to eightfold differences in the prevalence of enamel hypoplasias among different teeth types. It is therefore very likely that this source of variation has confounded many prior studies and confused comparison of studies that relied on dissimilar teeth. This source of variation may be accounted for by presenting tooth-specific rates.

In summary, a review of the literature suggests that such defects have great potential as indicators of stress during development and that they may provide a uniquely time-specific indication of stress. Studies in the 1960s and early 1970s have established that these defects are frequent in marginal populations and often anteceded the clinical appearance of malnutrition. However, further study of the utility of these defects may have been hindered by a lack of a reliable typology and method for estimating the age at development of these defects and a poor understanding of the confounding effects of intratooth differences in susceptibility to de-

fects. Based on recent advances in our understanding of these issues, the potential use of enamel defects in studies of marginal groups should be reconsidered.

In this article we present results of a study of the prevalence and time of development of enamel defects in children from five communities in the Solis Valley, Mexico. The specific purposes are 1) to test the interobserver and intraobserver reliability of these methods in a field situation, 2) to establish the frequency of defects in children by sex and toothtype, 3) to establish the age of these children at time of defect development, and 4) to consider the meaning of the above data in relationship to preliminary data on the current health and anthropometric status of these children and the socioeconomic level of their families and villages.

MATERIALS AND METHODS

The sample consists of 300 Ladino children, 5 to 15 years old, from five communities in the Solis Valley, Mexico. These communities are being studied by the Collaborative Research Support Program (CRSP), marginal malnutrition and function project.[1] The Solis Valley is located approximately 170 km northwest of Mexico City. These communities were selected by the CRSP because they met the following criteria: Previous research in these agricultural communities indicated that they would be receptive to an intensive research effort, diets were relatively stable, outmigration was minimal, and a diversity of economic and nutritional status existed.

The sample was derived from a list of families who were directly involved in the CRSP project by virtue of their having a "target" school-aged child (7 to 9 years old). Each of these children is followed intensively for a 1-year period, during which measurements are made of health, anthropometry, food intake, psychosocial and behavioral development, and socioeconomic and environmental conditions. Selection of target individuals ensured the availability of a wide array of supplemental data for later comparison with the dental data. Additional measures were made on the family unit (e.g., social and economic

conditions) or were performed on other family members (e.g., morbidity and anthropometry) at less frequent intervals. Thus, the list of potential candidates for the present study included both CRSP "target" individuals and their school-aged siblings.

The recruitment of students and the observation and recording of dental conditions were performed at the five community schools. Students were recruited from the above list by the headmasters of each school. The original list contained 474 possible students (112 "target" children and 362 siblings). Target children were called first and 98 (87.5%) were found and examined. At the one school where we were able to see all listed children, 74 out of 81 (91.3%) were examined. The high rate of matching of children on our list to those attending school lends support to the community social workers' view that almost all school-aged children attend their community schools. A possible bias exists in this sample toward healthier children (attending schools on the study days) and from better-off families (able to keep their children in school). However, given the high response rate, it is likely that this bias is minimal.

Defects were recorded on the anterior dentition (upper and lower canines and incisors) because these teeth are the easiest to examine, have been most often studied by other researchers, and have relatively high defect rates (Cutress and Suckling, 1982; Goodman and Armelagos, 1985b). As most individuals in this sample had mixed dentitions, data have been recorded for both deciduous and permanent anterior teeth, depending on which were present. Information on defects was derived from the most complete side.

The method used for identification and recording of enamel defects is a modification of the FDI method (FDI, 1982). The FDI suggests the recording of the following types of defects: 1) opacity, white/cream; 2) opacity, yellow/brown; 3) hypoplasia, pits; 4) hypoplasia, horizontal grooves; 5) hypoplasia, vertical grooves; 6) hypoplasia, missing enamel; 7) discolored enamel, not associated with opacity; 8) other defects; and 9) combinations of defects. Before recording we modified the FDI method so that combination defects were described in terms of the specific type of defects that they incorporated.

The FDI method was further modified to take into account the time of development of defects. The anterior tooth crowns were divided into six transverse zones of approximately equal width, from an incisal to a gingival zone. Since many permanent teeth had not completely erupted, a portion of the gingival end of these teeth was frequently missing. Similarly, many teeth exhibited severe attrition, thus eliminating a portion of the incisal border. In these cases one or more zones was recorded as missing. If any portion of a zone was judged to be missing, the entire zone was scored as missing.

Defects were recorded by either an anthropologist (A.H.G.) or a dentist (G.P.H.). After a training and standardization period, intraobserver and interobserver reliability were assessed. Reliability data are presented on the number of enamel zones lost to study because of incomplete eruption and attrition, presence or absence of enamel hypoplasias, and location of enamel hypoplasias.

In this study we consider two types of hypoplastic defects: pitting hypoplasias (FDI type 3) and horizontal grooves or lines (combined FDI types 4 and 6). Type 6 defects (missing enamel) have been combined with type 4 defects (grooves) because they could not be reliably distinguished. Enamel hypocalcifications (FDI defect types 1 and 2) will be considered elsewhere. Type 5 defects (vertical grooves) have not been considered because of low reliability (this is the only defect for which the FDI has not provided a standard).

The frequency of hypoplastic defects by transverse zone is converted to ages at time of development of enamel defects. This procedure involves estimating times of initiation and ending of enamel crown calcification and then dividing the crown into six equal-width zones. The calcification chronology is based on the data from Massler et al. (1941) for the permanent teeth and on Lunt and Law's revision (1974) of Massler and coworkers' data (1941) for the deciduous dentition. Thus, for example, the permanent mandibular incisors are estimated to begin crown calcification at birth and end crown calcification at 4 years of age. The 48-month period of development is then divided into six time periods of 8 months, with midpoints at 4, 12, 20, 28, 36, and 44 months. Finally, sex-specific frequencies of defects are presented for all teeth and by developmental zones of permanent incisors.

RESULTS
Interobserver and intraobserver reliability

Dental defect data were scored by both observers for the same 30 individuals, yielding 177 teeth for study. Ten individuals, with a

total of 59 teeth, were observed twice by the same investigator (A.H.G.)

In the interobserver study, there was agreement on the precise number of zones having attrition or being unerupted in 145 of 177 (81.9%) teeth (Table 1). A disagreement on one zone was found in 29 (16.4%), and a disagreement on two zones was found in the remaining three (1.7%) teeth. In total, agreement was reached on the presence for study of 1,027 of 1,062 zones (96.7%).

In the intraobserver study, there was agreement on the number of zones present for study in 51 of 59 (86.4%) teeth, while a disagreement on one zone was found in the remaining eight teeth (13.6%; Table 1). In total, agreement was reached on the presence of 346 of 354 (97.7%) zones. The intraobserver agreement is not significantly greater than the interobserver agreement ($x^2 = 0.9$).

Of 37 enamel hypoplasias scored by both observers in the interobserver study, there was agreement on their location in 30 (81.1%) cases. Similarly, of 18 hypoplasias scored both times in the intraobserver study, agreement on location was found in 16 (88.9%) cases (Table 1). All disagreements in both studies were on a single zone. The percentage of intraobserver agreement is not significantly greater than the interobserver agreement ($x^2 = 0.14$).

In the interobserver study 52 and 56 teeth were found to have defects by the first observer and second observers, respectively. Agreement on the presence or absence of a defect was found in 143 (80.8%) cases (Table 1). This agreement is significantly greater than chance (53.1% chance agreement, $\kappa = 0.628$ [SE = 0.157], z = 4.0, P <0.001) (Fleis, 1977:144–147).

Twenty-one and 23 teeth were found to have defects during the first and second observations in the intraobserver study. Agreement on the presence or absence of a defect was found in 51 of 59 (86.4%) cases (Table 1) and is significantly greater than by chance (57.6% chance agreement, $\kappa = 0.547$ [SE = 0.07], Z = 7.8, P <0.001). Again, the intraobserver agreement is not significantly greater than the interobserver agreement ($x^2 = 1.42$).

Prevalence of enamel hypoplasias

More than one third of the children (35.7%) have a transverse hypoplastic groove or missing enamel, and 15.3% have one or more hypoplastic pits (95% CI=30.2–41.2% and 11.1–19.5%, respectively). Nearly half of the children (46.7%) have one or more hypoplastic defects (95% CI=40.9–52.5%).

While the prevalence of hypoplastic defects per child is high, there is great variation in prevalence by tooth (Table 2). Among the permanent teeth, the highest prevalence of hypoplastic pits, hypoplastic grooves, and combined hypoplastic defects is found on the maxillary central incisor. Of the 162 complete permanent maxillary central incisors, 8.6% have a hypoplastic pit, 37.7% have a hypoplastic groove, and 44.4% have either a pit or groove (Table 2) (95% CI=38.7–50.1% for total defects).

Among the remaining complete permanent teeth (Table 2) the prevalence of hypoplastic pits varies between 0 and 6.2%, and the prevalence of hypoplastic grooves varies between 19.4 and 28.0%. Pits are slightly more frequent on incisors than on canines, and grooves are slightly more frequent on maxillary than on mandibular teeth. The prevalence of either pits or grooves varies between 22.2 and 28.0% for the remaining permanent teeth.

There are only 36 complete deciduous teeth in our sample. Two (6.1%) are hypoplastic. Therefore, comparisons were made by examining deciduous teeth with one or more zones present (Table 2). Compared to the permanent teeth, a lower frequency of defects is found on the deciduous teeth. Pits and grooves are about equally common, with prevalence rates ranging from 8.0% pits and 6.0% grooves on the maxillary central incisor to 0% for both pits and grooves of the mandibular lateral and central incisors. The frequency of combined pits or grooves varies from 14.0% for the maxillary central incisor to 0% for the mandibular incisors.

Age at development of defects: Permanent dentition

Enamel hypoplasias (either pits and grooves) are often found in all zones of all permanent anterior teeth (Table 3). The highest frequency of defects (28.3%) is found in the third zone of the maxillary central incisor, and the lowest frequency (3.8%) is found in the sixth zone of the mandibular central incisor. With the exception of a high frequency of defects in the sixth zone of the maxillary canine (23.3%), the highest frequency of defects tends to occur in the second through fourth zones of these teeth.

Based on frequencies of defects by transverse zones (Table 3), the age at development of enamel hypoplasias has been plotted for the four incisors (Fig.1). Relatively low frequencies of enamel hypoplasias are found during the first year, higher rates during the

TABLE 1. Inter- and intraobserver agreement in number of zones present (based on variation in agreement on the degree of attrition and eruption), location of enamel hypoplasias, and presence or absence of enamel hypoplasias

	Interobserver		Intraobserver	
Agreement	No.	%	No.	%
No. of zones present	145	81.9	51	86.4
Location of enamel hypoplasia	30	81.1	16	88.9
Presence or absence of enamel hypoplasia	143	80.8	51	86.4

TABLE 2. The prevalence (%) of enamel hypoplasias by tooth for teeth with all zones present and teeth with one or more zones present

		Type		
	No.	3	4–5	3–5
Complete teeth				
Perm. max. I1	162	8.6	37.7	44.4
Perm. max. I2	65	6.2	20.0	26.2
Perm. max. canine	25	0.0	28.0	28.0
Perm. man. I1	206	4.9	19.4	23.3
Perm. man. I2	124	4.0	21.0	24.2
Perm. man. canine	36	2.8	19.4	22.2
One or more zones present				
Perm. max. I1	249	8.4	34.5	40.6
Perm. max. I2	190	6.8	16.8	23.7
Perm. max. canine	61	1.6	18.0	19.7
Perm. man. I1	279	5.4	16.1	20.8
Perm. man. I2	238	2.5	14.3	16.4
Perm. man. canine	84	2.3	13.1	14.3
Decid. max. I1	50	8.0	6.0	14.0
Decid. max. I2	89	3.4	3.4	6.7
Decid. max. canine	228	2.6	1.7	3.9
Decid. man. I1	19	0.0	0.0	0.0
Decid. man. I2	56	0.0	0.0	0.0
Decid. man. canine	210	2.3	0.5	2.9

Type 3, hypoplastic pits; type 4–5, hypoplastic grooves or missing enamel. Note: The frequency of type 3 plus types 4 and 5 defects may be greater than the frequency of combined defects because both types may be present on the same tooth.

TABLE 3. Prevalence (%) of enamel hypoplasias by developmental zones for the permanent anterior teeth

	Developmental zones					
Permanent	1	2	3	4	5	6
Maxillary						
I1	1.37 (249)	27.1 (247)	28.3 (244)	27.3 (238)	13.0 (214)	11.1 (162)
I2	10.0 (189)	14.9 (188)	15.7 (184)	11.8 (161)	6.3 (127)	7.5 (67)
Canine	14.3 (56)	16.1 (56)	20.8 (53)	18.3 (49)	17.5 (40)	23.3 (30)
Mandibular						
I1	6.9 (276)	14.1 (279)	12.4 (276)	9.2 (273)	4.5 (246)	3.8 (211)
I2	8.4 (238)	13.0 (237)	11.2 (233)	8.3 (217)	4.8 (166)	6.5 (124)
Canine	12.0 (83)	11.1 (81)	14.6 (75)	13.4 (67)	13.2 (53)	12.8 (39)

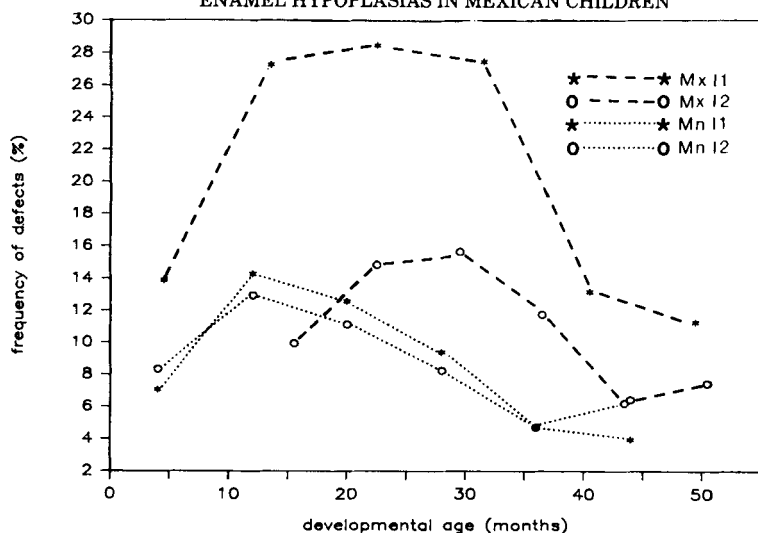Values in parentheses are numbers of teeth.

Fig. 1. The chronological distribution of enamel hypoplasias on the permanent dentition. MxI1, maxillary central incisor; MxI2 maxillary lateral incisor. Data are based on frequencies of enamel defects by transverse sixths (Table 3).

second and third years, and lower rates again after 36 months.

Among these teeth the highest frequency of enamel hypoplasias at all ages is found in the maxillary central incisor. The other three incisors have comparable frequencies of enamel hypoplasias, with rates equivalent to about one half of the maxillary central incisor rate (Fig. 1).

Age at development of defects: Deciduous dentition

Low frequencies (less than 10%) of enamel hypoplasias are generally found per transverse zone in the deciduous anterior teeth (Table 4). The highest frequency of hypoplasias is found in the fourth zone of the maxillary central incisor (9.4%). No defects were found in 18 of 54 zones (33.3%), including all of the first developing (incisal) zones of all teeth.

The age at development of enamel hypoplasias has been plotted for four deciduous teeth, maxillary lateral and central incisors, and both canines (Fig. 2). The highest frequency of enamel hypoplasias occurs during the last half of pregnancy. Moderately high frequencies are found during the middle trimester and around birth (or 42 weeks gestation), while low frequencies are found postnatally. If there is a high prevalence of premature births then the peak in the maxillary central incisor might be near the actual time of birth.

Male–female differences

Females have a higher frequency of hypoplasias on all permanent teeth, with the ex-

ception of the mandibular central incisor, where the male frequency is 3% greater (24.8 v 21.8%; $= -\chi^2$ 0.25; Table 5). The female rate is over twice as high (33.3 v 15.3%) on the mandibular lateral incisor ($\chi^2 = 4.90$, P $=0.03$).

Based on an analysis of sex differences in the rate of enamel hypoplasias by developmental zone (Table 6), it is unlikely that the increased frequency of defects in females is due to differential attrition or eruption. Female rates are significantly greater than male rates (P < 0.05) for six of 24 zones, four on the lateral mandibular incisor and two on the lateral maxillary incisor. Females remain at high risk for enamel defects in the third year, while the male rate drops by this time. As an example of this phenomenon, the male rate of hypoplasia falls to below 10% at all time periods in the mandibular lateral incisor. However, the female rate hovers near or above 10% for all zones. Differences are significant during the first zone (birth to 8 months), third zone (16 to 24 months), and fifth and sixth zones (32 to 48 months).

DISCUSSION
Reliability and methodological implications

This study of dental defects in a mild to moderately undernourished population incorporates several unique aspects: 1) enamel defects were assessed in a field situation in a nonwestern country using the FDI standard, 2) tooth-specific prevalence rates were collected for a population experiencing mild to

TABLE 4. *Prevalence (%) of enamel hypoplasias by developmental zones for the deciduous anterior teeth*

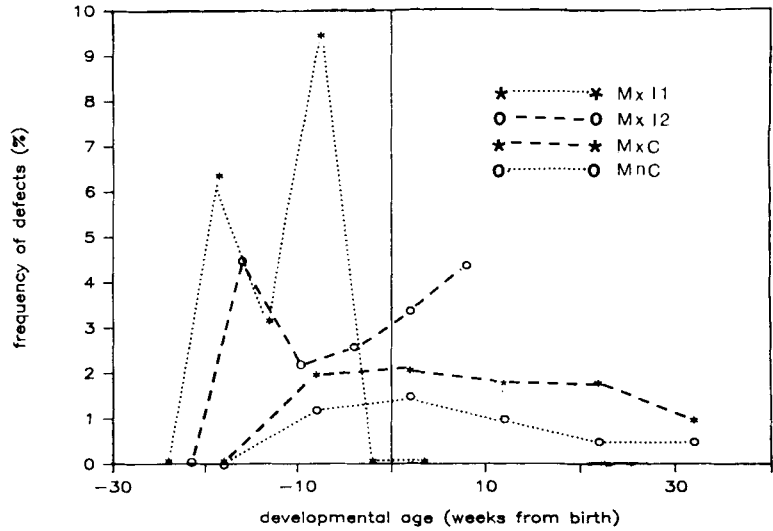| Deciduous	Developmental zones					
	1	2	3	4	5	6
Maxillary						
I1	0.0	6.3	3.1	9.4	0.0	0.0
	(9)	(16)	(32)	(43)	(50)	(50)
I2	0.0	4.5	2.0	2.6	3.4	4.4
	(5)	(22)	(51)	(78)	(88)	(89)
Canine	0.0	1.9	2.0	1.7	1.7	0.9
	(3)	(53)	(191)	(225)	(228)	(228)
Mandibular						
I1	0.0	0.0	0.0	0.0	0.0	0.0
	(5)	(10)	(17)	(18)	(18)	(18)
I2	0.0	0.0	0.0	0.0	0.0	0.0
	(4)	(17)	(44)	(55)	(55)	(55)
Canine	0.0	1.2	1.5	1.0	0.5	0.5
	(13)	(83)	(194)	(209)	(210)	(208)

Values in parentheses are numbers of teeth.

Fig. 2. The chronological distribution of enamel hypoplasias on the deciduous dentition. MxI1, maxillary central incisor; MxI2, maxillary lateral incisor; MxC, maxillary canine; MnC, mandibular canine. Data are based on frequencies of enamel defects by transverse sixths (Table 4). Birth is based on those carried to full term.

TABLE 5. *Comparison of the frequency of enamel hypoplasias found on the permanent dentition of males and females*

	Males		Females			
	Sample size	% Defects	Sample size	% Defects	χ^2	P
Max. I1	81	42.0	81	46.9	0.40	NS
Max. I2	31	19.4	34	33.4	1.42	NS
Max. canine	8	12.5	17	35.3	1.40	NS
Man. I1	105	24.8	101	21.8	0.25	NS
Man. I2	59	15.3	65	33.3	4.90	0.03
Man. canine	12	16.7	24	25.0	0.32	NS

χ^2 values are without continuity correction; P values are two-tailed.

TABLE 6. Comparison of the frequency of enamel hypoplasias by developmental zones
on the permanent incisors of males and females

Tooth and zone	Males		Females		χ^2	P
	Sample size	% Defects	Sample size	% Defects		
Max. I1						
1	121	11.6	128	15.6	0.86	NS
2	121	24.8	126	29.4	0.65	NS
3	118	26.3	126	30.2	0.45	NS
4	117	27.4	121	27.3	0.01	NS
5	104	13.5	110	12.7	0.02	NS
6	81	7.4	81	14.8	2.25	NS
Max. I2						
1	89	7.9	100	12.0	0.89	NS
2	89	11.2	99	18.2	1.78	NS
3	87	12.6	97	18.6	1.20	NS
4	77	10.4	84	13.1	0.28	NS
5	62	1.6	65	10.8	4.51	0.03
6	33	0.0	34	14.7	5.24	0.02
Man. I1						
1	136	4.4	140	9.3	2.56	NS
2	137	15.3	142	12.7	0.40	NS
3	137	11.7	139	12.9	0.10	NS
4	136	8.1	137	10.2	0.37	NS
5	122	3.3	124	5.6	0.80	NS
6	106	1.9	105	5.7	2.12	NS
Man. I2						
1	117	4.3	121	12.4	5.10	0.02
2	116	9.5	121	16.5	2.58	0.10
3	114	6.1	119	16.0	5.67	0.01
4	106	6.6	111	9.9	0.78	NS
5	82	1.2	84	8.3	4.58	0.03
6	59	1.7	65	10.8	4.22	0.04

χ^2 values are uncorrected; P values are two-tailed.

moderate malnutrition, 3) individual's ages at time of development of defects were assessed in a field situation, and 4) a training session has been adopted to improve intraobserver and interobserver reliability.

The FDI typology of defects showed relatively high intraobserver agreement in two other studies, both involving children studied in schools with the aid of a dental chair and artificial lights (Murray et al., 1984; Suckling and Pearce, 1984). Our intra- and interobserver agreement scores for identification of hypoplastic defects suggest that these defects may be scored under most field conditions with only minimal dental equipment (probes, mirrors), a chair, and natural lighting.

Interobserver agreement on the presence or absence of an enamel hypoplasia was reached on 80.8% of 177 teeth (Table 1). This rate of agreement is comparable with the intraobserver rate found in this study (86.4%) and rates noted by Murray et al. (1984) and Suckling and Pearce (1984) for all enamel defects. These results suggest that the publication of the FDI standard has increased re-

liability. Furthermore, this standard may be used by someone trained in dentistry, but with only limited (less than 2 weeks) training in the evaluation of dental defects. One potential use of enamel hypoplasias is that they may provide a relatively quick and inexpensive indication of the degree of stress experienced during tooth development. However, this potential will not be reached, and a large body of comparative data will be slow to develop, if the method can not be learned in a short amount of time.

Relatively high agreement was reached in both the intra- and interobserver studies on the number of zones present for assessment of defects and the location of defects. The high interobserver rates, which almost equal the intraobserver rates, were reached only after an initial training period, again suggesting the importance of adequate training.

To assess an individual's age at the time of defect development in Jordanian children, Alcorn and Goodman (1985) noted the degree of attrition and gingival emergence in the field and then took photographs of the anterior teeth. Defects were identified from pho-

tographs, and chronologies were constructed by measurement of the relative distance of defects from the gingiva and occlusal surface. This technique, however, is limited, since extensive time, expense, and expertise is required to take adequate photographs of a large sample. In general, the high degree of agreement on the location of defects by transverse sixths of enamel crowns suggests that this may be the method of choice in situations that do not allow for more objective techniques.

It is difficult to estimate the variation between the estimation of an individual's ages at the times of development of defects and his chronological ages. The standard of Massler and coworkers has been justified as the best available for studies of most anthropological populations (Goodman and Armelagos, 1985a). Both individual and population variations surely exist, especially under adverse living conditions. However, these variations may be relatively minor considering the high degree of genetic control over the time of tooth calcification (Garn et al., 1965; Garn, 1977). Longitudinal studies of dental calcification and defect development under adverse living conditions will certainly help to improve the validity of methods for estimating an individual's age at defect formation.

The recoding of defects by location on tooth crowns is the most important innovation of this study. The primary purpose in collecting data in this manner has been to estimate the individual's age at development of defects. While this procedure is not error-free, it may provide essential data. First, taking advantage of the chronological nature of dental defects provides a time-specific indication of stress. In many situations this may be the only method for estimation of the degree of stress by individual's age. Second, in a longitudinal study, location information may help in establishing both the cause of defects and the timing of dental calcification. Third, location information can help in understanding prevalence differences among teeth. For example, higher defect rates in a canine versus an incisor may be due to defect formation after the incisor crown has formed. This method has been presented as a first effort to provide information on the chronology of defects in a less-developed country. It is expected that future studies will refine this procedure.

The frequency of enamel defects

The 46.7% prevalence of enamel hypoplasias in this study is greater than that found in four of five other studies that have used the FDI method. Enamel hypoplasia rates of less than 5% have been noted by Murray et al. (1984) in a study of English children, by De Liefde and Herbison (1985) in New Zealand children, and by Cutress et al. (1985) in another study of New Zealand children. In a third study in New Zealand, Cutress and Pierce (1984) report that less than 10% of children have either missing enamel, hypoplastic pits, or horizontal lines. On the other hand, King and Brook (1984) note that 63.9% of dental students in Hong Kong have an enamel hypoplasia on at least one tooth. Interestingly, no individual had a defect on more than seven teeth, suggesting local and nonsystemic causes.

Variation in prevalence of enamel hypoplasias among these studies suggests the need to consider differences in environmental and genetic factors that may account for these rate differences. The high rate of hypoplasias found in our study is unlikely to be due to high fluoride concentrations in water. Two water samples were taken from the source serving the valley and were found to contain 0.20 ppm fluoride (Dr. N. Tinnanoff, personal communication), well below the level considered to cause enamel defects. It is also unlikely that genetic factors, local traumas, or drug toxicities are important causes. The high rate of hypoplastic defects found in this region suggests the need to test for the existence of a gradient of increased frequency of hypoplasias with decreased dietary adequacy and increased infant-childhood stress.

The increased prevalence of enamel defects on the permanent central incisors (Table 2) supports the results of prior studies of the pattern of defects among permanent teeth (Goodman and Armelagos, 1985b; Cutress and Suckling, 1982). These results specifically support the view that teeth that are under greatest genetic control, such as maxillary central incisors, may be the most susceptibile to enamel hypoplasias (Goodman and Armelagos, 1985a,b).

The implication of these intratooth variations in susceptibility to defects for future studies is that tooth-specific rates must be reported. Comparisons of results from studies based on different teeth are highly prob-

lematic. Whole mouth or individual rates, based on some combination of teeth, are equally difficult to interpret. Finally, differences within studies in the complement of teeth studied and their degree of attrition and eruption may influence rates and confound within-study comparisons.

The rate of defects on deciduous teeth is considerably lower than the rate of defects found on permanent teeth (Table 3). Among deciduous teeth, the maxillary central incisor is most often hypoplastic. A similar pattern of defects has been found by both Funakoshi et al. (1981) and Johnsen et al. (1984) in studies of low-birthweight infants. This increased susceptibility might also have been long-realized by others as this tooth has been the one most frequently included in studies of dental defects on primary teeth (e.g., Infante, 1974; Infante and Gillespie, 1974; Sweeney and Guzman, 1966; Sweeney et al., 1969, 1971). Finally, the increased frequency of defects on the deciduous antecedent of the maxillary central incisor indicates that the field of genetic control over development of the deciduous teeth is similar to the permanent dentition's field.

Comparison of the prevalence of hypoplasias on the deciduous maxillary central incisor with the rates and description of defects reported by Infante (1974; Infante and Gillespie, 1974) and Sweeney (Sweeney and Guzman, 1966; Sweeney et al., 1969, 1971) suggests that the defects found in this study are not as prevalent or as severe as those found in either Apache or Highland Guatemalan children. These differences may be due to better pre- and perinatal conditions for Mexican children.

Transverse groove hypoplasias are far more common than pitting defects. However, this pattern was not consistent between dentitions. Permanent teeth had more hypoplastic grooves but deciduous teeth had slightly more pitting defects. The cause of these pattern differences is unknown. However, if hypoplastic grooves are the result of disruptions in secretion of a complete front of ameloblasts and pitting defects are the results of disruptions to sporadic sections of the secretory front, then hypoplastic grooves may indicate a more severe stress.

Age at development of defects

In the permanent dentition, enamel hypoplasias appear to develop most often between 12 and 36 months (Fig. 2). This peak period is earlier than that which has been found in studies of archaeological populations (Goodman et al., 1984; Schulz and McHenry, 1975; Swardstedt, 1966) and occurs after the first year, which has long been proposed to be the time during which defects are most likely to occur (Sarnat and Schour, 1941). The peak period of hypoplasias is in accord with the Jordanian study of Alcorn and Goodman (1985). The high rate of hypoplasias during the second and third years suggests increased susceptibility with the loss of maternal antibodies from the decline and ending of breastfeeding. Women in the Solis Valley usually wean between the first and second years. Future studies of the chronology of defects in individuals now being studied during weaning may help to clarify the relationship between weaning, infant stress, and dental defect formation.

The peak for enamel hypoplasias in the deciduous dentition occurs during the last trimester and near birth (Fig. 2). This peak is near in timing to the peak frequency of hypoplastic defects found by Blakey and Armelagos (1985). Dental defects have a long history of association with the birth process (Kronfield and Schour, 1939; Via and Churchill, 1959).

Male–female differences

A review of the literature on sex differences in the frequency of enamel defects failed to reveal a consistent pattern. No significant sex differences in total defects were found by Sucking and Pearce (1984) or for linear hypoplasias by Infante and Gillespie (1974). However, Arkle (1962) noted a higher frequency of opacities and hypoplasias among Tasmanian girls than boys, while El—Najjar et al. (1978) identified more hypoplastic defects among males than females in the Hammon-Todd osteological collection (comprised of skeletons of lower–class individuals from the Cleveland area who died during the early 1900s).

In this study, females had a higher rate of enamel hypoplasias. Females were at a slightly increased risk for hypoplasias during the first 2 years and at a particularly high relative risk of defects during the third and fourth years. After 2 years, male rates dropped while female rates remained elevated. Data now being analyzed on the nutritional, anthropometric, and health status of

children in these communities may help to explain the meaning of this sex difference in frequency of enamel hypoplasias. At present, these data suggest the possibility that male children may be obtaining greater access to basic resources such as food, shelter, and health care.

CONCLUSIONS

The following conclusions are derived from a study of the prevalence and chronological distribution of enamel defects in 300 children from five rural agricultural communities.

1. Based on the FDI method for classification of defects, 46.7% of children have at least one enamel hypoplasia on their anterior teeth. This high rate, relative to other studies with similar methods of assessment, suggests that aspects of their marginal living conditions might be implicated.

2. The highest frequency of defects is found on the maxillary central incisors (44.4%). The increased frequency of defects on this tooth is consistent with prior studies in suggesting a field effect controlling susceptibility to dental defects. Tooth differences in susceptibility to defects may explain some of the inconsistencies in the results of prior studies. Future studies should report tooth-specific rates.

3. Enamel hypoplasias are relatively rare on deciduous teeth. Most deciduous tooth defects appear to have developed during the last trimester of pregnancy and neonatally.

4. The highest frequency of enamel hypoplasias occurs during the second and third years, suggesting a casual role for stress associated with weaning.

5. Females have a significant excess of hypoplastic defects. This sex difference may result from differential access to basic resources.

6. High intraobserver and interobserver reliability scores suggest that training for the identification and scoring of defects can be achieved in a relatively short period of time.

7. Future studies should aim at improving methods for the identification of defects. Most importantly, however, efforts should be directed toward understanding the conditions associated with and causative of enamel defects. As methods for nutritional assessment are few and generally inexact, development of assessment methods should be a research priority. Fifteen years after Jelliffe and Jelliffe (1971) concluded that research on the etiology and public health consequences of enamel hypoplasias is overdue. Researchers are now in a position to go ahead with this research.

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