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Enamel defects and its relation to life course events in primary dentition of Brazilian children: A longitudinal study

A.M.B. Chaves[1], A. Rosenblatt[1] and A.F.B. Oliveira[2]

[1]Department of Paediatric Dentistry, Faculty of Dentistry of Pernambuco, Camaragibe, Brazil; [2]Department of Morphology, Federal University of Paraiba, João Pessoa, Brazil

Objective: To evaluate the cumulative incidence of enamel defects (ED) and its correlation with life course events such as malnutrition and pre- and postnatal infections, in a cohort of children of low socioeconomic status. ***Methods:*** The children were registered at birth and examined from 12 to 36 months of age. At the baseline, 246 were examined and in the follow-up, 228. The teeth were examined under natural light and dried with gauze. ED was determined by the DDE index. ***Results:*** Results show that gender was not significant. The cumulative incidence of ED at the last recall examination was 78.9%. The most prevalent type of defect was diffuse opacity, present on the gingival half of the buccal surface ($p<0.001$). For logistic regression analysis maternal infections, intrauterine growth retardation (IUGR), malnutrition and postnatal infections were selected as predictor variables for the occurrence of enamel defects. ***Conclusions:*** The results indicate that life course events such as undernutrition and childhood infections during teeth development may be associated with enamel defects in socioeconomically underprivileged communities and may compromise the oral health-related quality of life.

Key words: Enamel defects; life course; longitudinal study, primary dentition

Introduction

Dental enamel is the most highly mineralized tissue of the body and is formed within a unique, extracellular matrix derived from the synthesis and secretion of proteins by the ameloblast cells of the inner enamel epithelium (Fincham *et al.*, 1999). Any disturbance in the process of apposition and mineralization may be manifested as enamel hypoplasias or opacities (Sato *et al.*, 1996; Seow, 1997).

Enamel hypoplasia is a quantitative defect associated with a reduced thickness of enamel, formed during the secretory phase of amelogenesis. The opacities involve a qualitative defect, namely an alteration in the translucency of the enamel, which is of normal thickness and, at eruption, has a smooth surface white, cream, yellow or brown in colour (FDI, 1992).

Enamel defects of human primary dentition have been reported to occur in 2% - 99% of children, depending on race, ethnicity, nutritional or socioeconomic status, birth weight, the type of classification system used, and the method of examination (FDI, 1992; Seow, 1997). Previous studies have shown a greater prevalence of enamel defects in undernourished children from underdeveloped countries (Pascoe and Seow, 1994; Rugg-Gunn *et al.* 1998).

Malnutrition is unquestionably a phenomenon of poverty. In the northeast of Brazil the prevalence of infant malnutrition is about 18%. Furthermore, a large proportion of this population has limited access to treated water and sanitation, which might be compromising their health and nutritional status (BEMFAM, 1997). In malnourished children, relative protein and mineral imbalance may be responsible for enamel hypoplasia either at the level of protein synthesis or during the rapid influx of calcium and phosphate ions or both (Agarwal *et al.*, 2003). Synergism between undernutrition and infection may also lead to a generalized defect in tooth development and its susceptibility to dental diseases (Agarwal *et al.*, 2003; Li *et al.*, 1995).

The purpose of the present study was to determine the cumulative incidence and distribution of enamel defects in a cohort of Brazilian children of low socioeconomic status from birth to 36 months of age and their association with life course factors such as nutritional status and childhood infections.

Materials and Methods

Subjects

This study was conducted from the year 2000 up to 2003 among 275 children in João Pessoa, capital of Paraíba, a state in Northeast Brazil. João Pessoa has 597,934 inhabitants and only about 148,000 have access to adequate water supply, whose level of fluoride is lower than 0.05ppm. Brazilian Northeast is poor, with the lowest per capita income of the country. About 39% of families survive with half of the legal minimum wage (IBGE, 2000).

The children in this study were all born at Cândida Vargas Maternity Hospital, a public maternity hospital for underprivileged people. Parents consented to participate in the study. Ethical approval was obtained from the Hospital's Ethics Committees.

The calculation of the sample was based on Li *et al.* (1995;1996), taking into consideration a 5.0% error and a 95% confidence interval. The power of the test was 80%.

Correspondence to: Dr. Ana Maria Barros Chaves, R. Joaquim Ferreira da Costa, 40 apto. 301 Manaíra João Pessoa - Paraíba/ Brazil 58038-040. E-mail: ambchaves@hotmail.com

Furthermore, the sample size was increased by 20% to allow for losses. Therefore, a total of 275 children born between January and February 2000 were recruited at birth in the Maternity Hospital. The baseline investigation commenced in February 2001 with 246 children.

Data collection

After the mothers gave their consent, the subjects were recruited at birth and were recalled every six months from 12 to 36 months of age. Ethical clearance was obtained from the Hospital's Ethics Committees. Two investigators conducted interviews and dental examinations. The first investigator examined children from 12 to 18 months of age and the second, those aged from 18 to 36 months.

The examination was performed in the knee-knee position outside the children's homes under natural light, by the visual method. The teeth were dried and cleaned with gauze (FDI, 1992) and a mirror and probe were used. Intraoral photographs were also taken. Developmental defects were scored according to the Developmental Defects of Enamel Index (DDE) proposed by the FDI Working Group (FDI, 1992) and recorded on a comprehensive chart (Table 1). Buccal and lingual surfaces of each anterior tooth and buccal, lingual, and occlusal surfaces of each posterior tooth were examined. The defect location was classified according to its position on the crown as gingival half, incisal or occlusal half, pit and fissure and cusp. At each clinical examination, partially erupted teeth were not recorded, except when defects were present on the erupted portion of the crown. Defects measuring less than one millimeter in diameter were excluded as well as those which were doubtful.

Nutritional status and relevant medical history

Maternal and neonatal medical histories were obtained from the hospital records. The prenatal nutritional status was assessed using the Intrauterine Growth Index Chart by Lubchenco *et al.* (1963). Percentiles of birth weight by gestational age were obtained by Denver reference curve (Lubchenco *et al.*,1963) and the infants were divided into two groups: (a) Small for Gestational Age (SGA), when the child was below the 10th percentile, and (b) Appropriate for Gestational Age, (AGA) when above the 10th percentile.

The relevant postnatal medical conditions, and dental history (hygiene habits, use of fluoride – dentifrice or supplements, dental trauma) were obtained from the parents. The postnatal nutritional status was ascertained by weight and height measurements at 24 months of age, according to the National Center for Health Statistics (Waterlow *et al.*, 1977) standards:

Normal: children of normal height-for-age (>95% of standard), and normal weight-for-height (>95% of standard).

Wasted: children who, by the age of 24 months, were of normal height-for-age but low weight-for-height (<90% of standard), indicating current acute malnutrition.

Stunted: children aged 24 months who were of normal weight-for-height and low height-for-age (<95% of standard), indicating past or chronic malnutrition.

Stunted and Wasted: children who, by the age of 24 months, were both low weight-for-height and low height-for-age, indicating that they were not only acutely but also chronically malnourished.

Calibration and Reproducibility

Consistency of diagnosis of the examiners was checked by inter- and intra-examiner variability tests. To evaluate the concordance between examiners, 24 children (10% of the sample) were randomly selected and examined by both examiners on separate occasions. During the fieldwork, the reproducibility examinations were carried out following the same examination procedures. For the intra-examiner test, about 10% of the children examined were randomly selected and reexamined on a separate occasion, 24 hours apart. The examiners had no access to the previous records. At 12 and 18 months, Cohen's Kappa values for enamel defects were 0.93 and 0.90, respectively, and at 24 and 36 months, it varied from 0.94 to 0.98. The interexaminer Cohen's Kappa value was 0.89 between the examinations at 18 and 24 months.

Data recording and analysis

All the data were organized and analyzed using the SPSS 10.0 statistical software package. Both descriptive and analytical techniques were used. The relationship between

Table 1. Definition of terms for the classification of enamel defects (DDE Index) according to FDI (1992).

Type of defects	*Definition*
Demarcated opacity	Defect involving an alteration in the translucency of the enamel, variable in degree. The defective enamel is of normal thickness with a smooth surface. It has a distinct and clear boundary with the adjacent normal enamel and can be white, cream, yellow or brown in color.
Diffuse opacity	Defect involving an alteration in the translucency of the enamel, variable in degree and white in color. The defective enamel is of normal thickness and can have a linear, patchy or confluent distribution but there is no clear boundary with the adjacent normal enamel.
Hypoplasia (reduced thickness)	Defect involving the surface of the enamel associated with a reduced localized thickness of enamel without dentinal exposition.
Hypoplasia (missing enamel)	Defect involving the surface of the enamel associated with a complete absence of enamel over a considerable area of dentine.
Opacity+ Hypoplasia	Defect involving the surface of the enamel with an alteration in the translucency of the enamel, diffuse or demarcated, associated with partial or complete absence of enamel over a considerable area of dentine.

enamel defects and the independent variables (infection during pregnancy, nutritional intrauterine status, post-natal nutritional status and infection in 2 - 6 months and in 7 - 12 month - old infants) were evaluated using nonparametric tests (Chi-square and Fischer's exact). A probability value of $p<0.05$ was considered statistically significant. A ninety-five percent confidence interval was considered for comparisons of different results within subgroups.

A logistic regression analysis model was built to assess the influence of some prenatal and postnatal factors, in the development of enamel defects. The models also took into account the hierarchical relationships of some independent variables that have great theoretical importance for the model and were moderately significant at the bivariate analysis ($p<0.25$). Two logistic regression models were developed for risk assessment of DDE, including all the representative variables. For the analysis, all enamel defects were combined in the category "presence". The post-natal malnutrition combined the categories wasted, stunted, stunted and wasted as "undernourished children". Backward elimination was used to reduce the number of variables in the models. The final model included only variables that were significant at $p<0.05$. In the first model, the relationship between prenatal nutritional status and neonatal infections and the occurrence of DDE at 18 months of age was tested. In the second model, the relationship between postnatal infections and nutritional status and the occurrence of DDE at 36 months of age was also verified.

Results

Cumulative Incidence of Enamel Defects.

Two hundred and twenty-eight (82.9%) children were seen at all cohort stages. Table 2 shows the cumulative incidence of DDE in this study. By the end of the study at the age of 36 months, 78.9% of the 228 infants had at least one tooth with enamel defects. The type of enamel defect did not differ according to gender ($p>0.05$).

The proportion of enamel defects increased with the number of erupted teeth. 25.1% of the teeth had at least one surface with enamel defects. The defect most commonly found was diffuse opacity (10.8%). The proportion of all types of hypoplasia (11.2%) was lower than that of both types of opacity (13.9%). Defects were observed more frequently on the buccal surface (18.4%) than on the lingual (0.8%) and occlusal (5.9%) surfaces, and they appeared more frequently on the gingival half (7.8%).

The percentage of various enamel defects for each tooth type is shown in Table 3. In the incisors, diffuse opacity and *opacity + hypoplasia* were more prevalent. Demarcated opacities occurred mostly in canines (6.3%). Diffuse opacity, followed by hypoplasia, was the most prevalent enamel defect in molars. Overall, enamel opacities were more prevalent than hypoplastic lesions for all tooth types.

Table 2. Cumulative incidence of Brazilian children with enamel defects followed from 12 to 36 months.

	Examinations (months)									
Enamel Defects	*12*		*18*		*24*		*30*		*36*	
	n	*%*	*n*	*%*	*n*	*%*	*n*	*%*	*n*	*%*
Present	68	33.7	140	61.4	167	73.3	176	77.2	180	78.9
Absent	134	66.3	88	38.6	61	26.7	52	22.8	48	21.1
TOTAL	202(1)	100.0	228	100.0	228	100.0	228	100.0	228	100.0

(1) – 26 children had no erupted teeth.

Table 3. Percentage of enamel defects for each tooth type among Brazilian children followed from 12 to 36 months.

	Type of Defect												Value of p
Tooth Group	Absent		Demarcated Opacity		Diffuse Opacity		Reduced Thickness		Hypoplasia (missing enamel)		Opacity + hypoplasia		
	n	%	n	%	n	%	n	%	n	%	n	%	
Central Incisor	709	77.7	15	1.6	91	10.0	18	2.0	28	3.1	51	5.6	p< 0.0001*
Lateral Incisor	777	85.2	9	1.0	68	7.5	9	1.0	17	1.9	32	3.5	
Canine	721	79.5	57	6.3	52	5.7	52	5.7	2	0.2	23	2.5	
1st Molar	561	61.6	25	2.7	159	17.4	20	2.2	138	15.2	8	0.9	
2nd Molar	580	70.1	31	3.8	114	13.8	2	0.2	90	10.9	10	1.2	
Total	3348	74.9	137	3.1	484	10.8	101	2.3	275	6.3	124	2.8	

(*) – There was a significant difference in each group ($p <0.0001$)

Nutritional Factors Related to Enamel Defects.

Table 4 shows the variables related to enamel defects during the first 18 months of age. The bivariate analysis showed that only the intrauterine nutritional status was statistically significant at $p<0.05$. Eighty-three per cent of enamel defects were related to children who were small for gestational age. However in the multivariate analysis, which included all variables under $p<0.25$, infections during pregnancy and infant's infections between two and six months were also selected for the model as well as the intrauterine and nutritional status.

Table 5 presents the proportion of enamel defects at 36 months, including the associated variables at 24 months of age. The cumulative incidence of enamel defects was higher among the children with some kind of nutritional disorder (91.4%). For the postnatal nutritional status group, 15.4% (n=35) of whom were in the undernourished category, 3.5% were classified as wasted, 1.3% stunted and 10.5% were classified as both stunted and wasted. These variables may act synergically to produce enamel defects then, another multivariate model was proposed.

The statistically significant factors at the bivariate analysis ($p<0.25$) were used to identify which variables had a significant impact on the occurrence of DDE in primary teeth. Multivariate logistic regression was carried out twice. Firstly, pre- and neonatal factors were considered; second by postnatal nutritional and environmental factors. Initially, at 18 months of age, all children were submitted to analysis; then again, at 36 months of age. Two prenatal factors were significantly related to the prevalence of enamel defects: *infection during pregnancy* (OR=1.43, 95% CI= 0.78-2.57) and *intrauterine nutritional status – small for gestational age* (OR=4.46, 95% CI= 0.95-21.04). Three postnatal related factors were significantly related to the prevalence of enamel defects: *presence of infection of 2 to 6 months (OR*=1.80, 95% CI= 0.83-3.90), *presence of infection between 7 and 12 months* (OR=2.48, 95% CI= 0.95-6.49) and *postnatal nutritional status – acute or chronic malnutrition* (OR=2.89, 95% CI= 0.84-10.03).

Discussion

In this study, the incidence of all types of enamel defects among the children examined (126 boys and 102 girls) was 78.9%. No significant association was observed between gender and the occurrence of defects. This

Table 4. Relationship between the independent variables and enamel defects of the children examined at 18 months.

	Enamel Defects							
	Present		Absent		Total		p value	OR (CI)
Independent variables	n	%	n	%	n	%		
Infection during pregnancy								
• Absent	93	53.1	82	46.9	175	100	0.245	1.16 (0.9-1.47)
• Present	40	61.5	25	38.5	65	100		
Infection from 2 thru 6 months								
• Absent	81	51.63	77	48.7	158	100	0.073	1.24(0.99-1.55)
• Present	52	63.4	30	36.6	82	100		
Infection from 7 thru 12 months								
• Absent	13	50	13	50	26	100	0.556	1.12(0.75-1.68)
• Present	120	56.1	94	43.9	214	100		
Intrauterine nutritional status								
• SGA	10	83.3	2	16.7	12	100	0.0459	1.54(1.17-2.04)
• AGA	123	53.9	105	46.1	228	100		

Table 5. Relationship between the independent variables and enamel defects of the children examined at 24 months.

	Enamel Defects							
	Present		Absent		Total		p value	OR (CI)
Independent variables	n	%	n	%	n	%		
Infection from 2 thru 6 months								
• Absent	118	77.1	35	22.9	153	100	0.3348	1.07(0.94-1.23)
• Present	62	82.7	13	17.3	75	100		
Infection from 7 thru 12 months								
• Absent	17	65.84	9	34.6	26	100	0.0715	1.23(0.93-1.65)
• Present	163	80.7	39	19.3	202	100		
Postnatal nutritional status								
• Undernourished	32	91.4	3	8.6	35	100	0.0490	0.84(0.74-0.95)
• Nourished	148	76.7	45	23.3	193	100		
Intrauterine nutritional status								
• SGA	10	83.3	2	16.7	12	100	0.0459	1.54(1.17-2.04)
• AGA	123	53.9	105	46.1	228	100		

finding is in agreement with data published by Fearne *et al.* (1990) and Slayton *et al.* (2001), but at variance with Li *et al*. (1995), who found a statistically significant percentage of enamel defects among boys.

Epidemiological studies on enamel defects have shown a great diversity of methodological procedures, giving rise to variations that merit consideration. For example, the criterion used to diagnose enamel defects is crucial when comparisons need be made. Furthermore, because teeth lacked previous cleaning and drying, the prevalence of enamel defects has probably been underestimated in some studies (Li *et al.*, 1995; Rugg-Gunn *et al.*, 1998; Vignarajah and Williams, 1992). The most relevant aspect of epidemiological investigations into enamel defects, however, resides in the kind of design selected for the study. According to Seow (1997), the rapid development of caries in teeth affected by enamel defects makes the diagnosis of a preexisting defect more difficult. The adoption of a longitudinal prospective design would minimize this problem.

In the present study, the cumulative incidence of enamel defects was 25.1% (Table 3), a percentile lower than that observed by Pascoe and Seow (1994) – 64.8%. The greater prevalence of enamel defects in Pascoe and Seow's study (1994) may be explained by the poorer socioeconomic conditions of the community the children lived in.

The teeth examined presented more qualitative (13.9%) than quantitative (11.2%) defects. According to Sato *et al.* (1996), it is possible that teeth suffered injury during calcification and maturation of the enamel, rather than during cell differentiation and matrix secretion.

As to location, enamel defects were more prevalent on the buccal surface (18.4%) and on the gingival half (7.89%), which is in agreement with Li *et al.* (1995) and Vignarajah and Williams (1992).

Higher incidence of enamel defects in the upper incisors than in the lower was observed in the present study, as well as in those of Li *et al.*(1995), Pascoe and Seow (1994) and Rugg-Gunn *et al.* (1998). These findings can be explained on the basis of the observations of Suga *et al.* (1989), who suggested that the difference in enamel thickness could be the reason for that. Suga *et al.* (1989) speculated that ameloblasts responsible for thick enamel are more susceptible to systemic disorders than are the ameloblasts associated with thin enamel. The diffusion of calcium ions from the ameloblasts to the matrix and the removal of organic substances from the matrix are slower in thick enamel than in thin enamel. Therefore, the teeth are exposed to systemic injuries for a longer period of time.

Because the greatest prevalence of enamel defects has been found in communities with low family income, severe malnutrition, rates of infection (Pascoe and Seow,1994; Rugg-Gunn *et al.,*1998), social and regional inequalities need be taken into consideration as they cause a strong impact on the health-related quality of life.

Regarding malnutrition and infection, the extreme poverty of the majority of the families might explain the fact that 78.9% of the children showed enamel defects. This percentage was lower than that found by Pascoe and Seow (1994) – close to 100% in an extremely poor Australian Aboriginal community. However, it was higher than that found by Rugg-Gunn *et al.* (1998), 51%, in rural communities. Nevertheless, in a study carried out with children that presented a satisfactory nutritional status and had fluoridated water, Slayton *et al.* (2001) observed only 6% hypoplasia and 27% opacity. It is important to bear in mind that the latter percentage relates to fluorosis, a condition that also promotes qualitative alterations in the enamel structure. Endemic fluorosis, another cause of enamel defects is unlikely to be present in this study because of the low level of fluoride in the water supply (<0.05ppm.).

The effects of poverty on child malnutrition are pervasive. According to Frankenberger (1996), absolute poverty means when one is unable to satisfy basic needs (adequate food, health care, water, shelter, primary education). In underdeveloped countries, children are more exposed to situations in which their health is at risk. When visiting their homes, highly unsatisfactory living conditions, such as no piped water, uncertainty as to how long they would be able to remain in their dwelling, which were overcrowded and low intake of essential nutrients were observed. Every one of them have probably contributed to the high level of recurrent infection registered. Life in such conditions since birth has given rise to the hypothesis that people's organism interacts with the environment; in which case, the ameloblast metabolism may have been affected. Besides, according to Lunt and Law (1974) the chronology of the deciduous dentition may also have been affected to a certain extent (Suga *et al.*, 1989).

It is impossible to distinguish each etiological factor, because perinatal variables often occur together and may interact. The defects may thus be the result of the cumulative severity, duration and timing of the insults rather than their specific nature (Fearne *et al.,*1990). After multivariate logistic regression, the presence of infections during pregnancy (OR=1.43) and the small child for gestational age status (OR= 4.46) may be factors that could explain the occurrence of enamel defects. Previous clinical studies (Agarwal *et al.,*2003; Lai *et al.,*1997) have also shown that low birth weight and premature birth are associated with the presence of enamel defects.

Regarding nutritional status, at 24 months of age, 15.4% of the children showed some nutritional disorder and 11.8% showed problems in the height/age ratio, which indicates chronic malnutrition (ACC/SCN, 2000). The nutritional disorder was statistically significant for enamel defects. This finding is of great importance, because it was associated with family income, and did not show improvement during the follow-up period. It is likely that these nutritional deficiencies appeared in the first year of life or even arose during the intrauterine period as a result of poor maternal nutrition during pregnancy (ACC/SCN, 2000).

In Brazil, according to the last epidemiological study by UNICEF (2005), 11% of the children under 5 years suffer from stunted malnutrition. This proportion is very close to the findings of this study (11.8%), but it is lower than that seen by BEMFAM (1997), for the same geographic region (17.9%).

In addition to caloric and protein malnutrition, the lack of important micronutrients must not be neglected. This is very common in underdeveloped countries, but it is not

identified by anthropometrics. In the analyses of diet, a low ingestion of fruit, vegetables, vitamins and mineral salts was observed, as well as a delay in introducing solid foods. Many children were fed exclusively by nursing bottle. The most common type of complementary food seen in this community was a thin gruel, low in protein and energy, made with water and manioc meal, to which salt or sugar was added. Another relevant feeding fact is over diluting of powdered milk, thereby lowering the concentrations of calcium and vitamin D, important elements for the amelogenesis process, as reported by Li *et al.*(1995). Thus, these factors must be also taken into account because they could be associated with the occurrence of occult nutritional deficiency, found in children classified as Eutrophic, who presented enamel defects.

It is known that children with inadequate dietary intake are more susceptible to disease. In turn, disease depresses appetite, inhibits the absorption of nutrients in food, and competes for a child's energy (ACC/SCN, 2000). Therefore, the synergism between malnutrition and infection justified the use of an additional logistic regression analysis. In this study, after multivariate analysis with logistic regression the infections that occurred during the first (OR=1.80) and second semesters (OR=2.48) of postnatal life, as well as malnutrition (OR=2.89), were selected to explain the occurrence of enamel defects in the studied population. Some authors have observed the association of malnutrition (Agarwal *et al.*, 2003; Rugg-Gunn *et al.*, 1998) and infections after birth (Rugg-Gunn *et al.*, 1998) in the development of enamel defects in deciduous dentition, but only in one of the mentioned studies (Rugg-Gunn *et al.*, 1998) the logistic regression was used.

Conclusions

Poverty was established as the main indicator of malnutrition. Poor housing conditions, overcrowding, the absence of piped water, low family income, all of which were associated with underdeveloped countries, affected the course of a child's life. These factors could explain the higher frequency of childhood illness associated with the high prevalence of enamel defects in this population. Therefore enamel defects may be a reflection of nutritional defects and other health problems in early life.

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