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REVIEW ARTICLE

Prenatal and neonatal risk factors for the development of enamel defects in low birth weight children

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OBJECTIVE: To analyse the influence of several prenatal and neonatal risk factors in the development of enamel defects in low birth weight children.
SUBJECTS AND METHODS: Children between 4 and 5 years of age (*n* = 102) were classified into: Group 1) 52 low birth weight (<2500 g); Group 2) 50 normal birth weight (≥2500 g). Medical history, prenatal and neonatal variables were collected. Enamel defects were evaluated with the modified Developmental Defects of Enamel Index.
RESULTS: The prevalence of hypoplasia and average number of affected teeth were significantly higher in group 1 than in group 2 (59.6% *vs* 16% and 1.6 *vs* 0.3 respectively). Low gestational age was linked to a higher prevalence of hypoplastic (*P* = 0.027) and combined defects (*P* = 0.001). Children with neonatal risk factors (low Apgar scores, parenteral nutrition, orotracheal intubation, mechanical ventilation and acidosis) developed defects more frequently (*P* < 0.05). Defects were symmetrically distributed in children who were not intubated; in those who required intubation they concentrated on the left maxillary teeth (*P* < 0.05). Smoking during pregnancy, young maternal age and multiple birth were significantly associated to developmental defects.
CONCLUSIONS: The prevalence of enamel defects in primary dentition is significantly influenced by birth weight, gestational age and several systemic factors. Orotracheal intubation probably plays an important role as a result of laryngoscope trauma on the maxilla.
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Keywords: enamel defects; enamel hypoplasia; low birth-weight; primary dentition; orotracheal intubation

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Introduction

As a result of the continuous improvement of Neonatal Medicine, survival rates of low birth weight (LBW) infants (< 2500 g) have greatly increased during recent years. Newborns, especially preterm and LBW, are exposed to several factors that can compromise their health in general and oral health in particular. Previous studies on LBW children have mainly focused on two aspects, enamel hypoplasia in primary dentition and palatal deformities. The reasons for these developmental dental defects are still unclear. While palatal deformities are related to local factors such as laryngoscope use and orotracheal intubation, enamel defects are additionally associated to systemic factors like immaturity, LBW (Noren, 1983; Seow *et al*, 1987, 1989; Fearne *et al*, 1990; Needleman *et al*, 1992; Pascoe and Seow, 1994; Seow, 1996; Lai *et al*, 1997; Agarwal *et al*, 2003; Lunardelli and Peres, 2006; Ferrini *et al*, 2008), respiratory distress (Johnsen *et al*, 1984; Franco *et al*, 2007), rickets of prematurity (Seow *et al*, 1984a, 1989), neonatal asphyxia, hyperbilirubinemia, neonatal infection and maternal conditions such as preeclampsia and diabetes (Seow, 1991).

Primary teeth have a long pre- and postnatal development period. Matrix formation and subsequent calcification begins on the 15th gestational week and continues until several months after birth (Moore, 1998). This process does not occur over the whole crown simultaneously; it begins at the highest point of the crown and progresses downwards towards the tooth neck in incremental layers. Since enamel is a stable structure that lacks natural repair mechanisms, any systemic circumstance that disrupts matrix formation or maturation will cause a permanent structural defect in the teeth under development at that moment. Likewise, enamel defects derived from local trauma will also leave a permanent mark, but usually on one or a few adjacent teeth only. Such defects may only be microscopic if the disturbance was mild, or they may be clinically evident if it was more severe. Consequently, after eruption, tooth enamel provides a unique window into the prenatal

period, as it may inform of early exposure to certain systemic and/or local factors (Turner, 1981).

The purposes of this study were, first, to determine the prevalence of enamel defects in a population of LBW children, and, second, to evaluate the influence of prenatal and neonatal risk factors in the development of enamel defects in primary dentition.

Subjects and methods

A sample of 102 children, all born at the same tertiary hospital where the study was conducted, the Hospital Clínico Universitario of Valencia (Valencia, Spain), was obtained. They were selected randomly from the hospital's database of children whose age would range from 4 to 5 years at the moment of evaluation. Informative letters were sent to the parents of 164 children, of which 102 accepted to participate in our study. They were classified as follows: Group 1 included 52 LBW children (<2500 g) and group 2 comprised 50 normal birth weight (NBW) children (≥2500 g). The study protocol was approved by the Committee on Ethical Practice of the Hospital Clínico Universitario of Valencia in accordance with the Declaration of Helsinki. Written parental informed consent was obtained.

Medical pre- and postnatal data were obtained from hospital records: (i) Pregnancy variables: Age, diseases, multiple pregnancy, daily consumption of alcohol, coffee, drugs and tobacco, and medication consumption; (ii) Cesarean *vs* vaginal delivery; (iii) Neonatal, perinatal and postnatal variables: Gestational age, birth weight, size for gestational age, gender, Apgar score, orotracheal intubation, mechanical ventilation, neonatal acidosis, phototherapy, parenteral nutrition, and perinatal and neonatal diseases. Besides, parents were surveyed about their child's relevant postnatal diseases and dental history including past dental treatment, fluoride supplementation, oral hygiene and dietetic habits.

Dental examinations were performed at the Dental School of the University of Valencia under ideal conditions by one previously trained examiner. The teeth were dried with gauze, and a mirror and probe were used to detect enamel defects and caries. The modified Developmental Defects of Enamel Index (Federation Dentaire Internationale (FDI) Commission on Oral Health RaE (1982) was used to register enamel defects. Qualitative change in enamel translucency without loss of enamel surface was categorized as enamel opacity. Enamel hypoplasia was diagnosed when an alteration in the enamel surface was identified. Finally, when an enamel defect presented both opacity and hypoplasia, it was classified as a combined defect. Every tooth and surface was examined, noting the severity and extent of each defect in a comprehensive chart. Dental caries were diagnosed following Radike's criteria (Radike, 1972) and plaque was scored using the plaque index introduced by Löe and Silness (Loe, 1967).

Data analysis was performed using SPSS® for Windows version 15.0 (SPSS Inc., Chicago, IL, USA). Descriptive statistics was used for quantitative analysis. Student's *t*-test was used to determine the association between a dichotomous variable and a normally-distributed interval variable. Analysis of variance (ANOVA) was performed to compare the association between a nominal variable and an interval parametric variable; if statistical difference was detected, a *post hoc* procedure was used to compare individual pairs of means. Pearson's correlation coefficient was used where appropriate to evaluate the strength of linear relationship between two variables. Differences were considered statistically significant at $P < 0.05$.

Results

Group 1 ($n = 52$ LBW children) comprised 26 boys and 26 girls, all preterm (mean gestational age 34 weeks, range 25–37), with a mean birth weight of 1950 g (range 590–2480). Fifteen (29%) were small-for-gestational age (SGA) and 37 (71%) had an appropriate size for gestational age (AGA). Group 2 ($n = 50$ NBW children) comprised 21 boys and 29 girls, all full-term, with a mean birth weight of 3383 g (range 2750–4650).

Prevalence of affected children and average number of affected teeth

The prevalence of enamel defects in the LBW and NBW groups is shown on Table 1. At least one enamel defect was found in 90.4% of the LBW children in group 1, while this percentage was 80% for group 2 (NBW). Both groups showed a similar prevalence of opacities (76.9% for group 1, 79.6% for group 2). However, the prevalence of hypoplasia was 59.6% in group 1 and 16% in group 2, and the prevalence of combined defects was 5.8% in group 1 and 4% in group 2.

The average number of teeth with enamel defects was globally higher in the LBW group (mean 5.7) than in the NBW group (mean 3.6) ($F = 3.891$, $P = 0.023$) (Table 1). Similarly, hypoplastic defects were more numerous in the LBW group (mean 1.6) than in the NBW group (mean 0.3) ($F = 15.131$, $P = 0.000$). No statistically significant differences were found when comparing opacities and combined defects between groups ($F = 1.558$, $P = 0.214$; $F = 0.178$, $P = 0.837$ respectively).

This study found no significant associations between developmental enamel defects and dental caries or plaque scores.

Table 1 Enamel defects in low-birth-weight and normal-birth-weight (NBW) groups

	Total enamel defects		*Opacity*		*Hypoplasia*		*Combined defects*	
	Prev	*Av*	*Prev*	*Av*	*Prev*	*Av*	*Prev*	*Av*
Group 1	90.4	5.7[a]	76.9	4.5	59.6	1.6[c]	5.8	0.2
Group 2 (NBW)	80.0	3.6[b]	79.6	3.2	16.0	0.3[d]	4.0	0.1
Mean	85.2	4.7	78.3	3.9	37.8	1.0	4.9	0.2

Prev, Prevalence of affected children (%); Av, Average number of affected teeth.
$P < 0.05$ for a *vs* b and c *vs* d.

Table 2 Relationship between maternal variables and developmental enamel defects

	Opacity	*Hypoplasia*	*Combined defects*
Young maternal age	$r = 0.136$	$r = 0.079$	$r = 0.349$
	$P = 0.096$	$P = 0.298$	$P = 0.000$*
Multiple birth pregnancy	$t = -0.29$	$t = 0.015$	$t = -0.641$
	$P = 0.631$	$P = 0.857$	$P = 0.007$*
Smoking during pregnancy	$r = -0.089$	$r = 0.179$	$r = 0.090$
	$P = 0.274$	$P = 0.028$*	$P = 0.246$
Coffee consumption during pregnancy	$r = -0.050$	$r = 0.046$	$r = 0.042$
	$P = 0.537$	$P = 0.546$	$P = 0.583$
Alcohol consumption during pregnancy	$r = -0.099$	$r = -0.036$	$r = -0.015$
	$P = 0.223$	$P = 0.632$	$P = 0.848$
Medication consumption during pregnancy	$r = -0.020$	$r = -0.062$	$r = -0.042$
	$P = 0.803$	$P = 0.418$	$P = 0.585$
Cesarean delivery	$t = 1.142$	$t = -2.303$	$t = 1.592$
	$P = 0.255$	$P = 0.024$*	$P = 0.114$

r, Pearson's correlation coefficient; *t*, Student's *t*-test.
*Statistical significance level < 0.05.

Relationship between maternal variables and developmental enamel defects in children

Analysis of mothers' medical and reproductive history evidenced that younger maternal age and multiple pregnancy were predictors of combined defects (Table 2). In the LBW group, 37 mothers (71%) had suffered at least one disease during pregnancy, compared to 15 mothers in the NBW group (29%).

In addition, a statistically significant association between tobacco use during pregnancy and the incidence of enamel defects was detected, with a positive linear relationship between the number of cigarettes smoked per day and the prevalence of hypoplasia. Similarly, children born by cesarean delivery were more likely to develop hypoplasia than those born by vaginal delivery.

No further statistically significant relationships were found between enamel defect occurrence and the other analysed variables (consumption of coffee, alcohol, or medication).

Relationship between neonatal variables and developmental enamel defects in children

Birth weight and gestational age were found to be associated with the occurrence of enamel defects (Table 3). In this sense, a statistically significant relationship between LBW and the development of dental hypoplasia was found. Likewise, more teeth were affected by hypoplasia and combined defects in low gestational age children. Our study did not find any statistically significant relationship between the prevalence of enamel defects and size-for-gestational age or gender.

Analysis of systemic variables led to the following findings (Table 3): Hypoplastic defects were more common in children with low 5-min Apgar scores and in those who received parenteral nutrition during the neonatal period. Combined defects were more frequent in children with low 1- and 5-min Apgar scores and neonatal acidosis. No associations between neonatal phototherapy and developmental enamel defects were found.

Table 3 Relationship between perinatal variables and enamel defects

	Opacity	*Hypoplasia*	*Combined defects*
Low gestational age	$r = 0.184$	$r = -0.306$	$r = -0.462$
	$P = 0.191$	$P = 0.027$*	$P = 0.001$*
Low birth-weight	$r = -0.260$	$r = -0.377$	$r = -0.132$
	$P = 0.753$	$P = 0.000$*	$P = 0.105$
Size for gestational age	$F = 0.062$	$F = 0.317$	$F = 0.048$
	$P = 0.940$	$P = 0.730$	$P = 0.953$
Gender	$t = 0.199$	$t = -0.954$	$t = 0.474$
	$P = 0.843$	$P = 0.342$	$P = 0.636$
1-min Apgar score	$r = -0.066$	$r = -0.195$	$r = -0.305$
	$P = 0.644$	$P = 0.166$	$P = 0.028$*
5-mins Apgar score	$r = -0.025$	$r = -0.228$	$r = -0.348$
	$P = 0.859$	$P = 0.038$*	$P = 0.012$*
Mechanical ventilation	$r = 0.007$	$r = 0.172$	$r = 0.316$
	$P = 0.959$	$P = 0.224$	$P = 0.023$*
Orotracheal intubation	$r = 0.009$	$r = 0.188$	$r = 0.308$
	$P = 0.950$	$P = 0.181$	$P = 0.026$*
Parenteral nutrition	$t = 0.851$	$t = -2.450$	$t = -0.958$
	$P = 0.339$	$P = 0.018$*	$P = 0.343$
Neonatal acidosis	$F = 1.389$	$F = 0.660$	$F = 3.864$
	$P = 0.258$	$P = 0.581$	$P = 0.015$*
Phototherapy	$t = 6.661$	$t = 0.127$	$t = 3.547$
	$P = 0.610$	$P = 0.911$	$P = 0.380$

r, Pearson's correlation coefficient; *t*, Student's *t*-test; *F*, analysis of variance.
*Statistical significance level < 0.05.

Concerning possible local causes of enamel defects, this study found that children who required orotracheal intubation and mechanical ventilation in the neonatal period showed a higher prevalence of severe defects (that is, combined defects) than children who did not undergo these procedures (Table 3). As shown on Table 4, maxillary teeth were globally more commonly and severely affected than mandibular teeth. Moreover, defects were symmetrically distributed in LBW children who were not intubated, whereas they were located asymmetrically in those who required intubation. In the latter, the left maxillary teeth were the most frequently involved teeth by hypoplasia (Table 4).

No relationship was found between intubation length and the development of enamel defects. In effect, visual analysis of intubated children's odontograms showed no differences in enamel defects when intubation lasted 1 to 3 days in comparison to when it lasted up to 1.5 months.

Discussion

The association between LBW and enamel defects is a widely acknowledged fact in the scientific literature (Noren, 1983; Johnsen *et al*, 1984; Seow *et al*, 1984a,b, 1987, 1989; Fearne *et al*, 1990; Seow, 1991, 1996; Needleman *et al*, 1992; Pascoe and Seow, 1994; Lai *et al*, 1997; Rugg-Gunn *et al*, 1998; Aine *et al*, 2000; Agarwal *et al*, 2003; Lunardelli and Peres, 2006; Franco *et al*, 2007; Ferrini *et al*, 2008). However, few authors have analysed other additional prenatal and neonatal risk factors.

Although enamel defects were more frequent than expected in both groups, our results confirm the risk of developmental enamel defects in LBW children is indeed

Table 4 Enamel defects distribution by quadrants in intubated and non-intubated low birth weight children (average number of affected teeth)

Quadrant	*Orotracheal intubation*	*Enamel defects (mean)*	*Opacity (mean)*	*Hypoplasia (mean)*	*Combined defects (mean)*
Right maxillary	No	0.7	1.6	0.3	0
	Yes	0.8	1.2	0.8[a]	0.3[c]
Left maxillary	No	0.7	1.7	0.3	0
	Yes	0.8	1.2	0.9[a]	0.2[d]
Left mandibular	No	0.4	0.9	0.3	0
	Yes	0.5	1	0.4[a]	0[d]
Right mandibular	No	0.4	1.1	0.2	0
	Yes	0.3	0.9	0.2[b]	0[d]

Hypoplasia: $P < 0.05$ for a *vs* b.
Combined defects: $P < 0.05$ for c *vs* d.

higher than in NBW children, concurring with previous studies. It is noteworthy that we found the prevalence of these defects to be 90.4% in the LBW population. This prevalence is higher than that reported by other studies. (Mellander *et al*, 1982; Johnsen *et al*, 1984; Seow *et al*, 1984a, 1987, 1989; Needleman *et al*, 1992; Pascoe and Seow, 1994; Lai *et al*, 1997; Aine *et al*, 2000; Chaves *et al*, 2007; Franco *et al*, 2007). One possible reason for these different prevalence rates is the different age period of children evaluation. In effect, while the age of children assessment ranged from 9 to 24 months in other studies (Johnsen *et al*, 1984; Seow *et al*, 1984a, 1987, 1989; Lai *et al*, 1997; Aine *et al*, 2000; Chaves *et al*, 2007), we chose to evaluate our children when they reached 4–5 years of age. This age of revision was selected on the basis that at this stage eruption of primary teeth is complete and satisfactory child-doctor cooperation can be expected (thus facilitating child examination and data compilation).

Other circumstances that might have given way to our higher prevalence of enamel defects are the conditions of dental assessment. In some studies, teeth were explored with a probe and mirror but without a dental chair light (Mellander *et al*, 1982; Seow *et al*, 1989; Pascoe and Seow, 1994). In others, children were evaluated with a torch while sitting on their parents' lap but without having dried the teeth (Johnsen *et al*, 1984). These conditions probably made the identification of enamel defects difficult especially in the case of opacities, which were the most prevalent defects in our study.

Furthermore, some studies considered enamel defects in deciduous teeth only (Johnsen *et al*, 1984; Needleman *et al*, 1992), hence disregarding possible anomalies in cuspids and molars. This obviously led to a lower prevalence of enamel defects than that in our study. Similarly, two studies conducted on 6 year-olds found a lower prevalence too, but it must be noted that these children's primary incisors had already exfoliated and were consequently not evaluated (Mellander *et al*, 1982). In our opinion, considering that at birth the formation of the deciduous incisor crown is almost complete and the middle third of the primary canine's crown and the first primary molar's occlusal surface are calcified, local and systemic factors interfering with these periods may materialize at these sites. If the child is born prematurely, defects will concentrate on the middle third of the primary incisors and cervical third of deciduous canines. Hence, we believe these sites must also be examined systematically.

Our study found birth weight and gestational age were significantly related to the occurrence of enamel defects. Actually, the lower the child's gestational age, the greater the number of teeth with hypoplastic and combined defects (Table 3). These findings support other authors' results (Fearne *et al*, 1990; Aine *et al*, 2000; Ferrini *et al*, 2008). Moreover, a statistically significant relationship between LBW and the development of dental hypoplasia was found (Table 3). The increased neonatal morbidity of LBW children could contribute to the development of enamel defects in primary dentition. In fact, various authors have linked enamel defects to systemic risk factors such as hypoxia (Seow *et al*, 1989), respiratory distress syndrome (Johnsen *et al*, 1984; Franco *et al*, 2007), cerebral damage, hyperbilirubinemia (Seow, 1991), neonatal rickets (Seow *et al*, 1984a, 1989), infection (Seow *et al*, 1989; Chaves *et al*, 2007), kidney and hepatic diseases (Koch *et al*, 1999), nutritional disorders (Rugg-Gunn *et al*, 1998; Chaves *et al*, 2007), gastroenteritis (Seow *et al*, 1989), and pneumonia (Seow, 1991).

In addition to these perinatal and neonatal conditions that tend to concur in LBW infants, maternal pregnancy care and diseases can also alter proper enamel formation. In agreement with Needleman *et al* (Needleman *et al*, 1992), our study found a statistically significant relationship between maternal age and enamel defects: the younger the mother, the higher the risk of enamel defects. These authors related this finding to the lack of adequate maternal care during pregnancy in the case of younger mothers. Also concurring with these authors, we found a significant association between smoking during pregnancy and the development of enamel defects. Besides, it is acknowledged that tobacco use during pregnancy is related to LBW. Hence, the three variables (smoking, LBW and developmental enamel defects) are implicated and interrelated.

In our study, children born by cesarean delivery were more likely to develop hypoplasias than those born by vaginal delivery. However, we believe this finding could be biased by the fact that LBW infants are at high risk for birth complications and hence are more likely to undergo cesarean delivery.

Concerning systemic variables, enamel defects were more prevalent in children with compromised immediate

perinatal outcomes: Combined defects and hypoplasia were more common in children with low 1- and 5-min Apgar scores respectively. These findings agree with other authors' (Fearne *et al*, 1990; Needleman *et al*, 1992). Also in accordance with previous studies, hypoplasia was found to be more frequent in children who required neonatal parenteral nutrition (Aine *et al*, 2000), and combined defects were more common in children who suffered mixed neonatal acidosis (Johnsen *et al*, 1984). The association between developmental enamel defects and respiratory diseases, orotracheal intubation and ventilation has already been pointed out (Mellander *et al*, 1982; Johnsen *et al*, 1984; Fearne *et al*, 1990). An accredited justification for this correlation is that ameloblastic cell function is altered by oxygen deprivation. These defects derived from systemic physiologic disruption tend to affect the teeth developing at the time of stress, and their specific location on the tooth's surface informs of the relative development of the involved teeth at that moment (Goodman, 1998).

Regarding possible local causes of enamel defects, we found that children who required mechanical ventilation and orotracheal intubation in the neonatal period were more commonly affected by severe defects (hypoplastic and combined). In these children, maxillary teeth showed more enamel defects than mandibular teeth. Besides, defects were located asymmetrically, being the left maxillary teeth more frequently involved. The possible explanation relies on the pressure exerted on the left maxillary bone by the laryngoscope during orotracheal intubation, which may disturb the normal development of local maxillary teeth (Seow *et al*, 1984b). The laryngoscope is held in the left hand and inserted into the right side of the patient's mouth, displacing the tongue to the left to gently insert the orotracheal tube into the trachea; no pressure should be applied on the teeth or oral tissues. No relationship was found between intubation length and the development of enamel defects, which suggests that the association between intubation and enamel defects is merely caused by the trauma caused by the laryngoscope on the maxillary bones if too much pressure is exerted on them during the procedure.

In conclusion, this study found children with LBW show a higher prevalence of enamel defects than NBW. Other neonatal factors that disturb normal enamel development are low gestational age, low Apgar scores, mixed acidosis and parenteral nutrition. Maternal variables such as smoking during pregnancy, young age and multiple birth pregnancy are also linked to a higher prevalence of enamel defects. Local causes include bone trauma linked to laryngoscope placement during endotracheal intubation. Further multidisciplinary studies conducted on larger samples of LBW infants with more extreme gestational ages may clarify the associations found in this study and thus help improve the quality of life and care of preterm infants.

Author Contributions

Mª Angeles Velló, Cecilia Martínez-Costa and Montserrat Catalá have participated in the study design, data collection, analysis and interpretation, drafting and revision. Jaime Fons and Juan Brines have participated in data analysis and interpretation. Raquel Guijarro-Martínez has participated in data analysis, drafting and revision.

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