

ORIGINAL ARTICLE

The relationship of enamel defects and caries: a cohort study

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Introduction

Early childhood caries (ECC) is a severe health condition found mostly in children living in socially disadvantaged communities in which malnutrition is a social

and health disparity (De Grauwe *et al*, 2004; Feldens *et al*, 2010).

Based on the available evidence, the etiologic factors related to ECC are high sugar intake (Rosenblatt and Zarzar, 2002; Harris *et al*, 2004; Milgrom *et al*, 2000; Seow *et al*, 2009; Thitasomakul *et al*, 2009; Uribe, 2009; Warren *et al*, 2009; Feldens *et al*, 2010); lack of oral hygiene (Seow *et al*, 1996; Harris *et al*, 2004; Uribe, 2009; Warren *et al*, 2009); lack of fluoride exposure (Seow *et al*, 1996; Peres *et al*, 2003; Leake *et al*, 2008); and enamel defects (Kanchanakamol *et al*, 1996; Seow *et al*, 1996; Ribeiro *et al*, 2005; Oliveira *et al*, 2006; Milgrom *et al*, 2000; Seow *et al*, 2009; Uribe, 2009).

Enamel defects are visible deviations from the normal translucent appearance of the tooth enamel resulting from early damage to the enamel organ (Suckling, 1989; FDI Commission on Oral Health, Research and Epidemiology, 1992). It is difficult to express the precise etiology of such defects because of its non-specific appearance and also to the limited data available on the chronology of the development of the human dentition (Seow, 1997). Some authors report that those defects are related to malnutrition and early childhood infections (Lai *et al*, 1997; Agarwal *et al*, 2003; Crombie *et al*, 2009).

Teeth showing enamel hypoplasia present irregular and retentive surfaces leading to increased risk for the adhesion and colonization of bacteria; therefore, infants presenting enamel hypoplasia have increased levels of *Streptococcus mutans* (Li *et al*, 1994; Uribe, 2009; Seow *et al*, 2009), and higher risk for dental caries (Pascoe and Seow, 1994; Lai *et al*, 1997; Seow *et al*, 2009; Uribe, 2009).

The current literature includes a vast number of cross-sectional studies reporting on the association between enamel defects and dental caries (Li *et al*, 1994; Pascoe and Seow, 1994; Kanchanakamol *et al*, 1996; Seow, 1997; Milgrom *et al*, 2000), however, longitudinal study represent the best study design to investigate the cause and effect relationship.

The purpose of this study was to assess the relationship between enamel defects and the development of

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dental caries in a cohort of children followed up for 54 months. This study also looks if there is any association of tooth brushing, diet and the use of fluoride toothpaste with the development of ECC.

Materials and methods

Subjects

The children were recruited at birth at a public maternity hospital in João Pessoa, a facility that provides Gynecologic and Obstetric care for poor families. This city is the capital of the state of Paraíba located in the northeastern part of Brazil, which has the lowest per capita income in the country. About 39% of the families survive with the half of the minimum legal wage and from a total of 597 934 inhabitants living in the city, only 148 000 have access to piped water, which fluoride content is lower than 0.2 ppm (IBGE, 2000). After recruitment, the children had their first dental examination at the age of 6 months, at their homes, in the suburban areas of extreme poverty.

The children were born in January/February 2000. Oliveira *et al* (2006) reported on the 24 months follow up, whereas Chaves *et al* (2007) reported on the 36 months follow up. Our study is based on the same population described by the two previous authors, however, at 12–54 months follow up.

The calculation of the sample size was based on a study by Li *et al* (1996) as described by Oliveira *et al* (2006). After 54 months, the dropout rate was 18%; however, a total of 224 children still remained in the trial, a sample large enough to provide statistically significant results.

Data collection

Ethical clearance was obtained from the Maternity Hospital Human Research Ethics Committee and the informed consent was obtained from parents at the time of birth.

The subjects were examined every 6 months from 12 to 54 months of age by three calibrated investigators. The first investigator examined children from 12 to 18 months of age, the second examiner, from 24 to 36 months and the third, from 42 to 54 months. Parents were interviewed at the same occasion.

The interviews took place prior to each dental examination and the questionnaire has been reported by Oliveira *et al* (2006) and Chaves *et al* (2007).

The children had their teeth visually examined at home, under natural light, with a dental mirror and a blunt probe, in the knee–knee position. The teeth were cleaned with gauze and examined for enamel defects using the development defects enamel (DDE) index proposed by the Fédération Dentaire Internationale (1992) and recorded on a comprehensive chart according to Oliveira *et al* (2006). We examined the buccal and lingual surfaces of each anterior tooth and the buccal, the lingual, and the occlusal surfaces of the posterior teeth. The enamel defects were classified according to their position on the crown, as gingival, incisal or occlusal half, pit and fissure and cusp. Defects measured < 1 mm in diameter were excluded from the sample and

any existing doubt concerning the presence of a defect was scored as normal.

Caries was evaluated following the World Health Organization (WHO) standard criteria for dental caries diagnosis (WHO, 1997) and if any tooth surface had a detectable softened area, the lesion was diagnosed as carious. Children were classified as presenting severe early childhood caries (ECC-S) when dental caries was present on any smooth surface, as reported by Drury *et al* (1999). However, white-spot lesions were not scored as carious lesions; they were differentiated from enamel opacities when placed adjacent to the gingival margin and extend along the buccal or lingual surfaces, while enamel opacities had no specific location on the tooth surface (Seow, 1997). The partially erupted teeth were not recorded, except when defects or caries were present on the erupted portion of the crown.

As there is a lack of access to health care for poor people living in this urban area, the children enrolled in this study were referred for treatment to the community dental clinics, to the University Dental Clinic and emergencies were treated free of charge by the dental examiners in their private dental clinic.

Calibration and reproducibility

Consistency in diagnosis among examiners was checked by inter- and intra-examiner variability tests. For the first examiner, calibration for enamel defect was conducted in a pilot study (Oliveira and Rosenblatt, 2004) when enamel defects were photographed and recorded. For the second and the third examiners, the calibration for enamel defects followed the same procedures of the pilot study. Each examiner examined the photographs twice on separate occasions, 24 h apart. Furthermore, to guarantee consistency of diagnosis for dental caries and enamel defects, the second examiner also took part in the 18-month examination, as well as the third examiner took part in the 36-month examination.

To evaluate inter-examiner agreement, 10% of the sample was randomly selected to be examined by both examiners on separate occasions, throughout the 5-year trial.

Data analysis

We used the SAS and SPSS 11.0 statistical software packages for the data analysis and both descriptive and analytical analyses were performed. We also used the chi-squared, Fischer's Exact test and the Pearson's test for categorical independent variables – enamel defects, daily tooth brushing frequency, exposure to fluoride toothpaste, sugar intake, breast-feeding, night breast-feeding, bottle-feeding, night bottle-feeding and sugar in the baby bottle (dependent variable – Table 1). *P* value < 0.05 was considered statistically significant. A 95% confidence interval was used for comparisons of different results within subgroups.

The cumulative incidence was calculated by the number of new cases during a period divided by the number of subjects at risk in the population at the beginning of the study.

Table 1 List of dependent and independent variables

| Dependent variable | Category | Variable definition | Logistic regression |
|--------------------------|-------------------------------|---|---------------------|
| Early childhood caries | Absent/present | Present when the child had ECC or ECC-S | Included |
| Independent variables | Categories | Variable definition | Logistic regression |
| Enamel defects | Absent/present | Present in one or more teeth | Included |
| Daily brushing habits | No/Yes | Present when helped by the parents at least once a day. | Included |
| Fluoride exposure | No/yes | Present when fluoride toothpaste was used at least once a day | Included |
| Dietary habits | Non-cariogenic/ cariogenic | A cariogenic diet was present when there were sugary snacks between meals | Excluded |
| Night breast-feeding | No/yes | Present when offered at least once per night | Included |
| Night bottle-feeding | No/yes | Present when used at least once per night | Included |
| Sugar in the baby bottle | No/yes | Present when the there was sugar added to the drink. | Included |

ECC, early childhood caries; ECC-S, severe early childhood caries.

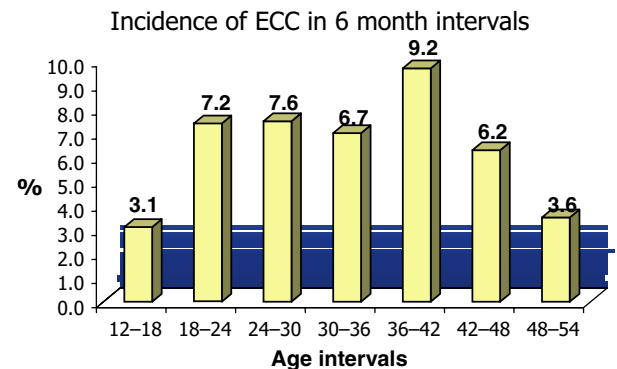
Logistic regression analysis was also carried out using the SPSS software to assess the importance of the co-variables in the model. The statistical significant variables ($P < 0.20$) for the model were selected by bivariate analysis. The variables were combined as shown in Table 1. Six logistic regression models were developed to predict caries experience at 54 months, including all the representative variables ($P < 0.20$) at 18, 24, 30, 36, 42, and 48 months of age. Stepwise elimination was used to reduce the number of variables in the models. The final model included the variables that were significant at $P < 0.05$. The lack of a model, including the representative variables at 12 months, is explained by the fact that 25 children did not show any erupted teeth at this age, yet. The Centre for Evidence-Based Medicine (Toronto; <http://www.cebm.utoronto.ca/practise/ca/statscal/>) calculator was used for calculating the NNH, which is an epidemiological measure that indicates how many patients need to be exposed to a risk-factor over a specific period to cause harm in one patient that would not otherwise have been harmed. Intuitively, the lower the number needed to harm (NNH), the worse the risk-factor.

Results

Inter- and Intra-examiner agreement for DDE and Caries scoring

Cohen's Kappa values for enamel defects for the inter-examiner agreement were 0.89 for the examinations carried out between 18 and 24 months and 0.78 for those carried out between 36 and 42 months. However, the levels of agreement for dental caries during the same period were 0.94 and 0.83, respectively.

The Cohen's Kappa values (intra-examiner agreement) varied from 0.90 to 0.98 for enamel defects and from 0.94 to 0.98 for dental caries between 12 and 18 months. At 24- to 36-month interval, Cohen's kappa value for enamel defects varied from 0.94 to 0.98 and for dental caries, from 0.96 to 0.98. In relation to the third examiner, Cohen's Kappa value for enamel defects varied from 0.96 to 0.90 and for dental caries, from 0.94 to 0.90.

**Graph 1** Incidence of ECC in six-month intervals

Association of dental caries with enamel defects

We examined a total of 224 children from the age of 12 to 54 months. At 12 months, none of the infants presented dental caries. There were 9.2% of children presenting carious teeth at 42 months and there was a decrease from 6.2% to 3.6% between 48 and 54 months, although there was an increase on active caries (Graph 1).

The present results suggested that at 54 months, 48.4% of the children presenting caries also had enamel defects ($P = 0.0091$). The risk of the children presenting enamel defects to develop dental caries was approximately two times greater than that of those without defective teeth. The NNH ranged from 3.0 at 24 months, to 5.0 at 54 months (Table 2). The incidence of dental caries in the exposed group compared with the group without enamel defects was greater along the cohort study. The cumulative incidence of caries was 0.54 from 18 to 54 months.

Twenty five children were excluded for the statistical analysis given that they had no erupted teeth until the child was 12 months of age.

Table 3 shows the relationship between different types of enamel defects and dental caries. The enamel hypoplasia was the most frequent type of enamel defect associated with dental caries. From the 10.6% of the

Table 2 Cumulative incidence of enamel defects and dental caries from 12 to 54 months of age

| Dental Caries | | | | | | | | | |
|-----------------|-------------------|---------|------|--------|-------|-------|-------|----------------|----------------------------|
| Age (months) | Enamel defects | Present | | Absent | | TOTAL | | P-Value | RR (95.0% CL) |
| | | n | % | n | % | n | % | | |
| 12 | Present | — | — | 67 | 100.0 | 67 | 100.0 | ND | ND |
| | Absent | — | — | 132 | 100.0 | 132 | 100.0 | | |
| Total | | — | — | 199 | 100.0 | 199 | 100.0 | | |
| 18 | Present | 6 | 4.4 | 131 | 95.6 | 137 | 100.0 | $P^a = 0.2546$ | 3.74 (0.46 a 30.53) |
| | Absent | 1 | 1.2 | 86 | 98.8 | 87 | 100.0 | | 1.00 NNH = 17.0 (9–167) |
| Total | | 7 | 3.1 | 217 | 96.9 | 224 | 100.0 | | |
| 24 | Present | 23 | 14.0 | 141 | 86.0 | 164 | 100.0 | $P^b = 0.0022$ | ND |
| | Absent | — | — | 60 | 100.0 | 60 | 100.0 | | 1.00 NNH = 3.0(3–5) |
| Total | | 23 | 10.3 | 201 | 89.7 | 224 | 100.0 | | |
| 30 | Present | 39 | 22.5 | 134 | 77.5 | 173 | 100.0 | $P^b = 0.0007$ | ND |
| | Absent | 1 | 2.0 | 50 | 98.0 | 51 | 100.0 | | 1.00 NNH = 5.0 |
| Total | | 40 | 17.9 | 184 | 82.1 | 224 | 100.0 | | |
| 36 | Present | 54 | 30.5 | 123 | 69.5 | 177 | 100.0 | $P^b < 0.0001$ | ND |
| | Absent | 1 | 2.1 | 46 | 97.9 | 47 | 100.0 | | 1.00 NNH = 5.0 |
| Total | | 55 | 24.6 | 169 | 75.4 | 224 | 100.0 | | |
| 42 | Present | 74 | 41.3 | 105 | 58.7 | 179 | 100.0 | $P^b < 0.0001$ | 6.20 (2.05 a 18.76) |
| | Absent | 3 | 6.7 | 42 | 93.3 | 45 | 100.0 | | 1.00 NNH = 3.0 |
| Total | | 77 | 34.4 | 147 | 65.6 | 224 | 100.0 | | |
| 48 | Present | 83 | 45.9 | 98 | 54.1 | 181 | 100.0 | $P^b = 0.0011$ | 2.46 (1.29 a 4.70) |
| | Absent | 8 | 18.6 | 35 | 81.4 | 43 | 100.0 | | 1.00 NNH = 4.0 |
| Total | | 91 | 40.6 | 133 | 59.4 | 224 | 100.0 | | |
| 54 | Present | 88 | 48.4 | 94 | 51.6 | 182 | 100.0 | $P^b = 0.0091$ | 1.85 (1.09 a 3.13) |
| | Absent | 11 | 26.2 | 31 | 73.8 | 42 | 100.0 | | 1.00 NNH = 5.0 |
| Total | | 99 | 44.2 | 125 | 55.8 | 224 | 100.0 | | |

ND – P value or RR not determined.

^a – Fischer's Exact chi-squared test.^b – Pearson's chi-squared test.

CL, confidence limits; NNH, number needed to harm.

teeth with dental caries, only 4.4% where present on teeth without enamel defects, which indicate the influence of enamel defects on the development of dental caries.

Factors influencing dental caries using a logistic regression model

Some etiologic factors are known as risk factors associated with the development of ECC. A multivariate analysis using a regression model was conducted to test the hypothesis that enamel defects are related to the development of ECC.

Two statistical models were used. At 18 months, night bottle-feeding, no fluoride exposure and enamel defects were among the selected variables that showed statistical significance ($P \leq 0.05$). At 24, 30, 36, and 42 months, enamel defects were the only variable proved to be statistically significant for the development of caries. At 48 months, the lack of exposure to fluoride toothpaste and enamel defects was the variables that showed the strongest relationship to the development of caries at 54 months (Table 4).

Discussion

The results of this longitudinal study show that ECC in preschoolers has a strong relationship to enamel defects, when compared with children who did not present those defects at that same age interval. Such association has been previously reported by Oliveira *et al* (2006) which is in agreement with other cross-sectional studies (Li *et al*, 1994; Pascoe and Seow, 1994; Kanchanakamol *et al*, 1996; Seow *et al*, 1996; Milgrom *et al*, 2000) and case-control study (Uribe, 2009). However, a longitudinal study may represent the best study design to check the influence of certain variables before the disease occurs, given that in cross-sectional studies untreated caries lesions may mask enamel defects.

As dental caries do occur in enamel hypoplasia sites, the first may mask these preexisting enamel defects, a fact already mentioned in previous cross-sectional studies (Johnsen, 1982). Likewise, when reporting on the occurrence of enamel hypoplasia and dental caries in 3–4 year olds, Vignarajah and Williams (1992)

Table 3 Relationship among different types of enamel defects and dental caries at 54 months of age

| Enamel defects | Dental caries | | | | | | P-value |
|--------------------------------|---------------|------|--------|------|-------|-------|-----------|
| | Present | | Absent | | Total | | |
| | n | % | n | % | N | % | |
| Absent | 146 | 4.4 | 3191 | 95.6 | 3337 | 100.0 | < 0.0001* |
| Demarcated opacity | 4 | 3.0 | 130 | 97.0 | 134 | 100.0 | |
| Diffuse opacity | 58 | 12.6 | 401 | 87.4 | 459 | 100.0 | |
| Hypoplasia (reduced thickness) | 5 | 3.8 | 125 | 96.2 | 130 | 100.0 | |
| Hypoplasia(missing enamel) | 181 | 65.3 | 96 | 34.7 | 277 | 100.0 | |
| Opacity + hypoplasia | 81 | 56.6 | 62 | 43.4 | 143 | 100.0 | |
| Total | 475 | 10.6 | 4005 | 89.4 | 4480 | 100.0 | |

*P-values obtained by Pearson's chi-squared test.

Table 4 Regression analysis model for dental caries at 54 months

| Variables | Model 1* | | Model 2** | |
|-------------------------------|------------------|----------|------------------|------------|
| | OR (95% CL) | P | OR (95% CL) | P |
| 18 months | | | | |
| Night bottle-feeding | 0.47 (0.3–0.8) | 0.007 | 0.49 (0.3–0.9) | 0.021*** |
| No fluoride exposure | 1.76 (1–3) | 0.039 | 2.16 (1.2–3.9) | 0.010*** |
| Enamel defects | 4.02 (2.21–7.30) | < 0.0001 | 4.10 (2.21–7.60) | < 0.001*** |
| Lack of daily brushing habits | 1.75 (1–3) | 0.038 | | |
| Sugar in the bottle | 0.47 (0.3–0.9) | 0.017 | | |
| 24 months | | | | |
| Night breast-feeding | 1.89 (1–3.7) | 0.060 | 1.84 (0.9–3.7) | 0.79 |
| Enamel defects | 2.82 (1.5–5.4) | 0.001 | 2.79 (1.5–5.4) | 0.002*** |
| Sugar in the baby bottle | 0.63 (0.3–1.2) | 0.16 | | |
| 30 months | | | | |
| Enamel defects | 2.89 (1.4–5.8) | 0.002 | 2.88 (1.4–5.8) | 0.003*** |
| Sugar in the bottle | 0.60 (0.3–1.1) | 0.092 | 0.61 (0.3–1.1) | 0.103 |
| 36 months | | | | |
| Enamel defects | 2.81 (1.4–5.8) | 0.003 | 2.74 (1.3–5.7) | 0.006*** |
| No fluoride exposure | 1.89 (0.7–5.2) | 0.20 | 1.88 (0.7–5.2) | 0.226 |
| Sugar in the bottle | 0.68 (0.4–1.2) | 0.17 | 0.66 (0.4–1.2) | 0.16 |
| 42 months | | | | |
| Cariogenic habits | 0.30 (0.1–1.6) | 0.14 | 0.26 (0.1–1.4) | 0.122 |
| Enamel defects | 2.99 (1.4–6.3) | 0.002 | 3.12 (1.5–6.6) | 0.003*** |
| 48 months | | | | |
| No fluoride exposure | 2.14 (1.1–4.1) | 0.016 | 2.23 (1.2–4.3) | 0.015*** |
| Enamel defects | 2.75 (1.3–5.8) | 0.006 | 2.85 (1.3–6.1) | 0.007*** |
| Daily brushing habits | 1.42 (1.1–1.9) | 0.018 | | |

*Model 1 – Variables included in the model after bivariate analysis $P \leq 0.20$.

**Model 2 – Variables that maintained $P \leq 0.20$ and submitted to logistic regression.

***Significant association ($P < 0.05$) after logistic regression.

OR, odds ratios; CL, confidence limits.

observed that enamel opacity occurred mostly on the gingival site.

Given that the gingival site which is also the enamel layer first to be attacked is located on the buccal surface of the tooth, we assume that it is caused by calcification damages occurring in the final phase of mineralization. This was previously mentioned by Matee *et al* (1994) and Lai *et al* (1997); however, in Lai's study, the first examinations were carried out at 30 months of age, when some defects had already been misdiagnosed as dental caries.

A logistic regression analysis was conducted to evaluate the correlation of ECC with their risk factors at 54 months of age. The association between enamel defects

and caries was observed in the two logistic regression models. This cohort study support the hypothesis that enamel defects predispose children to an increased caries risk as reported by other studies (Li *et al*, 1996; Lai *et al*, 1997; Oliveira *et al*, 2006; Chaves *et al*, 2007; Milgrom *et al*, 2000; Uribe, 2009). Other risk factors for dental caries were selected by the model, but they were not statistically associated with dental caries. This indicates a great influence of enamel defects on dental caries, especially in children living in socially disadvantaged communities as reported by Oliveira *et al* (2006).

The risk of children presenting enamel defects to develop dental caries was approximately two times

greater than those without these defective teeth. This risk varied along the study and at 24 months, NNH was 3.0. According to Oliveira *et al* (2006), children presenting enamel defects were 15 times more likely to develop dental caries than children showing no defects.

Among the types of defects found in this sample, enamel hypoplasia was found to be most frequently associated with dental caries, as described in previous studies (Matee *et al*, 1994; Kanchanakamol *et al*, 1996; Li *et al*, 1996; Milgrom *et al*, 2000).

The regression analysis models show that the following variables also influenced the development of dental caries at 54 months: the lack of use of fluoridated toothpaste at 18 and 48 months and the use of night feeding-bottle at 18 months. Twetman (2008, 2000), in two literature reviews on prevention of ECC, reported fluoride toothpaste as the most cost-effective home-care caries preventive treatment and Milgrom *et al* (2000) in a cohort of children aged from 6 to 36 months using semi-annual fluoride varnish applications indicated that it is the best professional method and found no association between dental caries and the use of night feeding-bottle.

In general, socioeconomic status has been considered as an indicator for caries risk assessment (Feldens *et al*, 2010; Agarwal *et al*, 2003; Oliveira *et al*, 2006; Li *et al*, 1996; Disney *et al*, 1992; Peres *et al*, 2005; Oliveira *et al*, 2008). The present results bring up the discussion on ECC prevention, which should imply on a holistic approach to health. Malnutrition plays an important role in dental development (Feldens *et al*, 2010; Li *et al*, 1996; Chaves *et al*, 2007; Peres *et al*, 2005; Oliveira *et al*, 2008; Ngoenwiwatkul and Leela-adisorn, 2009; Psoter *et al*, 2005) and the lack of basic nutrients in the intra-uterine phase could be a strong determinant for ECC (Uribe, 2009). The cumulative incidence of 0.54 indicates that it is high among the target population which suggests that there is a lack of social and health promotion policies for this target population. Therefore, the authors suggest that public policies should focus on the improvement of the quality of life and the general health of the less favored social groups.

Conclusion

There was a strong relationship between ECC and enamel defects in children aged from 18 to 54 months. The use of fluoridated toothpaste was found to be related to the decrease in the development of dental caries.

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References

Agarwal KN, Narula S, Faridi MMA, Kalra N (2003). Deciduous dentition and enamel defects. *Indian Pediatr* **40**: 124–129.

Chaves AMB, Rosenblatt A, Oliveira AFB (2007). Enamel defects and its relation to life course events in primary dentition of Brazilian children: a longitudinal study. *Community Dent Health* **24**: 31–36.

Crombie F, Manton D, Kilpatrick N (2009). Aetiology of molar-incisor hypomineralization: a critical review. *Int J Paediatr Dent* **19**: 73–83.

De Grauwe A, Aps JK, Martens LC (2004). Early Childhood Caries (ECC): What's in a name? *Eur J Paediatr Dent* **5**: 62–70.

Disney JA, Graves RC, Stamm JW, Bohannon HM, Abernathy JR, Zack DD (1992). The University of North Carolina Caries assessment study further developments in caries risk prediction. *Community Dent Oral Epidemiol* **20**: 64–75.

Drury TF, Horowitz AM, Ismail AI, Maertens MP, Rozier RG, Selwitz RH (1999). Diagnosing and reporting early childhood caries for research purposes. *J Public Health Dent* **59**: 192–197.

FDI Commission on Oral Health, Research and Epidemiology (1992). A review of developmental defects on dental enamel index (DDE index). *Int Dent J* **42**: 411–426.

Feldens CA, Giugliani ER, Duncan BB, Drachler MD, Vitolo MR (2010). Long-term effectiveness of a nutritional program in reducing early childhood caries: a randomized trial. *Community Dent Oral Epidemiol* **38**: 324–332.

Harris R, Nicoll AD, Adair PM, Pine CM (2004). Risk factors for dental caries in young children: a systematic review of the literature. *Community Dent Health* **21**(1 Suppl): 71–85.

IBGE (Brazilian Institute of Geography and Statistics) (2000): *Características da população e dos domicílios. Resultados do universo. Notas metodológicas*. IBGE: Rio de Janeiro.

Johnsen DC (1982). Characteristics and backgrounds of children with “nursing caries”. *Pediatr Dent* **4**: 218–224.

Kanchanakamol U, Tuongratanaphan S, Tuongratanaphan S *et al* (1996). Prevalence of developmental enamel defects and dental caries on rural preschool Thai children. *Community Dent Health* **13**: 204–207.

Lai PY, Seow WK, Tudehope DI, Rogers Y (1997). Enamel hypoplasia and dental caries in very-low-birthweight children: a case controlled, longitudinal study. *Pediatr Dent* **19**: 42–49.

Leake J, Jozzy S, Uswak G (2008). Severe dental caries, impacts and determinants among children 2–6 years of age in Inuvik Region, Northwest Territories, Canada. *Can Dent Assoc* **74**: 519–519g.

Li Y, Navia JM, Caufield PW (1994). Colonization by mutans streptococci in the mouths of 3- and 4-year-old Chinese children with or without enamel hypoplasia. *Arch Oral Biol* **39**: 1057–1062.

Li Y, Navia JM, Bian JY (1996). Caries experience in deciduous dentition of Rural Chinese children 3–5 years old in relation to the presence or absence of enamel hypoplasia. *Caries Res* **30**: 8–15.

Matee MIN, Van't Hof MA, Maselle SY, Mikx FHM, Van Palenstein Herderman WH (1994). Nursing caries, linear hypoplasia, and nursing and weaning habits in Tanzanian infants. *Community Dent Oral Epidemiol* **22**: 289–293.

Milgrom P, Riedy CA, Weinstein P, Tanner ACR, Manibusan L, Bruss J (2000). Dental caries and its relationship to bacterial infection, hypoplasia, diet, and oral hygiene in 6- to 36-month-old children. *Community Dent Oral Epidemiol* **28**: 295–306.

Ngoenwiwatkul Y, Leela-adisorn N (2009). Effects of dental caries on nutritional status among first-grade primary school children. *Asia Pac J Public Health* **21**: 177–183.

- Oliveira AFB, Rosenblatt A (2004). Prevalência de defeitos do esmalte e cárie dentária em crianças pré-escolares em João Pessoa/Brasil. *Rev ABO Nac* **12**: 107–110.
- Oliveira AFB, Chaves AMB, Rosenblatt A (2006). The influence of enamel defects on the development of early childhood caries in a population with low socioeconomic status: a longitudinal study. *Caries Res* **40**: 296–302.
- Oliveira LB, Sheiham A, Bönecker M (2008). Exploring the association of dental caries with social factors and nutritional status in Brazilian preschool children. *Eur J Oral Sci* **116**: 37–43.
- Pascoe L, Seow WK (1994). Enamel hypoplasia and dental caries in Australian Aboriginal children: prevalence and correlation between the two diseases. *Pediatr Dent* **16**: 193–199.
- Peres MA, Peres KG, Antunes JLF, Junqueira SR, Frazão P, Narvai PC (2003). The association between socioeconomic development at the town level and the distribution of dental caries in Brazilian children. *Rev Panam Salud Publica* **14**: 149–157.
- Peres MA, Oliveira LMR, Sheiham A et al (2005). Social and biological early life influences on severity of dental caries in children aged 6 years. *Community Dent Oral Epidemiol* **33**: 53–63.
- Psoter WJ, Reid BC, Katz RV (2005). Malnutrition and dental caries: a review of the literature. *Caries Res* **39**: 441–447.
- Ribeiro AG, Oliveira AF, Rosenblatt A (2005). Cárie precoce na infância: prevalência e fatores de risco em pré-escolares, aos 48 meses, na cidade de João Pessoa, Paraíba, Brasil. *Caderno de Saúde Pública* **21**: 1695–1700.
- Rosenblatt A, Zarzar P (2002). The prevalence of early childhood caries in 12- to 36-month-old children in Recife, Brazil. *Journal of Dentistry for Children* **69**: 319–24.
- Seow WK (1997). Clinical diagnosis of enamel defects: pitfalls and practical guidelines. *Int Dent J* **47**: 173–182.
- Seow WK, Amaratunge A, Bennett R, Bronsch D, Lai PY (1996). Dental health of aboriginal pre-school children in Brisbane, Australia. *Community Dent Oral Epidemiol* **24**: 187–190.
- Seow WK, Clifford H, Battistutta D, Morawska A, Holcombe T (2009) Case-control study of early childhood caries in Australia. *Caries Research* **43**: 25–35.
- Suckling GW (1989). Development defects of enamel – historical and present day perspectives of their pathogenesis. *Adv Dent Res* **3**: 87–94.
- Thitasomakul S, Piwat S, Thearmentree A, Chankanka O, Pithpornchaiyakul W, Madyusoh S (2009). Risks for early childhood caries analyzed by negative binomial models. *J Dent Res* **88**: 137–141.
- Twetman S (2008). Prevention of early childhood caries (ECC) – review of literature published 1998–2007. *Eur Arch Paediatr Dent* **9**: 12–18.
- Twetman S (2009). Caries prevention with fluoride toothpaste in children: an update. *Eur Arch Paediatr Dent* **10**: 162–167.
- Uribe S (2009). Early childhood caries – risk factors. *Evid Based Dent* **10**: 37–38.
- Vignarajah S, Williams A (1992). Prevalence of dental caries and enamel defects in the primary dentition of Antiguan pre-school children aged 3–4 years including an assessment of their habits. *Community Dent Health* **9**: 349–360.
- Warren JJ, Weber-Gasparoni K, Marshall TA et al (2009). A longitudinal study of dental caries risk among very young low SES children. *Community Dent Oral Epidemiol* **37**: 116–122.
- WHO (1997). *Basic methods*, 4th edn. World Health Organization: Geneva.

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