Scientific Article

Enamel hypoplasia and dental caries in very-low birthweight children: a case-controlled, longitudinal study

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Abstract

This longitudinal study investigated the sequelae of enamel defects in a group of 25 white, very-low birthweight (VLBW), preterm children (mean birthweight 969 ± 218 g, mean gestational age 27 ± 1.9 weeks). Twenty-five race-, age-, and sex-matched, full-term normal birthweight (NBW) control children born at the same hospital, were selected randomly from hospital records. The children were examined at approximate ages of 30, 44, and 52 months. At all examinations, VLBW children had significantly higher prevalence of enamel hypoplasia than did the NBW children. At the last recall examination, 96% of VLBW group, and 45% of the NBW group had at least one tooth with enamel defect, with a mean of 7.6 ± 4.9 affected teeth per VLBW child, and only 1.0 ± 1.3 affected teeth per control child ($P < 0.001$). A significant association of enamel defects with dental caries was observed only in the VLBW group on the second and third examinations ($P < 0.001$). The defect identified to be most significantly associated with dental caries was a variant showing both enamel hypoplasia and opacity. In spite of a high prevalence of enamel defects, the overall prevalence of dental caries in the VLBW children was not significantly different from that of NBW controls at all three examinations ($P > 0.1$). Other caries risk factors such as levels of Streptococcus mutans *infection, fluoride supplementation, plaque scores, toothbrushing frequency, and daily sugar exposures were examined but none was found to be related significantly to development of dental caries. (Pediatr Dent 19:42–49, 1997)*

Previous investigations on the dental complications of preterm and low birthweight children have all been cross-sectional.[1-15] In these studies, a high prevalence of developmental enamel defects had been found, with the highest frequency of more than 70% in very-low birthweight (< 1500 g, VLBW) children, and lower frequency of approximately 40% in the low birthweight groups (1500–2000 g, LBW).[1-11] The etiological factors associated with these defects include systemic metabolic changes associated with prematurity[1-11] as well as local traumatic forces resulting from laryngoscopy and intubation during the neonatal period.[12-13]

The clinical implications of the high prevalence of enamel hypoplasia in preterm children have not been well explored. In particular, the effects of enamel hypoplasia on the development of dental decay in preterm children are unclear. A few studies[3, 16] have suggested that LBW predisposes a child to increased dental caries but the relative importance of enamel hypoplasia compared with other caries risk factors is unknown. To the authors' knowledge, longitudinal studies to examine the sequelae of enamel defects in VLBW children have not been done before.

The aim of our investigation was to study a cohort of VLBW children and a matched, control group of full-term children longitudinally to determine whether enamel hypoplasia seen in VLBW children predisposes them to increased dental caries risk.

Subjects and methods

The subjects were preterm, VLBW children cared for at the Mater Mothers' Hospital.[17, 18] All VLBW children born between 1989 and 1992 were sent letters of invitation to participate.

For each VLBW subject included in the study, a full-term, normal birthweight (NBW) control patient matched for sex, and born at approximately the same time at the same hospital, was selected at random from hospital records and invited to participate in the study.

The consent rate for participation in the study was 97% in the group of patients who could be contacted. The subjects were examined at the pediatric dental clinic at the University Dental School. Signed informed consent was obtained from the parents.

The socioeconomic status of the subjects was assumed based on the parents' occupations and suburb of residence.[19, 20] Medical perinatal and neonatal histories were obtained from hospital records. Relevant postnatal histories were obtained from the parents. Dental histories, including past dental treatment, fluoride supplementation, and residence in towns with

TABLE 1. DEMOGRAPHIC DATA OF SUBJECTS IN STUDY.

	VLBW (*N* = 25)	*NBW* (*N* = 25)	*P-value*
Sex			
Boys	12	12	NS*
Girls	13	13	NS*
Mean Birthweight (g) ± SD	969 ± 218	3418 ± 415	$P < 0.001$
Range (g)	(652–1410)	(2810–4110)	$t = 25.7$, df = 44
Mean Gestational Age (wks) ± SD	27 ± 1.9	40 ± 1.2	$P < 0.001$
Range (wks)	(24–29)	(37–42)	$t = 34.2$, df = 44
Socioeconomic Status			
I (high)	12	8	NS*
II (middle)	3	2	NS*
III (low)	8	10	NS*
Unclassified	2	5	NS*
Mean Age at Exam (month ± SD)			
Exam I	27.5 ± 4.6	32.3 ± 2.8	NS†
Exam II	41.4 ± 6.5	46.2 ± 3.6	NS†
Exam III	51.5 ± 4.9	52.5 ± 4.2	NS†

* Chi-square tests.

† Student's *t*-test.

fluoridated water, oral hygiene habits, and frequency of daily of toothbrushing were obtained from the parents.

A three-day (including a weekend day) diet history form[21] was issued to each patient, and the parents were requested to fill in details of all foods and drinks consumed during and between meals.

The teeth were dried with gauze, and examined for enamel opacity, enamel hypoplasia, and dental caries. The modified DDE (Developmental Defects of Enamel) Index[26] was used to chart enamel defects. In brief, enamel hypoplasia

TABLE 2. PREVALENCE OF VARIOUS TYPES OF ENAMEL DEFECTS IN THE VLBW AND NBW CHILDREN

	Exam I		*Exam II*		*Exam III*	
	VLBW	*NBW*	*VLBW*	*NBW*	*VLBW*	*NBW*
Total no. of children	25	25	19	11	24	20
No. (%) of children affected with						
Enamel hypoplasia	17 (68)	1 (4)	10 (53)	0	16 (67)	2 (10)
(*P*-value)	$P < 0.001$, $\chi^2 = 19.5$, df = 1		$P < 0.001$, odds ratio = 0		$P < 0.001$, $\chi^2 = 12.2$, df = 1	
Enamel opacities	12 (48)	7 (28)	14 (74)	5 (46)	19 (79)	6 (30)
(*P*-value)	NS		NS		$P < 0.001$, $\chi^2 = 8.8$, df = 1	
Hypoplasia with opacity	0	2 (8)	3 (16)	2 (18)	13 (54)	2 (10)
(*P*-value)	NS		NS		$P < 0.005$, $\chi^2 = 7.6$, df = 1	
Total No. (%) of affected children	22 (88)	10 (40)	18 (95)	6 (54)	23 (96)	9 (45)
(*P*-value)	$P < 0.001$, $\chi^2 = 10.5$, df = 1		$P < 0.029$, $\chi^2 = 4.7$, df = 1		$P < 0.001$, $\chi^2 = 11.8$, df = 1	
Total no. of teeth	377	476	392	220	479	399
No. (%) of teeth affected with						
Enamel hypoplasia	43 (11)	4 (1)	36 (9)	0	51 (11)	5 (1)
(*P*-value)	$P < 0.001$, $\chi^2 = 43.1$, df = 1		$P < 0.001$, odds ratio = 0		$P < 0.001$, $\chi^2 = 30.6$, df = 1	
Enamel opacity	24 (6)	9 (2)	48 (12)	9 (4)	88 (18)	12 (3)
(*P*-value)	$P < 0.001$, $\chi^2 = 10.2$, df = 1		$P < 0.001$, $\chi^2 = 10.1$, df = 1		$P < 0.001$, $\chi^2 = 49.1$, df = 1	
Hypoplasia with opacity	0	3 (1)	6 (2)	3 (1)	49 (10)	3 (1)
(*P*-value)	NS	NS	NS	NS	$P < 0.001$, $\chi^2 = 33.4$, df = 1	
Total no. (%) of affected teeth	67 (18)	16 (3)	90 (23)	12 (6)	188 (39)	20 (5)
(*P*-value)	$P < 0.001$, $\chi^2 = 48.1$, df = 1		$P < 0.001$, $\chi^2 = 29.8$, df = 1		$P < 0.001$, $\chi^2 = 135.6$, df = 1	
Mean ± SD affected teeth/child	2.6± 2.5	0.6 ± 1.0	4.7 ± 4.1	1.1 ± 1.2	7.8 ± 4.8	1.0 = 1.3
(*P*-value)	$P < 0.001$, $t = 3.71$, df = 48		$P < 0.009$, $t = 2.8$, df = 28		$P < 0.001$ $t = 6.14$, df = 42	

was diagnosed if there was a deficiency of enamel in the form of pits, grooves, or other quantitative surface loss. Enamel opacity was diagnosed if there was a qualitative change in the translucency of enamel without loss of enamel surface. Enamel opacity may be white or stained. When an enamel defect had both hypoplasia and opacity, it was classified as a combination defect. The criteria of Radike[23] were used to diagnose dental caries. Only lesions with frank cavitation were scored as carious. All surfaces of each tooth were examined, and the severity and extent of each defect recorded.

The percentages of teeth affected by enamel hypoplasia and by dental caries were computed by dividing the number of affected teeth by the total number of teeth present.

A plaque score was obtained from each subject using the modified plaque score of Loe and Silness.[24, 25] In brief, plaque was noted to be either present (score of 1) or absent (score of 0) on the facial, lingual, and mesial surfaces of the following maxillary teeth: left central incisor, left first molar, right second molar and the following mandibular teeth: right central incisor, right first molar, and left second molar. This modified scoring system is simple and practical for use in young children. A percentage score was computed by dividing the obtained score by the maximum possible score. The maximum score was altered accordingly if one or more of the index teeth used for scoring were absent.

The dental examinations were performed by a single examiner (PYL). Intra-examiner consistency of the examination technique was demonstrated by comparing the results of plaque scores, enamel defects, and caries examination of three VLBW children three times on three different occasions. The kappa statistic[24] value was 0.7, which indicated good consistency.

Streptococcus mutans infection in each patient was assessed using Dentocult™ Strip mutans kits (Orion Diagnostica, Espoo, Finland).[27] A treated plastic strip supplied by the manufacturer was inserted into the mouth, and turned five times on each side to contact the tongue. The strip was then removed and placed in a vial containing bacterial culture medium. After incubation for 2 days at 37°C, the strip was removed, air-dried, and the number of *Streptococcus mutans* colonies counted and graded against standards provided on the manufacturer's charts. A score of 1 was given for colony counts of $<10^5$, 2 for counts of 10^5–10^6, and 3 for counts $> 10^6$. The second and third dental examinations were performed approximately 9 months and 18 months after the first one.

Statistical analysis

The student's *t*-test, chi-square test, or Fisher's exact test, was used for statistical analysis of the data where appropriate.

Results

Demography of subjects

Table 1 shows the demography of subjects in the study. There was a total of 25 in each group of VLBW and full-term control children. There were 12 boys and 13 girls in each group. The mean birthweight and gestational age for the VLBW group was 969 ± 218g and 27 ± 1.9 weeks, and 3418 ± 415 and 40 ± 1.2 weeks for the NBW group.

As shown in Table 1, there were no significant differences in the distribution of SES between the VLBW and NBW groups.

The mean ages (months) of preterm children at examinations I, II, and III were 27.5 ± 4.6, 41.4 ± 6.5, and 51.5 ± 4.9 respectively, and those for control children were 32.3 ± 2.8, 46.2 ± 3.6, and 52.5 ± 4.2, respectively. Differences in the corresponding mean ages between the VLBW and control groups were not significant ($P > 0.1$).

Prevalence of enamel defects in VLBW and NBW children

Table 2 shows the prevalence of enamel defects in the VLBW and NBW groups. The percentage of NBW children showing at least one type of enamel defect was 40% at exam I, 54% at exam II, and 45% at exam III.

In contrast, the percentage of VLBW children showing at least one type of enamel defect was 88% at exam

TABLE 3. DENTAL CARIES IN THE PRETERM AND CONTROL CHILDREN

	Exam I		*Exam II*		*Exam III*	
	VLBW (*N* = 25)	*NBW* (*N* = 25)	*VLBW* (*N* = 19)	*NBW* (*N* = 11)	*VLBW* (*N* = 24)	*NBW* (*N* = 20)
Mean age ± SD (months)	27.5 ± 4.6	32.3 ± 2.8	41.4 ± 6.5	46.2 ± 3.6	51.5 ± 4.9	52.5 ± 4.2
No. (%) of caries-free children	25 (100%)	24 (96%)	14 (78%)	10 (91%)	17 (71%)	16 (80%)
Mean no. of decayed teeth (*dt*) per child	0	0.04 ± 0	0.58 ± 2.0	0.45 ± 0.8	0.42 ± 1.1	1.15 ± 2.2
Mean no. of filled teeth (*ft*) per child	0	0	0	0	0.42 ± 1.1	0.25 ± 0.6
Mean dmft ± SD	0	0 ± 0.2	0.6 ± 1.4	0.5 ± 1.5	0.8 ± 1.5	1.4 ± 3.2

Student's *t* tests indicated that differences in mean dmft between the VLBW and NBW groups were nonsignificant at all dental examinations.

TABLE 4. ASSOCIATION OF DENTAL CARIES WITH ENAMEL DEFECTS BY THE NUMBER OF TEETH IN VLBW AND NBW CHILDREN

		Teeth Affected Number [%] Dental Caries			
	Enamel Defects	*Dental Caries Present*	*Dental Caries Absent*	*Total*	*P-value**
Exam I VLBW	Present	0	67 (19)	67 (19)	
					NS
	Absent	0	290 (81)	290 (81)	
	Total	0	357 (100)	357 (100)	
NBW	Present	0	16 (4)	16 (4)	
					NS
	Absent	1 (0.2)	423 (96)	424 (96)	
	Total	1 (0.2)	439 (99)	440 (100)	
Exam II VLBW	Present	10 (3)	78 (21)	88 (24)	
					$P < 0.001$,
	Absent	1 (0.3)	283 (76)	284 (76)	$\chi^2 = 24.7$
	Total	11 (3)	361 (97)	372 (100)	df = 1
NBW	Present	0	12 (6)	12 (6)	
					NS
	Absent	5 (2)	203 (92)	208 (94)	
	Total	5 (2.3)	215 (98)	220 (100)	
Exam III VLBW	Present	16 (3)	169 (35)	185 (38)	
					$P < 0.001$
	Absent	4 (1)	290 (61)	294 (62)	$\chi^2 = 13.3$
	Total	20 (4)	459 (96)	479 (100)	df = 1
NBW	Present	1 (0.3)	19 (5)	20 (5)	
					NS
	Absent	27 (7)	352 (88)	379 (95)	
	Total	28 (7)	371 (93)	399 (100)	

* *P*-values were calculated using the chi-square test.

NS = Not significant.

I ($P < 0.001$), 95% at exam II ($P < 0.029$), and 96% at exam III ($P < 0.001$).

In relation to the total number of teeth present at each examination (Table 2), in the NBW control children, the percentage of teeth showing at least one type of enamel defect was 3% at exam I, 6% at exam II, and 5% at exam III. In contrast, the figures for the VLBW group were 18% at exam I ($P < 0.001$), 23% at exam II ($P < 0.009$), and 39% at exam III ($P < 0.001$).

Prevalence of dental caries in VLBW and NBW children

Table 3 shows the prevalence of dental caries in the VLBW and NBW control groups of children. In Exam I, all but one child in the NBW group were caries-free. At exams II and III, more children were caries-free in the NBW group than in the VLBW group, but the results were not statistically significant. The mean number of decayed, missing and filled teeth (dmft) increased with increasing ages of the children. At the last recall at mean age of approximately 52 weeks of age, the NBW group had a mean dmft of 1.4 ± 3.2 compared with 0.8 ± 1.5 in the VLBW group. This difference was not significant ($P > 0.1$).

Distribution of enamel defects and dental caries

The distributions of teeth affected by enamel defects and dental caries are shown in Fig 1. In the VLBW group, the most common primary tooth series affected by enamel defects are, in order of decreasing prevalence, second molars (52%), first molars (43%), incisors (31%), and canines (31%). With regard to dental caries, the most common primary tooth groups affected are, in order of decreasing prevalence, second molars (14%), first molars (3%), incisors (1%), and canines (1%).

In contrast, in the case of NBW children, enamel defects were most commonly observed in the canines (13%), followed by the incisors (4%) and second molars (4%) in decreasing order. Dental caries in this group was most common in the first molars (15%), followed in fairly even distribution by the second molars (8%), canines (5%), and incisors (4%).

TABLE 5. THE ASSOCIATION OF VARIOUS TYPES OF ENAMEL DEFECTS TO DENTAL CARIES IN NBW AND VLBW CHILDREN (AT EXAM III)

	Teeth Affected			
Enamel Defects	*Caries Present No. (%)*	*Caries Absent No. (%)*	*Total No. (%)*	*P-value**
NBW				
None	27 (7)	352 (88)	379 (95)	
Hypoplasia	1 (0)	4 (1)	5 (1)	NS
Opacities	0 (0)	12 (3)	12 (3)	NS
Hypoplasia with opacity	0 (0)	3 (1)	3 (1)	NS
Total	28 (7)	371 (93)	399 (100)	
VLBW				
None	4 (1)	287 (60)	291 (61)	
Hypoplasia	0 (0)	51 (11)	51 (11)	NS
Opacities	4 (1)	84 (17)	88 (18)	NS
Hypoplasia with opacity	12 (2)	37 (8)	49 (10)	$P < 0.001$, $\chi^2 = 50.8$, df = 1
Total	20 (4)	459 (96)	479 (100)	

* Using chi-square tests.

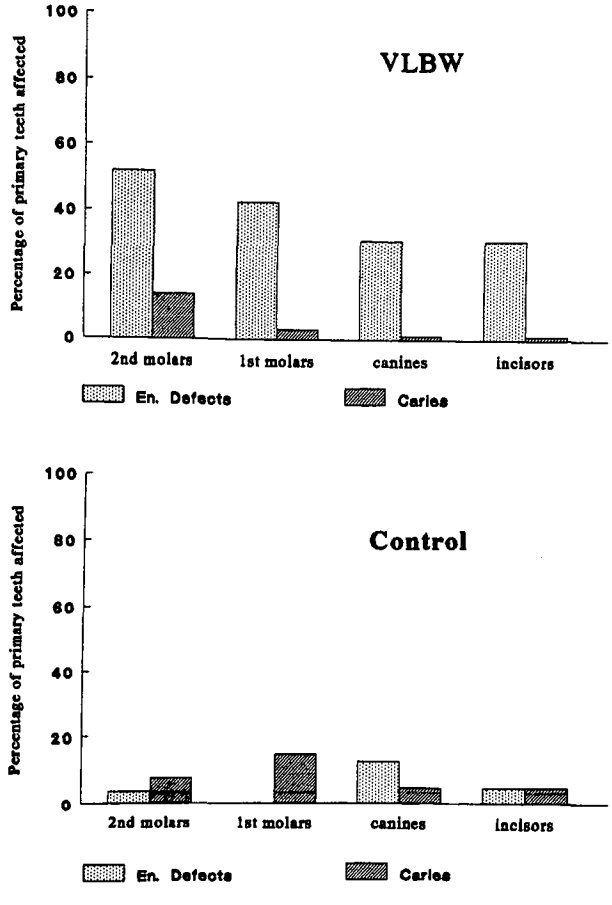

Fig 1. Distributions of enamel (En) hypoplasia and dental caries in preterm and control children. The data were obtained from the final examination at mean age of 51.5 ± 5.1 months for the VLBW children and 52.5 ± 4.2 months for the control children.

Association of dental caries with enamel defects

The number of enamel defects with and without dental caries was analyzed to determine their association. The results, as shown in Table 4, indicated that in the case of the VLBW group, there were strong associations between enamel defects and dental caries only at exam II ($P < 0.001$) and exam III ($P < 0.001$). In contrast, in the case of the NBW control children, there were no relationships between enamel defects and dental caries at any examinations.

Type of enamel defects associated with dental caries

The data for this analysis were taken from exam III. Table 5 shows the different types of enamel defects associated with dental caries. In the VLBW group, only the type of defect charted as "hypoplasia with opacity" was significantly associated with dental caries ($P < 0.001$). This type of defect was most commonly found in the primary molars, and involved loss of full thickness of the enamel in the cuspal-occlusal regions. Dental caries was diagnosed if these defects appeared soft to probing.

Factors influencing dental caries

Factors other than enamel hypoplasia that may influence dental caries in the VLBW and NBW children were examined also (Table 6). These include *Streptococcus mutans* scores, plaque scores, daily brushing frequency, fluoride exposure, and daily sugar intake. Regression tests indicated that none of the factors showed significant association with dental caries ($P > 0.1$).

In addition, the socioeconomic status of the children also was analyzed with respect to their dental caries status (Table 7). In the VLBW group, four of seven (57%) children with dental caries had high socioeconomic status (SES), compared to only one of five (20%) of the NBW group. However, these results did not reach statistical significance ($P > 0.1$).

Discussion

In this study, the prevalence of enamel defects of 96% found in VLBW children was very high compared with our previous investigations,[8–14] which showed prevalences of approximately 50–70%. The main reason for this could be that most of the subjects in this study had extremely low birthweights of < 1000 g, whereas those of the previous studies were < 1500 g. The greater systemic derangement associated with the extremely low birthweight, together with greater need

TABLE 6. FACTORS INFLUENCING DENTAL CARIES

	Number of Children (%)			
	VLBW		NBW	
	Caries Present	Caries Free	Caries Present	Caries Free
Strep. Mutans Score				
0	4 (24)	13 (76)	0	10 (100)
1	0 (0)	3 (100)	0	1 (100)
2	3 (75)	1 (25)	3 (100)	0
3	0	1 (100)	1 (100)	0
Plaque Score				
0–25%	5 (33)	10 (67)	0	13 (100)
More than 25–50%	2 (25)	6 (27)	3 (33)	6 (67)
More than 50–75%	1 (50)	1 (50)	1 (100)	0
More than 75%	0	0	0	1 (100)
Brushing History (mean frequency/day)				
Less than 1	0	2 (100)	0	0
1 to less than 2	1 (10)	9 (90)	4 (29)	10 (71)
2 to less than 3	6 (46)	7 (54)	0	10 (100)
3 and more	0	0	1 (100)	0
*Fluoride Exposure**				
Yes	5 (33)	10 (67)	1 (17)	5 (83)
No	2 (20)	8 (80)	4 (21)	15 (79)
Sugar Intake (mean daily frequency)				
1 to less than 2	0	5 (100)	0	2 (100)
2 to less than 3	3 (50)	3 (50)	0	3 (100)
3 to less than 4	3 (38)	5 (62)	1 (33)	2 (67)
4 to less than 5	0	2 (100)	0	1 (100)

Chi-square tests showed no signficant differences between caries-present and caries-free children in both VLBW and NBW groups.

* Fluoride exposure indicates history of residing in town with fluoridated water or has used fluoride supplements.

TABLE 7. SOCIOECONOMLC STATUS (SES) AND DENTAL CARIES IN VLBW AND NBW CHILDREN

	Number of Children (%)			
	VLBW		NBW	
	Caries Present N (%)	Caries Free N (%)	Caries Present N (%)	Caries Free N (%)
SES				
I [high]	4 (16)	8 (32)	1 (4)	7 (28)
II [middle]	0 (0)	3 (12)	0 (0)	2 (8)
III [low]	1 (4)	7 (28)	3 (12)	7 (28)
Unclassified	2 (8)	0 (0)	1 (4)	4 (16)

Chi-square tests indicated that differences in the percentage of caries-present and caries-free children between the VLBW and NBW groups were nonsignificant.

for prolonged endotracheal intubation are likely etiological factors in the high prevalence of enamel hypoplasia.

The results of our longitudinal study have provided insight into the sequelae of the high prevalence of enamel hypoplasia found in VLBW children. Speculation is that these enamel defects predispose affected children to increased caries risk, but no definitive longitudinal data were previously available. An early report by Rosenzweig and Sahar[3] suggested that preterm children with enamel hypoplasia had increased dental caries. Also, Fadavi et al.[16], in a study on inner-city, black preterm children reported a higher prevalence of dental caries compared with full-term children. However, in that study, the employment of low birthweight subjects from inner city population groups with underlying high dental caries rates associated with other social factors may have introduced confounding variables not related to prematurity and low birthweight. In contrast, Curzon et al.,[28] in a cross-sectional study on a small group of preterm children, reported that they did not have a higher rate of dental caries than those born full-term.

We hypothesized that a case-controlled, longitudinal study designed to monitor the sequelae of the enamel defects would provide clearer information on the role of these defects in the development of dental caries. Despite the fact that some children did not attend all the examinations, our results showed a strong association of some enamel defects with dental caries in the latter two recall examinations in the VLBW group, but not in the NBW control children. The defects most significantly associated with caries were usually severe and involved loss of entire enamel thickness in the cuspal-occlusal regions of the first and second primary molars. These lesions became carious (soft to probing) very soon after emergence of the teeth. As the defects usually show opacity around the areas of enamel loss, they were designated as "hypoplasia with opacity". Our finding that this is the most common type of enamel defect associated with decay concurs with previous investigations of other pediatric populations.[29,30] Other developmental enamel defects such as pits, grooves and shallow areas of enamel loss

were not associated significantly with dental caries.

In this study, the overall susceptibility to dental caries of all the children was low. At the mean age of approximately 4 years, the mean number of dmft in the preterm and control groups was 0.8 ± 1.5 and 1.4 ± 3.2, respectively, compared with an overall mean of 1.7 for all 4-year-olds in the country in general.[31] Thus, the VLBW children did not appear to be more predisposed to dental decay than the control children. At the third examination, at an approximate age of 5 years, the NBW control children, in fact, had a higher mean number of decayed teeth per child than did the VLBW group. The reason for the lack of increased risk to dental caries, in spite of a high prevalence of enamel hypoplasia in the VLBW group may be that most of the enamel defects were of the types that were not likely to become carious within the period of study. The only enamel defect found to be significantly associated with dental caries was a form of severe enamel hypoplasia that was found in only 10% of the VLBW teeth.

Previous clinical studies[32–34] have generally shown that detectable salivary levels of *Steptococcus mutans* in young children are associated with dental caries, although the results are not always consistent[35], probably due to relatively low sensitivity of the detection tests available. Other risk factors associated with dental caries such as poor oral hygiene, and increased frequency of sugar exposure have also been shown to be significant risk factors in caries development.[32–34] In addition, low socioeconomic status,[36] particularly in relation to the mother's educational level,[32] and characterized by lower levels of oral hygiene and more cariogenic diet, is associated with increased caries risk. However, in our study, none of these showed significant trends in the association with dental caries. One reason for this may be the relatively small number of subjects in the study. Another possibility may be "halo" as well as "hard" effects of increased oral hygiene caused by enrollment in the study, resulting in reduction in plaque and oral bacterial counts. However, these effects would be observed to the same extent in both the VLBW and control groups.

As in other industrialized societies, differences in health practices among the different SES classes[36] would also be found in the Australian population. It was, therefore, not surprising, that in the control group, the majority of children with dental caries was found to have low SES. In contrast, the majority of children with dental caries in the VLBW group was found to have high SES, indicating that perhaps, caries risk factors other than the usual were operating in this group of children. It is likely that in the VLBW children, the presence of the severe type of enamel defects predisposes even the high SES groups to increased caries risk despite their better health practices.

Conclusions

1. VLBW preterm children showed a higher prevalence of enamel defects than NBW controls.
2. Only one type of severe enamel hypoplasia involving full thickness loss of enamel, and commonly found in newly erupted primary molars, was strongly associated with dental decay.
3. Mild enamel defects did not increase caries prevalence in the group of white, predominantly, middle- and upper-class VLBW children compared to matched, full-term controls.

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This study was funded in part by the Australian Dental Research Fund Inc.

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