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Enamel hypoplasia in the primary dentition of monozygotic and dizygotic twins compared with singleton controls

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Background. The study of enamel hypoplasia (EH) and opacity in twins provides insights into the contribution of genetic and environmental factors in the expression of enamel defects.

Aim. This study examined prevalence and site concordance of EH and opacity in the primary dentition of 2- to 4-year-old twins and singleton controls to assess the relative contribution of genetics and the environment to the aetiology of these defects.

Design. The study sample consisted of 88 twin children and 40 singletons aged 2–4 years of age. Medical histories were obtained and the children examined for enamel defects.

Results. The prevalence of EH by teeth was 21% in monozygotic twins (MZ), 22% in dizygotic twins (DZ), and 15% in singleton controls. Twins showed a higher prevalence of EH compared with singletons ($P < 0.05$). Factors contributing to increase EH in twins were neonatal complications including intubation. There were no significant differences in site concordance of EH within the MZ twin pairs compared with DZ twin pairs when only presence of EH was considered, whereas a greater concordance was noted between MZ twin pairs compared with DZ twin pairs when both presence and absence of EH were considered.

Conclusions. The results suggest that both genetic and environmental factors contribute to observed variation of EH, although it is likely that environmental factors exert a greater influence.

Introduction

Developmental abnormalities of enamel may be acquired or inherited and can be broadly categorized into enamel hypoplasia or enamel opacities[1–3]. Enamel hypoplasia is a defect associated with a reduction in the thickness of the enamel, either in a localised or more widespread area, but the enamel matrix that is present is mineralised normally[3]. These defects can present clinically as pits, grooves, or partial or total lack of surface enamel[1,2]. In the case of enamel opacities, the matrix is thought to be secreted to a normal thickness but areas within it fail to mature or mineralise properly, so there are regions where the mineral content is deficient and these present clinically as diffuse or demarcated opacities that may be yellow or brown in colour[4]. Such defects are thus considered a qualitative anomaly[4] and can range in severity from very mild to severe. In many cases, enamel hypoplasia and enamel opacities may be present on the same tooth surface.

Estimates of the prevalence of acquired enamel defects in the primary dentition vary from 2%[5] to 99%[6], depending on the population studied, the teeth examined and the criteria utilized for their assessment[7]. A possible genetic predisposition, together with environmental and systemic factors, have been suggested to be involved in the formation of enamel defects[8]. The aetiological factors associated with acquired enamel defects may act prenatally, perinatally or postnatally, and they may be systemic or localised. The systemic factors can be classified generally as birth trauma, infections, nutritional disorders, metabolic diseases, and chemicals[2,9].

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Despite a potential underlying genetic link to the aetiology of localised enamel hypoplasia, research in this field is scarce. In a study using dental casts of Australian twins, Taji *et al.*[8] examined twin concordance for hypoplastic lesions on the labial aspect of primary canines. Slightly higher concordance levels in monozygotic twin pairs, together with the presence of some mirror imaging in the size and location of lesions among twins, led the researchers to suggest cautiously an underlying genetic predisposition to the development of localised enamel hypoplasias, with the developmental environment being the main determinant of trait expression[8].

Evidence for a genetic basis to the aetiology of localised enamel hypoplasia can be obtained from historical samples as well as from animal studies. In a study of enamel defects of mediaeval and modern Danes, Jorgensen[10] found no difference between the presentations or prevalence of enamel defects over time. He suggested that this consistency was indicative of at least some genetic basis. Further to these studies, in an investigation by Elwood (1970) that explored hypoplastic enamel defects in Guinea pigs, the researchers suggested that a genetic influence could account for the observed increase in susceptibility of certain strains of the animals to hypoplastic enamel defects, as well as the presence of variation in the occurrence of defects within a species[11].

The study of twins and utilisation of the twin model allow assessment and quantification of the relative contribution of genes and the environment to phenotypic variation of particular traits[12]. Monozygotic twins, or so-called identical twins, share all their genes whereas dizygotic twins share only half of their genes on average[13]. If a particular trait is more highly correlated between monozygotic co-twins than dizygotic co-twins, and assuming that both types of twins are sampled from the same gene pool and that similar environmental factors affect them[14], it can be concluded that there is a genetic contribution to observed variation[15].

The aim of this study was to investigate enamel defects in a group of twin children and singleton controls, aged 2–4 years to clarify the relative contribution of genetic and environmental influences to the aetiology of these defects.

Materials and methods

Subjects

Ethical approval for the research project was obtained from the relevant institutions. Signed informed consent was obtained from the parents or guardians prior to the dental examination.

Twins. This investigation is part of a longitudinal study of Australian twins that has been ongoing since 2005[16–18]. All parents of twin children, aged 2–4 years and living in the state of Queensland since birth, were sent a letter of invitation to participate in the proposed study. Parents who consented to their children participating in the study were given a dental appointment. A total of 88 of 96 twin children (including two sets of triplets) responded. The consent rate for participation was thus 91%. All participants received oral hygiene instruction, a free toothbrush and toothpaste, and reimbursement of travel costs for the study.

Singleton children. A total of 40 singleton control children, aged 2–4 years and matched for socioeconomic status and age with the twins, were recruited from childcare centres. The directors of these childcare centres were approached to obtain consent for the study subjects to be derived from their facilities. A letter of purpose and invitation was forwarded to all parents/carers of children aged 2–4 years at the centres.

Socio-demography, medical and dental histories

Socio-demographic data, as well as medical and dental histories, were obtained through the use of a simple questionnaire. The questionnaire included questions about parents' occupation and level of education. A complete medical history, including gestational age, birth weight, difficulties at birth, intubation at birth and medical problems such as number of ear infections and fevers since birth, was obtained for each child.

Dental examination

The twin and singleton children were examined by the one dentist (ST) in the local government dental clinic using disposable dental mirrors. The teeth were dried with gauze and all visible surfaces of the teeth were examined. Surfaces of all teeth present were scored for enamel hypoplasia and enamel opacities, utilizing a modified Developmental Defects of Enamel (DDE) Index[4]. Enamel hypoplasia was charted as present for a tooth surface where enamel was missing, pitted or grooved. Missing enamel denotes enamel that is developmentally absent, namely, enamel that is not formed (i.e., not from enamel missing due to post-eruptive loss). Enamel opacity was charted as present when there was a distinct change in the translucency of enamel. All data were recorded on standardised forms. If a tooth showed both enamel opacity and hypoplasia, it was recorded as displaying hypoplasia to prevent double-counting of the lesions on a tooth.

Intra-examiner variability

The dentist who performed the examinations had previously undergone training in the use of the DDE Index. To determine intra-examiner consistency, duplicate examinations were carried out on five individuals 1 week apart. The kappa value for intra-examiner consistency, based on the statistical model recommended by Fleiss *et al.*[19] was found to be 0.92.

Statistical analysis

Data from the examination and questionnaire were entered into an electronic database. Statistical analysis was performed using chi-square tests with GraphPad InStat® computer software (GraphPad Software, San Diego, CA, USA). Statistical significance was set at $P < 0.05$.

Concordance testing

The degree of concordance within monozygotic twin pairs and dizygotic twin pairs was determined for the presence and absence of enamel hypoplasia on the labial surface of the mandibular primary canines as these teeth provide enamel defects that can be scored most reliably[8]. In the first part of the analysis, each pair of teeth was assigned to one of two possible groups, one group being pairs in which both members of the twin pair showed the enamel defect (concordance for presence), whereas the other group consisted of twin pairs in whom only one member had the enamel defect or both members lacked the enamel defect (considered as nonconcordant for presence in this part of the analysis). In the second part of the analysis, the absence of enamel defects in co-twins was also accepted as concordance, i.e., concordance for either presence or absence. The phi coefficient and concordance percentages were calculated. The theoretical maximum expected concordance values are 100% for MZ twin pairs and 50% for DZ twin pairs[8].

Results

Socio-demography, medical and dental histories

Table 1 shows the demography and medical histories of the subjects, comprising a total of 88 twins (49 males, 39 females) and 40 singletons (22 males, 18 females). The mean age at examination was 2.93 (±0.61) years for twins and 3.02 (±0.82) years for singleton controls, (range 2–4 years). Overall, 93% of singleton subjects and 96% of twin subjects were from Caucasian background. Although socioeconomic status did not differ among the groups, more mothers of twin children had completed tertiary levels of education compared with mothers of singletons ($P < 0.01$). Significant differences in both birth weight and gestational age were observed between twin (2.4 ± 0.5 kg, 35.3 ± 2.6 weeks) and singleton subjects (3.5 ± 0.8 kg, 38.3 ± 2.6 weeks), ($P < 0.01$), but not between MZ (2.3 ± 0.5 kg, 35 ± 2 weeks) and DZ (2.4 ± 0.5 kg, 35.3 ± 3.0 weeks) twins. The frequency of intubation at birth was also significantly higher in twin children (35%) compared with singleton controls (13%), ($P < 0.01$). Evaluation of medical problems at birth or within the first 6 months following birth, showed a significant

Table 1. Demography and medical history of twins and singleton 2- to 4-year-old subjects.

	Monozygotic	Dizygotic		Unknown zygosity	All twins	Controls	
	n (%)	*n* (%)	*P*	*n* (%)	*n* (%)	*n* (%)	*P*
Gender							
Boys	18 (58)	29 (58)	–	2 (29)	49 (56)	22 (55)	–
Girls	13 (42)	21 (42)	–	5 (71)	39 (44)	18 (45)	–
Total subjects	31 (100)	50 (100)	–	7 (100)	88 (100)	40 (100)	–
Mean age at examination (y)	2.8 ± 0.6	3.1 ± 0.6	–	2.4 ± 0.2	2.9 ± 0.6	3.1 ± 0.8	–
Mean birth weight (kg)	2.3 ± 0.5	2.4 ± 0.5	0.76	2.4 ± 0.4	2.4 ± 0.5	3.5 ± 0.8	<0.001 $t = 9.25$ d.f. = 119
Mean gestational age (weeks)	35.0 ± 2.0	35.3 ± 3.0	0.81	37.0 ± 2.0	35.3 ± 2.6	38.3 ± 2.6	<0.001 $t = 5.27$ d.f. = 118
Mother's highest level of education							
Primary	0 (0)	0 (0)	NS	0 (0)	0 (0)	1 (3)	$P < 0.01$
High	9 (29)	12 (24)		3 (43)	24 (27)	22 (56)	
Tertiary	22 (71)	38 (76)		4 (57)	64 (73)	16 (41)	
Occupation of mother							
Professional	4 (13)	4 (8)	NS	0 (0)	8 (9)	2 (3)	NS
Semi professional	26 (84)	40 (80)		7 (100)	73 (83)	67 (84)	
Unskilled	1 (3)	4 (8)		0 (0)	5 (6)	9 (11)	
Intubation at birth							
Yes	13 (42)	16 (32)	NS	2 (29)	31 (35)	5 (13)	$P < 0.01$
No	17 (55)	31 (62)		5 (71)	53 (60)	35 (87)	$\chi^2 = 7.83$ d.f. = 1
Medical problems at birth							
Yes	21 (68)	31 (62)	NS	5 (71)	57 (65)	13 (33)	$P < 0.01$
No	10 (32)	18 (36)		2 (29)	30 (34)	26 (65)	$\chi^2 = 11.29$ d.f. = 1
Ear infections since birth							
Nil	15 (48)	26 (52)	NS	4 (57)	45 (51)	17 (43)	NS
1 to 3	10 (32)	17 (34)		3 (43)	30 (34)	16 (40)	
4 to 6	1 (3)	3 (6)		0 (0)	4 (5)	5 (13)	
>6	5 (16)	2 (4)		0 (0)	7 (8)	2 (5)	
Fever since birth							
Nil	11 (35)	19 (38)	NS	2 (29)	32 (36)	13 (33)	$P < 0.01$
1 to 3	4 (13)	11 (22)		4 (57)	19 (22)	24 (60)	$\chi^2 = 15.96$
4 to 6	8 (26)	4 (8)		1 (14)	13 (15)	1 (3)	d.f. = 3
>6	6 (19)	5 (10)		0 (0)	11 (13)	2 (5)	
Antibiotic courses since birth							
Nil	2 (6)	4 (8)	NS	0 (0)	6 (7)	2 (5)	NS
1 to 3	14 (45)	17 (34)		5 (71)	36 (41)	14 (35)	
4 to 6	10 (32)	12 (24)		2 (29)	24 (27)	13 (33)	
>6	3 (10)	6 (12)		0 (0)	9 (10)	6 (15)	

difference between all twins (65%) compared with singleton controls (33%), ($P < 0.01$).

Medical histories related to prevalence of enamel hypoplasia

Associations between the prevalence of enamel hypoplasia and several variables were tested using 2 × 2 chi-square tests. These variables included birth weight, gestational age, socioeconomic status (mother's highest level of schooling and mother's occupation), intubation at birth, medical problems at birth, ear infections since birth, fever since birth and number of antibiotic courses since birth. As shown in Table 1, statistically significant differences were found between all twin groups and the singleton control group for birth weight, gestational age, intubation at birth, medical problems at birth and episodes of high fever since birth ($P < 0.01$). In contrast, there were no significant associations between the prevalence of EH and taking antibiotics or having ear infections.

Table 2. Prevalence of enamel hypoplasis in monozygotic and dizygotic twins compared with singleton controls by teeth affected.

	Monozygotic	Dizygotic		Unknown zygosity	Twins	Singletons	Total	
	n (%)	*n* (%)	*P*	*n* (%)	*n* (%)	*n* (%)	*n* (%)	*P*
Affected	120 (21)	209 (22)	0.73	20 (16)	349 (21)	114 (15)	463 (19)	*$P < 0.01$
Not Affected	452 (79)	753 (78)		106 (84)	1311 (79)	661 (85)	1972 (81)	
Total	572 (100)	962 (100)		126 (100)	1660 (100)	775 (100)	2435 (100)	

**P*-value comparing all twins and singletons.

Table 3. Total counts of opacities and enamel hypoplasia combined, by number of subjects affected.

	Monozygotic (%)	Dizygotic (%)	Unknown zygosity (%)	All twins (%)	Singletons (%)
Affected	26 (84)	44 (88)	7 (100)	77 (88)	35 (88)
Not affected	5 (16)	6 (12)	0 (0)	11 (12)	5 (12)
Total	31 (100)	50 (100)	7 (100)	88 (100)	40 (100)
P-value	–	NS	–	–	NS

Prevalence of enamel hypoplasia

Table 2 shows the prevalence of enamel defects in MZ and DZ twins compared with singleton controls by the number of teeth affected. As shown in the table, the prevalence of enamel hypoplasia was 21% for monozygotic twins, 22% for dizygotic twins and 15% for singleton controls. The observed difference in prevalence between the twins and singleton controls was highly significant ($\chi^2 = 13.680$, d.f. = 1, $P < 0.01$). In contrast, no statistically significant difference was observed between the monozygotic and dizygotic twin groups.

The percentage of individuals with at least one tooth affected with enamel hypoplasia/opacity is presented in Table 3. Both twin and singleton control groups showed similar percentages of affected children (88%). Furthermore, no statistically significant difference was observed between the twin groups.

Distribution of the enamel defects

A total of 10791 surfaces from 2435 teeth were scored for the presence of enamel hypoplasia. Fig. 1 shows the site specific distribution of enamel hypoplasia within all the subjects. For the entire primary dentition, the teeth with the largest number of sites affected were (in descending order) primary second molars (44%), primary first molars (35%), primary canines (12%), primary central incisors (6%) and primary lateral incisors (3%). No significant differences were observed between sites affected by enamel hypoplasia or enamel opacity or between the distribution of the enamel defects occurring in either the maxillary or mandibular dental arches.

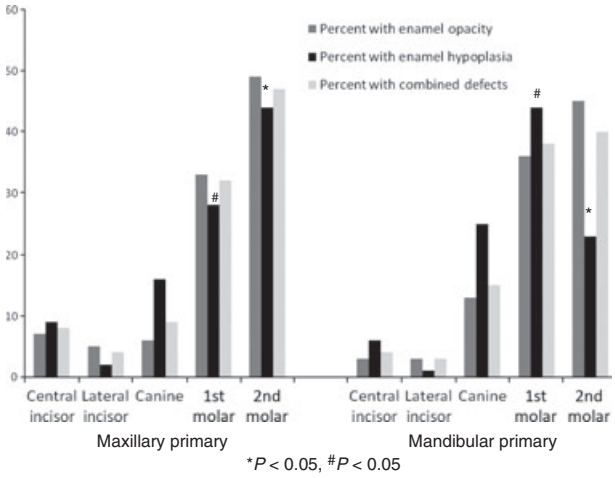

Fig. 1. Site specific distribution of surfaces affected by enamel hypoplasia and opacity in all subjects.

Concordance and discordance in expression of EH between monozygotic and dizygotic twins

Concordance for presence of enamel hypoplasia within co-twins. Twin concordances were measured for the presence of enamel hypoplasia on the labial surface of the mandibular left

Table 4. Twin concordance for frequency of enamel hypoplasia on the labial surfaces of the mandibular canines.

Enamel hypoplasia present on labial surface of primary tooth	Concordance		Discordance		Total	
	MZ	DZ	MZ	DZ	MZ	DZ
	n (%)	*n* (%)	*n* (%)	*n* (%)	*n* (%)	*n* (%)
Mandibular right primary canines	4 (27)	1 (7)	11 (73)	14 (93)	15 (100)	15 (100)
Mandibular left primary canines	1 (4)	2 (8)	24 (96)	23 (92)	25 (100)	25 (100)
Concordance (MZ vs DZ)	–	NS	–	–	–	–

MZ, monozygotic twins; DZ, in dizygotic twins.

and right canine and these are presented in Table 4. Concordance rates for affected teeth were found to be similar in both monozygotic and dizygotic groups.

Concordance for presence and absence of enamel hypoplasia within co-twins. Twin concordances were also assessed taking into consideration both concordance for presence and concordance for absence of enamel hypoplasia. This approach has been described in a previous twin study[20]. If concordance for both the presence and absence of enamel hypoplasia on the labial surface of the mandibular left canine was considered, no significant difference was observed in the concordance between MZ co-twins (86.7%) and DZ co-twins (60%). For the mandibular right primary canine, the percentage concordance between MZ co-twins was 80% and between DZ co-twins it was 44%, a statistically significant difference ($P < 0.05$). The value of the phi coefficient for expression of EH on the mandibular left canine in MZ co-twins was found to be 0.70 compared with 0.09 in DZ co-twins. A similar trend was found for the mandibular right canine, with the phi coefficient for MZ co-twins being 0.55 compared with 0.29 for DZ co-twins.

Discussion

To the best of the authors' knowledge this is the first clinical study of enamel hypoplasia in the primary dentition of twin children. Twin studies have long served as a means of evaluating the relative contribution of genetic and environmental factors to the development of specific traits[21]. The comparison of similarities between MZ and DZ twins allows estimation of the relative contribution of genes and the environment to variation observed in any given trait. This model assumes that the co-twins within each twin pair have been subjected to the same common environmental factors[14].

In this study, the prevalence of EH when assessed by teeth affected was found to differ significantly between all twins when compared with singleton controls. No discernable difference was observed, however, between the monozygotic twins and dizygotic twins. Our findings suggest that environmental factors, or possibly epigenetic factors[22,23], may exert a greater influence than genetic factors on the formation of these defects.

There were some limitations in the twin analysis that we performed, including relatively small sample sizes of participating twin pairs, some uncertainty about whether there were similar environmental factors operating between the MZ and DZ twin pairs, and unknown amounts of gene-gene and gene-environmental interactions that could have altered the phenotype and distorted the outcome of analyses[20]. When twin concordances were assessed only for the presence of enamel hypoplasia on the labial surface of the mandibular and maxillary primary canines, the differences observed between the two groups were not significant. The numbers of cases where both co-twins were affected by the lesion was very low. In order to further clarify the relative contributions of genetic and environmental influences to expression of enamel defects, concordance percentages were also calculated by including absence of the feature in both co-twins as an example of concordance. These assessments yielded higher percentage concordances in the MZ

co-twins compared with those in DZ co-twins, suggesting some genetic influence. Values of phi coefficients support this conclusion. Nevertheless, these results need to be interpreted with caution given the small sample sizes available, the relatively simple analyses performed and their underlying assumptions. Future studies utilising more sophisticated modelling approaches based on a larger sample of twins may offer more detailed insights into the influence of genetic factors on the formation of EH defects compared with environmental factors.

The findings in our study showed that low birth weight and gestational age were significantly related to the occurrence of EH which is consistent with findings in other studies[2,9,24–26] In a study by Seow *et al.*[27] a correlation between both birth weight and gestational age was observed with the prevalence of enamel defects. In their study, the very low birth weight group had a high prevalence of enamel defects (over 70%) in comparison with 50% for the low birth weight group and 20% for the normal birth weight group[27]. Prevalence rates as high as 96% have been reported in very low birth weight children[28]. The resulting defects can present as either generalised or localised depending on the associated aetiological factors. Preterm children often experience one or more systemic illnesses, which on their own can lead to enamel hypoplasia. Also, hypoxia which is often present in premature infants, has been suggested as a factor in the high prevalence of hypoplastic enamel defects observed[29]. It is also believed, however, that bone mineral loss which is exacerbated by many of the metabolic conditions experienced by preterm babies may be the leading cause of enamel hypoplasia within this group[9]. It has been suggested that as the mineral stores are depleted within a preterm infant, alteration occurs in the entry of calcium and phosphorus into the developing tooth germ, thus affecting enamel formation[9]. The results of our study showed the twin children experienced significantly more medical problems at birth compared with singleton controls which may have led to the significantly greater prevalence of EH observed in the twin group.

Twin children are more predisposed to neonatal medical conditions compared with singleton children and tend to suffer greater perinatal morbidity and mortality rates than singletons[30,31]. Metabolic diseases that have been found to contribute to enamel hypoplasia include toxaemia of pregnancy, maternal diabetes, hyperbilirubinemia, neonatal asphyxia, hypocalcemia, hypothyroidism, hypoparathyroidism, cardiac disease, gastrointestinal malabsorption, nephritic syndrome, chronic renal failure, biliary atresia, and birth prematurity[2,32–34]. Factors contributing to the disruption of ameloblasts in children with kidney and liver conditions include hypocalcaemia, decreased serum levels of 1,25-dihydroxycholecalciferol, and raised serum levels of inorganic phosphate and serum parathyroid hormone[32]. Furthermore, an elevated serum fluoride level due to the kidney's inability to remove excess fluoride may contribute to formation of fluorosis[35]. Children with gastrointestinal and malabsorption conditions are likely to have a deficiency in supply of calcium and phosphorus, leading to enamel hypoplasia[2]. It is notable that even though numerous systemic factors have been associated with enamel hypoplasia, it is difficult to isolate the relative contribution of each due to their potential synergistic effects[36].

In our study, a higher proportion of children who had been intubated at birth showed EH. This observation is consistent with other studies that have reported a prevalence of EH that is up to 4 times greater in children with a history of intubation at birth, compared with children with no history of laryngoscopy or endotracheal intubation[37,38].

Our findings showed a significantly higher proportion of twin children had experienced a high number of fevers since birth compared with the singleton children. Ameloblastic activity tends to be affected through the presence of infections and high fevers that occur during amelogenesis. This damage may be attributed to direct cellular damage by the infecting microorganism, derangement due to increase in body temperature, or due to secondary systemic insults[2]. As the twins in our study were part of a longitudinal project since birth, it is possible that the recollection of

medical history information by parents of the twin children may have been more accurate than that of the parents of singleton subjects who have not part taken in any previous part of this ongoing project. This possibility should be taken into account when considering differences in medical histories.

In addition to the above factors, recent research has suggested possible causative links between the use of amoxycillin and development of EH[39,40]. Hong *et al.*[40] have suggested the mechanism behind this is associated with amoxycillin's effect on delaying the removal of enamel matrix proteins. Our results, however, did not show any correlation between the use of amoxycillin after birth and the expression of EH.

It is interesting to note that a recent histological study[41] has suggested that the period of damage to the enamel may be relatively short, suggesting that long-duration insults such as hypocalcaemia, hypoxia and other factors may exert their influence by increasing the sensitivity of the ameloblasts rather than by damaging the cells directly. The short duration of abnormal ameloblast activity also suggests that the period of sensitivity to insults is relatively brief, and that these cells are able to recover.

The clinical presentation of enamel hypoplasia and opacities can range in severity from very mild to severe. In many clinical situations, enamel hypoplasia and enamel opacities may occur concurrently or in association with one another. Some studies[1,5,7,41–45] have differentiated between enamel hypoplasia and enamel opacity, whereas others[46,47] have made no differentiation between the two. We included data for both enamel hypoplasia and enamel opacity in our analyses. This is consistent with the fact that many defects may only be evident microscopically and thus not be represented when diagnosis is carried out at a clinical level. For example, Seow *et al.*[48] found in a study of primary dental enamel, that 52% of the teeth with no clinically detectable defects demonstrated surface enamel hypoplasia detectable at the scanning electron microscopy level.

The prevalence of enamel hypoplasia (EH) in the primary dentition has been reported to range from 48% in pre-historic populations[49], to between 2%[5] and 99%[6] in contemporary populations. In our study, 88% of all subjects were found to have at least one tooth affected by EH. Numerous possible reasons could be postulated to explain these differences in prevalence rates. Inclusion of subjects from different ethnic groups and from different age groups in previous studies probably accounts for much of the variability. In some children, the defects may become masked by erosion, attrition or caries. Other difficulties that may have affected diagnosis include use of different examination techniques and different indices, and different selection criteria for examination. Difficulties in diagnosis can also result from failure to dry adequately tooth surfaces under examination and from the presence of dental plaque masking a defect.

What this paper adds

- This paper contributes new information about the prevalence of enamel hypoplasia in the primary dentition of twins and the relative contribution of genetic and environmental factors to the expression of enamel defects.

Why this paper is important to paediatric dentistry

- Knowledge of the contribution of genetic and environmental factors to the aetiology of enamel defects will help in diagnosis and risk assessment of affected children and also in counselling families about associated complications such as dental caries.

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