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Localised enamel hypoplasia of human deciduous canines: genotype or environment?

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Abstract

A discrete area of defective enamel formation that appears on the labial surface of the crowns of deciduous canine teeth has been described in both recent and prehistoric human populations, with reported frequencies varying from 1 to 45 per cent. Suggestions about the aetiology of this localized hypoplasia range from genotypic factors to environmental conditions and systemic effects. The major aims of this study were to describe the frequency of occurrence and pattern of expression of the lesion in Australian Aboriginal and Caucasian ethnic groups, and to clarify the role of genetic factors by examining a sample of twins.

The study sample consisted of dental casts of 181 pairs of Australian Caucasian twins, 215 Aborigines and 122 Caucasian singletons, together with 253 extracted deciduous canines. Examination of dental casts and extracted teeth was undertaken under 2× magnification with emphasis being placed upon location and expression of the lesion.

The defect was observed in 49 per cent of twins and 44 per cent of Aborigines, but only 36 per cent of singletons. The percentages of affected teeth in each group were: 18 per cent in twins, 17 per cent in Aborigines and 13 per cent in Caucasians. A significant proportion of the defects occurred on the mesial aspect of the labial surface, in the middle area incisocervically, with the majority in the lower jaw. Anumber of significant differences in frequency were observed between groups, sexes, arches and sides.

The results confirm some of the findings of previous studies, but also suggest that none of environmental, genetic or systemic factors can be ruled out as being involved in aetiology of the defect. The higher incidence of the lesion occurring on the mesial aspect of the labial surface is suggestive of physical trauma. Also, the vulnerability of the prominent developing mandibular canine, with its thin or missing labial covering of bone, would be expected to lead to higher prevalence of the lesion in the lower jaw. Although not definitive, the results of concordance analyses in twins were suggestive of a possible genetic predisposition in the formation of the lesion. Further research with a greater clinical orientation and emphasis on determining specific aetiological factors within any given environment in different ethnic groups may provide better insight into the ambiguous aetiology of the hypoplastic enamel defect.

Key words: Enamel defects, trauma, genetics, twins.

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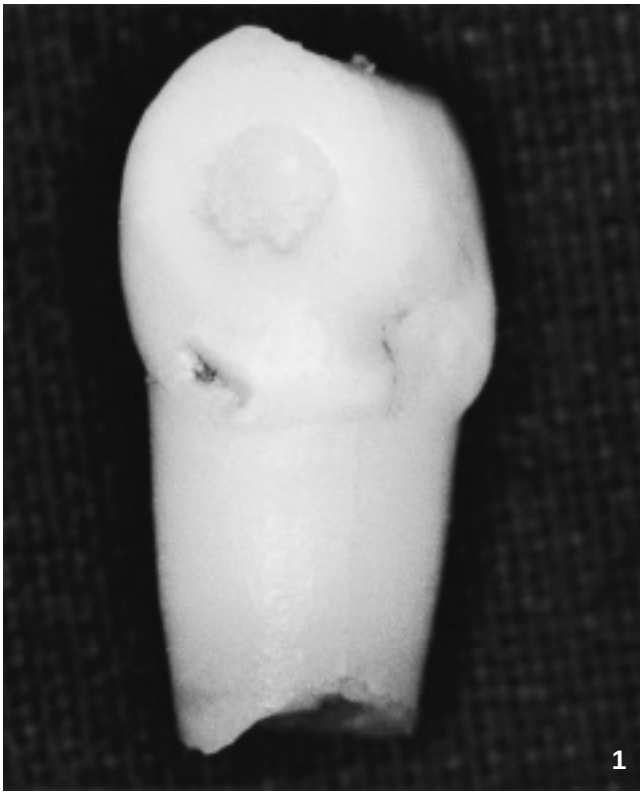
Introduction

A discrete area of defective enamel formation on the labial surface of the crowns of deciduous canine teeth has been described in both recent and prehistoric human populations.[1] The lesion usually manifests as a flat-bottomed crater covering an area of about 1-2 mm, extending either partially or completely through the enamel to the dentine (Fig. 1). A solitary lesion is most frequent, although multiple defects have been observed also on individual teeth.[2]

Many suggestions have been proposed to explain the aetiology of the lesion. Jorgensen,[2] the first to report the defect, found no difference between medieval and modern Danes with respect to the frequency of the defect. He believed that this consistency over time indicated at least a partial genetic basis. However, no other studies appear to have been carried out to verify whether genetic factors play a role.

Skinner and Hung[3] have used the location of the lesion, most commonly at mid-crown level, to support their view that the defect is initiated shortly after birth. Skinner[4] further suggested that minor trauma to the developing canine crown of neonates (for example, sharp objects placed in the mouth) is an important environmental factor. At birth the cortical bone forming the labial bulge of the crypt of the primary canine is either thin or absent, and thus the crown may not receive adequate protection from early physical trauma (Fig. 2). Skinner[4] has argued that this differentiates the canine with respect to other adjacent primary teeth, which are either less labially positioned, or have largely completed crown



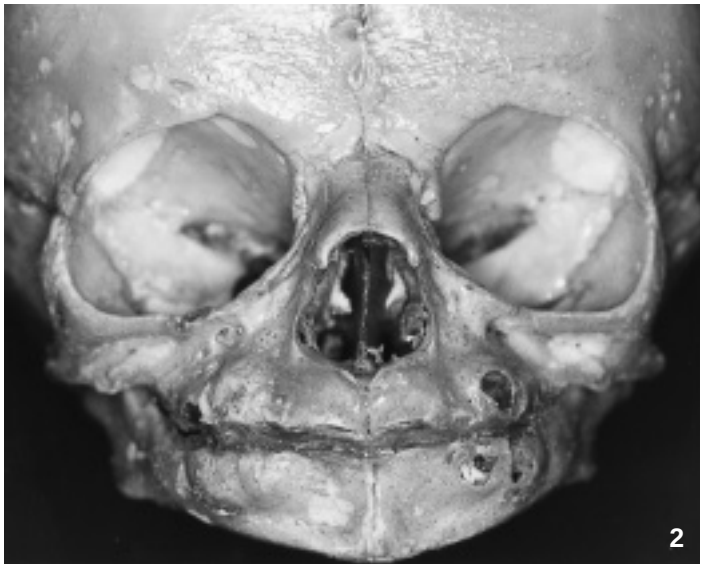


Fig. 1. – Extracted deciduous mandibular canine showing a distinct hypoplastic defect on the labial surface of the crown.

Fig. 2. – Neonate skull showing distinct bulges in the regions of developing deciduous canines, with deficiency of labial bone on the left side.

formation at birth. Nation *et al*.[5] have hypothesized that the high prevalence of the defect in mandibular canines at a level corresponding to the perinatal period of tooth formation may be due to the protruded position of these teeth during formation. They have suggested also that, during the act of birth, trauma of the canines is induced by compression of the lower labial alveolar wall due to pressure from the maxilla.[5]

Nutritional status of the expectant mother has also been implicated as a possible contributing factor to the defect. Skinner and Hung[6] have attributed reduced cortical bone in the jaws of the infant to nutritional factors, involving calcium deficiency of the mother and/or developing infant. Calcium deficiency may either be due to unavailability of calcium-rich food or lactose intolerance. It has been proposed that diets composed of high fibre and phytate contribute directly to low calcium levels by interfering with absorption of vitamin D required for phosphorous and calcium deposition.[1] Deficiency of such nutritional elements can affect epithelial cell function and the mineralization process.[7] Such a situation may produce a developmental setting conducive to formation of the hypoplastic defect.[1] Other studies have reported a strong association between socio-economic status and prevalence of the defect together with differences in frequencies between developing and developed countries. This suggests that differences in nutritional status, or obstetric and paediatric care, may be important aetiological factors.[1,6,8]

Skinner[4] has looked at the onset and duration of the lesion in detail and notes that the time interval during which a specific defect formed can be determined by relating the height of the defect to rates of deciduous canine formation. An intermediate formation rate for the deciduous canine is 0.33 mm per month, corresponding to an average time of 5.2 months for the formation of the defect.[4] To determine the time of onset and cessation of the lesion, its vertical extent is drawn on an outline of a representative deciduous canine, adjusting the data to allow for differences in observed tooth heights. Both Jorgensen[2] and Nation *et al.*[5] commented on the presence of apparent prenatal defects in Caucasian subjects. However, Skinner[4] suggested that defects assumed to occur prenatally may result from an individual being born at an early stage of canine formation due to retarded crown formation and/or premature birth. A link between size of the hypoplastic defect and apparent prematurity has also been demonstrated.[4] Lesions commencing close to the crown tip appear significantly larger than those initiated near the cervical margin.

The prevalence of labially located enamel hypoplasia of the primary canine was first reported by Jorgensen,[2] with lesions observed in 21 per cent of modern Danish and 28 per cent of medieval Danish primary canine teeth. Other studies in modern populations have found a varied prevalence ranging from 45 per cent or more[1,4] to less than 1 per cent.[3] Such variation depends on the population studied, the sample size of teeth examined and the

criteria used for diagnosis.[7] The lesion occurs twice as commonly in the lower jaw.[4] Jorgensen[2] noted that the hypoplastic areas appeared to occur within or on the boundary of the mesiogingival quadrant of the labial surface. Variation in the defect has been observed between ethnic groups. Nation *et al*.[5] have suggested that variation in occurrence of the defect between different ethnic groups may be due to differences in susceptibility to trauma of the cells taking part in amelogenesis or to a difference in the tendency to develop general disorders or diseases affecting indirectly amelogenesis. Population differences in the prevalence of the lesion may reflect innate and acquired variation in thickness of the alveolar bone and soft tissues overlying the bulging crypt of the deciduous canine.[4]

In studies conducted previously, the lesion has most commonly presented as a solitary defect in the cervical half of the deciduous canine crown. However, little is known about the shape of the defect or the relevance of its location to its aetiology. Previous studies also lack any examination of the lesions over time (longitudinal assessment).

The current study aimed to address many of these issues. The main aims of the study were:

1. To determine the range of expression of the lesion by examination of a sample of extracted teeth.

2. To compare the frequency of occurrence and range of expression between large samples of Australian Caucasians and Aborigines.

3. To examine a sample of twins in order to determine the relative contributions of genetic and environmental influences to variation in expression of the lesion.

4. To examine serial dental casts of selected Aboriginal subjects to determine whether any trends in expression of the lesion were evident with time.

Materials and methods

The sample consisted of dental casts of 181 pairs of Australian Caucasian twins, 122 Caucasian singletons and 215 Australian Aborigines. The twins were part of an ongoing study of dentofacial variability and ranged in age from around 4 to 9 years. The dental casts of Caucasian singletons, aged from 3 to 6 years, were collected during an earlier study of deciduous tooth size variability.[9] The dental casts of Aborigines belonging to the Wailbri tribe were obtained during a longitudinal growth study that was undertaken in the 1960s and 70s.[10] Their ages ranged from approximately 6 to 10 years. A total of 253 extracted deciduous canines collected in South Australia during the early 1900s were also examined.

The extracted deciduous canines were examined initially to assess the extent of variation in expression of the lesion and to confirm that the dental casts provided a valid representation of the various forms, ranging from pinpoint to larger defects. Dental casts were examined for evidence of the defect on the labial surface of deciduous canines from the four dental quadrants. Presence or absence of the defect and its position on the tooth crown were recorded (Fig. 3). Crowns were subdivided into thirds in two planes. These were denoted incisal, middle and cervical in the incisocervical plane, and mesial, middle and distal in the mesiodistal plane, following Wheeler.[11]

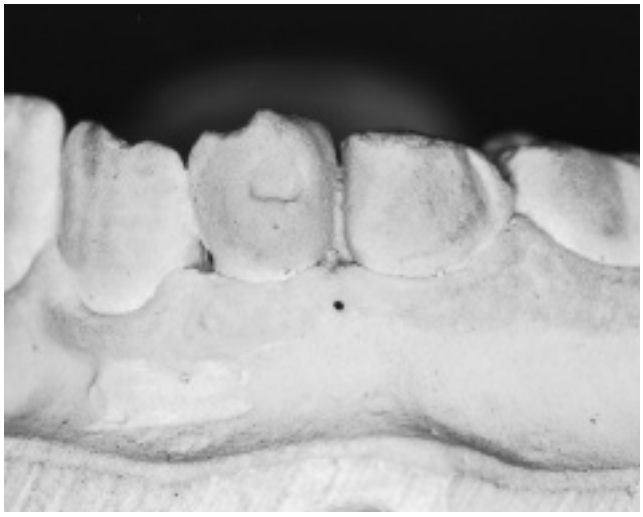


Fig. 3. – Dental cast showing a distinct hypoplastic defect on the labial surface of a mandibular deciduous canine.

Detection was based on the presence of a depressed area on the labial surface of the deciduous canines presenting as pinpoint, oval, circular or triangular in shape. Observation was carried out initially under 2× magnification adjusting the light angulation for clear demarcation of the border of the lesion. Further observations were conducted using a stereo-microscope to aid detection of pinpoint defects. Apart from their characteristic position and shape, localized hypoplastic defects in the extracted teeth were differentiated from possible carious lesions by confirming with a dental explorer that the enamel or dentine at the base of the lesion was hard. Given that it was impossible to use tactile methods of discrimination on the dental models, hypoplastic lesions were distinguished solely on the basis of their appearance and location.

Frequency of occurrence was calculated on an individual-by-individual basis and also on a tooth-by-tooth basis. Due to the availability of serial dental casts for the Aborigines, it was possible to determine whether lesions were always present from the youngest age or whether they became apparent at later ages.

Genetic analysis was conducted on the twin samples by comparing percentage concordances in

monozygotic (MZ) twins with dizygotic (DZ) twins. Under the assumption that MZ twins share the same genes, the theoretical maximum expected concordance is 100 per cent, whereas the DZ twins share only half of their genes, leading to maximum values of 50 per cent.

The ethical guidelines issued by the National Health and Medical Research Council (NHMRC) of Australia were followed in the study and informed consent was obtained from human subjects.

Chi-square tests were used to examine associations between presence or absence of the defect and several variables, including ethnicity, sex, twin zygosity, age, dental arch, side and location of the lesion.

Double determination tests were carried out on 10 per cent of the sample to assess the reliability of the scoring method. Concordances between repeated measures were high (80 per cent) for all measured traits.

Results

Extracted teeth

Nineteen per cent of the extracted teeth demonstrated some form of hypoplastic lesion. There was a significant difference ($\chi 2=10.96$, $p<0.001$) in frequencies between arches. Only 11 per cent of maxillary canines were affected, compared with 28 per cent of the mandibular canines.

Incisocervically, 10 per cent of lesions were in the incisal third, 29 per cent were in the middle third and 61 per cent were in the cervical third. Mesiodistally, 65 per cent were in the mesial third, 2 per cent were in the middle third and 33 per cent were in the distal third. No significant arch or side differences were observed in lesion distribution. Fifty-eight per cent of lesions were round, 35 per cent were ovoid and 6 per cent were triangular or irregular in shape.

Dental casts

Forty-seven per cent of all individuals were affected by at least one detectable hypoplastic lesion. Of these, 64 per cent had only a single affected tooth, 26 per cent had two, 8 per cent had three and only 2 per cent had lesions on all four canines. Overall, 17 per cent of all canines were affected.

There was a highly significant difference ($\chi 2=82.35$, $p<0.001$) between maxillary and mandibular frequencies (11 per cent and 24 per cent respectively); however, there was no difference between left and right sides.

Overall, lesions occurred predominantly (59 per cent) in the middle region of the incisocervical plane, followed by the cervical (32 per cent) and the incisal (9 per cent) regions. Mesiodistally, lesions occurred predominantly (58 per cent) in the mesial region, followed by the middle (34 per cent) and distal (8 per cent) regions. Fifty-six per cent of all lesions were round, 37 per cent were oval, 4 per cent were triangular and 3 per cent were irregularly shaped.

Table 1. Frequencies of occurrence of hypoplastic lesions on human deciduous canines within three distinct populations

	Twins n %	Aborigines n %	Singletons n%
Individuals with no lesions	184 (50.8)	121 (56.3)	78 (63.9)
Individuals with one tooth affected	115 (31.8)	62 (28.8)	30 (24.6)
Individuals with two teeth affected	43 (11.9)	23 (10.7)	12 (9.8)
Individuals with three teeth affected	16 (4.4)	6 (2.8)	2 (1.6)
Individuals with four teeth affected	4 (1.1)	3 (1.4)	0 (0.0)
Individuals with at least one lesion	178 (49.2)	94 (43.7)	44 (36.1)

Frequency of occurrence (at least one lesion) differed significantly between the three groups ($\chi 2 = 6.89, p<0.05$).

Group differences

Table 1 presents frequencies of occurrence of the lesion for the three groups. There was a significant difference in frequencies between the groups ($\chi 2=6.89$, $p<0.05$); Aborigines and twins showed similar frequencies of individuals with at least one lesion (44 per cent and 49 per cent respectively) but singletons displayed lower frequencies (36 per cent).

On a tooth-by-tooth basis, there was also a significant difference between groups ($\chi 2=8.81$, $p<0.05$). Twins had the highest frequency at 18 per cent, closely followed by Aborigines (17 per cent), with the Caucasian singletons having the lowest frequency at 13 per cent.

Table 2 presents frequencies of occurrence of the lesion for the three groups by arch and side.All three groups exhibited a frequency in the mandible greater than that in the maxilla. Furthermore, there were group differences in frequency, averaged for right

Table 2. Frequencies of occurrence of hypoplastic lesions by arch and by side

	Twins n %	Aborigines n %	Singletons n %
Maxilla			
Right	48 (13.3)	8 (3.9)	12 (9.8)
Left	46 (12.8)	25 (12.2)	6 (4.9)
Mandible			
Right	99 (27.4)	54 (29.7)	21 (17.2)
Left	72 (19.9)	48 (26.1)	22 (18.0)

Frequency in mandible significantly greater than maxilla for all three groups: twins ($\chi 2=26.65$, $p<0.001$), Aborigines ($\chi 2=52.86$, $p<0.001$),singletons ($\chi 2= 11.71$, $p<0.001$).

Table 3. Frequency distribution of localized enamel hypoplasia incisocervically

	Twins			Aborigines			Singletons		
	Inc %	Mid %	Cer %	Inc %	Mid %	Cer %	Inc %	Mid %	Cer %
Maxilla									
Right	20.8	47.9	31.3	0.0	37.8	62.5	0.0	66.7	33.3
Left	15.0	70.0	30.0	8.0	48.0	44.0	16.7	16.7	66.6
Mandible									
Right	10.2	66.3	23.5	7.4	61.1	31.5	9.5	61.9	28.6
Left	5.6	70.8	23.6	6.2	41.7	52.1	0.0	72.7	27.3

Inc=incisal third;Mid=middle third; Cer=cervical third.
No significant difference in distribution of lesions between the three groups.

and left sides, within both the maxilla (8 per cent, 7 per cent and 13 per cent for Aborigines, singletons and twins respectively; $\chi 2=10.05$, $p<0.01$) and the mandible (28 per cent, 18 per cent and 24 per cent; $\chi 2=8.52$, $p<0.05$). However, the relative differences between arches were similar between groups (14 per cent higher in the mandible).

Tables 3 and 4 present frequencies of occurrence of the lesion with regard to vertical and horizontal position respectively. In all three groups the lesions occurred predominantly in the middle region incisocervically, followed by the cervical and incisal regions. With respect to mesiodistal location, again a similar pattern of expression was noted in all three groups, with mesially positioned lesions tending to be most common, particularly in the mandible, followed by those located in the middle, then distal thirds of the crown.

There were no significant differences between sexes in the frequency of occurrence of the lesion, either on an individual-by-individual basis or on a tooth-by-tooth basis.

Twin concordances

Twin concordances are presented in Table 5. Dizygous twin pairs had a higher concordance for presence of at least one lesion than their monozygous counterparts, whereas concordances for number of teeth affected were similar in both groups.

Concordances for vertical and horizontal location were determined by accumulating the pair-wise comparisons of each of the four canine types (upper right, upper left, lower right and lower left). This was after removal of any canine pairs that did not demonstrate concordance for lesion presence. Subsequent sample sizes were relatively low (33 for dizygous twins, 19 for monozygous twins) and the results should be considered accordingly. Monozygous twins had slightly higher concordance for incisocervical location but were similar to their dizygous counterparts for mesiodistal location.

Discussion

Most of this study's findings with regard to location, occurrence and the influence of gender on the hypoplastic defect were similar to those observed by other investigators. However, some new insights into the defect's aetiology were gained.

Observation of 253 extracted Caucasian deciduous canines indicated that around 19 per cent of the teeth showed the defect. Results from examination of dental casts were similar overall (17 per cent) and for all groups studied (18, 17 and 13 per cent for twins, Aborigines and singletons respectively), supporting the use of casts as an appropriate medium for detecting the defect. It should be acknowledged, however, that there are limitations in using dental casts for studies of hypoplastic lesions, as mild forms of hypoplasia including opacities cannot be distinguished and differential diagnosis of hypoplastic lesions from carious lesions is generally not possible.

The results derived from dental casts were comparable to those found by Lukacs[1] but were lower than other studies such as Jorgensen's[2] Danish data (21 per cent and 28 per cent for modern and medieval Danes respectively), and Skinner's[4] Calcutta data (29 per cent).

Table 4. Frequency distribution of localized enamel hypoplasia mesiodistally

	Twins			Aborigines			Singletons		
	Mes %	Mid %	Dis %	Mes %	Mid %	Dis %	Mes %	Mid %	Dis %
Maxilla									
Right	39.6	47.9	12.5	62.3	37.5	0.0	25.0	66.7	8.3
Left	26.1	50.0	23.9	44.0	52.0	4.0	66.7	33.3	0.0
Mandible									
Right	57.1	33.6	9.2	75.9	18.5	5.5	61.9	33.3	4.8
Left	70.8	27.8	1.4	81.3	14.6	4.2	68.2	31.8	0.0

Mes=mesial third; Mid=middle third;Dis=distal third.
No significant difference in distribution of lesions between the three groups.

Table 5. Twin concordances for frequency, position and size of hypoplastic lesions

Trait	Concordance %	
	Monozygous	Dizygous
Presence of at least one lesion	55	73
Number of teeth affected	43	54
Incisocervical position	42	30
Mesiodistal position	53	48

Frequency

In the current study, the defect has been shown to occur more frequently in the lower jaw with no detectable difference in prevalence between sexes. This has also been reported in previous studies.[1,3,4] Skinner[4] suggested that this may be a reflection of the presence of a thicker layer of soft tissue, especially fat cheeks, overlying and protecting the upper canines from trauma, or alternatively, it may result from a reduction or total absence of protective alveolar bone in the lower jaw. An increased frequency in the mandible may also reflect the marked protrusion of the lower canines during early growth and development.This is illustrated in Fig.2. Such exposure may make mandibular canines more susceptible to physical trauma. This is an example where environmental influences may be interacting with an underlying genetic predisposition to produce a phenotypic effect.

Localized trauma as an aetiological factor in formation of the hypoplastic defect has been supported by the majority of previous studies.[1,3-5,7] However, none of these studies have employed the three very distinct groups of subjects who were included in this study.

Ethnic differences in frequency may reflect either genetic or environmental differences, or more likely a combination of the two. It is difficult to quantify the relative contributions of these two components based only upon interpopulation comparisons, however some inferences may be drawn about the impact of various socio-economic and cultural differences.

If the aetiology were dictated by environmental factors alone, in particular physical trauma, one may have expected a greater frequency of affected teeth within the Aboriginal population, who were exposed to harsher environmental conditions and a greater likelihood of developmental trauma. More importantly, one would have expected a greater frequency of individuals with multiple teeth affected, but this was not the case. The results in this study suggest that the large differences between the two groups in lifestyle, diet and socio-economic status are having only a small impact on the frequency of occurrence of the lesion.

It has been suggested that an acquired hypoplastic defect may be a result of a systemic effect on the ameloblasts.[4] In the case of a systemic insult to the ameloblasts, a bilateral expression of the defect and its presence in both jaws is expected. However, as ameloblasts do not become secretory simultaneously, presence of a lesion in less than all four quadrants need not rule out systemic influence.[4] Of the subjects studied, only 17 per cent of the twins, 15 per cent of the Aborigines and 11 per cent of the singletons showed two or more affected teeth (Table 1). Furthermore, absence of the defect on other tooth types whose crown formation overlaps that of the canines, and the observation of a greater frequency of occurrence of the defect in the lower jaw, lessen the possibility of a systemic cause.

The susceptibility of deciduous canines to display the defect may indeed have some genetic basis. In the current study involving the twin data set, seven pairs of MZ twins and six pairs of DZ twins showed mirror-imaging for size and location of lesions. This indicates subjectively that there may be an underlying genetic predisposition to lesion development. However, the concordance value for presence of at least one lesion in MZ twin pairs was only 55 per cent, indicating substantial environmental variation. This is further evidence that lesion development reflects a complex interplay between genetic and environmental factors. Further analysis using more advanced genetic modelling is warranted to quantify the relative contributions of genotype and environment.

The highest percentage of subjects with at least one lesion was noted in twins. Twinning has long been associated with both prenatal and perinatal mortality and morbidity, with perinatal mortality estimated to be 11 times that of normal singleton births.[12] Twins have a much higher incidence of premature birth, and gestation is on average three weeks shorter than singletons. Parity is also important.The second-born twin is at greater risk of oxygen deficiency and birth trauma due to abnormal uterine positioning.[13] A higher frequency of low birth weight resulting from nutritional deficiency has also been observed.[14] Perinatal and prenatal growth retardation and prematurity observed in twins may be factors associated with the aetiology of the hypoplastic lesion. Seow[15] has commented on the high prevalence of enamel hypoplasia (both localized and generalized) in low birth weight and premature children with a range of 43-96 per cent. Such dental defects have been variously associated with low bone mineral stores, traumatic laryngoscopy and prolonged endotracheal intubation.[15] With respect to the greater risk of nutritional and oxygen deficiency in twins, the chance of an abrupt cessation of enamel formation through sudden ameloblast death, and

thus development of the hypoplastic lesion, may be much greater.

Location

The deciduous canine crown forms from the cuspal tip to the cervix at linear rates.[4] It is known that ameloblasts do not become secretory at the same time, cells in the presumptive cuspal regions being more differentiated than those placed more laterally and cervically. Correspondingly, the cuspal region contains the advanced organic front in which mineralization will subsequently follow. The pattern of mineral deposition and growth appears to follow the incremental lines of Retzius, laid down by the matrix-secreting ameloblasts, thus transversing the cuspal zones in an arc-like fashion; ending bisymmetrically at the cervical regions of the dento-enamel junction with a decreasing gradient of mineralization.[16] Kraus and Jordan[17] observed that both maxillary and mandibular deciduous canines have one-third of their enamel formed at birth with crown formation being completed by nine months after birth.The vertical position of the lesions in our study was mostly in the middle third of the tooth incisocervically, followed by the cervical third and then the incisal third, suggesting a perinatal commencement of the defect, with some defects commencing up to a few months after birth.The few examples of the lesion being positioned in the incisal third may in fact reflect a premature birth and/or a retarded crown formation.

Of note was evidence of two or more pinpoint lesions presenting in close proximity on some teeth. This was observed in approximately 5 per cent of the dental casts that displayed lesions and 23 per cent of affected extracted primary canine teeth. This suggests that the crater-like appearance of at least some lesions may result from contiguous pinpoint lesions uniting through enamel wear or breakdown.

The high prevalence of the lesion on the mesial aspect of the labial surface may be indicative of the action of physical trauma in the aetiology of the defect.This is demonstrated in part by the data from the different ethnic groups studied. The Aboriginal tribe from whom dental casts were obtained lived in harsher environmental conditions compared with the twins or Caucasians who participated in the study. Such conditions may have had an impact in terms of early postnatal access to damaging agents in a non-urban environment. Finding a higher incidence of the mesially located lesions in the Aboriginal data than in twins or singletons supports this hypothesis.

Expression

While the aetiological agent of the hypoplastic defect remains enigmatic, evidence from a variety of observations made throughout the course of the study provides some further insights. Where serial dental models were available for Aborigines, changes in lesion appearance were noted over time. In five cases, defects were only evident on casts representing older age, suggesting the possibility of a subclinical lesion early in development that deteriorates with age due to factors within the local environment of the tooth. Two or more pinpoint lesions in close proximity were observed in 23 per cent of affected extracted canines. It is possible that these may unite in some teeth, through wear or enamel breakdown, leading to the formation of typical crater-like defects.

Conclusion

The results of this study have shown a specific trend in the location of the hypoplastic defect. A significant proportion of the lesions occur on the mesial aspect of the labial surface of the crown, and are centralized vertically. There is a higher frequency of occurrence in the mandibular canines relative to the maxillary canines, but there is no effect of gender. Overall, there is some suggestion of an underlying genetic predisposition to lesion formation but the developmental environment seems to be the main determinant of trait expression. A more comprehensive analysis involving genetic modelling of twin data is warranted to elucidate the relative contributions of genotype and environment. Further research with a clinical orientation may also provide insight into the ambiguous aetiology of the hypoplastic enamel defect.

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