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Linear enamel hypoplasia and historical change in a central Australian community

J Littleton,* GC Townsend†

Abstract

Background: The current study extends the use of linear enamel hypoplasia (LEH) to examine the historical changes in living conditions encountered by Aboriginal people at Yuendumu who were born between 1890 and 1960. LEH provides health information beyond written records and gives insight into the relationship between individual health and living conditions during initial and ongoing contact with Europeans.

Materials and Methods: The dental casts of 446 people, collected as part of the University of Adelaide longitudinal study of growth and development, were recorded for the presence of hypoplastic defects. Defects were recorded according to the Development Defects of the Enamel (DDE) standards and assigned to developmental units based on their crown position.

Results: The frequency of LEH on the permanent dentitions increased five-fold from the 1890-1929 birth cohort to the 1955-1960 cohort. LEH also affected earlier developing enamel units. Deciduous defects did not show a strong temporal trend but overall prevalence was comparable to other disadvantaged groups.

Conclusion: The changes in permanent LEH frequency and age distribution correspond to altered living conditions with the worst hypoplasia recorded after settlement of Aboriginal people at Yuendumu. Prior to that period LEH was comparable to pre-contact Australian populations indicating that resettlement had a dramatic impact on childhood morbidity.

Key words: Developmental defects of enamel, Australian Aboriginal health, Yuendumu.

Abbreviations and acronyms: DDE = Developmental defects of enamel; LEH = linear enamel hypoplasia; UM2A = second molar.

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INTRODUCTION

Australian Aboriginal health has been profoundly affected by European settlement. Increasing rates of infectious diseases, changed nutritional patterns, and violence were all immediate consequences of colonization.[1] Land dispossession, change of life styles, and poverty have played further roles resulting in the current situation where Aboriginal life expectancy is 20 years less than the Australian average. Unsurprisingly recent studies of oral health[2] reflect the pattern seen in health more generally: higher rates and severity of dental disease.

Linear enamel hypoplasia (LEH) is routinely recorded in oral health surveys[3] and in anthropological studies of prehistoric and modern populations.[4] In both instances LEH is used as a basis from which to make more general inferences about health. A recent survey of work on LEH revealed it has been linked to 72 specific causes as well as to socio-economic circumstances and even to specific events such as the Chinese famine.[5] No single precipitating cause has been identified and, in general, LEH of the permanent dentition has been accepted as a marker of non-specific physiological disruption during the secretory phase of amelogenesis.[6] In the current study, we examined a long sequence of hypoplasic defects, as recorded on historic dental casts from Yuendumu, Central Australia, in relation to known historical change among a specific population.

We tested the hypothesis that changes in Aboriginal living conditions over the period from 1890 to 1970 had a direct reflection in the prevalence and age distribution of LEH thus providing a correct and timely record of childhood development. Furthermore, the study provides a baseline of hypoplasia frequency for comparison with modern populations.

MATERIALS AND METHODS

This study was only possible because of the unique series of dental casts of Aboriginal people resident at Yuendumu, Central Australia, from 1950-1970. Yuendumu settlement, located approximately 290km north-west of Alice Springs in the Northern Territory,

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Table 1. Permanent dentitions from the Yuendumu casts with more than 75 per cent of developmental units observable, divided by cohort and sex

Cohort	Female	Male	Total
1890-1929	8	4	12
1930-1939	23	25	48
1940-1944	36	39	75
1945-1949	26	29	55
1950-1954	46	50	96
1955-1960+	28	40	68
Total	167	187	354

was established by the Australian Government in 1946. The casts are of 446 Aboriginal people who were born between 1890 and 1960. They were collected as part of a longitudinal study of growth and development by Dr Murray Barrett and Professor Tasman Brown, Adelaide Dental School.[7] The age and sex distribution of the sample used in this analysis is shown in Table 1.

In 1961 the scope of the study was extended to include a wider range of observations and, over the next decade, teams from the University of Adelaide Dental School travelled to Yuendumu at least once a year. The overall aim of the longitudinal study was to carry out correlated studies of morphological features, functional relationships, and patterns of growth and development. A series of reports and articles emerged both at the time and since on aspects of growth and development, inheritance of dental characteristics, and oral health.[7] Apart from their value in studies of the development of occlusion, the dental casts also preserve a history of changing childhood developmental circumstances among a group of people in whom the oldest grew up in near traditional circumstances, the youngest of whom were born at a Government settlement in the 1950s and very early 1960s.

The recording of LEH was based on the developmental defects of enamel (DDE) index (Table 2).[8] Hypoplasia is defined as a quantitative deficit of the tooth's enamel, identified as a groove, single pit, vertical or diffuse pitting, or missing enamel. Lack of enamel was confirmed by running a fingernail down the cast surface to confirm the existence of a deficit of enamel and could be readily distinguished from caries or traumatic loss of structure by the intact external surface and lack of exposure of dentine. Opacities or hypocalcifications were not visible on the casts although in a small number of circumstances there has been post-eruptive enamel loss along the line of a hypocalcification. This was distinguished from hypoplasia by the ragged edge of the groove. Vertical pitting and diffuse pitting across the tooth's surface, possibly related to fluorosis or localized hypoplastic amelogenesis imperfecta,[8-9] were found in two individuals occurring in a non-chronological pattern. These non-chronological defects were not included in the analysis.

Table 2. Classification of enamel defects recorded in this study, based on the DDE index[8]

Types of defects (hypoplasia only)	Number and demarcation
Pits	Single
Grooves: horizontal	Multiple
Grooves: vertical	Diffuse: fine white lines
Missing enamel	Diffuse: patchy

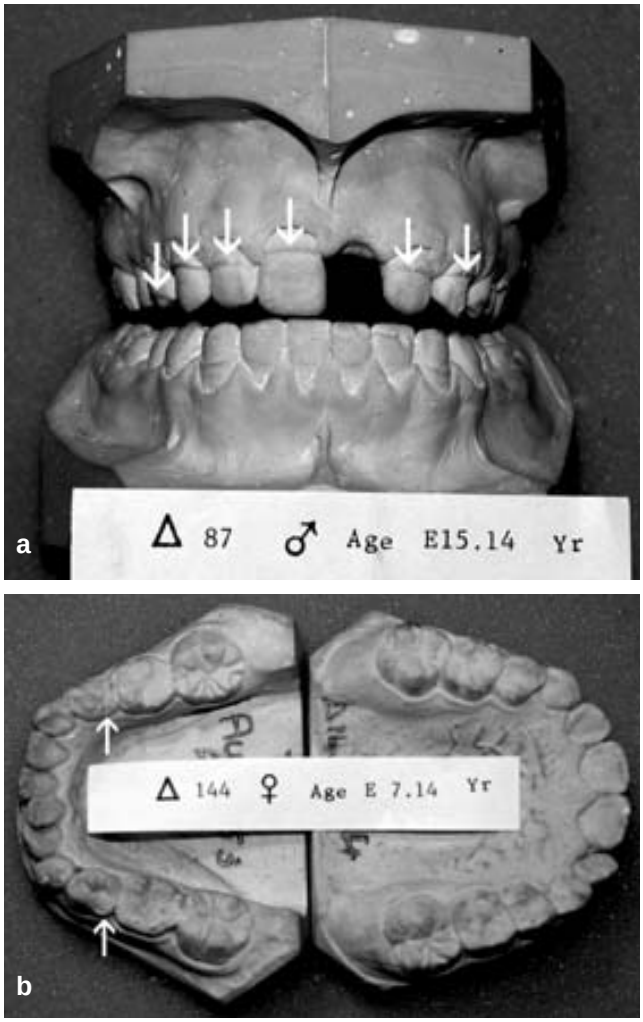

Fig 1. Dental casts from Yuendumu showing: a) marked hypoplasic groove on all anterior teeth; b) symmetrical pitting on deciduous mandibular molars from Yuendumu.

To avoid including defects caused by trauma or other non-systemic causes, a hypoplasic defect was only recorded if it occurred on both antimeres and if it occurred in a tooth other than the pair in a similar chronological position, i.e., on a tooth segment known to be developing at the same time (Fig 1).

All permanent teeth were observed. Defect location on the crown was recorded and matches made between defects based on a chronological scheme adapted from Hillson[6] in order to accommodate the accelerated development of the later-developing teeth in Aboriginal children (Fig 2).[10] Matching defects to a developmental unit (comprising segments of enamel developing over the same time period) allowed the age distribution of defect occurrence to be compared over cohorts. The seven developmental units were labelled from A to G and associated with approximate chronological age ranges (allowing for 6-8 months of hidden enamel on the crown surfaces).[11]

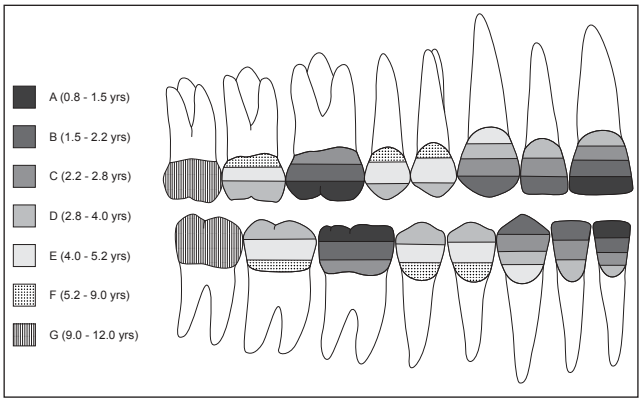

Fig 2. Diagram showing the enamel defect matches used in this analysis and the approximate age associated with each unit.

Among primary teeth, defects were recorded as grooves or pits (no other defects were observed) and a note was made of whether the defect was symmetrical or not (Fig 1b).

Only one observer (JHL) recorded LEH. A repeat observation of 750 enamel segments was undertaken three years after the initial recording in order to assess interobserver error. The rate of concordance was 0.80 which includes errors of assignment to neighbouring crown units (0.05). All differences were randomly distributed and paired *t* tests indicate no significant differences between the two sets of results.

However, there are two potential sources of error: the quality of the dental stone material and degree of tooth wear arising from attrition, abrasion and erosion.

Three types of dental stone, varying in degree of coarseness, were used between 1950 and 1970. Occasional casts were too coarse to be used for recording and were not included in the analysis. Among the remainder there were no statistically significant differences between cast type and hypoplasia frequency after controlling for cohort effect.

The other potential bias was tooth wear. If a segment of tooth enamel was no longer visible due to wear it was recorded as missing. However, it is possible that wear could remove LEH defects from the lingual or labial surfaces. A two-way ANOVA comparison of mean number of defects among those with more than 75 per cent of developmental units was undertaken using wear on the maxillary second molar (UM2A) and cohort as factors. The final model indicated no significant contribution of UM2A to the mean number of defects (F=0.860, p=0.525, d.f.=6).

RESULTS

The distribution and type of defects

As observed elsewhere[12] the frequency of LEH varies by tooth type. Canines and central incisors were the most frequently hypoplastic teeth with particularly high numbers of LEH visible on the mid-third of the crown. Defects did vary by type in relation to position of the crown so that while grooves predominated on the mid-crown, pits were the most common defect observed on the occlusal surface (Fig 3).

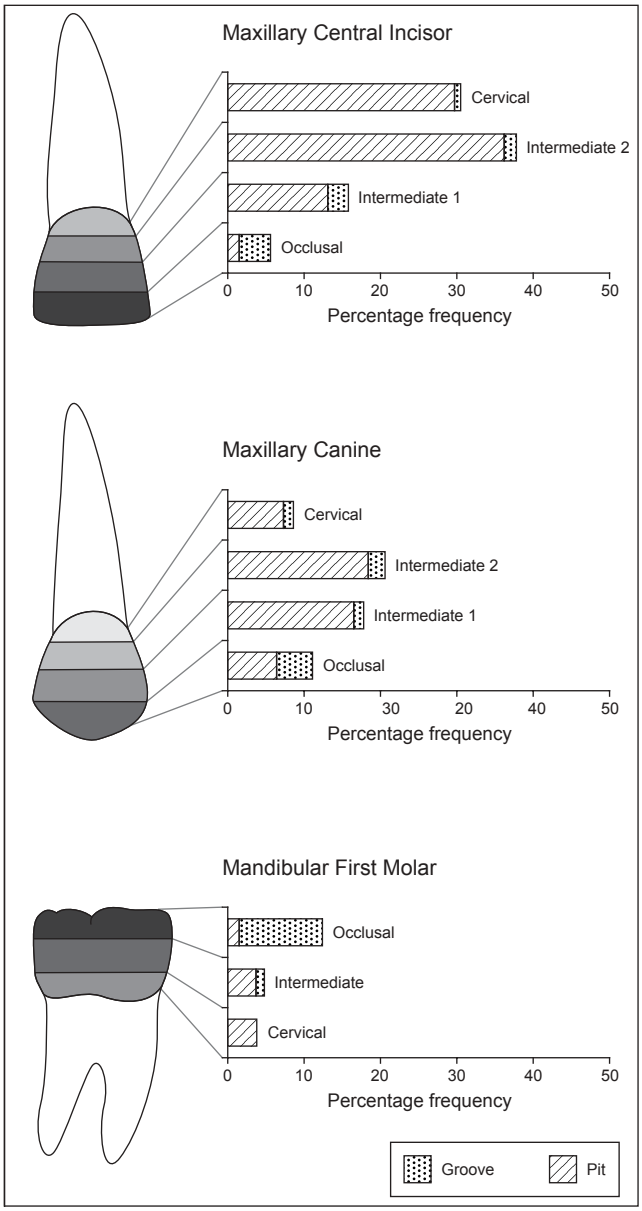

Fig 3. The distribution of grooves and single pits by location on the crown on selected teeth.

LEH by cohort

Overall frequency of LEH was high ranging from 33 to 88 per cent, but varied significantly by cohort (Table 3). The percentage of individuals without LEH declined markedly between the 1930-1939 and 1940-1944 cohort, and then further with successive cohorts. At the same time the number of people with three or

Table 3. Number (N) and percentage of individuals with at least one symmetrical defect within developmental units of the permanent dentition

Cohort	No hypoplasia	1 defect	2 defects	3+ defects	N
1890-1929	66.7	25.0	–	8.3	12
1930-1939	60.4	22.9	12.5	4.2	48
1940-1944	38.7	29.3	17.3	14.6	75
1945-1949	23.6	30.9	25.5	20.0	55
1950-1954	14.6	22.9	19.8	42.7	96
1955-1960+	11.7	22.1	26.5	39.7	68

Chi-square=89.636, d.f.=12, p=0.000

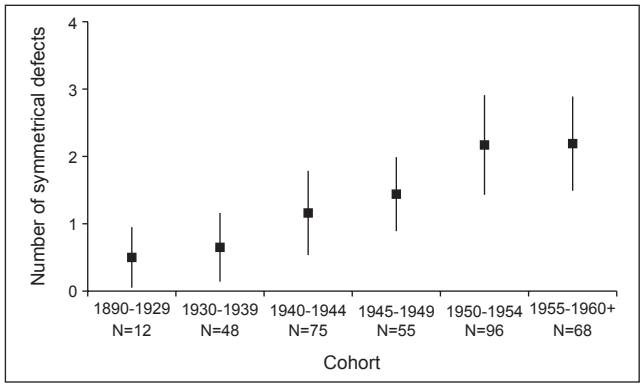

Fig 4. A box and whisker plot of the mean number of symmetrical defects (occurring on more than one tooth pair) by cohort at Yuendumu.

more episodes (i.e., sum of defects by developmental unit not the sum of defects on each crown) increased particularly in the last two cohorts.

Examining this in terms of the mean number of defects per cohort (Fig 4) it is clear that as the number of hypoplastic episodes increased, variation among the population, as measured by the coefficient of variation, actually decreased because the number of individuals who escaped LEH diminished dramatically. Effectively at the same time more children experienced hypoplastic defects they became more homogeneous.

Defect frequency and sex

When all casts were considered together, there was no significant difference between the frequency of defects among males and females. However, in the youngest cohort, 1955-1960, the frequency of defects among males was significantly higher than among females (male: av.=2.50, SD=1.43, n=40; female: av.=1.75, SD=1.24, n=28; t=2.278, p=0.03).

Age distribution of defects

The age distribution of defects also varied over time (Fig 5a). In the earliest cohort (1890-1940) defects occurred in a distinct unimodal pattern with most occurring between two to four years of age. Very few defects occurred prior to two years of age (Units A and B) and there was a marked decrease after four years of age. The 1940-1944 cohort follows a similar base pattern but with higher frequencies of defects in each age group. In the 1945 and later cohorts there is a marked increase in earlier occurring defects. Effectively children were experiencing many more repeated episodes over a longer, less compressed period of infancy and childhood.

The shift to earlier occurrence was even clearer if the age of first defect was compared (Fig 5b). In the first two cohorts the modal stage of first onset was unit B (c 1.5-2.2 years) and many still experienced their first defect later, between 2.2 and 5.2 years of age. There was a trend to earlier occurrence in the 1940-1944 cohort but this became increasingly pronounced so that in the youngest cohort most children who experienced a defect did so within the first year of life.

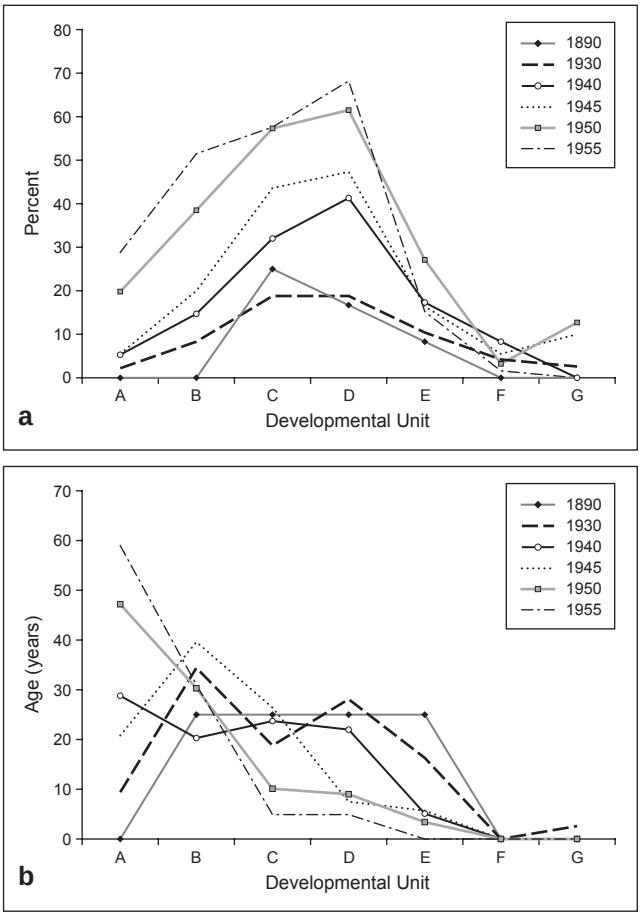

Fig 5. Percentage distribution by development units of: a) all LEH within the permanent dentition; b) age of onset, by cohort.

Defects of primary teeth

All hypoplastic defects of the primary teeth were recorded: pits, lines, missing enamel along with whether or not the defect occurred symmetrically. The frequency of defects varied by tooth with the highest frequency recorded on the canines. Defects were two times more common on lower canines than uppers (Table 4). The majority of these defects were asymmetrical and took the form of a plane-like defect on the labial surface.[13]

Primary defects could only be recorded for the post-1940 cohorts. Unlike defects of the permanent dentition there were no statistically significant differences between the cohorts either in the frequency of hypoplastic defects although there is a slight increase from 1940 to 1945 and an increase with the last cohort (Fig 6).

Cross-tabulation of the presence/absence of primary defects with presence/absence of permanent LEH indicated no relationship between the two sets of defects nor did comparisons involving age of onset nor mean number of defects with one exception. Individuals who had primary defects were significantly more likely to have higher rates of LEH in developmental unit A (48.3 vs. 38.4 per cent, n=92, chi-square=12.57, d.f.=1, p<0.000).

Table 4. Distribution of deciduous hypoplasic defects by tooth type

Tooth	% individuals with defects (n)	% of teeth with defects (n)	% of defects asymmetrical (n)
Ui1	5.3 (57)	2.7 (113)	33.3 (3)
Ui2	1.2 (85)	1.2 (170)	0 (2)
Uc	12.1 (190)	10.4 (376)	24.3 (37)
Um1	2.7 (225)	2.7 (448)	0 (12)
Um2	3.7 (268)	3.7 (534)	0 (18)
Li1	2.0 (51)	1.9 (104)	0 (2)
Li2	1.6 (64)	0.8 (128)	100.0 (1)
Lc	28.8 (177)	23.8 (353)	20.5 (83)
Lm1	3.1 (225)	3.1 (447)	7.7 (13)
Lm2	4.8 (270)	4.7 (533)	0 (22)
Total	27.1 (273)	6.0 (3206)	

DISCUSSION

History and LEH

The history of European movement into Central Australia and increased control over Aboriginal resources can be seen as involving three stages.[14] Firstly, from the 1860s to 1920s there was little direct interaction between Warlpiri people and Europeans. As livestock were moved through Warlpiri country and short-lived mining ventures were established, only some Aboriginal people came into intermittent contact with Europeans.[15] At this stage the hypoplasia rate was low and comparable to the rate observed on human remains from this area.[16] Many people escaped LEH altogether and its occurrence tended to be late.

A disastrous drought in the late 1920s ushered in a second stage of closer contact between Aboriginal people and Europeans.[15] Government and station owners circulated rations (mainly dry) to Aboriginal people both as payment for Aboriginal labour (an important resource for station owners) and as a way of avoiding conflict.[14] The result was much greater sustained contact and higher levels of sedentism. LEH rates reflect this: there was a significant increase in the numbers affected, although the age of onset did not change.

During World War II, many Aboriginal people were employed in Alice Springs while others congregated around ration points. The poor health of these people was recorded by army doctors at the time.[14] Thus, marked changes were found among children born between 1940 and 1944. Many more experienced multiple growth disruptions which occurred slightly earlier than previously. On average, higher rates of LEH and earlier onset reflected these changing circumstances.

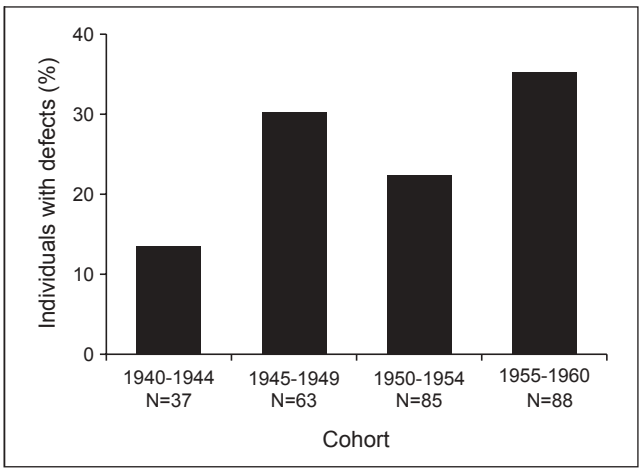

Fig 6. Percentage of individuals with at least one deciduous defect by cohort (chi-square=7.66, d.f.f=3, p=0.05).

In the third stage, post-World War II, the earlier protectionist policies were replaced by a focus on assimilation of the no longer declining Aboriginal population with the Europeans.[14] Aboriginal people were to be placed on government settlements (away from European contact except for trainers) and taught European customs. Yuendumu became one of the first of these settlements in 1946, initially as a Baptist mission with very little infrastructure. During the 1950s, as the number of people on the settlement increased from about 300 to around 1000, the number of European government employees also increased with more infrastructure including a health clinic and communal dining hall.[17] However, housing and provision of water and sewage facilities remained poor.

The settlement effectively congregated large numbers together exposing them to a variety of gastro-enteric and respiratory infections in an unhealthy environment.[17] At this stage the frequency of LEH rose dramatically and variation between children collapsed. Along with increased prevalence of LEH, defects started earlier and occurred over a longer period in infancy and childhood.

Among the Yuendumu children, those born before 1940 had mothers who were probably on average less sedentary and these children had a later age of LEH onset. As sedentism increased, LEH onset occurred earlier and when, post-1955, the nurses introduced infant feeding and supplementation of infants by six months at the latest,[17] LEH onset moved even earlier to between 6-12 months. This is probably a direct link with feeding practices and their impact is seen in the decreasing variation around age of onset.

Thus, as children's environments became more uniform, so did their experience of LEH. This is despite the availability of nursing staff at the settlement, attempts to regulate infant feeding, and hospital visits by residents on average every two weeks for children.[17]

Mortality and morbidity

The relationship between morbidity, as indicated by LEH, and mortality probably altered over the time period discussed. While morbidity worsened from pre- to post-settlement conditions yet mortality rates remained constant from 1955 (when first records were collected) to c 1970. Of the 594 children born on the settlement between 1953 and 1970, 24.7 per cent died before five years of age, primarily from respiratory and gastrointestinal disease.[17]

For earlier cohorts there is no indication of mortality rates but it is unlikely that mortality exceeded the post-

Table 5. Comparative rates of LEH among historic and modern populations

Location	Population	Time period	% of individs with LEH
*Primary dentition**			
Various	Developed nations[22]	Modern	4-5
Nigeria	Mixed[23]	Modern	21.0
Guatemala	Rural and urban[24]	Modern	22-73
Tiwi Islands	Aboriginal children[21]	Modern	99.0*
Brisbane	Aboriginal children[2]	Modern	98.0*
Central Aust.	Yuendumu	1940-70	27.1
Permanent dentition			
Australia	Australian Aborigines[16]	Pre-contact	28.2†
Jordan[25]	Pella	Iron Age	92.0
	Bedu	Modern	78.7
	Villagers	Modern	70.5
	Urban	Modern	42.9
USA[25]	Slaves	19th c AD	89.0
	Colorado	AD 500-700	90.0
	Pueblo Grande	AD 1050-1400	99.0
China[5]	Shanghai (pre-famine)	1949-1953	49.0
	Shanghai (famine)	1954-1961	55.9
	Shanghai (post-famine)	1962-1974	45.5
Brazil[26]	Zoro – pre-permanent contact	1939-1950	c.27
	Zoro – at permanent contact	1970s	c.36

*Includes enamel hypocalcification and opacities.
†Based only upon canines.

1940s cohorts. In the early years of contact it may be that ill children failed to survive so that the LEH here represents a subset of survivors. However, the variation amongst earlier cohorts indicates that while some children developed LEH others escaped entirely which suggests a distinctly different pattern of disease and causes of death. It is probable that up to 1940 children were more affected by occasional epidemics and accidental death than the pervasive ill health that impacted upon the settlement's young children.

The relationship between LEH and changed patterns of infectious and nutritional disorders among Aboriginal children indicates the usefulness of hypoplasia as a marker of childhood circumstances. Since it is an indicator that, barring extreme wear or tooth removal, remains throughout life, LEH is demonstrably a permanent record of these circumstances.

Primary defects

In the Yuendumu population, primary enamel defects follow a distinctly different pattern of occurrence to permanent LEH. The aetiology of such defects is still debated. Linear defects have been related to a similar range of infectious and nutritional conditions as in the permanent dentition.[18] However, it has been suggested that localized hypoplasia of the primary canines is the result of osteopenia in infancy, either because the poor mineralization of the teeth makes them susceptible to trauma or that it contributes to fenestration of the developing primary canine crypt making the tooth germ more vulnerable to physical trauma.[19] Hence the relationship of localized primary defects to socio-economic status or growth indicators is potentially more indirect.[20]

In the current sample the disproportionately high prevalence of asymmetrical defects on the canines, particularly mandibular canines, supports the suggestion that plane form defects have a distinctly different aetiology from pits and grooves. The distribution maps closely that observed by Lukacs *et al.*[20] among children from North Indian villages.

There is a lack of association between hypoplasic defects of the primary and permanent dentitions, again supporting the idea of underlying differences in aetiology. However, these are not entirely unrelated conditions since presence of primary LEH was associated with a higher likelihood of a defect in the earliest development unit in the permanent dentition. The question remains is this because the same conditions create risk for both conditions or that a child who has experienced developmental disturbance in infancy is weakened and more susceptible to developmental disruptions in early childhood?

Comparative rates of defects

Although care needs to be taken since methods of recording often vary, a comparison of the rates of defects recorded here with other studies demonstrates how harsh conditions were at Yuendumu (Table 5). The rates of primary hypoplasic defects recorded here fall within a group living in stressful conditions with low socio-economic status. However, they are much lower than the rates recorded by Seow *et al.*[2,21] for recent rural and urban Aboriginal schoolchildren. The difference is likely to be methodological. The very high rate among modern Aboriginal children reflects the inclusion of hypocalcification and opacities among the defects. Obviously these could not be recorded among the Yuendumu dental casts.

The comparison of permanent defects (Table 5) indicates that the pre-World War II rates are comparable with other hunter-gatherer populations while the 1940-1950 rates compare with relatively disadvantaged modern populations (e.g. Bedu and rural populations in Jordan). The post-1950 rates are higher

and similar to those of historic groups who experienced nutritional and infectious stress or severe disadvantage, e.g., USA slave populations of the 19th century AD.

CONCLUSION

The rates of LEH among Australian Aboriginal people born between 1890 and 1960 reflect changing life conditions for these people and extend their record of health back beyond the period for which written records survive. As sedentism and the risks of gastrointestinal, respiratory and nutritional disorders increased so did the frequency of LEH. As efforts were made to enforce supplementary feeding under unhygienic circumstances and change breastfeeding patterns, LEH simply began to occur earlier. For this population LEH is a marker of changing morbidity, particularly infectious disease. It demonstrates that the chronic and long-term effects of European contact were not visible within the initial phase of contact (violence and epidemic diseases are not reflected in the LEH record) but that increasing control and forced sedentism with the attendant increases in nutritional and infectious disease, seriously impacted upon the life chances of children.

In many prehistoric populations it has been demonstrated that high rates of LEH are associated not only with other markers of childhood stress but also with lowered life expectancy among adults.[4] While these relationships are dependent upon the particular circumstances of population, they do mean that LEH is an important indicator not only of dental health but of more general child morbidity. LEH also provides a potentially useful indicator of childhood circumstances amongst adult populations for whom medical records may never have been collected. The current study demonstrates just how fine the LEH record may actually be.

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