Enamel hypoplasia and dental caries in Australian Aboriginal children: prevalence and correlation between the two diseases

L. Pascoe, BDS W. Kim Seow, BDSc, MDSc, DDSc, PhD, FRACDS

Abstract

This study investigated the prevalence of enamel hypoplasia and dental caries and the relationship between the two diseases in all 4- to 6-year-old Australian Aboriginal children of the Tiwi tribe on Bathurst Island. Seventy-nine of 80 children (99%) had enamel hypoplasia, with a mean of 12.0 ±4.1 hypoplastic teeth per child. Dental caries was noted in 66 (83%) of children and the mean number of decayed teeth per child was 3.9 ±3.3. A strong association between enamel hypoplasia and dental caries ($P < 0.01$) suggests that enamel hypoplasia may be a significant caries risk factor in this group. Furthermore, while high levels of medical morbidity were found, the relative importance of each medical condition in the pathogenesis of enamel hypoplasia could not be determined because nearly all patients with enamel hypoplasia had the full range of medical problems. It is likely that all the medical conditions commonly encountered contribute to enamel hypoplasia and may act synergistically. (Pediatr Dent 16:193–99, 1994)

Introduction

Enamel hypoplasias may be inherited as primary defects of enamel[1] or may be acquired as the result of childhood medical problems such as infections,[2,3] metabolic derangements,[4–6] premature birth,[7–9] and nutritional disorders.[9,10] Ameloblasts, the specialized end cells that form enamel, are very sensitive to insults, and the locations of enamel defects on the teeth provide permanent records of the injury during particular periods of tooth development.[11] The abnormal discoloration and tooth morphology associated with enamel hypoplasia may compromise esthetics and predispose the affected teeth to dental caries.

The prevalence of enamel hypoplasia in many indigenous populations has been reported to be high, and reflects the high levels of infant perinatal morbidity often encountered in these communities. The Guatemalan Indians of South America, for example, are reported to have a prevalence of enamel hypoplasia of approximately 73%.[12] Equally high prevalences observed in many prehistoric populations around the world[13,14] suggest high levels of biological stress.

The study of enamel hypoplasia in population groups highly predisposed to this condition would provide better insight into the etiological factors involved in this condition. Furthermore, identifying predisposing factors might encourage preventive methods. Clinical complications of enamel hypoplasia, as well as possible prevention methods, may be effectively studied in these predisposed groups.

It is hypothesized that Australian Aboriginal children represent a population group at high risk for enamel hypoplasia due to high levels of infant morbidity. Although early work by Barrett and Williamson[15] indicated that enamel hypoplasia is common in the permanent dentition of these children, no previous report describes the primary teeth. Similarly, little published information reports on the prevalence of dental caries in the primary dentition of Aboriginal children. The aims of this investigation are to examine the prevalence and etiological factors associated with enamel hypoplasia and dental caries in a group of Australian Aboriginal children and to determine possible relationships between the two dental diseases.

Patients and methods

Patients

The study group consisted of the entire population of Australian Aboriginal children of the Tiwi tribe, aged 4–6 years at the time of study, residing on Bathurst Island situated off the northern coast of Australia, approximately 80 kilometers from Darwin. Eighty children (38 males and 42 females) with a mean age (± SD) at the time of dental examination of 5.1 ± 0.7 years (range 4.0–6.5 years) were available for examination.

Dental examination

Children were examined at the dental clinic in the community medical center at the settlement. Mirror and probe were used to determine the presence of enamel hypoplasia and dental caries. All clinically visible surfaces were examined, and the findings entered into standard examination forms. A modified DDE (Developmental Defects of Enamel) Index[16] was used to classify the enamel defects. Enamel hypoplasia was considered present if a tooth surface showed enamel to be either pitted, grooved, or missing. Enamel opacity was considered to be a distinct change in translucency of enamel. Dental caries was diagnosed and classified using WHO criteria.[17]

Table 1. Prevalence of enamel hypoplasia and dental caries

	Number Affected	*Percent Affected*
Enamel hypoplasia		
Teeth (N = 1524)	969	64
Patients (N = 80)	79	99
Mean ± SD hypoplastic teeth/patient = 12.0 ± 4.2		
(Range = 0–18)		
Dental caries		
Teeth (N = 1524)	309	20
Patients (N = 80)	66	83
Mean number ± decayed teeth/child = 3.9 ± 3.3		
(Range = 1–14)		

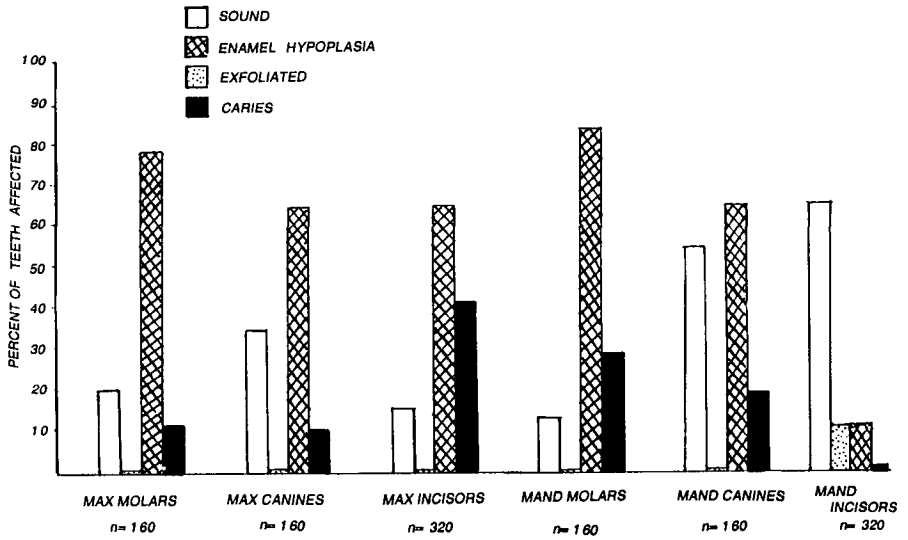

Fig 1. Distribution of enamel hypoplasia and dental caries in the primary dentition of Aboriginal children.

The dental examinations were performed by both authors, who were standardized using established techniques.[18] Consistency of diagnosis between examiners was checked by inter- and intraexaminer variability tests. Six nonparticipating patients were examined by both authors on three separate occasions, and paired *t*-tests revealed no significant intra- and interexaminer differences ($P > 0.1$).

Dental and medical histories

For 75 (94%) subjects no previous dental records were available. Medical histories, including prenatal and neonatal histories, were obtained from community medical center records. Low birthweight, premature birth, respiratory and gastrointestinal illnesses, anemia, and failure to thrive were noted. The body weights of the children at 2 years of age were obtained and related to percentile curves on standard growth charts. Previous studies[19–22] have indicated that standard growth charts for Caucasian Australian children are appropriate for Aboriginal children, as similar growth potential has been observed in both racial groups.

Statistical analysis

The Student's *t*-test, and Chi-square tests where appropriate, were used for statistical analysis of the data.

Results

Prevalence of enamel hypoplasia and dental caries (Table 1)

In the 80 subjects, 1524 of 1600 primary teeth were available for study, the remainder having been exfoliated. Seventy-nine patients (99%) had enamel hypoplasia of at least one tooth. Of the teeth available for study, 969 (64%) had at least one surface with enamel hypoplasia. The mean number of hypoplastic teeth per child was 12.0 ± 4.2 (range 0–18).

Sixty-six (83%) patients had at least one decayed tooth, with a mean of 3.9 ± 3.3 decayed teeth per child (range 1–14). A total of 309 of 1524 (20%) teeth were decayed. The dental examinations for this study were the first for most of the children (75 of 80), so no previous dental treatment had been rendered to the majority.

Distribution of enamel hypoplasia and dental caries

The distribution of enamel hypoplasia and dental caries in the primary dentition is shown in Fig 1. The highest frequency of enamel hypoplasia was observed in the primary molars; 85% of mandibular and 78% of maxillary primary molars. These were followed by the maxillary primary incisors (76%), and the maxillary and mandibular canines (64% each). The mandibular incisors were the least affected (12%).

Table 2. Association of dental caries with enamel hypoplasia

	Dental Caries Number of Teeth (%)		
	Absent	*Present*	*Total*
Enamel hypoplasia			
Absent	515 (42)	40 (13)	555 (36)
Present	700 (58)	269 (87)	969 (64)
Total	1215 (100)	309 (100)	1524 (100)

The association of dental caries with enamel hypoplasia was statistically significant, $P < 0.01$ ($x^2 = 92.1$, df = 1).

Table 3. Relationship between different types of enamel hypoplasia and dental caries in the maxillary primary incisors

Enamel Defect	*Number of Teeth (%) Dental Caries None*	*Demineralization With No Cavitation*	*Decayed With Cavitation*	*Total*
None	433 (89)	3 (1)	51 (10)	487 (100)
Opacity	18 (78)	2 (9)	3 (13)	23 (100)
Opacity with staining and enamel hypoplasia*	164 (54)	61 (20)	81 (26)	306 (100)
Hypoplasia — pits or grooves	28 (85)	2 (6)	3 (9)	33 (100)
Missing enamel	6 (86)	0 (0)	1 (14)	7 (100)
Total	649 (76)	68 (8)	139 (16)	856

* Enamel defects which presented as opacity with staining and enamel hypoplasia are significantly associated with decay, $x^2 = 96.2$, df = 2, $P < 0.001$. By contrast, all other defects are not significantly associated with decay.

Dental caries was most frequent in the maxillary incisors (41%), followed by the mandibular molars (26%), mandibular canines (19%), maxillary molars (12%), and mandibular incisors (5%).

Association of enamel hypoplasia with dental caries

To determine if there was an association between enamel hypoplasia and dental decay, hypoplastic lesions presenting with and without dental caries were compared. The results indicate that dental caries occurred predominantly in teeth with enamel hypoplasia, and that carious lesions without the presence of enamel hypoplasia occurred infrequently (Table 2). The association between enamel hypoplasia and caries was statistically significant ($P < 0.01$, $x^2 = 92.1$, df = 1).

Relationship between different types of enamel hypoplasia and dental caries

To determine the type(s) of enamel hypoplasia significantly associated with dental caries, the enamel lesions on the maxillary primary incisors were analyzed for their highest frequency of dental caries. As shown in Table 3, the type of hypoplastic lesion most significantly associated with dental caries was that classified as "opacity with staining and enamel hypoplasia" ($P < 0.001$, $x^2 = 96.2$, df = 2). By contrast, no other type of enamel defect (enamel opacity, hypoplastic pits or grooves, or missing enamel) was associated significantly with dental caries (Table 3).

Prenatal (maternal) health factors and enamel hypoplasia

The influence of various prenatal (maternal) health factors such as pregnancy age, number of previous pregnancies, perinatal infections, and anemia on the development of enamel hypoplasia was studied by comparing the mean numbers of hypoplastic teeth in children born of mothers with different frequencies of each condition. As shown in Table 4, children born of older mothers, or those with a higher number of previous pregnancies, as well as those who suffered severe infections and anemia, did not show greater numbers of hypoplastic teeth compared with those who were born of mothers without these conditions.

Prevalence of medical conditions in the perinatal and postnatal periods

The prevalence of various medical conditions in the perinatal and infancy periods that may be associated with enamel hypoplasia was surveyed. As shown in Fig 2, 14 of 80 children (18%) had low birthweight

Table 4. Prenatal (maternal) medical conditions and enamel hypoplasia

Maternal Condition	*Number of Patients*	*Hypoplastic Teeth Mean ± SD*	*% Teeth Affected*
Age at pregnancy			
< 16	5	10.8 ± 3.2	52
16–29	52	12.5 4.1	62
> 30	22	11.8 3.9	59
Number of previous pregnancies			
0	22	11.4 4.3	55
1–2	23	12.6 3.2	70
3–5	20	12.1 3.2	65
5	15	12.2 3.7	60
Infection			
Yes	36	12.2 3.5	61
No	35	12.5 3.4	67
Anemia			
Yes	39	12.8 3.9	69
No	32	11.4 ± 4.2	60

There was no significant association of any particular maternal health factor with enamel hypoplasia, $P > 0.1$.

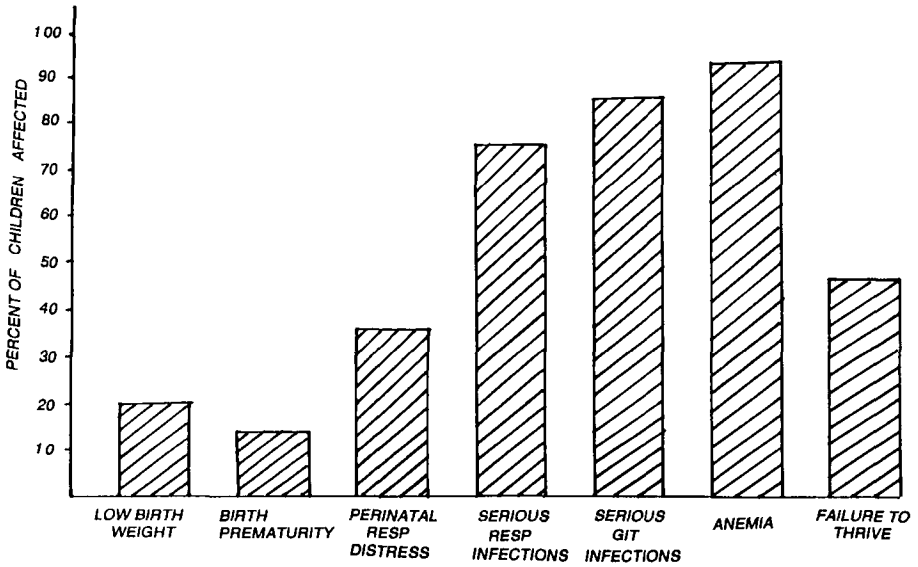

Fig 2. Prevalence of medical conditions in infancy.

Table 5. Perinatal medical conditions and enamel hypoplasia

Perinatal Condition	*Number of Patients*	*Hypoplastic Teeth* Mean ± SD	% Teeth Affected
Birthweight < 2500 g			
Yes	14	11.4 ± 3.1	66
No	66	12.3 4.0	66
Intubation			
Yes	3	10.0 4.2	53
No	75	12.6 3.8	67
Respiratory distress			
Yes	30	10.9 3.3	57
No	38	13.4 4.1	71
Infections			
Yes	12	10.6 2.9	56
No	66	12.7 ± 3.8	67

There were no significant differences in the mean numbers of hypoplastic teeth among the various infant medical conditions, $P > 0.1$.

(<2500 g), 11 (14%) were premature, and 29 (36%) had perinatal respiratory distress. In the first 18 months of life, serious respiratory and gastrointestinal infections were suffered by 60 (75%) and 68 (85%), respectively. Chronic iron deficiency anemia affected 74 (93%) of the children, and 37 (46%) were diagnosed as failing to thrive.

Perinatal medical conditions and enamel hypoplasia

The mean numbers of hypoplastic teeth in children with and without various perinatal medical conditions were compared. As shown in Table 5, no significant differences in the mean numbers of hypoplastic teeth could be detected in children with or without low birthweight, intubation, respiratory distress, or perinatal infections ($P > 0.1$).

Postnatal medical conditions and enamel hypoplasia

To determine the putative effects of various medical illnesses on enamel hypoplasia, the mean numbers of hypoplastic teeth in patients who suffered each medical condition were compared with those who did not have the corresponding illnesses. As shown in Table 6, children who suffered recurrent gastroenteritis, chronic respiratory infections, anemia, and repeated hospitalizations in the first 18 months of life, did not differ significantly in the mean numbers of affected teeth compared with those children who did not have the conditions ($P > 0.1$).

Body weight of children at age 2 and enamel hypoplasia

The body weight of each child at 2 years of age was determined from medical records and plotted against standard growth charts to determine its location on percentiles of normal. This age was selected because it coincided with the usual age of weaning from breastfeeding.[19] The results as shown in Table 7 indicated that the mean numbers of hypoplastic teeth in each child were similar, regardless of body weight, suggesting that low body weight is not associated with increased enamel hypoplasia prevalence ($P > 0.1$).

Discussion

Australian Aborigines

The Australian aborigines have lived in the country for at least 40,000 years — these original colonists most likely having arrived from southeast Asia through the then-connecting land mass between Australia and New Guinea.[23] Until the arrival of European settlement in Australia in the late-18th century, the Aborigines led mainly nomadic hunter-gatherer lifestyles, which were remarkably well adapted to the continent's many environments, including the severe desert conditions of central Australia.[24] In modern times, the alienation of their tribal lands and deprivation of the freedom to hunt and gather freely have caused them to drift toward settlements or reserves created for them where

Table 6. Postnatal medical conditions and enamel hypoplasia

Infant Condition	*Number of Patients*	*Hypoplastic Teeth* Mean ± SD	*% Teeth Affected*
Recurrent gastroenteritis			
Yes	67	12.6 ± 3.8	66
No	11	11.6 4.1	65
Respiratory infections			
Yes	60	12.5 3.7	65
No	18	12.4 4.1	66
Other infections			
Yes	54	12.8 3.5	66
No	24	10.9 4.1	61
Anemia			
Yes	77	12.4 3.1	67
No	1	16	80
Hospitalizations			
0	22	13.1 3.1	69
1	19	12.0 3.8	64
2	15	12.9 3.6	67
3 or more	20	13.7 2.9	72
Failure to thrive			
Yes	37	14.2 3.1	62
No	41	13.2 ± 3.3	70

There were no significant differences in the mean numbers of hypoplastic teeth among the various infant medical conditions, $P > 0.1$.

they have access to assistance provided by the government or religious missions.[23]

The impact of European settlement on Aboriginal health has been devastating. In an early survey of mortality and morbidity in Queensland Aboriginal settlements, the death rate for children aged 12 months to 5 years was 13 times that of the general population in the state.[25] The commonest causes of deaths were gastroenteritis and pneumonia, with severe nutritional deficiencies as precipitating factors. Other well-documented diseases among Aboriginal children include infections, skin diseases, intestinal parasitic infections, anemia, and eye and ear infections.[26] Low birth weight and growth retardation are extremely common. Those who survive have poorer educational and employment records, which help to perpetuate the cycle of deprivation.[21] Among adult Aborigines, there is a high prevalence of obesity, diabetes mellitus, alcoholism, hypertension, and coronary heart disease.[25, 26] These disease patterns, which are often associated with poor nutrition, reflect the problems of social inequality experienced by the Aborigines.

The Tiwi Aboriginals

The Tiwi tribes on Bathurst and Melville Islands, off the northern coast of Australia, have distinct social and cultural characteristics that differentiate them from the other Aborigines of the mainland. The main settlements on the islands started as Catholic missions established during the early part of the century.[27] Today, these settlements are administered by councils comprising mainly elected Tiwi people. On Bathurst Island, the population of approximately 2000 live mainly in the settlement of Nguiu. Medical care is available at a small medical center staffed by a resident doctor and a few nurses and Aboriginal health care workers. A mobile dental clinic is present at the medical center, and free dental care is provided by an itinerant government dental officer.

While there remains some hunting, fishing, and food gathering, Tiwi Aborigines now mainly buy their basic

Table 7. Body weight of children and prevalence of enamel hypoplasia

Body Weight at 2 Years of Age (Percentile of Normal)	*Number of Children*	*Total Number of Teeth*	*Hypoplastic Teeth* Number/child (Mean ± SD)	*Number of Teeth Affected*	*Percent Affected*
< 3	30	600	11.5 ± 4.9	344	57
3–10	27	540	12.6 3.5	340	63
11–25	18	360	12.4 3.9	223	62
26–50	3	60	13.6 3.8	41	68
> 50	2	40	10.5 4.5	21	53
Total	80	1600	12.0 ± 4.2	969	64

There were no significant differences in the mean number of hypoplastic teeth among the groups with different body weights at 2 years of age, $P > 0.1$.

foodstuffs from the local store run by their council. The costs of food items are usually a few times higher than the mainland equivalent, and fresh meat, fruit, and vegetables are very expensive, and are not always available. Compounding the problem of high food costs[28] is poor knowledge about food and health, and undeveloped cooking and budgeting skills. Furthermore, a large proportion of the family income is spent on alcohol and cigarettes. These undesirable practices commonly encountered in Aboriginal settlements,[28] make Aboriginal families, particularly children, vulnerable to malnutrition. As is the case with other Aboriginals, the general health of the Tiwi Aborigines has deteriorated since abandoning their traditional lifestyles. This fact is demonstrated in the children in this study, 20% of whom had low birthweight. As well, serious respiratory infections, gastrointestinal infections, and anemia were reported in 75, 85, and 93%, respectively. Furthermore, nearly half the children suffered failure to thrive.

Enamel hypoplasia

Our hypothesis that extremely poor general health is likely to lead to derangements of enamel formation is proven in this study in which 99% of the 80 children showed enamel hypoplasia. This figure is extremely high compared to the 3–10% reported for other Western population groups.[8, 29] Furthermore, the percentage of teeth affected was also high (64%), with a mean number of 12.0 ± 4.2 hypoplastic teeth per child. Although as early as 1956, Barrett[30] mentioned in passing in his study of Aboriginal children in Yuendumu in central Australia, that the prevalence of enamel hypoplasia in the primary dentition was high, no previous data on this aspect of dental health are available. To the authors' knowledge, this study represents the first comprehensive investigation of enamel hypoplasia in the primary dentition of Australian Aboriginal children.

While the adverse health of the children provides insight into possible etiological conditions associated with enamel hypoplasia, in this study it was not possible to statistically determine the relative importance of each medical condition in the etiology of enamel defects. This was because there were uniformly large numbers of affected teeth occurring in the children, who were nearly all similarly affected by the medical conditions in question. It is most likely that many medical factors such as malnutrition and infections act synergistically to produce the enamel defects. In this regard, a central mechanism of low calcium stores[9] resulting from chronic malnutrition may be likely, although infective organisms or high fevers can also cause ameloblastic dysfunction or death directly.[11]

Other possible causes of enamel defects such as endemic fluorosis are unlikely as the community uses rain water, and regular 3-month water analysis by the state government has consistently revealed fluoride levels to be below 0.1 ppm.

Dental caries

Previous data of caries prevalence of the primary dentition of Australian Aboriginal children are scarce. To date, only two early reports of the caries prevalence in the primary dentitions are available, and they are parts of other dental investigations. In an early study by Barrett and Williamson[15] in Yuendumu in central Australia, 30 children with primary dentitions were included as part of an overall study of caries prevalence. Sixty percent of the children were caries free, while 40% had dental decay, the number of decayed teeth per child being 2.5 ± 0.6. Kailis[31] in 1971 reported large differences in caries rates between two Aboriginal communities that had different diets and water fluoride levels. In the community with low fluoride levels and high refined carbohydrate intake, the mean dmf index was 4.05 compared with 0.66 in the community with high fluoride levels and a more traditional diet.

In this study, dental caries was noted in 83% of the patients, with each child having a mean of 3.9 ± 3.3 decayed teeth. This figure is more than twice that of the dmf per child recently reported in other Australian children of similar age groups in the same state.[32] However, the mean dmf per child is comparable to that of 2.7 reported by Kailis[31] of Aboriginal children residing in a low-fluoride community of Cundeelee in Western Australia. On the other hand, it contrasts markedly to the dmf of 1.01 reported in the community of Warburton,[31] which had high fluoride levels in the water.

Although the differences in dmf may be related to differences in fluoride exposure, it is important to determine risk factors predisposing to dental caries in the Aboriginal children. It has already been well documented that the change from a traditional bush diet of healthy, natural foods to a Western diet high in refined carbohydrates is one of the most important risk factors for caries development in Australian Aborigines.[33] In addition, in this study, we have found enamel hypoplasia to be significantly associated with dental decay. This association was observed in both anterior and posterior teeth, and the type of hypoplastic defect most commonly associated with dental caries is one that shows missing enamel with staining and opacity. These results thus show clearly that developmental enamel defects are an important risk factor in the high caries prevalence in the primary dentition of Aboriginal children. It is reasonable to hypothesize that the absence of an intact enamel surface highly predisposes a tooth to caries attack when the diet is high in refined carbohydrates and fluoride levels are low in the drinking water.

In conclusion, the results of our study have provided further insight into the reasons for the high dental caries susceptibility of Australian Aboriginal children and similar groups of indigenous peoples around

the world. While the introduction of western diets high in refined carbohydrates has been thought to be a major factor causing dental caries in these populations, the additional risk factor of underlying, highly prevalent enamel hypoplasia should be considered.

Conclusions

1. The group of Australian Aborigines studied have high prevalences of enamel hypoplasia and dental caries in the primary dentition.
2. There is a strong association of enamel hypoplasia with dental caries.
3. High prevalences of medical morbidity in the perinatal and infancy periods may be important etiological factors in the pathogenesis of enamel hypoplasia, although the relative importance of these factors is difficult to determine.

This study was funded in part by the Australian Institute of Aboriginal and Torres Straits Islander Studies. The authors thank the Tiwi people for their extended cooperation and support, the Northern Territory Department of Health for granting Dr. Pascoe time for this research study, the staff of St. Gerard Health Centre for assistance with the medical records, and Emeritus Professor G.N. Davies for advice and help with data analysis.

Dr. L. Pascoe is a dental officer with the Northern Territory Department of Health, Katherine, Australia. Dr. W. Kim Seow is associate professor in pediatric dentistry, University of Queensland Dental School, Brisbane, Australia and visiting professor, department of pediatric dentistry, Harvard School of Dental Medicine, Boston, Massachusetts and the Children's Hospital, Boston.

1. Witkop CJ: Amelogenesis imperfecta, dentinogenesis imperfecta, and dentin dysplasia revisited: problems in classification. J Oral Pathol 17:547–53, 1989.
2. Fiumara NJ, Lessell S: Manifestations of late congenital syphilis—an analysis of 271 patients. Arch Dermatol 102:78–83, 1970.
3. Stagno S, Pass RF, Thomas JP: Defects of tooth structure in congenital cytomegalovirus infection. Pediatrics 69:646–48, 1982.
4. Woodhead JC, Nowak AJ, Crall JJ: Dental abnormalities in children with chronic renal failure. Pediatr Dent 4:281–85, 1982.
5. Seow WK, Shepherd RW, Ong TH: Oral changes associated with end-stage liver disease and liver transplantation: implications for dental management. ASDC J Dent Child 58:474–80, 1991
6. Seow WK: Enamel hypoplasia in the primary dentition: a review. ASDC J Dent Child 58:441–51, 1991.
7. Seow WK, Brown JP, Tudehope DI, O'Callaghan: Developmental defects in the primary dentition of low birthweight infants: adverse effects of laryngoscopy and prolonged endotrachael intubation. Pediatr Dent 6:28–31, 1984.
8. Seow WK, Humphreys C, Tudehope DI: Increased prevalence of developmental dental defects in low birthweight, prematurely-born children: a controlled study. Pediatr Dent 9:221–23, 1987.
9. Seow WK, Masel JP, Weir C, Tudehope DI: Mineral deficiency in the pathogenesis of enamel hypoplasia in prematurely-born, very low birthweight children. Pediatr Dent 11:297–301, 1989.
10. Sweeney EA, Saffir AJ, De Leon R: Linear enamel hypoplasia of deciduous incisor teeth in malnourished children. Am J Clin Nutr 24:29–31, 1971.
11. Stewart RE: Developmental defects of enamel. In Pediatric Dentistry. Stewart RE, Barber TK, Troutman K, Wei SHY, EDS. St Louis: CV Mosby Co, 1982, pp 57–110.
12. Infante PF, Gillespie GM: An epidemiologic study of linear enamel hypoplasia of the deciduous anterior teeth in Guatemalan children. Arch Oral Biol 19:1055–61, 1974.
13. Blakey ML, Armelagos GL: Deciduous enamel defects in prehistoric American from the Dickson Moulds: Prenatal and postnatal stress. J Phys Anthropol 66:371–80, 1965.
14. Cook DC, Buikstra JE: Health and differential survival in prehistoric populations: prenatal dental defects. Am J Phys Anthropol 51:649–64, 1979.
15. Barrett MJ, Williamson JJ: Oral health of Australian Aborigines: survey methods and prevalence of dental caries. Aust Dent J 17:37–50, 1972.
16. Federation Dentaire Internationale Commisssion on Oral Health, Research and Epidemiology: An epidemiological index of developmental defects of dental enamel (DDE Index). Int Dent J 32:159–62, 1982.
17. World Health Organization. Oral Health Surveys. Basic Methods. 2nd Ed. Geneva: WHO, 1977.
18. Cleaton-Jones P, Hargreaves JA, Fatti LP, Chandler HD, Grossman ES: Dental caries diagnosis calibration for clinical field surveys. Caries Res 23:195–99, 1989.
19. Gracey M: Nutrition of Australian Aboriginal infants and children. J Paediatr Child Health 27:259–71,1991.
20. Hitchcock NE, Gracey M, Malley RA, Spargo RA: Physical size of 1887 Aboriginal school children in the Kimberley region. Med J Aust 146:415–19, 1987.
21. Gracey M, Spargo M, Malley RA, Spargo RA: The state of health of Aborigines in the Kimberley region. Med J Aust 146:200–4,1987.
22. Cockington RA: Growth of Australian Aboriginal children related to social circumstances. Aust NZ J Med 10:199–208, 1980.
23. Flood J: Archaeology of the Dreamtime. Sydney: Collins Publishers, 1989, pp 30–154.
24. Kirk RL: Aboriginal man adapting: the human biology of Australian Aborigines. New York: Oxford University Press, 1981, pp 110–67.
25. McIlraith S, Reid J, Franklin M: Aboriginal health and lifestyle. Canberra: Australian Medical Association, 1982, pp 3–18.
26. Thompson N: Aboriginal health — current status. Aust NZ Med 14:705–18, 1984.
27. Pye J: The Tiwi Islands. Canberra: National Library, Australian Commonwealth Teritory, 1987, p 25.
28. Sullivan H, Gracey M, Hevron V: Food costs and nutrition of Aborigines in remote areas of northern Australia. Med J Aust 154:294, 1991.
29. Grahnen H, Larsson PG: Enamel defects in the deciduous dentition of prematurely born children. Odontol Revy 9:193–204, 1958.
30. Barrett MJ: Dental observations on Australian Aborigines: water supplies and endemic fluorosis. Aust Dent J 16:87–92, 1956.
31. Kailis DG: Dental conditions observed in Australian Aboriginal children resident in Warburton and Cundeelee missions, Western Australia. Aust Dent J 16:44–52, 1971.
32. Australian Commonwealth Department of Health: Dental Health of Children in Australia 1977–1986. Canberra: Australian Government Publishing Service, 1987, pp 110–14.
33. Campbell TD, Barrett MJ: Dental observations on Australian Aborigines: a changing environment and food pattern. Aust J Dent 57:1–6, 1953.