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Dental enamel defects and dental caries of primary teeth among Indigenous children in Western Australia

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ABSTRACT

Background: Teeth affected with enamel defects (EDs) are at an increased risk of dental caries. In spite of improving oral health status overall in Australia, Indigenous Australians still experience higher rates of dental caries than non-Indigenous Australians. This study reports on the prevalence of EDs and dental caries experience among Indigenous children in the Kimberley region of Western Australia.
Methods: Health status of all the primary teeth (ICDAS II criteria) and the presence of EDs on index teeth (modified Dental Defects of Enamel index; DDE) of young Indigenous children who participated in a 2-arm intervention trial was recorded. Generalized estimating equations were used to estimate the association between EDs and dental caries and effect estimates were presented as odds ratios and associated 95% confidence intervals.
Results: Person-level prevalence, from 237 children (mean age 3.6 years, standard deviation 1.7) assessed for EDs, was 58% and tooth-level prevalence was 24%. Teeth affected with demarcated or diffuse defects were associated with a two-fold higher odds of having caries experience, odds ratio (OR) 2.5, 95% confidence interval (CI) 1.7–3.7 and OR 2.7, 95% CI 1.7–4.0 respectively.
Conclusions: The presence of EDs among young Indigenous children was associated with a higher odds of caries experience among affected teeth.

Keywords: Dental caries, dental defects of enamel, Indigenous Australians, molar incisor hypomineralization, primary teeth.

Abbreviations and acronyms: CI = confidence interval; DDE = Dental Defects of Enamel; dmfs = decayed, missing, filled tooth surfaces; ED = enamel defects; OR = odds ratio; PEB = post-eruptive breakdown; SD = standard deviation; SE = standard error.

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INTRODUCTION

The presence of teeth affected with enamel defects (EDs) imposes a significant burden on affected individuals. Affected teeth are at an increased risk of dental caries, sometimes hypersensitive and the friable enamel which breaks down easily makes for difficulties in their repair, and impair oral health-related quality of life in children.[1,2] The prevalence of demarcated EDs of the first permanent molars in Western Australia (WA) children has been estimated at 22%.[3] The global prevalence of EDs defined as molar incisor hypomineralization (EDs of the first permanent molars and incisors) has been estimated to be 13% with over 840 million prevalent and incident cases in 2015–2016.[4] The worldwide prevalence of hypomineralized EDs of the primary molars has been reported to range from 0% to 41% at the individual level with a pooled prevalence of 7%, and ranged from 0% to 30% at the tooth level, with a pooled prevalence of 4%.[5] The occurrence of dental EDs, particularly demarcated opacities, has been reported to be associated with an increased risk of caries in permanent and primary teeth[6–12] and findings from a few studies suggest that hypomineralized EDs might predispose teeth to a more rapid progression of caries, thus leading to poorer carious outcomes where access to care may be limited.[7,8] Also, hypomineralized EDs of second

primary molars have been reported to be associated with poorer oral health-related quality of life.[13]

There is limited information available on the occurrence of EDs among the Indigenous population in Australia. The national Australian child oral health study of 2012–2014 recorded the presence of enamel hypoplasia (detectable loss of enamel) in permanent and primary teeth, and reported a higher prevalence among the Indigenous than non-Indigenous children, 13.3% and 10.1% respectively.[14] An Australian study, which used archived data of dental study casts of residents born between 1890 and 1960 in an Indigenous settlement in Central Australia, reported a prevalence of EDs in the form of linear enamel hypoplasia in permanent teeth ranging from 33% to 88%, and that the prevalence increased over time.[15]

A high prevalence of EDs, primarily hypoplasia, in primary teeth of young Australian Indigenous children compared with non-Indigenous children has also been reported.[16,17] The studies hypothesized that the high prevalence of EDs might also be associated with the high prevalence of carious lesions among Indigenous children. In spite of the improving oral health among the Australian population, Indigenous children still have higher caries experience than non-Indigenous children.[18,19] There is limited contemporary information on the prevalence of EDs and the potential association of EDs with the occurrence of dental caries in Australian Indigenous children. This study used data collected as part of a clinical trial in the Kimberley region of Western Australia (WA) to report on the prevalence of dental EDs among young Indigenous children and their potential association with the occurrence of caries. The study's hypothesis was of an association between the occurrence of dental EDs and increased occurrence of dental caries with the null hypothesis being no association between EDs and dental caries experience.

METHODS

Study participants were young Indigenous children who participated in a randomized clinical trial which tested a minimally invasive dental treatment model of care in remote Indigenous communities in the Kimberley using a cluster-randomized delayed-intervention design. The principal aim of the clinical trial was to test the feasibility of delivering comprehensive primary dental care using portable dental equipment and using the 'no needle, no drill' Atraumatic Restorative Treatment and the Hall Technique for restorative treatment.[20,21] Pulp therapy and dental extractions were also provided, where indicated. Ethical approval for the study was provided by the WA Country Health Service Human Research Ethics Committee (WACHS HREC Project Reference 2017/01), the WA Aboriginal Health Ethics Committee (Project Reference: 790) and the University of Adelaide Health Research Ethics Committee (H-2017-015).

The details of the study design, including sample size, and population selection and clinical findings have been reported.[22,23] Briefly, parents of young children (<6 years of age) living in selected remote Indigenous communities in the Kimberley region of WA provided signed informed consent to participate in the study. Participating children and their parents completed a questionnaire which elicited demographic information as well as information on child dental anxiety and child oral and general health-related quality of life at baseline. Each participating child was also examined by two calibrated examiners and the tooth status recorded at baseline. The children were examined either under standard dental lighting or natural light, depending on where the clinical area was set up. Each child was examined either lying on their back on a portable dental chair or in a knee to knee position with the child lying on the parent's/carer's lap. All examinations were undertaken without prior tooth cleaning. The teeth were dried using cotton rolls or gauze and visually assessed using a plane mirror and periodontal probe (the probe was only used to remove debris, and for assessing enamel integrity, but not for tactile detection of lesion softness). The teeth were assessed for the presence of dental caries using the ICDAS II and the modified Dental Defects of Enamel (DDE) criteria for the presence of EDs.[24,25] Oral hygiene was determined using the Silness-Löe index through visual assessment of the presence of plaque on index teeth (55, 65, 75, 85) coded at four levels; 0 = no plaque, 1 = thin film of plaque which can be seen by scraping with a periodontal probe, 2 = moderate amount of plaque visible with the naked eye, 3 = abundance of plaque, and an overall score obtained by summing the scores of the four teeth.[26]

All surfaces of all the teeth present were assessed for dental caries (without dental radiographs), and DDE was assessed on the occlusal and buccal/labial surfaces of index primary teeth (53, 63, 73, 83, 55, 65, 75, 85). The index teeth were chosen based on the close timing to the commencement and completion of enamel calcification to each other and to the enamel formation of the first permanent molars. A tooth surface was coded as having caries experience for lesions classified at ICDAS code 4 or higher or where a tooth has been filled or extracted due to caries. Caries experience of the primary teeth was aggregated at the person and tooth level as decayed, missing and filled surfaces (dmfs). Teeth were classified for EDs as sound (normal enamel); demarcated opacity (well defined opacity with a clear boundary between normal and affected enamel); diffuse opacity (more generalized opacity with ill-defined boundary

between affected enamel and normal enamel); hypoplasia (defects where enamel is missing, or reduced amount of enamel is present); or a combination of the three types. Hypoplasia was differentiated from post-eruptive breakdown (PEB) by characteristics of the enamel and the occurrence of PEB associated with enamel opacities.[27] The tooth surface level information was aggregated into tooth-level information as follows; any tooth surface with demarcated opacity or demarcated opacity in combination with other types of defects (diffuse, hypoplasia) or presented with atypical restorations was coded as demarcated; any tooth with diffuse opacity and hypoplasia was coded as diffuse opacity; and any tooth with hypoplastic defect alone was coded as hypoplasia. The ED data were further aggregated at the person level using the same logic as for tooth-level aggregation.

Analysis

Descriptive statistics using means and proportions are presented for the presence of EDs. Bivariate analyses for associations between the presence of EDs and measured factors were evaluated using Chi-square test of proportions for categorical variables (Fisher's exact test when expected cell size <5) and non-parametric Kruskal–Wallis rank test for continuous variables that were not normally distributed, and *t*-test and analysis of variance for normally distributed variables. Generalized estimating equations with logit link and robust standard error estimation was used to undertake multivariable analysis to evaluate the association between the presence of EDs at the tooth and person level and dental caries at the tooth level (caries experience = 0 if dmfs = 0, caries experience = 1 if dmfs > 0). The effect estimates are presented as odds ratios and the associated 95% confidence intervals, controlling for possible confounding factors and the intra-individual correlation. All statistical analyses were undertaken using Stata 17 (Stata Corp LLC, College Station, TX, USA), and *P* values less than 0.05 were considered statistically significant.[28]

RESULTS

Three hundred and thirty-eight children participated in the study, however, one child was unable to be examined and was excluded. The mean age at baseline was 3.6 years (standard deviation (SD) 1.7). The overall caries experience (mean dmfs) of the children was 8.1 (SD 10.8). Two hundred and thirty-seven individuals were able to be assessed for EDs; those unable to be assessed were principally because of the index teeth being unerupted. The level of agreement of the examiners for dental caries was excellent (kappa = 0.81) The person-level prevalence of any ED was 58% (45% demarcated opacity, 11% diffuse opacity and 2% hypoplasia). The baseline distribution of demographic and clinical measures by type of EDs at person level is shown in Table 1. Children with EDs had higher caries experience and plaque levels, were more likely to have been allocated into the intervention group, and lived in areas with higher levels of community water fluoride level than children without any defects.

The distribution of tooth type by ED type is shown in Table 2; teeth unable to be assessed for EDs either because of restored surfaces or extensive caries were excluded. The overall tooth prevalence of EDs was 24%, with demarcated opacities being the most prevalent at 13%, diffuse opacities 9% and hypoplastic defects 2%. The distribution of tooth type by EDs, including the combination of defects present is shown in Table S1.

Generalized estimating equation with logit link for any caries experience at the tooth level controlling for independent factors and EDs classified at the individual (person) level is shown in Table 3. A 35% higher odds of caries experience was associated with an increase in age by 1 year (odds ratio (OR) 1.35) and

Table 1. Baseline factors by enamel defect type

Factors	Normal (n = 99)	Demarcated opacity (n = 106)	Diffuse opacity (n = 28)	Hypoplasia (n = 4)	*P* value
Sex n (%)					Fisher's exact
Female = 126	58 (46.0)	51 (40.5)	15 (1.9)	2 (1.6)	
Male = 111	41 (36.9)	55 (49.5)	13 (11.7)	2 (1.8)	0.50
Age, mean (SD)	4.1 years (1.5)	4.5 years (1.3)	4.7 years (1.4)	4.5 years (0.7)	0.11
dmfs, mean (SD)	7.8 (10.9)*	11.9 (11.9)*	12.0 (10.6)	15.3 (7.4)	0.04
Plaque, mean (SD)	4.8 (2.4)*	5.7 (2.2)	6.2 (2.0)*	5.0 (2.6)	0.02
Group allocation n (%)					Fisher's exact
Test = 133	42 (31.6)	73 (54.9)	15 (11.3)	3 (2.2)	
Control = 104	57 (54.8)	33 (31.7)	13 (12.5)	1 (1.0)	0.001
Fluoride levels (mg/L), mean (SD)	0.36 (0.28)**	0.42 (0.29)*	0.61 (0.25)*, **	0.62 (0.31)	<0.001

dmfs = decayed, missing, filled tooth surfaces; SD = standard deviation.
*Statistically significant, $P < 0.05$.
**Statistically significant, $P < 0.01$.

Table 2. Tooth type by type of enamel defects, n (%)

Tooth (n)[a]	Sound	Demarcated opacity	Diffuse opacity	Hypoplasia
55 (n = 209)	142 (67.9)	39 (18.7)	24 (11.5)	4 (1.9)
65 (n = 206)	145 (70.4)	30 (14.6)	25 (12.1)	6 (2.9)
75 (n = 208)	126 (60.6)	50 (24.0)	31 (14.9)	1 (0.5)
85 (n = 208)	128 (61.5)	44 (21.2)	28 (13.5)	8 (3.9)
53 (n = 230)	189 (82.2)	22 (9.6)	15 (6.5)	4 (1.7)
63 (n = 229)	201 (87.8)	13 (5.7)	13 (5.7)	2 (0.9)
73 (n = 226)	187 (82.7)	21 (9.3)	11 (4.9)	7 (3.1)
83 (n = 226)	198 (87.6)	15 (6.6)	8 (3.5)	5 (2.2)
Total (n = 1742)	1316 (75.5)	234 (13.4)	155 (8.9)	37 (2.2)

[a]The number of teeth do not equal the number of persons because of unerupted or absent teeth.

Table 3. Generalized estimating equation regression of caries experience at the tooth level (dmfs > 0) with logit link and enamel defect classified at the person level

	Odds ratio	SE	95% CI	*P* value
Age	1.35	0.09	1.18–1.55	<0.000
DDE_person				
Ref – Sound				
Demarcated	1.68	0.33	1.14–2.48	0.009
Diffuse	1.45	0.38	0.87–2.41	0.155
Hypoplasia	2.83	1.82	0.80–10.00	0.106
Plaque	1.20	0.05	1.10–1.31	<0.000
Sex				
Ref – female				
Male	0.70	0.11	0.51–0.97	0.029
Group allocation				
Ref – test				
Control	0.85	0.14	0.61–1.19	0.349
Fluoride levels	0.92	0.27	0.52–1.64	0.782

CI = confidence interval; DDE = Dental Defects of Enamel; dmfs = decayed, missing, filled tooth surfaces; SE, standard error.

a 20% increased odds of caries experience with one increment in the plaque score (OR 1.2). The presence of demarcated EDs at the individual level was associated with a 68% higher odds of caries experience (OR 1.68). Boys were at 30% lower odds of caries experience.

Generalized estimating equation with the logit link for any caries experience and EDs at the tooth level, controlling for independent factors and intra-individual correlation is shown in Table 4. Being older and having high accumulation of plaque was associated with higher odds of having caries experience, OR 1.19 and 1.36 respectively. The presence of demarcated and diffuse EDs was also associated with two and a half times higher odds of caries experience. Males were at a lower odds of caries experience, OR 0.69. Exposure to higher levels of community water fluoride levels was associated with lower odds of caries experience, OR 0.95, but this difference was not statistically significant.

Table 4. Generalized estimating equation regression of caries experience (dmfs > 0) with logit link and enamel defect classified at the tooth level

	Odds ratio	SE	95% CI	*P* value
Age	1.36	0.10	1.18–1.57	<0.001
DDE_tooth level				
Ref – Sound				
Demarcated	2.51	0.50	1.69–3.73	<0.001
Diffuse	2.65	0.56	1.73–4.04	<0.001
Hypoplasia	2.12	0.96	0.90–4.99	0.085
Plaque	1.19	0.05	1.09–1.30	<0.001
Sex				
Ref – female				
Male	0.69	0.12	0.50–0.96	0.028
Group allocation				
Ref – test				
Control	0.85	0.15	0.60–1.20	0.348
Fluoride levels	0.95	0.28	0.53–1.67	0.852

CI = confidence interval; DDE = Dental Defects of Enamel; SE, standard error.

DISCUSSION

This study evaluated the prevalence of EDs in primary teeth of Indigenous children and tested the hypothesis that there was an association between the occurrence of dental EDs and the of dental caries experience. The findings showed that the prevalence of EDs in the primary teeth of young Indigenous children in the Kimberley was higher than the prevalence reported of Australian non-Indigenous children, and the null hypothesis of no association between EDs and dental caries was rejected. An overall person-level prevalence of 58% of any ED was lower than the 98% prevalence of any ED reported among Indigenous children in Brisbane by Seow *et al.*[17] and the 99% prevalence reported by Pascoe and Seow of children of the Tiwi tribe on Bathurst Island,[16] but was higher than the prevalence of hypoplasia (13.3%) and non-fluoride opacities (6.8%) found among Indigenous children in the 2012–2014 national child oral health survey.[14] The differences may be due to variations in study sample recruitment and clinical assessment procedures. The children in the Brisbane study were attendees at six local Indigenous pre-schools and were older, and the children in the national study were a random sample of children 5 years of age or older. Participants in the current study were recruited for a randomized controlled trial, and were not a random sample of Indigenous children in community, thus, the findings are not generalizable to the wider Indigenous child population. Seow *et al.* used the modified DDE index as used in this study,[17] however, all teeth present were evaluated for the presence of EDs while only the primary second molars and canines were evaluated in this study for EDs, and the national study evaluated non-fluorotic opacities only on the

two upper permanent central incisors, and hypoplasia on all teeth present.[29]

This study found a higher prevalence of EDs than that reported for non-Indigenous children in Australia.[11] The person-level prevalence reported by Owen *et al.* of non-Indigenous children was 14% and tooth-level prevalence of 6%, which was lower than the prevalence found in this study. This study also found a strong association between caries experience of teeth affected with EDs; a twofold higher odds of caries experience among teeth affected with either demarcated or diffuse opacities, unlike the study by Owen *et al.* which did not find a statistically significant association between the presence of EDs and dental caries. The authors of that study speculated that the lack of association may have been due to the low caries prevalence in their study sample.

The finding in this study of the occurrence of EDs and its association with caries experience is similar to the findings of Seow *et al.* and Pascoe and Seow.[16,17] The association of the presence of EDs at the person level with caries experience was statistically significant for demarcated opacities, but not for other defects. The association was found to be stronger and was statistically significant when evaluated at the tooth level with higher odds of caries experience of teeth with demarcated and diffuse opacities, after controlling for age and plaque levels. There was also a higher odds of caries experience among teeth classified as having hypoplastic defects, although it was not statistically significant at the 0.05 level. It is likely that the low numbers of teeth with hypoplastic defects limited the capacity to reach statistical significance. A longitudinal evaluation of incidence of caries in primary teeth in relation to the presence of EDs of Australian children reported an overall prevalence of EDs of 10%.[12] The authors reported increased risk of caries over the observation interval among children with EDs, with the highest teeth at risk being the second primary molars. That study, however, classified EDs at the level of hypoplasia only, thus does not provide information on the association with hypomineralization defects. This study found a lower prevalence of hypoplastic defects than reported elsewhere and it is possible that the difference may be due to differences in the diagnostic criteria used among the studies and also could be due to the different time periods in which the studies were undertaken.

Another longitudinal study, among Brazilian children 2–5 years of age, found person-level and tooth-level prevalence of EDs of 28% and 4% respectively. They reported an unadjusted higher risk of caries with demarcated and hypoplastic defects, but only hypoplastic defects remained a significant risk factor for caries increment after adjusting for other baseline factors, including baseline caries experience.[30] It is possible that children with demarcated defects in that study, which are more prone to PEB, may be misclassified as having hypoplastic defects. In that study, baseline caries was a significant risk factor for caries incidence and it is also possible that teeth with demarcated defects may have already experienced caries at baseline and the extent of caries prohibited the classification of teeth for EDs, thus, masking the presence of EDs.

This study found a higher prevalence of EDs and caries experience than reported for non-Indigenous Australian children in other studies. This study does not explain the prevalence of EDs among the Indigenous children in the Kimberley. While the causes of some EDs are relatively well-known, hypomineralization EDs associated with molar incisor hypomineralizations and hypomineralizations of the second primary molars are still relatively unknown although there have been many purported causal factors and mechanisms reported.[31] Our study sample can be broadly classified as living in remote locations, disadvantaged, and are likely to be exposed to the broad range of factors experienced by disadvantaged groups in Australia, which includes prenatal, neonatal and postnatal risk factors potentially impacting on enamel development. Our study does not explain the findings of relative protection from dental caries among boys, after controlling for age and plaque levels, in multivariable analyses. It can only be speculated upon as to whether boys in our study were less exposed than girls to other unmeasured caries promoting factors, such as the extent of sugar sweetened beverage consumption.

The study findings reflect the higher level of caries experience among Australian Indigenous children reported elsewhere.[32–34] By restricting caries experience to those lesions with ICDAS score 4 or greater means that only the more severe lesions likely to require restorative interventions were included and potentially underestimate the caries prevalence and caries experience. The finding in our study of the association of higher caries experience in primary teeth with the presence of EDs supports the results that have been reported.[35] The finding that the presence of ED was independently associated with higher caries after controlling for age and plaque levels suggests that the EDs may place teeth at risk of caries independent of oral hygiene practices. This finding suggests that interventions beyond strategies to improve oral hygiene levels may be required to reduce the higher caries experience among Australian Indigenous children. The association of demarcated opacities at the person level and at the tooth level with caries experience might also be an indication that factors operating at the systemic level for the occurrence of EDs may play a role in caries risk beyond the

observed role of defects at the tooth level, and explain the higher caries experience of Indigenous peoples more broadly.

The study has limitations which should be taken into consideration when interpreting the findings. Few children examined in the examiner calibration training presented with dental EDs, thus, formal evaluation of examiner agreement was not possible. However, the risk of misclassification of EDs was minimized by the fact that both examiners were calibrated for evaluation of EDs for the Australian National Child Oral Health Survey of 2012–2014, in which EDs were evaluated using the DDE index for the WA-specific component. It is also possible that the use of index teeth may underestimate the number of teeth affected with EDs, and reduce the effect estimates. However, the aim of the study was not to estimate the overall prevalence of EDs, but to evaluate the association between the presence of EDs and dental caries, thus, the internal validity of the study is not compromised. There was no radiographic evaluation for dental caries and thus it is possible for there to be an underestimation of carious lesions. Also, the evaluation of EDs only considered the presence or absence of three ED types, without detailed evaluation of its extent or specific characteristics, such as colour or types of hypoplasia, thus, the specific characteristics of EDs and their association with caries occurrence were not able to be evaluated.

This cross-sectional study also does not enable a causal association between EDs and dental caries to be established, and the sample selection limits the generalizability of the findings to children in other settings. Although no measurements were taken of purported prenatal, neonatal or postnatal risk factors for EDs nor of any potential caries causing factors, the study findings suggest that the higher odds of dental caries experience, after controlling for oral hygiene status among Indigenous children, may be due to the presence of dental EDs. The findings suggest that strategies to improve access to timely care and improve the oral hygiene levels of Indigenous children should be implemented. The findings of sex differences in caries occurrence are of interest and the reason for the difference warrants further investigations. The overall findings point to the need for further longitudinal studies evaluating the incidence of caries and categorizing the prevalence of EDs and potential risk factors before the teeth have suffered the ravages of early childhood caries among Indigenous children.

CONCLUSIONS

In this study there was a 58% and 24% prevalence of EDs in the primary teeth of Indigenous children at the person and tooth level respectively. The presence of EDs was associated with higher odds of dental caries experience in affected children and teeth after controlling for oral hygiene level and community water fluoride levels.

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SUPPORTING INFORMATION

Additional Supporting Information may be found in the online version of this article:

Table S1. Tooth type by enamel defect type, n (%).

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