Community Dental Health (2016) **33,** 297–302
Received 2 February 2016; Accepted 2 May 2016

doi:10.1922/CDH_3937Claudia06

The relationship between maternal smoking during pregnancy and parental-reported experience of dental caries in Indigenous Australian children

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Objective: This study aimed to test the association between maternal smoking during pregnancy and parental-reported experience of dental caries in Indigenous Australian children. ***Methods:*** Data were from the Longitudinal Study of Indigenous Children (LSIC); a population-based cohort study in Australia. Participants were 1,687 Indigenous Australian children aged 5 or less. Biological, social and behavioural variables were tested using log-linear modelling with binomial regression to determine the association with parental-reported experience of dental caries. Markov Chain Monte Carlo methods were used for multiple imputation of missing data. ***Results:*** Overall 25.8% of Indigenous Australian children had dental caries as reported by a carer. In the multivariable model, increased prevalence of parental-reported caries was significantly associated with low maternal education levels (RR=1.60, 95%CI 1.17,2.20) and high sugar consumption (RR= 1.60, 95%CI 1.26,2.02). In the group of children whose mothers smoked tobacco during pregnancy, the association with parent-reported dental caries approached the threshold of significance, but was not significantly associated with caries status in children (RR=1.19, 95%CI 0.99,1.43). After multiple imputation, the most significant association was evident in children of the least educated mothers (RR=1.57, 95%CI 1.25,1.95), breastfeeding more than 12 months (RR=1.26, 95%CI 1.01,1.56), sweet intake more than 30% (RR=1.42, 95%CI 1.15,1.74) and 20-30% (RR=1.29 95%CI 1.04,1.59) and residing in outer regional (RR=1.56, 95%CI 1.19,2.05) or inner regional locations (RR=1.50, 95%CI 1.19,1.88). Mothers' tobacco smoking status showed a weak association with parent-reported dental decay (RR=1.42, 95%CI 1.20,1.68). ***Conclusion:*** This study suggests there is a weak association between maternal smoking during pregnancy and prevalence of parentally-reported dental caries in Indigenous Australian children.

Key words: *dental caries, children, smoking, pregnancy, Indigenous Australian*

Introduction

During the last 30 years, Indigenous Australians have experienced deterioration in their oral health (Hopcraft and Chow, 2007). Indigenous Australian children are twice as likely to develop dental decay as non-Indigenous Australian children. Data from The Child Dental Health Survey 2007 in Australia indicated that Indigenous Australian children aged 5 years had double the rate of dental decay compared with non-Indigenous Australian children, 63.1% and 31.1% respectively (Jamieson *et al*., 2007a). Therefore, particular concerns have been raised that Indigenous Australian children are at higher risk of developing early childhood caries (ECC). A number of underlying risk factors are of concern; experience of colonisation, biological factors, socio-economic characteristics and parental behaviour (Jamieson *et al*., 2007b).

There is increasing interest in dental research on how tobacco smoking can negatively impact children's oral health, especially caries development. Williams and colleagues were one of the earliest groups to expose the effects of parental smoking practices on caries in pre-school children. They found that maternal smoking practices increase caries susceptibility in children (Williams *et al*., 2000). Other examples exploring the relationship between smoking and dental caries in children have considered second-hand smoke to be a risk factor for ECC (Aligne *et al*., 2003; Haniokaa *et al*., 2008; Leroy *et al*., 2008; Shenkin *et al*., 2004; Tanaka *et al*., 2009; 2010). In a systematic review, Hanioka and colleagues concluded that the strength of this association was weak to moderate (Hanioka *et al*., 2011). Recent studies indicate that there is a significant association between postnatal second-hand smoking exposure in the household and dental caries (Tanaka *et al*., 2015a;b; Nakayama and Mori, 2015). The most significant factor involved in this association is maternal smoking. The reason is likely because mothers generally spend more time with their children than other family members (Nakayama and Mori, 2015).

A cross-sectional study in Japan showed a positive association between maternal smoking during pregnancy and a significant increased prevalence of dental decay in 3-year-old Japanese children (Tanaka *et al*., 2009). A cohort study of Italian children aged 24-30 months also indicated a relationship between smoking during pregnancy and high prevalence of caries in children (Majorana *et al*., 2014). The researchers found that smoking 5 or more cigarettes/day while pregnant exposed the unborn child to an increased risk of caries development in early childhood (Majorana *et al*., 2014). Other studies have supported this association (Schroth *et al*., 2013; Tanaka *et al*., 2015a).

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A recent cross-sectional Japanese study in 3-year-old children has suggested that the risk of dental decay in children is significant if pregnant women smoke during the first trimester of pregnancy (Tanaka *et al*., 2015a). Some studies could not establish whether the increased risk of caries in children is the result of smoking during pregnancy, after pregnancy or both (Lida *et al*., 2007).

Perinatal insults can adversely affect deciduous teeth that are forming intrauterine (Caufield *et al*., 2012). It is well documented that children with low birth weight and low gestational age are at higher risk of developing enamel defects (Caufield *et al*., 2012; Vello *et al*., 2010). Rates of low birth weight and premature birth are high in Australian Indigenous populations (Laws and Hilder, 2008). Smoking during pregnancy is considered one of the main contributors to these conditions (Laws and Hilder, 2008). These negative impacts result in hypoplastic defects of enamel that promote early colonisation of the deciduous teeth by cariogenic bacteria, for example *Streptococcus mutans* (Caufield *et al*., 2012). However, it is important to highlight that a diet high in sugar must be present for enamel hypoplasia to progress to decay (Caufield *et al*., 2012). These types of events are strongly associated with low-socioeconomic status (Caufield *et al*., 2012).

In a 2006 population survey, the prevalence of smoking during pregnancy in Indigenous Australian women was 52% (Laws and Hilder, 2008). We postulate that there is a relationship between maternal smoking during pregnancy and dental caries in Indigenous Australian children. Therefore, the aim of this research was to examine whether maternal smoking during pregnancy was associated with an increased prevalence of dental caries in Indigenous Australian children.

Methods

We used a population-based cohort design. This study was conducted with data from the Longitudinal Study of Indigenous Children (LSIC), which collected information about Indigenous Australian children through self-reported questionnaires. LSIC is an accelerated, cohort cross-sequential study that uses two different cohort groups of Indigenous Australian children. One group is the Baby Cohort that includes Indigenous children aged from 6 months to 18 months, and the second group, the Child Cohort that includes Indigenous children from 3½ years to 5 years. A cluster sampling technique was used for LSIC to select geographic sites. Eleven sites were chosen based on Aboriginal and Torres Strait Islander people concentrations, ensuring equal representation of urban and rural areas. The sample was designed to provide 150 children for each of the 11 sites; however, in some places it was difficult to reach the number due to small population size. Reasons for non-participation were relocation of the families or participants deciding to withdraw from the study (Kneebone *et al*., 2012).

LSIC received ethical approval from the Australian Government Department of Health and Ageing Departmental Ethics Committee, which was the nominated Human Research Ethics Committee for the study. Permission to use the LSIC dataset was obtained from the Department of Social Science under the University of Adelaide's organisational licence. No ethical approval was required for this secondary analysis of existing data.

For the purposes of this study, only data from Waves 1 to 5 were included. Eligible Indigenous Australian children for the present study were LSIC participants aged 5 or less to limit the analysis to children with deciduous dentition only. Only information from the primary caregiver was considered. A flow chart showing the selection of participants for this study is presented in Figure 1. A total of 1,687 children were included in the analysis.

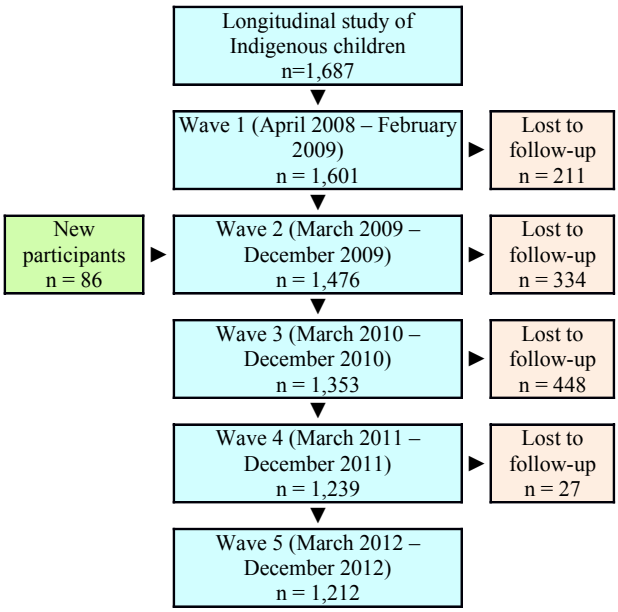

Figure 1. *Flow chart of combined child and baby participating cohorts from the LSIC Waves 1 to 5 selected for this study*

To identify if maternal smoking during pregnancy is a predictor of dental caries in Indigenous Australian children, the outcome variable was parental reporting of dental decay. Smoking during pregnancy was the main explanatory variable. Other independent variables were cohort, gender, breast feeding duration, average percentage of sweet food/beverage intake over a period of 5 years, mother's educational level, family income status and location. A composite variable was constructed to elicit the combination of factors that might contribute to caries development – birth weight and gestational age. It was deemed necessary to create this composite variable due to the literature suggesting a close relationship between a series of events that lead to dental decay.

The outcome variable (parental-reported decay) was assessed by the questions 'Has *child's name* ever had any of the following problems with (his/her) teeth or gums?' and 'Since the interview last year, has *child's name* had any of the following problems with (his/her) teeth or gums? Any cavities, holes or tooth decay, teeth pulled out because of decay' and dichotomised into two groups, yes/no. The exposure variable was also dichotomised into two categories, yes/no and ascertained by the question 'After finding out you were pregnant with *child's name* did you smoke any cigarettes during the pregnancy?'

Age was classified into two categories, Baby Cohort (6 to 18 months) and Child Cohort (3½ to 5 years), encompassing classification selected for this category in the LSIC. Low birth weight and preterm infants were those children born less than 2500 grams and with less than 37 weeks of gestation respectively. To assess breastfeeding duration, the answer to the question 'How old was *child's name* when (s/he) completely stopped being breastfed?' was used. The classification of this category was based on a 2015 systematic review that evaluated the association between breastfeeding and risk of dental caries. After analysis of eligible studies, the authors concluded that prolonged breastfeeding beyond 12 months exposed children to an increased risk of developing dental decay (Tham *et al*., 2015). Therefore, breastfeeding duration comprised two groups: 'breastfed 12 months or less' and' breastfed beyond 12 months'. As the actual value of sugar consumption could not be measured directly, aggregated nutrition information on fresh fruit and vegetables, grains, protein foods, dairy and sweet food consumption was used to estimate the average percentage of children's energy intake (including snack-savoury and sweet food, sweetened carbonated drinks) across the study duration. The percentage of sweet consumption was divided into three categories (<20%, 20-30% and >30%). For birth mother characteristics, education was categorised into two categories ('high school or less' and 'trade or university'). Family income status was evaluated based on financial strain information assessed by the question 'Which words best describe your family's money situation?' This variable was collapsed to four categories, 'low' (we run out of money before payday or we are spending more money than we get), 'medium' (we have just enough money to get us through to the next pay day or there is some money left over each week but we just spend it), 'high' (we can save a bit every now and then or we can save a lot) and 'fluctuating'. The fluctuating category was considered to reflect changes in family's finances across the five Waves studied. For the purpose of the present study the Australian Standard Geographical Classification was used and collapsed to form four categories: 'Remote/Very remote', 'Outer regional', 'Inner regional' and 'Major city'.

Statistical analysis was performed using SAS v.9.3 (SAS Institute Inc., Cary, NC, USA). The variables of interest were measured to determine the distribution of the study participants and the prevalence of parent-reported caries. Bivariate analysis was conducted to determine associations between the outcome variable (parent-reported dental caries) with the variables of interest. Unadjusted analysis was carried out to obtain the crude relative risk (RR) and 95% confidence interval (CI). An exploratory Log–linear model was tested with binomial regression to analyse associations between all variables. The adjusted relative risk (RR) and 95% confidence interval (CI) were calculated for children with parental-reported caries. The Pearson Chi-square test was used to test the model fit. Because tobacco smoking and education are both socially patterned, we examined effect modification by stratification and the Breslow-Day test. No effect modification was observed. The Markov Chain Monte Carlo with the fully conditional method (FCS) was used to generate multiple imputed (MI) data. The function of 'Proc mianalyze' was used as the diagnostic statistic to examine if it was suitable for using the MI model. All variables, excluding the outcome variable, with more than 3% missing values were imputed (10 iterations).

Results

Over one-quarter of children (25.8%) had parentally-reported dental caries. The prevalence of parent-reported caries at each time point is presented in Table 1. The prevalence of parent-reported dental decay increased from 10.6% in the Wave 1 to 31.4% in Wave 5 (same children in each wave).

Table 1. Number, mean (SD) and % (95%CI) of dental decay in from the LSIC project, 2008-2012

Wave	*(year)*	*N*	*Age (months) Mean (SD)*	*% of decay (95%CI)*
1	(2008)	1,601	31 (18)	10.6 (9.0,12.1)
2	(2009)	1,476	43 (18)	15.7 (13.8,17.5)
3	(2010)	1,353	55 (18)	22.2 (20.0,24.4)
4	(2011)	1,239	67 (18)	24.4 (22.0,26.8)
5	(2012)	1,212	79 (18)	31.4 (28.8,34.1)

The data in Table 2 indicates that among 1,687 children, 985 (58.4%) were from the Baby Cohort and 702 (41.6%) from the Child Cohort. A total of 857 were boys and 830 girls. Around 4% of the children had low birth weight and were born prematurely. In terms of nutrition, 79% of the children had been exclusively or partly breastfeed, while 19% of participants were fed something other than breast milk. Approximately 13% of children were breastfed beyond 12 months. With regard to sugar consumption the highest percentage of children was evident in the group with more than 30% sugar intake, with almost 35% of the population reported in this category. Only 16% of mothers had completed a higher education course while the remaining 71% had completed high school or less. Almost half (48%) the mothers reported smoking throughout the pregnancy.

Bivariate analysis is shown in Table 2. Indigenous children who were exposed to tobacco smoke during pregnancy had a higher prevalence of parent-reported dental decay than children whose mothers did not smoke during the pregnancy (31% and 21% respectively). The association with maternal smoking during pregnancy was statistically significant. There was not a significant difference in relation to gender, cohort and family income status. Children breastfed beyond 12 months had a higher prevalence of parent-reported caries (31%) when compared with children breastfed ≤12 months (25%). The confidence intervals overlap, hence the difference is not statistically significant. In relation to sweet intake, the prevalence of parent-reported caries was higher in children who reported consumption of 30% or more of sugar-containing products (31%) than in children who reported low sweet intake (22%), 43% higher as indicated in the unadjusted model. The greatest difference was in the education groups. Participants having mothers with high school or less education experienced almost twice the prevalence of parent-reported dental decay than their counterparts with mothers who had obtained tertiary education (29% and 16% respectively). The unadjusted model indicates the outcome was 1.8 times as great as in Indigenous children having mothers with higher education or more. With regard to geographic distribution, the prevalence of parent-reported dental caries was unequal in the four groups. The highest prevalence was obtained in the outer regional area (46%) and the lowest in the major cities (34%).

Table 2. Sample distribution, proportion of caries according to study variables and RR (95%CI) for associations between dental caries with study variables (unadjusted model, adjusted model and multiple imputation results)

		Sample		*Proportion of caries*	*Unadjusted models*[b]	*Adjusted model (CC*[c]*)*	*Adjusted Model (MI*[d]*)*
		N	*(%*[a]*)*	*% (95%CI)*	*RR (95 % CI)*	*RR (95 % CI)*	*RR (95 % CI)*
Children's characteristics	Baby cohort	985	(58.4)	24.4 (21.7, 27.1)	0.87 (0.74, 1.03)	0.90 (0.74, 1.09)	0.87 (0.73, 1.03)
	Child cohort	702	(41.6)	27.9 (24.6, 31.2)	1.00	1.00	1.00
	Boy	857	(50.8)	26.1 (23.2, 29.1)	1.02 (0.87, 1.20)	1.03 (0.86, 1.23)	1.06 (0.90, 1.24)
	Girl	830	(49.2)	25.5 (22.6, 28.5)	1.00	1.00	1.00
	Low birth weight & preterm	75	(4.4)	20.0 (10.7, 29.3)	0.78 (0.49, 1.24)	0.71 (0.41, 1.20)	1.00 (0.71, 1.42)
	Normal birth weight & term	1503	(89.1)	25.6 (23.4, 27.8)	1.00	1.00	1.00
	Breastfeeding >12 months	220	(13.0)	30.9 (24.8, 37.1)	1.23 (0.99, 1.53)	*1.30 (1.03, 1.66)	*1.26 (1.01, 1.56)
	Breastfeeding ≤12 months	1441	(85.4)	25.1 (22.9, 27.4)	1.00	1.00	1.00
	Sweet intake >30%	586	(34.7)	31.1 (27.3, 34.8)	**1.43 (1.16, 1.76)	**1.60 (1.26, 2.02)	**81.42 (1.15, 1.74)
	Sweet intake 20-30%	507	(30.1)	28.4 (24.5, 32.3)	*1.31 (1.05, 1.62)	*1.31 (1.02, 1.68)	*1.29 (1.04, 1.59)
	Sweet intake <20%z	492	(29.2)	21.7 (18.1, 25.4)	1.00	1.00	1.00
	Decay Yes	436	(25.8)				
	Decay No	1251	(74.2)				
Mothers' characteristics	Education, High school or less	1,192	(70.7)	29.2 (26.6, 31.8)	***1.87 (1.39, 2.51)	**1.60 (1.17, 2.20)	**1.57 (1.25, 1.98)
	Education, Trade or University	262	(15.5)	15.6 (11.2, 20.1)	1.00	1.00	1.00
	Family income, Low	182	(10.8)	27.5 (20.9, 34.0)	1.13 (0.79, 1.61)	0.92 (0.59, 1.42)	0.74 (0.51, 1.08)
	Family income, Medium	612	(36.3)	27.5 (23.9, 31.0)	1.13 (0.83, 1.52)	1.03 (0.72, 1.45)	0.83 (0.61, 1.12)
	Family income, Fluctuation	648	(38.4)	25.0 (21.7, 28.3)	1.03 (0.76, 1.38)	0.99 (0.71, 1.39)	0.83 (0.62, 1.10)
	Family income, High	164	(9.7)	24.4 (17.7, 31.0)	1.00	1.00	1.00
	Location, Remote/very remote	190	(11.3)	37.9 (30.9, 44.9)	1.02 (0.71, 1.47)	1.12 (0.73- 1.70)	0.94 (0.65, 1.34)
	Location, Outer regional	267	(15.8)	46.4 (40.4, 52.5)	***1.73 (1.32, 2.26)	**1.75 (1.30, 2.35)	**1.56 (1.19, 2.05)
	Location, Inner regional	808	(47.9)	45.3 (41.9, 48.7)	***1.66 (1.32, 2.09)	**1.44 (1.13, 1.84)	**1.50 (1.19, 1.88)
	Location, Major city	422	(25.0)	34.4 (29.8, 38.9)	1.00	1.00	1.00
	Smoking in pregnancy	805	(47.7)	30.9 (27.7, 34.1)	***1.46 (1.24, 1.73)	1.19 (0.99, 1.43)	***1.42 (1.20, 1.68)
	Not smoking in pregnancy	852	(50.5)	21.1 (18.4, 23.9)	1.00	1.00	1.00

Note:
***p-value <0.0001
**p-value <0.01
* p-value <0.05
[a], Percentages may not sum to 100% due to missing data
[b], Each explanatory variable has its own model: in total, there are 9 unadjusted models
[c], Complete case
[d], Multiple imputations

In the adjusted model (Table 2), low maternal education, breast feeding more than 12 months, residing in inner or outer regional locations, and sugar intake >30% were significantly associated with parent-reported dental caries. After multiple imputation (10 iterations) (Table 2), sugar consumption in the group with more than 30% of sugar intake remained significant but was slightly attenuated (RR= 1.42, 95%CI 1.15,1.74, p=0.0009). The most significant association was for children of mothers with lower education levels. Based on the imputed data, the proportion of parent-reported dental decay was 1.5 times as great as in Australian Indigenous children having mothers with higher education or more.

In the group of children whose mothers smoked during pregnancy the association with parent-reported dental caries approached but did not reach the threshold of significance after adjusting for other variables in the model. The RR was 1.19 (95%CI 0.99,1.43, p=0.0696). After multiple imputation, children whose mothers smoked during pregnancy were more likely to experience parent-reported dental decay than children whose mothers did not smoke through pregnancy (RR=1.42; 95%CI 1.20,1.68, p<0.0001). After multiple imputation, the most significant associations were evident among children who were breastfed more than 12 months (RR=1.26, 95%CI 1.01,1.56, p=0.0396) and who resided in outer (RR=1.56, 95%CI 1.19,2.05, p=0.0013) or inner (RR=1.50, 95%CI 1.19,1.88, p=0.0005) regional locations.

Discussion

This study suggests a weak association between maternal smoking during pregnancy and parent-reported dental caries in Australian Indigenous children. Additionally, this study shows that sugar consumption and low maternal educational levels were the strongest factors associated with the development of parent-reported dental caries in this population.

A major strength of this study is that it is the largest dental study that has been conducted among pre-school Indigenous Australian children with nationally-geographic representation. Hopefully, future Waves will include a dental examination. Limitations include the use of self-reported data that were not validated with clinical outcomes. Also the assessment of tobacco smoking was only during pregnancy, with no consideration of the smoking environment to which the infant was exposed in his/her first months of life. However, it is important to acknowledge that parental reporting has been found to be a reliable method of obtaining information when logistics and costs prohibit the collection of clinical data (Jamieson *et al*., 2004). The sample is not representative as no randomisation procedures were used in the initial recruitment phases. Sampling bias could be present due to the non-probability sampling method used. An important aspect of this approach is that the participants tended to share similar characteristics and therefore the ability to generalise the results from the sample to the entire Indigenous population is limited (Magnani *et al*., 2005).

The present study provides further evidence that maternal smoking during pregnancy may be associated with caries prevalence in children. To our knowledge this is the first study that has examined this relationship among Indigenous Australian children. Based on the literature, our results are in agreement with studies carried out in other countries (Lida *et al*., 2007; Majorana *et al*., 2014; Schroth *et al*., 2013; Tanaka *et al*., 2009; 2015a). Our findings corroborate evidence from Jamieson and colleagues who have suggested that consumption of sugar-containing food is one of the most probable explanations for high caries prevalence in Indigenous Australian children (Jamieson *et al*., 2007a). Additionally, and consistent with previous studies, our findings indicate that children whose mothers have low educational levels are at a significantly increased risk of developing dental decay (Seow, 2012).

Our results also provide evidence for a relationship between duration of breastfeeding and dental caries. A previous systematic review of this association showed that prolonged breastfeeding beyond 12 months exposes children to an increased risk of developing dental decay (Tham *et al*., 2015). With regard to geographic distribution, the highest prevalence of dental decay was observed in the outer regional area and was lowest in the major cities. In agreement with our results, an Australian study reported that regional-dwelling Indigenous children had higher dental disease prevalence than Indigenous children living at a state level (Jamieson *et al*., 2007c).

The most plausible explanation of the relationship between maternal smoking and dental caries in Indigenous Australian children might be linked to developmental defects of enamel. Tobacco smoking during pregnancy contributes to the development of enamel defects (Vello *et al*., 2010). This kind of defect on tooth structure increases the risk of dental decay (Salanitri and Seow, 2013).

However, it is important to highlight that a diet high in sugar must be present for enamel hypoplasia to progress to ECC (Caufield *et al*., 2012). This association has been substantiated among Indigenous children and hence it has been suggested that enamel hypoplasia, a diet high in sugar and dental decay are interrelated (Seow and Pascoe, 1994).

Further studies should be conducted to explore other risk factors that may contribute to caries development in children exposed to maternal smoking during pregnancy, for example enamel defects. Taking advantage of the longitudinal nature of the study, future studies could also use causal analyses to examine etiologic hypothesis from observational data (Robins *et al*., 2000).

In conclusion, this study found a positive but weak association between maternal smoking during pregnancy and prevalence of parent-reported dental decay in Australian Indigenous children. The role of recognised risk factors for dental decay was confirmed, such as sugar intake and mothers' low education level

Acknowledgements

We thank the Department of Social Services for access to Longitudinal Study of Indigenous Children data, the research team from the Australian Research Centre for Population Oral Health and especially the Australian Indigenous families who agreed to be part of the study.

This paper uses unit record data from the Longitudinal Study of Indigenous Children (LSIC). LSIC was initiated and is funded and managed by the Australian Government Department of Social Services (DSS). The findings and views reported in this paper, however, are those of the authors and should not be attributed to DSS or the Indigenous people and their communities involved in the study.

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