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# Heavy maternal alcohol consumption and cerebral palsy in the offspring

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## PUBLICATION DATA

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## ABBREVIATIONS

FAS Fetal alcohol syndrome  
ICD International Classification of Diseases

**AIM** The aim of this study was to investigate the association between heavy maternal alcohol consumption and pre- peri- and postneonataly acquired cerebral palsy (CP).

**METHOD** The records of all mothers with an International Classification of Diseases, revision 9 or 10 (ICD-9/-10) alcohol-related diagnostic code, indicating heavy alcohol consumption, recorded on population-based health, mental health, and drug and alcohol data sets from 1983 to 2007, and their children were identified through the Western Australian Data-linkage System. This 'exposed' cohort was frequency matched with mothers without an alcohol-related diagnosis and their offspring (comparison group). Cases of CP were identified through linkage with the Western Australia CP Register. Analyses were undertaken using multivariate logistic regression.

**RESULTS** There were 23 573 live births in the exposed group (58.6% non-Aboriginal; 41.4% Aboriginal) and 292 cases of CP. The odds of pre/perinatally acquired CP were elevated for children of non-Aboriginal mothers with an alcohol-related diagnosis recorded during pregnancy (adjusted odds ratio 3.32; 95% confidence interval [CI] 1.30–8.48) and for Aboriginal children when an alcohol-related diagnosis was recorded up to 12 months before the mother's pregnancy (adjusted odds ratio 2.49; 95% CI 0.99–6.25). Increased odds of postneonataly acquired CP following any alcohol-related diagnosis were found for non-Aboriginal children (adjusted odds ratio 7.92; 95% CI 2.23–28.14).

**INTERPRETATION** These results suggest that heavy maternal alcohol consumption is a direct cause of pre/perinatally acquired CP, and an indirect cause of postneonataly acquired CP, in non-Aboriginal children. The lack of an association for Aboriginal children requires further investigation but may be due to under ascertainment of alcohol use disorders during pregnancy and other aetiological pathways.

Maternal alcohol use disorders, indicating alcohol abuse, harmful use, or dependence,<sup>1</sup> are biological, psychological, and environmental risk factors for the offspring.<sup>2</sup> Children are at risk of being exposed to a number of interacting factors stemming from the environment and the family which increase the risk of developing adverse health and cognitive and psychosocial problems.

Heavy maternal alcohol use occurring during pregnancy increases the risk of a range of fetal effects, many of which are included under the umbrella term fetal alcohol spectrum disorders.<sup>3</sup> Cerebral palsy (CP) is among the possible diagnostic features of structural or neurological brain damage related to prenatal alcohol exposure.<sup>4,5</sup> However, the evidence to support this association is not strong.<sup>6</sup> A handful of studies have reported a prevalence of CP in children with fetal alcohol syndrome of between 2% and 10%, but these are based on small numbers<sup>7–10</sup> and many studies lack scientific rigour.<sup>6</sup>

The prevalence of CP has remained relatively stable over the past decade<sup>11</sup> and, although the majority (82–91%) of cases of CP are thought to be due to prenatal or perinatal factors, few prenatal causes have been identified.<sup>12</sup> Therefore, identifying modifiable risk factors is a priority; however, CP is a rare condition<sup>11,12</sup> and health professionals do not routinely ask mothers about their alcohol use during pregnancy.<sup>13</sup> Therefore, research into the relationship between prenatal alcohol exposure and CP is difficult to conduct.

The aim of this study was to investigate the association between heavy levels of maternal alcohol use before, during, and after pregnancy and CP in the children of these mothers, using a population-based cohort.

## METHOD

### Participants

The population at risk were all women who gave birth in Western Australia between 1983 and 2007. All mothers with

an alcohol-related diagnosis, used as a proxy for heavy levels of alcohol consumption, were identified along with their children through linked, population-based data from the Western Australian Data-linkage System.<sup>14</sup> The exposed cohort comprised all mothers with an alcohol-related diagnosis (International Classification of Diseases, revision 9 or 10 [ICD-9 and/or ICD-10]) recorded on one or more of the Western Australian Hospital Morbidity data sets (which include information on hospital in-patient admissions), the Midwives Notification System, or the Mental Health Outpatients data set, or who had been treated for an alcohol problem at the Perth-based government Drug and Alcohol Office treatment services, and all offspring whose birth was recorded on the Midwives Notification System (1983–2007). The ICD-9 and/or ICD-10 codes included alcohol-related mental and behavioural disorders, an alcohol-related disease with a 100% attributable fraction, and other alcohol codes. (Appendix 1, supporting information published online).

The exposed cohort was frequency matched on maternal age within maternal race (Aboriginal: 2–3 unexposed: 1 exposed; non-Aboriginal: 3–4 unexposed: 1 exposed) and year of birth of the child to mothers in the Midwives Notification System without an alcohol-related diagnosis (ICD-9 and/or ICD-10) recorded on the Hospital Morbidity data sets or the Mental Health Outpatients or Drug and Alcohol Office data sets. The selection of the comparison group also excluded women with an ICD-8 alcohol diagnostic code occurring in the Hospital Morbidity data sets from 1970 to 1983 and the Mental Health Outpatients data set between 1966 and 1983. The records of the selected women and all their children whose births are recorded on the Midwives Notification System are referred to as the comparison group.

Records from the exposed and comparison cohorts were linked by the Western Australia Data-linkage Branch using probabilistic matching,<sup>14</sup> and de-identified data files were provided to the researchers. The data from both cohorts were linked with the Western Australian CP Register, which includes all cases of CP in Western Australia occurring from 1956 onwards.<sup>11,12</sup> Cases are actively ascertained from multiple sources, and all information is updated at 5 years of age and classified as either pre-, peri-, or postneonally acquired.<sup>11</sup>

Children in both cohorts with a diagnosis of fetal alcohol syndrome (FAS) were identified through linkage with the Western Australian Birth Defects Register, which collects information for the Western Australia population on birth defects diagnosed in stillbirths, terminations of pregnancy, and live births up to 6 years of age, using multiple sources of ascertainment.<sup>15</sup> The Birth Defects and CP Registers are now combined and known as the Western Australian Register of Developmental Anomalies.

### Statistical analysis and covariates

Maternal alcohol-related diagnosis was coded into (1) a binary variable (yes/no), and (2) the timing of recording of the alcohol-related diagnosis in relation to pregnancy using a hierarchical coding with the order as follows: (a) any record during preg-

### What this paper adds

- Children of non-Aboriginal and Aboriginal mothers with an alcohol-related diagnosis are at increased risk of pre/perinatally acquired CP.
- Increased odds of postneonally acquired CP were observed only in children of non-Aboriginal mothers.
- These results suggest that a maternal alcohol use disorder is both a direct and indirect risk factor for CP in the offspring.

nancy, which may also include an alcohol-related diagnosis before and/or after pregnancy; for women without a diagnosis during pregnancy, the groups were (b) up to 1 year before pregnancy and may include exposure of more than 1 year before pregnancy or any exposure after pregnancy, (c) up to 1 year after pregnancy and may include exposure for more than 1 year before or after pregnancy, (d) more than 1 year before pregnancy and may include exposure more than 1 year after pregnancy, and (e) more than 1 year after pregnancy (Table I). The pregnancy period was estimated by subtracting gestational age<sup>16</sup> at birth from date of birth to give the date of conception.

The proportion of CP cases for the exposed and comparison groups was calculated for all CP and for pre-, peri-, and postneonally acquired CP for Aboriginal and non-Aboriginal children separately and presented per 1000 live births, along with the odds ratio and with 95% CIs (Table II). The proportion of CP in children with FAS is presented per 1000 children with FAS.

Multivariate logistic regression analysis was used to examine the odds of CP in live births of mothers with an alcohol-related diagnosis compared with children of mothers in the comparison group. All analyses were stratified by Aboriginal status and adjusted for year of birth and maternal age used in the frequency matching. To examine whether the relationship between exposure and CP was causal or due to postnatal environmental factors, the analyses were repeated separately for pre-, peri-, and postneonally acquired CP. Postneonatal analyses included only children alive at 29 days of age. Preterm birth and intrauterine growth restriction are known to increase the risk of CP<sup>17</sup> and may be associated with prenatal alcohol exposure,<sup>18</sup> so the binary analyses examining pre/perinatally acquired CP were further restricted to examine the association for (1) term births, defined as births at 37+ weeks' gestation (non-Aboriginal,  $n=4573$  [8.5%] excluded; Aboriginal,  $n=5193$  [17%] excluded) and (2) appropriate fetal growth (non-Aboriginal,  $n=5677$  [10.5%] excluded; Aboriginal,  $n=5980$  [19.7%] excluded). The proportion of optimal birthweight was used as a measure of fetal growth, where optimal birthweight was determined after taking into account infant sex, gestational age, maternal height, and parity.<sup>19</sup> A proportion of the optimal birthweight score on the 10th centile or higher was used to define appropriate fetal growth.

Maternal characteristics, behaviours, and pregnancy complications that were considered potential confounders were entered individually into the univariate model and those that altered the univariate odds ratios (ORs) by 20% or greater were included as possible confounders in the multivariate logistic analyses. Variables assessed as possible confounders included marital status, parity, maternal illicit drug use (any

**Table I:** Coding of timing of maternal alcohol-related diagnosis in relation to pregnancy

	Timing of maternal alcohol-related diagnosis in relation to pregnancy				
	During	≤1y before	≤1y after	>1y before	>1y after
During pregnancy	Yes	Possible	Possible	Possible	Possible
≤1y before pregnancy	No	Yes	Possible	Possible	Possible
≤1y after pregnancy	No	No	Yes	Possible	Possible
>1y before pregnancy	No	No	No	Yes	Possible
>1y after pregnancy	No	No	No	No	Yes

**Table II:** Maternal demographic characteristics at each live birth 1980–2007, *n* (%)

Characteristic	Non-Aboriginal ( <i>n</i> =53 955)		Aboriginal ( <i>n</i> =30 409)	
	Exposed ( <i>n</i> =13 807)	Comparison ( <i>n</i> =40 148)	Exposed ( <i>n</i> =9766)	Comparison ( <i>n</i> =20 643)
<b>Maternal age (y)</b>				
<20	1676 (12.1)	4755 (11.8)	2587 (26.5)	5421 (26.3)
20–24	4089 (29.6)	11 764 (29.3)	3232 (33.1)	6909 (33.4)
25–29	4062 (29.4)	11 944 (29.7)	2245 (23.0)	4743 (23.0)
30–34	2689 (19.5)	7869 (19.6)	1202 (12.3)	2520 (12.2)
35–39	1086 (7.9)	3211 (8.0)	448 (4.6)	933 (4.5)
40+	205 (1.5)	605 (1.5)	52 (0.5)	117 (0.6)
<b>Marital status</b>				
Married	10 168 (73.9)	34 700 (86.5)	5680 (58.6)	13 455 (65.5)
Never married	3065 (22.3)	5056 (12.6)	3807 (39.2)	6726 (32.8)
Single <sup>a</sup>	520 (3.8)	356 (0.9)	213 (2.2)	355 (1.7)
Illicit drug use <sup>b</sup>	2891 (20.9)	551 (1.4)	916 (9.4)	436 (2.1)
<b>Socio-economic status</b>				
Most advantaged >10%	557 (4.0)	2711 (6.8)	24 (0.2)	108 (0.5)
10 to ≤25%	1215 (8.8)	5071 (12.6)	83 (0.8)	358 (1.7)
25 to ≤50%	2507 (18.2)	8623 (21.5)	471 (4.8)	1404 (6.8)
50 to ≤75%	3372 (24.4)	9458 (23.6)	1274 (13.0)	3410 (16.5)
75 to ≤90%	2595 (18.8)	5993 (14.9)	1831 (18.7)	4448 (21.6)
10%	1983 (14.4)	3478 (8.7)	2903 (29.7)	6110 (29.6)
Unknown	1578 (11.4)	4814 (12.0)	3180 (32.6)	4784 (23.2)
<b>Pregnancy complications</b>				
Threatened abortion <20wks	756 (5.5)	2008 (5.0)	154 (1.6)	386 (1.9)
Threatened preterm labour	288 (2.1)	431 (1.1)	261 (2.7)	401 (1.9)
Antepartum haemorrhage/placenta abruption	145 (1.1)	226 (0.6)	69 (0.7)	111 (0.5)
<b>Health service region</b>				
Perth metro	9891 (71.6)	28 986 (72.2)	2874 (29.4)	6631 (32.1)
Rural/remote	3858 (27.9)	10 893 (27.3)	6873 (70.4)	13 956 (67.6)
Other	58 (0.4)	269 (0.7)	19 (0.2)	56 (0.3)
<b>Maternal smoking<sup>c</sup> (1998–2007)</b>	<i>n</i> =5531 2757 (49.8)	<i>n</i> =16 255 3266 (20.1)	<i>n</i> =3556 2410 (67.5)	<i>n</i> =8329 3764 (45.2)

<sup>a</sup>Includes widowed or divorced. <sup>b</sup>Any ICD-9 and/or ICD-10 code for illicit drugs present on either the Hospital Morbidity or Mental Health Outpatients data sets, or illicit drug use recorded on the Drug and Alcohol Office data set. <sup>c</sup>Collected on the Midwives Notification System since 1998.

ICD-9/ICD-10 code for illicit drugs present on either the Hospital Morbidity data sets or the Mental Health Outpatients data sets, or illicit drug use recorded on the Drug and Alcohol Office data set), threatened abortion at less than 20 weeks, threatened preterm labour, antepartum haemorrhage–placental abruption, socio-economic status at time of birth based on Australian census data collected at the census district level (approximately 200 households)<sup>20</sup> stratified into quintiles, and maternal smoking during pregnancy. The only potential confounding variables to alter the OR by 20% or more, and hence included in the multivariate analyses, were illicit drug use for all CP and pre/perinatally acquired CP and socio-economic status for postneonatally acquired CP in non-

Aboriginal children. No variables altered the odds by more than 20% in the analyses in Aboriginal children. Logistic regression analyses were conducted using SPSS version 17.0 (SPSS Inc., Chicago, IL, USA). Results are presented as ORs with 95% CIs.

The population-attributable fraction and 95% CI was calculated for significant findings for exposure during pregnancy<sup>21</sup> using whole Western Australian population numbers for the comparison numerators and denominators.

Ethics approval for the conduct of this study was granted by the Princess Margaret Hospital Research Ethics Committee, the Western Australia Department of Health Confidentiality of Health Information Committee (now called the Health

Research Ethics Committee), the Western Australia Aboriginal Health Information Ethics Committee, and the Curtin Human Research Ethics Committee. A requirement of Western Australia ethics committees is that researchers using linked data are not permitted to report numbers where the number of cases within a stratum is less than 5 to prevent the likelihood of identification of individuals.

## RESULTS

There were 84 364 children in the study; 53 955 (64.0%) were non-Aboriginal, of whom 13 807 (25.6%) were exposed, and 30 409 (36.0%) were Aboriginal, of whom 9766 (32.1%) were exposed (Table II). Mothers with an alcohol-related diagnosis were less likely to be married and more likely to use illicit drugs and smoke cigarettes than those in the comparison group, and the percentage of exposed mothers using illicit drugs was higher in non-Aboriginal than in Aboriginal mothers, while maternal smoking was more prevalent in non-Aboriginal mothers. Non-Aboriginal mothers were older than Aboriginal mothers, had more pregnancy complications, and were more affluent, with 31% of exposed and 41% of comparison mothers in the top 50% of socio-economic status compared with 6% and 9% (respectively) of Aboriginal mothers. Over 70% of non-Aboriginal mothers and 30% of Aboriginal mothers lived in Perth at the time of birth (Table II).

There were 292 children with CP, of whom 59 non-Aboriginal children were exposed and 96 were comparison children, and 39 Aboriginal children were exposed and 98 were comparison children. The proportion of all CP in non-Aboriginal offspring was 4.3 per 1000 for exposed and 2.4 per 1000 for comparison offspring, giving a ratio of 1.8 (95% CI 1.58–2.08), with a similar distribution for pre/perinatally acquired CP. In Aboriginal children, the proportion of all CP was 3.99 per 1000 in the exposed and 4.70 in the comparison groups, with a ratio of 0.84 (95% CI 0.70–0.98), with a similar proportion for pre/perinatally acquired CP. The ratio for postneonally acquired CP in non-Aboriginal children was 5.8 (95% CI 3.12–8.48) compared with 0.79 (95% CI 0.56–1.02) for Aboriginal children (Table SI, supporting information published online only). There were 124 live born children diagnosed with FAS on the Western Australian Register of Developmental Anomalies and nine had pre/perinatally acquired CP (72.6/1000 children with FAS; results not shown).

The timing of the alcohol-related diagnosis in relation to pregnancy for cases of CP is shown in Table SII (supporting information published online only). The majority of children had a mother with a diagnosis recorded more than 1 year after pregnancy (59.4% of non-Aboriginal and 63.2% of Aboriginal mothers), followed by a diagnosis more than 1 year before pregnancy (27.1% of non-Aboriginal and 16.7% of Aboriginal mothers). The percentage of Aboriginal mothers with a diagnosis recorded during pregnancy (8.5%) was over twice that of non-Aboriginal mothers (3.8%). There were six cases of CP (3.8%) in which the non-Aboriginal mother had an alcohol-related diagnosis recorded during pregnancy and for Aboriginal mothers five cases had a diagnosis recorded within the 12

months before pregnancy and fewer than five cases had a diagnosis recorded during pregnancy.

The severity of CP varied by ethnicity, with a higher percentage of non-Aboriginal children (52.6%) than Aboriginal children (30.7%) having either minimal or mild CP (classification based on clinical notes equates approximately to Gross Motor Function Classification System/Manual Ability Classification System levels I or II),<sup>22,23</sup> and there was little variation in the distribution of severity by exposure status. Causes were documented for all 63 children with postneonatal CP. Head injury was the cause in half (50.0%) of the non-Aboriginal and 22.7% of the Aboriginal children and infection for 22.2% of non-Aboriginal and over half (63.6%) of Aboriginal children. Causes of head injury were passenger or pedestrian motor vehicle accident ( $n=8$ ; 12.9%), non-accidental head injury ( $n=6$ ; 9.7%), and fall or other non-specified head injury ( $n=5$ ; 8.0%; results not shown).

The results of the multivariate logistic regression examining CP (yes/no) as the outcome variable and maternal alcohol diagnosis (yes/no) as the exposure variable showed a small increase in all CP for non-Aboriginal children (adjusted OR 1.40; 95% CI 0.97–2.02), with similar odds following stratification for term births and optimal birthweight (Table III). Adjusting for maternal smoking (1998–2007) did not alter this association by more than 20% (results not shown). An eightfold increased odds of postneonally acquired CP (adjusted OR 7.92; 95% CI 2.23–28.14) was observed for non-Aboriginal children. In Aboriginal children, there was no association between the binary alcohol variable and CP in any of the models (Table III). The population-attributable fraction for non-Aboriginal CP when a maternal alcohol-related diagnosis was recorded during pregnancy was 0.28 (95% CI –0.00002 to 0.70; results not shown).

Multivariate logistic regression examining the timing of maternal alcohol diagnosis in relation to pregnancy showed a threefold increased odds of non-Aboriginal CP when a diagnosis was recorded during pregnancy (adjusted OR 3.72 [95% CI 1.57–8.81]), which remained in the analyses examining pre/perinatally acquired CP (adjusted OR 3.32; 95% CI 1.30–8.48; Table IV). The increased odds for all CP when an alcohol diagnosis was recorded more than 1 year before pregnancy (adjusted OR 1.83; 95% CI 1.07–3.13) reduced when pre/perinatally acquired CP was examined (adjusted OR 1.51; 95% CI 0.83–2.73). Small numbers of CP in Aboriginal children in some alcohol-exposure groups limited investigation of the timing of maternal alcohol-related diagnoses. However, twofold increased odds of pre/perinatally acquired CP were observed when an alcohol-related diagnosis was recorded within 12 months of pregnancy (adjusted OR 2.49; 95% CI 0.99–6.25).

## DISCUSSION

This study demonstrates that a maternal alcohol-related diagnosis, indicating heavy maternal alcohol drinking, increases the risk of both pre/perinatally and postneonally acquired CP in non-Aboriginal children. The threefold increased odds of pre/perinatally acquired CP (adjusted OR 3.32; 95% CI

**Table III:** Adjusted odds ratio (and 95% confidence interval) of all cerebral palsy (CP) and pre/perinatally and postneonatally acquired cerebral palsy for children of mothers with an alcohol-related diagnosis, by Aboriginal status

All CP	Pre/perinatal <sup>a</sup>									
	All		Term births <sup>b</sup>		Optimal birthweight <sup>c</sup>		Postneonatal <sup>d</sup>			
Non-Aboriginal (n=53 955)										
Alcohol-related diagnosis										
No	1.00 <sup>e</sup>	1.00 <sup>f</sup>	1.00 <sup>e</sup>	1.00 <sup>f</sup>	1.00 <sup>e</sup>	1.00 <sup>f</sup>	1.00 <sup>e</sup>	1.00 <sup>f</sup>	1.00 <sup>e</sup>	1.00 <sup>g</sup>
Yes	1.79 <sup>e</sup>	1.40 <sup>f</sup>	1.51 <sup>e</sup>	1.21 <sup>f</sup>	1.63 <sup>e</sup>	1.37 <sup>f</sup>	1.62 <sup>e</sup>	1.31 <sup>f</sup>	4.34 <sup>e</sup>	7.92 <sup>g</sup>
	(1.29–2.48)	(0.97–2.02)	(1.06–2.15)	(0.81–1.80)	(1.04–2.56)	(0.83–2.27)	(1.10–2.39)	(0.85–2.02)	(1.45–12.98)	(2.23–28.14)
Aboriginal (n=30 409)										
No	1.00 <sup>e</sup>	– <sup>h</sup>	1.00 <sup>e</sup>	– <sup>h</sup>	1.00 <sup>e</sup>	– <sup>h</sup>	1.00 <sup>e</sup>	– <sup>h</sup>	1.00 <sup>e</sup>	– <sup>h</sup>
Yes	0.83 <sup>e</sup>	– <sup>h</sup>	0.87 <sup>e</sup>	– <sup>h</sup>	0.81 <sup>e</sup>	– <sup>h</sup>	0.80 <sup>e</sup>	– <sup>h</sup>	0.68 <sup>e</sup>	– <sup>h</sup>
	(0.57–1.20)		(0.56–1.37)		(0.43–1.53)		(0.44–1.43)		(0.28–1.68)	

<sup>a</sup>Live births. <sup>b</sup>37+ weeks' gestation. <sup>c</sup>Includes the top 90% of optimal birthweight. <sup>d</sup>Only children alive at 29 days are included in the denominator. <sup>e</sup>Adjusted for variables used in frequency matching: maternal age, and year of birth. <sup>f</sup>Adjusted for variables used in frequency matching and illicit drug use. <sup>g</sup>Adjusted for variables used in frequency matching and socio-economic status. <sup>h</sup>No potential confounder altered the odds by ≥20%.

**Table IV:** Adjusted odds ratio (and 95% confidence intervals) of cerebral palsy for non-Aboriginal and Aboriginal children by timing of maternal alcohol-related diagnosis in relation to pregnancy

Timing of alcohol-related diagnosis	All CP <sup>a</sup>		Pre/perinatally acquired CP <sup>a</sup>	
Non-Aboriginal (n=53 955)				
No alcohol-related diagnosis	1.00 <sup>b</sup>	1.00 <sup>c</sup>	1.00 <sup>b</sup>	1.00 <sup>c</sup>
During pregnancy	5.01 <sup>b</sup> (2.16–11.60)	3.72 <sup>c</sup> (1.57–8.81)	4.33 <sup>b</sup> (1.74–10.82)	3.32 <sup>c</sup> (1.30–8.48)
≤1y before pregnancy	<5	<5	<5	<5
≤1y after pregnancy	<5	<5	<5	<5
>1y before pregnancy	2.16 <sup>b</sup> (1.28–3.65)	1.83 <sup>c</sup> (1.07–3.13)	1.75 <sup>b</sup> (0.98–3.13)	1.51 <sup>c</sup> (0.83–2.73)
>1y after pregnancy	1.49 <sup>b</sup> (0.98–2.26)	1.11 <sup>c</sup> (0.70–1.76)	1.27 <sup>b</sup> (0.81–2.02)	0.98 <sup>c</sup> (0.59–1.62)
Aboriginal (n=30 409)				
No alcohol-related diagnosis	1.00 <sup>b</sup>	– <sup>d</sup>	1.00 <sup>b</sup>	– <sup>d</sup>
During pregnancy	0.59 <sup>b</sup> (0.14–2.41)	– <sup>d</sup>	0.81 <sup>b</sup> (0.20–3.33)	– <sup>d</sup>
≤1y before pregnancy	1.76 <sup>b</sup> (0.71–4.39)	– <sup>d</sup>	2.49 <sup>b</sup> (0.99–6.25)	– <sup>d</sup>
≤1y after pregnancy	<5	– <sup>d</sup>	<5	– <sup>d</sup>
>1y before pregnancy	<5	– <sup>d</sup>	<5	– <sup>d</sup>
>1y after pregnancy	0.84 <sup>b</sup> (0.54–1.30)	– <sup>d</sup>	0.85 <sup>b</sup> (0.49–1.46)	– <sup>d</sup>

<sup>a</sup>Live births. <sup>b</sup>Adjusted for variables used in frequency matching: maternal age, and year of birth. <sup>c</sup>Adjusted variables used in frequency matching and illicit drug use. <sup>d</sup>No potential confounder altered the odds by ≥20%.

1.30–8.48) for non-Aboriginal children was demonstrated only when the maternal alcohol diagnosis was recorded during pregnancy. However, the population-attributable fraction, assuming causality, is low and imprecise, indicating that prevention of heavy maternal alcohol consumption in this group of mothers would prevent less than 0.5% of CP cases. The eightfold increased odds of postneonatally acquired CP in non-Aboriginal children for any alcohol-related diagnosis (adjusted OR 7.92; 95% CI 2.23–28.14) supports the theory that heavy maternal alcohol consumption is both a direct and an indirect risk factor for the offspring.<sup>2</sup> The small numbers of postneonatally acquired CP prevented investigation of whether a postnatal alcohol diagnosis was associated with higher odds than diagnoses recorded either during pregnancy or prenatally. However, it is recognized that heavy maternal alcohol use continuing after pregnancy exposes the offspring to interacting risk factors stemming from the environment and the family, which increases the risk of health, mental health,

behaviour, development and educational problems, and child abuse and neglect.<sup>2</sup>

The lack of an association between an alcohol-related diagnosis recorded during pregnancy and pre/perinatally acquired CP in Aboriginal children was unexpected and may reflect the high proportion of Aboriginal children with CP in the comparison group. However, the increased odds when the diagnosis was recorded within 12 months before pregnancy (adjusted OR 2.49; 95% CI 0.99–6.25) suggests that these mothers continued to drink heavily during pregnancy and there may be under ascertainment of maternal alcohol problems during pregnancy. This may have masked an association. The lack of alcohol service data for Aboriginal-specific and rural services, the high percentage of Aboriginal mothers in this study living in rural/remote regions, and the evidence that high-risk drinking and alcohol-related harm are more common in rural/remote regions than in metropolitan regions in Australia<sup>24</sup> support this assumption.

We did not find an association between maternal alcohol consumption and postneonataly acquired CP in Aboriginal children, which may be due to multiple health and environmental contributors to risk in these children. For example, the Aboriginal population is more likely to have complex, chronic health problems than the non-Aboriginal population,<sup>25,26</sup> is less likely to access antenatal services,<sup>27</sup> and is more likely to be of low socio-economic status. The findings that 63.6% of postneonataly acquired CP in Western Australia was associated with infection for Aboriginal children and that 50.0% of CP in non-Aboriginal children was attributable to head injury were not unexpected as these have been documented previously.<sup>12</sup> These indicate different aetiological pathways for postneonataly acquired CP, leading us to question whether this may also be the case for pre/perinataly acquired CP.

We need to exercise caution in our interpretation of the data as an alcohol-related diagnosis was recorded in the mother during pregnancy in only a small number of cases. However, the proportion of pre/perinataly acquired CP in children with FAS (73/1000) is consistent with the findings of other studies<sup>7-10</sup> and lends support to the validity of the findings from this study. The small number of cases of CP in our study limited our analyses of many categories and highlights the difficulty in examining the association between prenatal alcohol exposure and CP even in a large population-based study such as this one. CP is a rare outcome,<sup>11,12</sup> and pregnant women<sup>13</sup> and individuals admitted to general hospital and psychiatric facilities<sup>28</sup> are not routinely asked about their alcohol consumption. The women in this study have serious alcohol-related problems and require non-judgemental treatment and support. Routinely asking women of childbearing age and pregnant women about their alcohol use should become standard practice and be supported with appropriate brief interventions and referral to alcohol treatment and family support services where indicated.

While we are confident that the mothers with an alcohol-related diagnosis in this study were drinking heavily, many women drinking at heavy levels will not have been identified.<sup>29</sup> So it is likely that at least some of the comparison mothers will also have alcohol-related problems unreported in the data sets used in this study, resulting in an underestimate of the true level of association. In spite of this, the two- to threefold increased odds of CP found for non-Aboriginal children in this study are on a parallel with other identified CP risk factors such as preterm birth, low birthweight, and infections.<sup>17</sup>

Finally, it is possible that CP in Aboriginal children is underascertained and that the true number of children with CP is higher than reported here. Changes in the privacy legislation in Western Australia in 2001 resulted in a decrease in the response rate from clinicians, particularly in regional areas

where a higher proportion of the population is Aboriginal. However, the CP Register uses multiple sources of ascertainment to identify children with CP and the stability of trends over time supports the robustness of the CP Register. Also, there is no evidence to indicate that underascertainment of CP would occur more frequently in the children of mothers with an alcohol-related diagnosis than in the comparison group. In 2011, mandatory reporting of birth defects and CP was legislated in Western Australia and so validation of the prevalence of CP on the Western Australian Register of Developmental Anomalies will be possible in a few years.

Although CP is rare, it was estimated that around 34 000 Australians in 2007 had CP, and CP carries higher disability burden and costs than many other forms of disability.<sup>30</sup> The findings of this study add strength to the evidence of an association between prenatal alcohol exposure and CP, which is one of only a few potentially modifiable risk factors that have been identified for both pre- and postneonataly acquired CP.

## CONCLUSIONS

Harmful maternal alcohol use is a potentially modifiable risk factor for CP. Children of non-Aboriginal mothers with an alcohol-related diagnosis recorded during pregnancy have increased odds of pre/perinataly acquired CP, indicative of a direct/causal association, and postneonataly acquired CP, indicative of an indirect/environmental risk factor. However, the percentage of non-Aboriginal children with CP attributable to heavy prenatal alcohol use in this cohort is less than 0.5%. The 2.5-fold increased odds of pre/perinataly acquired CP for Aboriginal children when a maternal alcohol-related diagnosis was recorded in the 12 months before pregnancy suggests a lack of identification of alcohol use disorders during pregnancy.

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## ONLINE MATERIAL/SUPPORTING INFORMATION

Additional material and supporting information may be found in the online version of this article.

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